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The Emotion Dysregulation Model of Anxiety: A preliminary path analytic examination

Cynthia Suveg*, Diana Morelen, Gene A. Brewer, Kristel Thomassin

Psychology Building, Department of Psychology, University of Georgia, Athens, GA 30602, United States

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ABSTRACT

Both temperamental (e.g., behavioral inhibition) and environmental (e.g., family emotional environment) factors are associated with etiology and maintenance of anxiety; however, few studies have explored mechanisms through which these risk factors operate. The present study investigation of a developmental model of anxiety (i.e., the Emotion Dysregulation Model of Anxiety; EDMA) that hypothesizes that emotion dysregulation is the mechanism through which temperamental and emotion parenting variables relate to anxiety. Emerging adults ($N = 676$, M age = 19.5) retrospectively reported on behavioral inhibition and emotion parenting factors in childhood, and current emotion regulation skills and symptoms of anxiety. Results of path analyses provide initial support for the EDMA. Emotion dysregulation fully mediated the relationship between behavioral inhibition and anxiety and partially mediated the relationship between family emotional environment and anxiety.

Given the impairing nature of anxiety (Woodward & Ferguson, 2001), research has attempted to identify factors related to the etiology and maintenance of these disorders. Research suggests that genetic factors account for a small percentage of variance in anxiety and that environmental factors play a critical role (Eley, 2001; Kendler, Neale, Kessler, Heath, & Eaves, 1992). Importantly, risk factors for anxiety disorders (ADs) are thought to have both genetic and environmental influences. In particular, temperamental factors (e.g., behavioral inhibition) and environmental variables (e.g., family emotional environment) have been strongly related to anxiety (Biederman et al., 2001; Caspi, Henry, McGee, Moffitt, & Silva, 1995; Hibbs et al., 1991; Muris, Bogels, Meesters, van der Kamp, & van Oosten, 1996; Stubbe, Zahner, Goldstein, & Leckman, 1993; Suveg, Sood, Comer, & Kendall, 2008; Suveg, Zeman, Flannery-Schroeder, & Cassano, 2005).

Despite strong relations between child temperament, family emotional environment, and anxiety, relatively scant research has examined these variables in tandem and little is known about the specific mechanisms by which such effects operate. The goal of this study is to address such gaps in the literature by collectively examining the influence of child temperament and family emotional environment on anxiety levels, and examine emotion dysregulation as a mechanism of effect. Specification of mechanisms through which temperamental and family environment variables influence anxiety has the potential to contribute to the refinement of etiological models of anxiety and the development of more focused prevention and intervention programs.

Using an emotion regulation framework, this study conducts a preliminary investigation of a model, the Emotion Dysregulation Model of Anxiety (EDMA), which examines the impact of both temperamental (behavioral inhibition) and environmental (family emotional environment) variables on current anxiety levels. Behavioral inhibition is a temperamental style characterized by reticence towards new people and situations, withdrawal, and high reactivity to novel stimuli (Garcia-Coll, Kagan, & Reznick, 1984). In the mediational EDMA, high child temperamental reactivity (measured via behavioral inhibition) is expected to contribute to emotion dysregulation because high reactivity likely impedes the use of helpful emotion regulation strategies, thus making adaptive emotion regulation difficult (Suveg, Payne, Thomassin, & Jacob, 2009). For instance, a child in the midst of a tantrum is likely to have greater difficulty implementing emotion regulation strategies to help himself calm down.

Empirical research suggests that arousal beyond a certain level may interfere with an individual's ability to respond adaptively in an emotionally evocative situation (i.e., engage in adaptive emotion regulation; Bradley, 1990; Calkins, 1994; Cole, Michel, & O'Donnell-Teti, 1994). Therefore, the ability to modulate arousal is likely to result in more constructive methods of responding to an emotionally arousing event (Saarni, Mumme, & Campos, 1998). Further, prolonged arousal may cause physiological changes at the biochemical or neuronal level that may ultimately sensitize the individual to respond in maladaptive ways (Bradley, 2000; Bremner & Vermetten, 2001).

* Corresponding author. Tel.: +1 706 583 5544, fax: +1 706 542 8048.
E-mail addresses: csuveg@uga.edu (C. Suveg), dmorelen@uga.edu (D. Morelen), gabrewer@uga.edu (G.A. Brewer), Kristel@uga.edu (K. Thomassin).

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The EDMA also proposes that a family emotional environment characterized by general truncated emotional expressivity and high levels of expressed negative affect will influence emotion dysregulation through a failure to appropriately socialize the emotion understanding and regulation skills that are necessary for emotionally competent functioning. Dunsmore and Halberstadt (1997, p. 53) suggest that the "overall frequency, intensity, and duration of positive and negative emotional expressiveness in the family is important in the child's formation of schemas about emotionality, about expressiveness, and about the world." Youth who are not exposed to facilitative emotional environments are not likely to develop the emotion regulation skills that are necessary to successfully negotiate stressful emotional situations (Denham, Mitchell-Copeland, Strandberg, Auerbach, & Blair, 1997). Additionally, research suggests that negative emotions tend to produce greater levels of arousal than positive emotions, thus requiring greater resources to regulate (Bradley, 2000; Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000).

In the EDMA, emotion dysregulation is hypothesized to emerge as the mechanism through which both high temperamental reactivity and family emotional environment influence anxiety levels. When individuals do not successfully negotiate emotion-provoking situations, they may choose maladaptive strategies (e.g., avoidance, aggression) to reduce their emotional arousal in the moment. Engagement in maladaptive emotion regulation leads to unsuccessful management of arousal, which in turn serves to maintain anxiety levels. Note that the EDMA proposes to examine both high temperamental reactivity (sometimes referred to as emotional reactivity in the literature) and emotion regulation. There is considerable debate regarding the relationship between temperamental reactivity and emotion regulation (see special issue of Child Development v. 75). For the purposes of this study, temperamental (emotional) reactivity is considered distinct from emotion regulation. As Rothbart and Sheese (2007) emphasize, temperamental reactivity refers to "responses to change in the external and internal environment, including a broad range of reactions . . ." (p. 332), whereas emotion regulation "is the modulation of a given emotional reaction . . ." (p. 333). Thus, emotional reactivity might be conceptualized as one's initial, unmodulated response to an emotion-provoking event, whereas emotion regulation involves the modification of the reactivity through a variety of means (e.g., cognitive interpretation of the arousal, use of distraction, support seeking). Though distinct, the constructs are related – when reactivity is high, regulation is likely to be difficult (Santucci et al., 2008). Temperamental reactivity will be assessed via a measure of behavioral inhibition because of the high reactivity associated with this construct. Research reviewed below provides a strong rationale and empirical foundation for testing the EDMA.

1. Links between high child temperamental reactivity and anxiety

Research drawn from various literatures supports the notion that individuals with anxiety disorders experience elevated levels of reactivity. Several studies by independent research groups have demonstrated links between behavioral inhibition (i.e., the tendency to display heightened physiological arousal and withdrawal when exposed to unfamiliar people, places, or situations) and anxiety problems (Biederman, Rosenbaum, Chaloff, Kagan, & March, 1995; Coplan, Wilson, Fohlick, & Zelenski, 2006; Kagan, Reznick, Snidman, & Gibbons, 1988; Muris & Meesters, 2002; Turner, Beidel, & Wolff, 1996). For example, Gladstone, Parker, Mitchell, Wilhelm, and Malhi (2005) examined the relation between self-reported childhood behavioral inhibition and lifetime anxiety disorders in a clinical sample of depressed adults ranging in age from 17 to 68 years. Retrospective reports of behavioral inhibition were related to lifetime rates of social phobia, specific phobias, and multiple anxiety disorders.

Research on the tripartite model of depression and anxiety also supports the notion that individuals with anxiety disorders are often restless or keyed up. Clark and Watson (1991) found that high physiological arousal was distinctly related to anxiety using an adult population, and subsequent research found support for the model with children (Jacques & Mash, 2004; Joiner, Catanzaro, & Laurent, 1996; Lonigan, Carey, & Finch, 1994). In a community sample of youth aged 6–17, Cannon and Weems (2006) found that physiological hyperarousal was distinctly related to anxiety symptoms – a finding that held for boys and girls of all ages in the sample used. Though it is not the case that physiological hyperarousal is necessarily temperamentally-based, research on the tripartite model nonetheless suggests that individuals with anxiety are often "keyed up," which may make regulation efforts difficult.

Collectively, much research supports the notion that individuals with ADs experience elevated temperamental reactivity, though there is less support linking the experience of high reactivity to emotion regulation difficulties. Our study will expand upon this research base by examining whether the relation between temperamental reactivity and anxiety can be accounted for by emotion dysregulation.

2. Links between family emotional environment and anxiety

In the EDMA, family emotional environment is expected to influence emotion dysregulation through a failure to appropriately socialize the emotion understanding and regulation skills that are necessary for adaptive emotion functioning. Suveg et al. (2005) found that children with an AD and their mothers independently indicated lower levels of family emotional expressivity in comparison to non-clinical children and their mothers (cf., Noguchi & Ollendick, in press). During discussions about times the child felt different emotions, mothers of youth with an AD discouraged the discussion of negative emotional experiences and used fewer positive emotion words during the discussion than did mothers of control children. Further, in contrast to mothers of non-clinical children who spoke the majority of the time, mothers of children with an AD used fewer words overall than their children during the emotion discussion. Additional research has provided commensurate findings. Hudson, Comer, and Kendall (2008) found that parents of youth with ADs were less warm than the control group when discussing certain emotions. In the context of a negative emotion scenario, parents of AD youth were more likely to respond to the youth with nonsupportive responses than were parents of the control children. Findings about fathers in particular were commensurate with those by Suveg et al. (2008a) and Suveg, Sood, Comer, and Kendall (2008) who found that fathers of AD children engaged in less explanatory discussion of emotion overall and exhibited less positive affect and more negative affect when interacting with sons than did fathers of non-clinical children.

Collectively, findings suggest that parents of youth with anxiety disorders may encourage the suppression or restriction of emotional expression through both direct (e.g., discussions regarding emotion) and indirect means (e.g., home emotional climate). AD youth's emotion learning may further be impeded by the lack of pleasant interactions with their parents during emotion discussions. This study moves beyond this literature by examining how family emotional environment relates to anxiety.

3. Links between emotion dysregulation and anxiety

Finally, the EDMA proposes that emotion dysregulation is the mechanism through which both high temperamental reactivity and family emotional environment exert their effects on anxiety. Preliminary research has linked emotion regulation difficulties to anxiety in both youth and adult populations and suggests that emotion regulation difficulties extend *beyond* the ability to manage anxiety specifically. Suveg and Zeman (2004) found that children with ADs reported less adaptive emotion regulation methods, experienced their emotions with greater intensity, and perceived themselves as less efficacious in managing emotional experiences than their peers. This study is consistent with other research that has also found that anxiety in youth is globally associated with dysregulated emotions (i.e., emotions beyond anxiety; Blumberg & Izard, 1986; Suveg et al., 2008a, 2008b, 2009).

Deficits in emotional functioning have also been linked to anxiety in adult populations. In one study, Mennin, Holaway, Fresco, Moore, and Heimberg (2007) identified components of emotional functioning specific to GAD (i.e., heightened intensity of emotion, maladaptive management of emotions) and to SoP (i.e., low levels of emotion intensity, poor understanding of emotions, negative reactivity to emotions). Other research has likewise identified emotion functioning deficits related to anxiety in adult populations (Mennin, Heimberg, Turk, Fresco, 2005; Roemer et al., 2009; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006).

Research to date has contributed to our understanding of potential etiological or maintaining factors in anxiety, yet significant gaps remain. No studies could be located that have examined both temperamental and family emotional environment variables simultaneously. Despite the central focus on the role of emotion regulation in psychopathological processes, no studies were found that examined emotion regulation as a mediating factor. The current study addresses these gaps by examining temperamental and environmental factors in tandem and examining their mechanisms of effect in a sample of emerging adults. In particular, the study examined behavioral inhibition, family emotional expressivity, and frequency of negative affect expressed in the family. These variables were chosen for study because of the previously reviewed literature that found each of them to be implicated, directly or indirectly, in anxiety. Based on the EDMA, it was expected that: (a) High child temperamental reactivity (i.e., behavioral inhibition) would be positively related to anxiety symptoms and the association would be mediated by emotion dysregulation and (b) Family emotional environment (i.e., low family emotional expressivity, high frequency of negative affect expressed in the family) would be positively related to anxiety symptoms and the relation would be mediated by emotion dysregulation.

4. Method

4.1. Sample

Participants included 676 students between the ages of 18 and 29. Undergraduate students from a large university located in a small city volunteered in exchange for partial credit toward a class research participation requirement. The sample included 242 males (36%) and 434 females (64%) with a mean age of 19.5 (SD = 1.22). With respect to the diversity of the sample, 552 (81.7%) were Caucasian, 53 (7.8%) were Asian, 46 (6.8%) were African American, 14 (2.1%) were Hispanic, and 11 (1.6%) indicated "other."

4.2. Measures

4.2.1. Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1994)

The SCL-90-R is a 90-item questionnaire that assesses current symptoms of adult psychopathology (Derogatis, 1994). Participants were instructed to indicate on a 5-point Likert scale (0 = *not at all*, 4 = *extremely*) how much they were distressed by a variety of symptoms in the last week. The ten-item Anxiety Subscale, which includes items such as "Suddenly scared for no reason" and "The feeling that something bad is going to happen to you," was used for the current study. Adequate reliability and validity have been previously established (Derogatis, 1994). Alpha for the Anxiety Subscale in this study was .84.

4.2.2. Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

The DERS is a 36-item self-report measure of current, clinically-relevant difficulties with emotion regulation. Participants were asked to rate items according to the frequency that the statement applied to them by using a 5-point Likert scale (1 = *almost never*, 4 = *almost always*). The items in the questionnaire center around six subscales: Nonacceptance (e.g., "When I'm upset, I feel ashamed with myself for feeling that way)", Goals (e.g., "When I'm upset, I have difficulty getting work done"), Impulse (e.g., "When I'm upset, I lose control over my behaviors"), Awareness (e.g., "When I'm upset, I take time to figure out what I'm really feeling"), Strategies (e.g., "When I'm upset, I believe that there is nothing I can do to make myself feel better"), and Clarity (e.g., "I am confused about how I feel"). A total DERS score comprised of all items was used in this study, with higher scores being indicative of poorer ER. High reliability (e.g., internal consistencies ranging from .80 to 89) and acceptable construct and predictive validity have been established for the overall scale (Gratz & Roemer, 2004). Internal consistency for the DERS total score in the current study was .81.

4.2.3. Family Environment Scale (FES; Moos & Moos, 1994)

The FES is a measure of one's perceptions of the family climate at home. A retrospective version of the FES was used to assess participants' family climate during their childhood (Fassler et al., 2005). For the purpose of this study, the 10-item Expressiveness Subscale of the FES was used. Participants were asked to rate items (e.g., "Family members often kept their feelings to themselves") according to the frequency that the statement applied to them by using a 5-point Likert scale (1 = *not at all with my family*, 5 = *frequently in my family*). This subscale assessed participants' perceptions of the level of expressiveness in their home during childhood. Moderate to good reliability has been previously established with internal consistency ranging from .67 to.78 and test-retest reliability ranging from .73 to .86 (Moos & Moos, 1981). The internal consistency in the current study was .69.

4.2.4. Family Expressiveness Questionnaire (FEQ; Halberstadt, 1986)

The FEQ is a 40-item measure of one's perceptions of the degree of emotional expressiveness, both negative and positive, in the home. A retrospective version of the FEQ was created and participants were asked to rate items reflecting their perceptions of the degree of negative emotional expressiveness in their home during their childhood. (e.g., "Showing dislike for someone," "Praising someone for good work") using a 9-point Likert scale (1 = *not at all in my family*, 9 = *very frequently in my family*). Adequate reliability and validity have been previously established with internal consistency ranging from .75 to .88 and test-retest reliability ranging from .89 to .91 (Halberstadt, 1986). The internal consistency for the Negative Subscale in this study was .84.

Table 1
Correlations, means, standard deviations, and Skew and Kurtosis estimates for the measures.

	1	2	3	4	5
1. RMBI	1.00				
2. FES	−0.25	1.00			
3. FEQ - Neg	0.11	−0.14	1.00		
4. DERS	0.25	−0.22	0.21	1.00	
5. SCL-90 ANX	0.18	−0.15	0.19	0.53	1.00
M	0.75	3.10	4.44	2.09	0.43
SD	0.33	0.62	1.00	0.54	0.47
Skew	0.31	−0.08	0.48	0.63	1.95
Kurtosis	−0.34	0.18	0.41	0.50	4.73

Note. RMBI = Retrospective Measure of Behavioral Inhibition, FES = Family Environment Scale, FEQ - Neg = Family Environment Questionnaire Negative Subscale, DERS = Difficulties in Emotion Regulation Scale, SCL- 90 ANX = Symptom Checklist-90 Anxiety Subscale. Correlations exceeding $r = .08$ are significant at $\alpha = .05$.

4.2.5. *Retrospective Measure of Behavioral Inhibition (RMBI; Gladstone & Parker, 2005)*

The RMBI is an 18-item self-report measure designed to retrospectively assess aspects of a behaviorally-inhibited temperament style during primary school years (e.g., "When you went on outings with your family to new places, would you tend to become quiet and freeze up?" "When unfamiliar visitors came to your home did you feel fearful or nervy?"). Participants were asked to rate items using a 3-point Likert scale (0 = *no/hardly ever*, 1 = *some of the time*, 2 = *yes/most of the time*). Previous research has indicated adequate test-retest reliability (i.e., .56–.77) and adequate internal consistency (i.e., .40–.90; Gladstone & Parker, 2005). The internal consistency in this study was .85.

4.3. *Procedure*

Participants in the current study volunteered in exchange for partial credit toward a research participation requirement. The study was completed using Survey Monkey, a website designed to enable the completion of online surveys. Of note, Survey Monkey meets the United States Department of Commerce's Safe Harbor Privacy Standards. Participants provided consent and completed the measures online in the order presented in Section 4.2. All procedures were in accordance with the mandates of the sponsoring university's Institutional Review Board.

5. Results

Path analyses were conducted using Lisrel v.8.80 (Jöreskog & Sörbom, 1996). The correlations between the measures and their means, standard deviations, and estimates of skew and kurtosis can be found in Table 1. A preliminary inspection of Table 1 reveals a positive association between the anxiety measure (SCL-90 ANX) and the temperamental reactivity (RMBI), family emotional environment (FES and FEQ Negative), and emotion dysregulation (DERS) measures. Path analyses were conducted on the correlations to further understand the direct and indirect effects of these variables on anxiety (James, Mulaik, & Brett, 2006). To examine our hypothesis that DERS would mediate the relations between RMBI, FES, and FEQ and SCL-90 ANX, all variables were entered into the preliminary model shown in Fig. 1. In Model 1, DERS mediates the paths from RMBI, FES, and FEQ to SCL-90 ANX. As can be seen in Table 2, this model fit the data well (χ^2 (3) = 8.22, $p = .04$, RMSEA = .05, CFI = .99, AIC = 32.22).[1]

[1] There were no qualitative differences in this and all subsequent models as a function of gender. Thus, all of the models in the present study reflect data from the complete group of 676 participants.

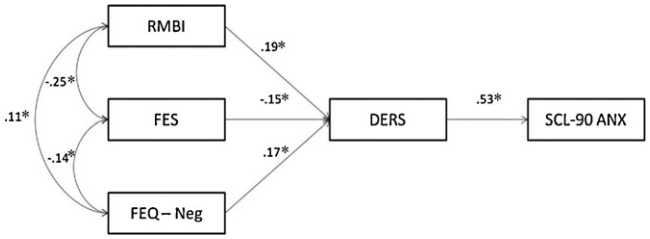

Fig. 1. Baseline model: DERS mediates paths from RMBI, FES, and FEQ to SCL-90 ANX.

Using this model as our baseline model, we fit several additional models to assess whether DERS completely, or only partially, mediated the relations between RMBI, FES, and FEQ and SCL-90 ANX. In Model 2, a direct pathway was specified leading from FEQ to SCL-90 ANX. As can be seen in Table 2, this model fit the data well and resulted in an improved fit as compared with Model 1 ($\Delta\chi^2 = 6.06$, $p < .05$). The path from FEQ to SCL-90 ANX was significant ($\beta = .08$, $p < .05$), suggesting direct and indirect effects of FEQ on SCL-90 ANX. In other words, DERS only partially mediated the relation between FEQ and SCL-90 ANX. Due to the improved fit, this model was used in further model testing of direct pathways between RMBI and FES and SCL-90 ANX. In Model 3, the direct effect of RMBI on SCL-90 ANX was tested by specifying an additional path in Model 2. This model fit the data well, but failed to significantly improve the fit from Model 2 ($\Delta\chi^2 = 1.86$, $p = .17$). Thus, there does not appear to be a direct effect of RMBI on SCL-90 ANX due to the DERS fully mediating the relation. In Model 4, a direct pathway was specified from FES to SCL-90 ANX. This model fit the data well, but failed to significantly improve the fit over Model 2, $\Delta\chi^2 = .66$, $p = .42$. Therefore, the relation between FES and SCL-90 ANX was fully mediated by DERS. Taken together, DERS fully mediated the relations between RMBI and FES and SCL-90 ANX whereas the DERS partially mediated the relation between FEQ and SCL-90 ANX (see Fig. 2).

6. Discussion

Research has implicated both temperamental and environmental factors in anxiety (Caspi et al., 1995; Hibbs et al., 1991; Suveg et al., 2005, 2008a, 2008b). The goal of this study was to extend previous research by examining mechanisms by which three particular variables (i.e., high temperamental reactivity measured via behavioral inhibition, restrictive family expressiveness, frequency of negative affect expression in the family) influence anxiety symptoms. Based on the EDMA, it was expected that all three variables would influence anxiety levels through emotion dysregulation. Overall, the results are consistent with previous research and offer new insights in support of the EDMA.

The first prediction of the EDMA suggested that high child temperamental reactivity (measured via behavioral inhibition) would exert its effects on anxiety through emotion dysregulation. This prediction was supported in that emotion dysregulation fully

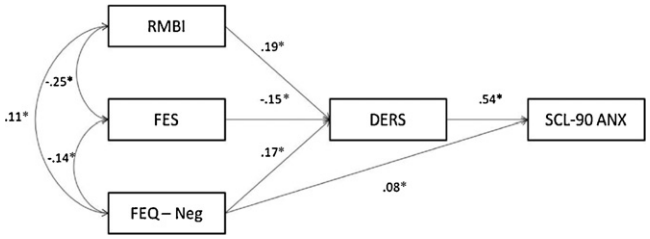

Fig. 2. Comparison model: DERS fully mediates paths between RMBI and FES to SCL-90 ANX but partially mediates the paths between FEQ and SCL-90 ANX.

Table 2
Fit indices for all models.

Model	X^2	df	χ^2/df	RMSEA	CFI	AIC
Model 1: Full mediation	8.22	3	2.74	.05	.99	32.22
Model 2: FEQ-Neg partial	2.16	2	1.08	.01	1.0	28.16
Model 3: RMBI partial	.30	1	.30	.00	1.0	28.30
Model 4: FES partial	1.50	1	1.50	.03	.99	29.50

Note. FEQ-Neg = Family Environment Questionnaire Negative Subscale, RMBI = Retrospective Measure of Behavioral Inhibition, FES = Family Environment Scale. The Chi-square statistic reflects whether there is a significant difference between the observed and reproduced covariance matrices and nonsignifcant values are desirable. The ratio of Chi-square to the number of degrees of freedom is also reported. Ratios of two or less usually indicate acceptable fit. Also reported are the root mean square error of approximation (RMSEA) which reflects the average squared deviation between the observed and reproduced covariances. In addition, comparative fit index (CFI) reflects the proportion of the observed covariance matrix explained by the model are reported. CFI values greater than .90 are indicative of acceptable fit (Kline, 1998). Finally, the Akaike information criterion (AIC) reflects the fit between models in which the model with the smallest AIC is preferred.

mediated the relation between behavioral inhibition and anxiety levels. High child temperamental reactivity was expected to contribute to child emotion dysregulation because high reactivity in response to novel situations and people is likely to make adaptive emotion regulation difficult. Commensurately, previous research found that individuals with anxiety exhibit high physiological arousal (Cannon & Weems, 2006; Clark & Watson, 1991) and longitudinal studies identified a longitudinal link between behavioral inhibition and anxiety (Coplan et al., 2006; Kagan et al., 1988, 1997). Research suggests that arousal beyond a certain level may interfere with an individual's ability to respond adaptively in an emotionally evocative situation (Bradley, 1990; Calkins, 1994; Cole et al., 1994) and empirical studies have found links between emotion intensity and difficulties in regulation (Santucci et al., 2008; Suveg et al., 2009). There are many ways in which high temperamental reactivity might contribute to emotion dysregulation. High reactivity may make emotions difficult to differentiate from one another, contributing to poor awareness of emotional experience (Baker, Holloway, Thomas, Thomas, & Owens, 2004). Poor awareness of emotional experience is a key ingredient of competent emotional functioning, and without it, regulation efforts are likely to be difficult and ineffective (Halberstadt, Denham, & Dunsmore, 2001). Further, high reactivity may interfere with the ability to access and generate cognitive strategies to adaptively modulate the emotional experience, limiting one's ability to modify an emotional experience via behavioral means. In sum, behavioral inhibition contributes to emotion dysregulation, which in turn influences anxiety symptoms potentially due to the inability to manage the emotionally-arousing experience.

The EDMA also predicted that emotion dysregulation would fully mediate the relation between family emotional environment factors (i.e., restrictive family expressiveness and frequency of negative affect expression in the family) and symptoms of anxiety. This prediction was partially supported—the relation between restrictive family expressiveness and anxiety was fully mediated, but the association between frequency of negative affect expression and anxiety was only partially mediated. Family factors have been implicated in the development of anxiety symptoms (McLeod, Wood, & Weisz, 2007), but few investigations have examined the mechanisms of effect. The current study found that one way in which aspects of the family emotional environment influence anxiety levels is through emotion dysregulation. Halberstadt (1986) suggests that the family emotional environment, including the expression of particular types of emotions, contributes to youth's formation of schemas about appropriate emotional expression. Youths reared in family environments in which expressiveness is low are likely to develop schemas that overall emotion expression is not appropriate. Low emotional expressiveness is not maladaptive itself, but research suggests that chronically low family emotional expressiveness can have significant implications for the development of emotion regulation (Denham et al., 1997). Expressiveness within the family likely provides opportunities for youth to discuss emotional experiences and in so doing learn adaptive methods of emotion regulation. Youths from low expressive homes are not likely to have this benefit. Low expressiveness may become particularly problematic when it leads to youth suppressing emotional experiences. Chronic suppression of emotional experience might lead to increased sympathetic arousal and subjective distress (Campbell-Sills, Barlow, Brown, & Hofman, 2006; Gross & Levenson, 1997) and further contribute to regulation difficulties. Anxious youth in particular are at high risk for continued maladaptation given a predisposition to high reactivity and because research has identified low expressiveness in their families (e.g., Noguchi & Ollendick, in press; Suveg et al., 2005). It should be noted that the correlation between the two family factors used in the model was surprisingly low (i.e., −.14). One possible explanation is that the FEQ and FES-E measure two relatively different constructs. Specifically, the FEQ subscale used in the present study emphasized displays of negative emotions in the home whereas the FES-E may have captured a broader concept of familial emotional expressivity given that both positive and negative emotions were included. Though surprisingly low, the correlation suggests the distinctiveness of the constructs studied in the model.

The EDMA also hypothesized that the relation between high frequency of expressed negative emotion in the family and anxiety symptoms would be fully mediated by emotion dysregulation. This hypothesis was supported in part in that the relation was partially mediated. A high frequency of expressed negative affect was proposed to contribute to emotion dysregulation because of direct and indirect implications associated with chronic expression of negative affect. Family interactions laden with negative affect influence youth through the direct modeling of dysregulated emotions (Muris et al., 1996). Negative affect within the household may also lead to the development of schemas about emotional functioning (Denham, Zoller, & Couchoud, 1994). More specifically, emotion interactions in the household may provide youth with models about how emotion-laden interactions should occur. Additionally, research suggests that negative emotions cause more arousal and may be more challenging to regulate than positive emotions (Bradley, 2000; Cacioppo et al., 2000). As a result of this greater level of arousal, a preponderance of negative emotion is likely to make regulation more difficult. Despite the links between negative affect expression, emotion dysregulation, and anxiety, only partial mediation was found in these analyses. Partial mediation allows for various interpretations of this model. Though it could be that the frequency of negative affect has a direct effect on symptoms of anxiety, it may also be that a more comprehensive model is necessary to explain the exact mechanisms by which negative emotion expression leads to higher levels of anxiety. In other words, additional factors may also mediate the relation between negative affect expression in the family and anxiety. Nonetheless, emotion dysregulation does partially mediate this relation, thus providing

support for the EDMA—a necessary first step in understanding the relations between risk factors for the development of anxiety symptoms.

Taken together, results of this study provide preliminary support for the EDMA. A large literature exists linking temperamental and environmental factors to anxiety, yet much less research has examined the mechanisms by which the variables relate to anxiety. This study identifies emotion dysregulation as one way in which high temperamental reactivity and family emotional environment influence anxiety. Our results have implications for conceptual models of anxiety and suggest that the consideration of multiple variables is necessary to fully understand the development and maintenance of anxiety disorder. The findings also lend support to the notion that emotion dysregulation is a key factor to consider in psychopathology. Clinical implications are relevant as well – greater specification of these conceptual issues has great potential to inform the prevention and intervention literature. Numerous empirically-supported intervention programs for anxiety exist (see APA Task Force on the Promotion and Dissemination of Psychological Procedures, 1995). Nevertheless, given the number of cases left with clinical levels of anxiety following treatment (e.g., Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008), researchers need to consider moderators of outcome and ways to enhance existing treatments for individuals with significant emotion regulation difficulties (i.e., those that extend beyond the experience of anxiety/worry). Preliminary results of treatments that have incorporated emotion components have been positive for both youth and adults with anxiety (Barlow, Allen, & Choate, 2004; Ehrenreich, Goldstein, Wright, & Barlow, 2009; Mennin, 2006; Suveg, Kendall, Comer, & Robin, 2006, see also Suveg et al., 2008b and Trosper, Buzzella, Bennet, & Ehrenreich, 2009). Information gained from this project can be considered in the further refinement of anxiety treatments and would directly address the call from the National Advisory Mental Health Council's Behavioral Science Workgroup (2000) to use basic research to inform the development of prevention and treatment programs. Lastly, some treatments for child anxiety currently include parents in the treatment (e.g., Barrett, Dadds, & Rapee, 1996; Bogels & Siqueland, 2006; Kendall et al., 2008), although the ways in which parents have been included has varied widely (see Barmish & Kendall, 2005). This research may help identify particular ways in which it might be most beneficial to include parents.

6.1. Limitations and future directions

Strong support was found for the conceptual model proposed in this investigation. Nonetheless, the study utilized retrospective reports of behavioral inhibition and family emotional environment. Although previous research has found validity for retrospective reports of childhood experiences generally (Yancura & Baldwin, 2009), and behavioral inhibition (Gladstone & Parker, 2005) and family functioning specifically (Negy & Snyder, 2006), prospective research is necessary to deduce causal relationships among the study variables. Although the current study included a set of constructs that have been shown to be empirically related to childhood anxiety, other variables could have been included. Additional research including other variables would allow for the examination of individual, additive, and interactive contributions of multiple variables, thus further delineating the most comprehensive model of anxiety. A clear pattern of findings support the EDMA in a community sample of individuals with anxiety symptoms; it will be necessary to determine whether this model holds for clinical samples. It is not known whether the identified relations are specific to anxiety or whether they would apply to other forms of psychopathology. Future research should include comparison groups.

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