Distribution Agreement

In presenting this thesis as a partial fulfillment of the requirements for a degree from Emory University, I hereby grant to Emory University and its agents the non-exclusive license to archive, make accessible, and display my thesis in whole or in part in all forms of media, now or hereafter now, including display on the World Wide Web. I understand that I may select some access restrictions as part of the online submission of this thesis. I retain all ownership rights to the copyright of the thesis. I also retain the right to use in future works (such as articles or books) all or part of this thesis.

Rachel Diane Brenner April 02, 2018

Mood Repair and Respiratory Sinus Arrhythmia in Children of Depressed Mothers

by

Rachel D. Brenner

Sherryl H. Goodman
Adviser

Department of Psychology

Sherryl H. Goodman

Adviser

Yuk Fai Cheong

Committee Member

Paul Bhasin

Committee Member

2018

Mood Repair and Respiratory Sinus Arrhythmia in Children of Depressed Mothers

By

Rachel D. Brenner

Sherryl H. Goodman

Adviser

An abstract of
a thesis submitted to the Faculty of Emory College of Arts and Sciences
of Emory University in partial fulfillment
of the requirements of the degree of
Bachelor of Arts with Honors

Department of Psychology

2018

Abstract

Mood Repair and Respiratory Sinus Arrhythmia in Children of Depressed Mothers
By Rachel D. Brenner

Depression often runs in families, and children of depressed mothers face an especially high risk for developing the disorder. Among children of mothers with depression, those whose mothers have chronic and severe depression are at greater risk for developing the disorder than those whose mothers have mild and infrequent depression. Children's difficulties with regulating negative emotions is a likely contributor to this heightened risk. Past studies suggest that familial risk for depression is linked to difficulties with emotion regulation on a strategic level, as indexed by mood repair, and a physiological level, as indexed by respiratory sinus arrhythmia. However, these relationships are relatively unexplored among children in middle childhood and children of depressed mothers. As first depressive episode onset often occurs in adolescence, studies on high-risk children's emotion regulation in middle childhood may inform early intervention efforts aimed at preventing or mitigating depression in youths. The current study sought to clarify these relationships by examining differences in mood repair and respiratory sinus arrhythmia among a sample of 131 children ages 8 to 10 with and without a maternal history of depression. We predicted that children of mothers with a history of depression would demonstrate attenuated mood repair and respiratory sinus arrhythmia compared to children of mothers without a history of depression. Furthermore, we examined the extent to which duration of maternal depressive episodes was associated with children's mood repair and respiratory sinus arrhythmia. Analyses revealed no significant group differences regarding mood repair or respiratory sinus arrhythmia scores, and no significant associations between mood repair and respiratory sinus arrhythmia scores. Duration of maternal depressive episodes was not significantly associated with either mood repair or respiratory sinus arrhythmia. The developmental and methodological implications of these results are discussed along with directions for future research.

Mood Repair and Respiratory Sinus Arrhythmia in Children of Depressed Mothers

By

Rachel D. Brenner

Sherryl H. Goodman

Adviser

A thesis submitted to the Faculty of Emory College of Arts and Sciences
of Emory University in partial fulfillment
of the requirements of the degree of
Bachelor of Arts with Honors

Department of Psychology

2018

Acknowledgements

Many thanks to Katherine A. Cullum, Judy Garber, and the personnel of the Children's and Mother's Emotions Lab at Emory for their guidance and support.

Table of Contents

List of Tables

Depression is a disorder that often goes hand in hand with other health problems, financial burden, and suicidality (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). These consequences are far-reaching and can impact not only afflicted individuals, but also their family members (Goodman et al., 2011). And, while depression is highly prevalent in today's world and has gained a lot of attention in various fields of research, there is still much to be discovered about the etiology of the disorder (Beauchaine & Thayer, 2015). For these reasons, the importance of investigating risk factors that drive the development of depression cannot be overstated.

One risk factor for the development of depression that has been consistently backed by scientific evidence is family history of the disorder. Children with a significant family background of depression are an estimated three times more likely to develop depression themselves relative to peers who come from families without a history of depression (Williamson, Birmaher, Axelson, Ryan, & Dahl, 2004). This familial influence is linked most strongly to maternal depression history, which serves as a significant predictor of the onset of a child's first depressive episode (Williamson et al., 2004). Among families of depressed individuals, having a family member with a more chronic form of depression was found to be a stronger predictor of an individual's likelihood to develop depression than presence of family history of the disorder alone (Bland, Newman, & Orn, 1986; Zubenko, Zubenko, Spiker, Giles, & Kaplan, 2001). Notably, depression chronicity in these studies was defined as recurrence, or having two or more episodes, and did not discriminate between individuals who had low and high numbers of depressive episodes within this category (Bland et al., 1986; Zubenko et al., 2001). As children's development and risk for depression is likely impacted by the extent of their

exposure to maternal depressive episodes (Goodman & Gotlib, 1999), it is worth examining the chronicity of maternal depressive episodes in greater detail.

While studies on heritability help us to identify who is the most at risk, examining early vulnerabilities to depression may provide clues as to how and why certain groups are at risk for the development of the disorder. In a meta-analytic review, researchers noted that emotion regulation (ER) may be one such process (Goodman et al., 2011). They found depression in mothers to be associated with children's more negative emotions, and the effect size of this association to be inversely related to children's age (Goodman et al., 2011). As inability to pull oneself out of a sad or irritable mood is a cardinal feature of depression, this suggests that children of depressed moms may begin to deviate from a healthy developmental course at an early age (Goodman et al., 2011). And indeed, when researchers consider regulation of negative emotions separately from the broader construct of ER, they find that it characterizes not only children with depression but also their non-depressed siblings (Bylsma et al., 2015). More broadly, depressed individuals and their relatives consistently demonstrate difficulties with regulating negative emotions (Bylsma et al., 2015; Sarıtaş, Grusec, & Gençöz, 2013).

Depressed individuals and their relatives may experience difficulty with regulating negative emotions because they are less likely to use effective regulatory strategies and more likely to use regulatory strategies that are ineffective or worsen their mood (Joormann & Stanton, 2016; Joormann & Vanderlind, 2014). Mood repair is a measure that examines such strategies in detail by indexing behavioral, cognitive, and social tactics that people may use to regulate negative emotions (Kovacs, Rottenberg, & George, 2009). Adaptive mood repair is thought to facilitate healthy emotional processing in the face of unpleasant stimuli, while maladaptive mood repair may prolong or worsen negative emotions (Kovacs et al., 2009). Researchers have found

deficits in adaptive mood repair and heightened maladaptive mood repair use in depressed adults and adolescents, indicating that these emotional difficulties may be observed at various developmental stages across the lifespan (Bylsma et al., 2015; Kovacs et al., 2009). Notably, these associations are not confined to those with mood disorders. Never-depressed siblings of depressed adolescents also show deficits in mood repair repertoires in comparison to low-risk controls, suggesting that problems with negative ER may be a contributor to the development of depression rather than just a symptom (Bylsma et al., 2015). As first onset of depression often occurs during adolescence, it is worth studying children in earlier developmental stages to see if they, too, demonstrate a relationship between familial risk for depression and mood repair deficits (Avenevoli et al., 2015). Such a study could contribute to preventative efforts by shedding light on the potential role of mood repair in children's risk for developing depression.

While indices such as mood repair give us a better understanding of what may be going on in response to emotional stimuli, it is also important to consider the role of physiological processes in emotions. Porges' Polyvagal Theory posits that emotions are linked to physiological processes such as respiratory sinus arrhythmia (RSA) via branches of the parasympathetic vagus nerve (Porges, 1995, 2007). The vagus nerve works by signaling the heart to slow down, which promotes both energy conservation and a state of calm in the individual (Porges, 1995, 2007). Due to these periods of slowing, this process can explain the fluctuations in heart rate that individuals will experience (Porges, 1995). Accordingly, RSA indexes the functioning of the parasympathetic nervous system by measuring how much variation in heart rate occurs during a respiration cycle (Porges, 1995). By measuring RSA, researchers can gain insight into an individual's level of arousal at rest (baseline RSA) as well as in response to negative emotional stimuli (RSA reactivity; Porges 2007).

In a study of children ages 8-12, researchers found that low baseline RSA was associated with an increased risk for depression while high baseline RSA may be protective against exposure to mother's melancholic depressive symptoms (Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Attenuated development of baseline RSA may be linked to risk for depression, as another study found that children of parents with childhood-onset mood disorders (COMDs) did not experience the same increase in baseline RSA across childhood as did their low-risk peers (Gentzler, Rottenberg, Kovacs, George, & Morey, 2012). As baseline RSA has been found to be a consistent biomarker of emotional regulation and parasympathetic nervous system functioning, it appears that the emotional development of these high-risk children is veering off course and rendering them susceptible to depressive tendencies over time (Beauchaine, 2015).

RSA reactivity also has implications for depressive tendencies, as it appears to be related to maladaptive physiological and emotional tendencies (Porges, 2007). High-risk children of depressed mothers demonstrate blunted development of emotion regulation (indexed by RSA reactivity) across childhood when compared to controls (Blandon, Calkins, Keane, & O'brien, 2008). Another study on high-risk children found that RSA reactivity was negatively associated with adaptive mood repair and positively associated with depressive symptoms (Gentzler, Santucci, Kovacs, & Fox, 2009). This study did not find risk for mood disorder to be a moderator, though this may be because the high-risk group was heterogeneous and included children of parents with any COMD instead of depression specifically (Gentzler et al., 2009). Heterogeneity is also important to consider even within a diagnosis of depression, as researchers discovered that abnormalities in RSA reactivity were more prominent among children of

chronically depressed mothers compared to controls and children of mothers with milder, more stable, and remitted depression (Ashman, Dawson, & Panagiotides, 2008).

Recent findings suggest that the best approach to predicting emotional difficulties in adolescents and adults with juvenile-onset depression is to consider baseline RSA and RSA reactivity jointly (Cribbet, Williams, Gunn, & Rau, 2011; Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013; Yaroslavsky et al., 2016; Yaroslavsky, Rottenberg, & Kovacs, 2013). More specifically, researchers found that individuals with high baseline RSA and RSA withdrawal had the lowest depression rates, while RSA augmentation across all levels of baseline RSA was associated with depression (Yaroslavsky, Rottenberg, et al., 2013). As depression is known to be quite heterogeneous, this approach may be useful in identifying and accounting for specific RSA repertoires as they relate to differences in depressive symptoms and tendencies among depressed and high-risk individuals (Yaroslavsky, Rottenberg, et al., 2013). And, combining this new approach with mood repair in pre-adolescent children may provide insight about what goes awry in the emotional development and regulation of high-risk individuals in the years preceding typical depression onset.

In the present study, we sought to examine mood repair and RSA repertoires in high-risk children prior to the typical age of onset for depression. Based on previous findings in the literature, we have several hypotheses:

1) Children of depressed moms will show high maladaptive and/or low adaptive mood repair relative to controls.
2) Children of depressed moms will demonstrate low resting RSA relative to controls and/or RSA augmentation in response to emotional stimuli.

3) Adaptive mood repair will be positively associated with baseline RSA and negatively associated with RSA reactivity, while maladaptive mood repair will have a negative association with baseline RSA and a positive association with RSA reactivity.
4) Mother's duration of depressive episodes will be positively associated with maladaptive mood repair and RSA reactivity, and negatively associated with adaptive mood repair and baseline RSA. Also, mother's duration of depressive episodes will add predictive power to the relationship between baseline RSA and RSA reactivity.

Method

Recruitment

Mothers and their children ages 8-10 were recruited by Emory University and Vanderbilt University. Emory recruited through the Emory University Child Study Center database and Kaiser Permanente-Georgia. Vanderbilt recruited through the Vanderbilt University research listserv, referrals, and a birth record database. Participants were told that the aim of the study was to learn about children's and mother's emotions.

In order to determine eligibility for the study, research assistants administered a phone screen to women who expressed interest in participating with their child. The phone screen included a modified version of the Structured Clinical Interview for DSM-IV (SCID-IV) to determine whether or not they met DSM-IV criteria for depression in their child's lifetime (First, 2002). Dyads with a mother who reported current suicidality, psychosis, substance abuse, or a lifetime history of bipolar I or schizophrenia were excluded from the study. Dyads were also excluded if the child had a pervasive developmental disorder, intellectual disability, psychosis, autism, bipolar disorder, or had ever met criteria for major depressive disorder. Children who did not speak English fluently were also excluded from the study. In families where more than one

child was eligible, one child was chosen at random to participate. For respondents who were determined to be a danger to themselves or others, see appendix for the Critical Incidents Protocol. The phone call typically took between 15 and 30 minutes.

Participants

Our sample was composed of 131 children of mothers with depression (n = 65) and without a lifetime history of depression (n = 66). Roughly half (53.4%) were girls, and the average age was 9.4 years. The racial breakdown was 55% Caucasian, 31% African American, 4% Asian American, 1% Hispanic or Latino, 9% other/unspecified. About 74.2% of children had mothers who had obtained a four-year college degree or higher. About 79% came from a household where the mother was married. Median household income was between $90,000 and $110,000.

Procedure

Data for this study were collected as part of a larger study. Here we describe the procedures and measures tied to the current hypotheses. Prior to data collection, participants were informed that they could choose not to answer a question if they preferred, and that their responses would be kept confidential unless they or their child were in immediate danger of harming themselves or others (see Appendix for the Critical Incidents Protocol). After completing the phone screening (described earlier), children of mothers who had depression during the child's lifetime were assigned to the high-risk group, while children of mothers without a history of depression during the child's lifetime were assigned to the control group. Prior to the lab visit, mothers filled out several online questionnaires. In the lab, children and mothers were provided with blocks and crafts and were instructed to choose amongst them and

play together for 10 minutes. After the play session, mothers left the room and research assistants read a series of self-report questionnaires aloud (to control for reading ability) while children entered their responses on a computer. While the children were entering these responses, research assistants attached electrodes to the children's chests for collecting the data needed to calculate RSA.

Next, children were seated in front of a video monitor and instructed to pay attention to a series of 4-minute video and audio segments for the mood induction procedure. The procedure began with a neutral baseline audio clip, and then children watched a sad video clip (e.g. Lion King) and a happy video clip (e.g. Happy Feet) (order of these was randomized), each of which were followed by a neutral affect video. While the presentation order of the happy and sad clips were randomly assigned, there was always a neutral affect video after each clip. The procedure ended after a neutral affect audio clip. Children were instructed to focus on their feelings as they watched/listened and rate their mood after each subsequent clip. Heart rate data needed for the calculation of RSA were collected throughout the mood induction procedure, in separate segments for each clip.

After data collection was completed, the research assistant gave the mother a small amount of money and the child a toy.

Measures

Sociodemographic variables. Mothers self-reported information about a number of sociodemographic characteristics including child's sex, age, race, as well as mother's highest level of education completed, marital status, and annual household income.

Depression in mothers. Lifetime history of depression in mothers was assessed using the SCID-IV. To capture depression recurrence, mothers were asked to estimate the total amount of

time that they have been depressed during their child's lifetime (e.g. a few weeks, a few months, about 6 months, about a year, about 2 years, about 3 years, more than 3 years). To standardize the units in this variable, all duration responses were converted to the total number of weeks that the mothers reported as having experienced depression.

Mood repair in children. The Feelings and Me - Child Version (FAM-C) provides an index of mood repair (Kovacs, 2000). Children respond to 54 statements pertaining to specific strategies they would use if they were feeling sad or upset and were asked to rate each statement as 0 "not true of me", 1 "sometimes true of me", or 2 "often true of me." The FAM-C items describe either adaptive or maladaptive emotion regulatory responses. Adaptive items include emotion regulatory strategies that have been shown to facilitate an improvement in mood (e.g. talk about it with a friend or family member), while maladaptive items include strategies that have been shown to hinder an improvement in mood (e.g. throw, kick, or hit things). There are 30 items for the adaptive scale with potential scores ranging from 0-60, and 24 items for the maladaptive scale with potential scores ranging from 0-48. Evidence for the reliability of the FAM-C includes a Cronbach's alpha of .89 for the adaptive scale, and .87 for the maladaptive scale (Tamás et al., 2007). In the present study, Cronbach's alpha was .93 for the adaptive scale and .87 for the maladaptive scale. One year test-retest reliability yielded an intraclass correlation coefficient of 0.44 for both the adaptive and maladaptive scales (Tamás et al., 2007). The maladaptive scale of the FAM-C was found to be correlated with symptoms of depression ($r = .64$, $p < .05$) as well as rumination ($r = .71$, $p < .05$), providing evidence of construct validity (Tamás et al., 2007).

RSA in children. Child electrocardiograms (ECGs) were used to assess RSA during the mood induction procedure. Event markers were used to partition the RSA data into segments that

coincided with the mood induction segments (e.g. resting, negative, etc.) Trained research assistants edited the ECG signals using CardioEdit, and RSA was computed using CardioBatch (Brain-Body Center, 2007). Following the protocol of previous researchers, baseline RSA was computed using average RSA across the first neutral segment and RSA reactivity was calculated by subtracting baseline RSA from averaged RSA during the sad mood induction (Alkon et al., 2003).

Data Analytic Strategy

We conducted preliminary analyses to assess for significant relationships between sociodemographic variables and maternal depression, mood repair, and RSA. These analyses informed whether it was necessary to control for each sociodemographic variable.

Main analyses assessed the relationships between maternal depression, mood repair, and RSA. To test the first hypothesis, that children of depressed mothers would score lower on adaptive mood repair and higher on maladaptive mood repair, we conducted t-tests to assess whether children from high-risk and control groups differed in adaptive and maladaptive mood repair. For the second hypothesis, that children of depressed mothers would differ from controls regarding baseline RSA and RSA reactivity, we conducted t-tests to assess each RSA measure individually and we also conducted an analysis of covariance (ANCOVA) to determine whether history of maternal depression accounted for significant variance among RSA reactivity scores when accounting for baseline RSA as a covariate. For the third hypothesis, that baseline RSA and RSA reactivity are significantly associated with adaptive and maladaptive mood repair, we used Pearson's correlations to determine the strength and direction of these associations. The fourth hypothesis, specific to the subsample of children with depressed mothers, predicted that duration of mothers' depressive episodes during the child's lifetime would be associated with

children's mood repair and baseline RSA and RSA reactivity. To test this, first, we ran correlations to assess the relationship between each of the two mood repair scores and the duration of time that mothers were depressed during their child's lifetime. Second, we ran correlations to assess the relationship between each of the two RSA scores and the duration of time that mothers were depressed during their child's lifetime. Third, we ran a linear regression model to determine if the duration of mothers' depression during the child's lifetime added predictive power to the relationship between baseline RSA and RSA reactivity.

Results

Preliminary Analyses

An initial step was to examine the distribution of scores and identify potential outliers. Among women with histories of depressive episodes, the distribution of scores for duration of maternal depressive episodes was positively skewed and violated the assumption of normality for parametric data. Upon further inspection, although the mean duration was 107.79 weeks (SD = 131.45), approximately 25% of mothers with depression were depressed for between 2 and 12 weeks, 25% were depressed for 13 to 36 weeks, 25% were depressed for 37 to 180 weeks, and the top 25% were depressed for 180 to 572 weeks. Accordingly, we used nonparametric tests to examine relations between duration of maternal depressive episodes and other variables.

Not all participants provided data for all variables. In particular, data were missing on mother's depressive episode duration (n =8; 12.3%) and children's baseline RSA (n = 18; 13.7%), which precluded the calculation of a score for RSA reactivity for those children. Missing RSA scores were attributable to random problems with equipment or bad data.

We conducted chi-square tests to compare children of mothers with a history of depression to controls with no history of depression regarding dichotomous sociodemographic variables. As shown in Table 1, on average, mothers of high-risk children, relative to controls, were more likely to identify as a race other than Caucasian, and less likely to be currently married, but did not differ from controls regarding child sex or maternal education level. We also conducted t-tests to compare mean scores between groups regarding continuous sociodemographic variables. We found that mothers with depression history had a significantly lower gross household income than control mothers, though the two groups did not differ regarding child age (see Table 2).

We used t-tests, Mann-Whitney tests, Pearson's correlations, and Spearman's correlations to assess whether sociodemographic variables needed to be controlled when examining relations between children's FAM-C scores (adaptive and maladaptive mood repair), RSA scores (baseline and reactivity), and mother's duration of depressive episodes during the child's lifetime. The first of the two FAM-C scores, adaptive mood repair, was significantly associated with only one of the sociodemographic variables: child sex (see Table 3). Adaptive mood repair was not significantly associated with child age or family income (see Table 4). The second of the two FAM-C scores, maladaptive mood repair, was significantly associated with only one sociodemographic variable: race/ethnicity (see Table 5). Children who identified as other than Caucasian scored significantly higher on maladaptive mood repair than Caucasian children. Maladaptive mood repair was not significantly associated with child age or family income (see Table 4).

Baseline RSA and RSA reactivity were not significantly associated with child age or family income (see Table 4). Baseline RSA was not associated with child sex, race, mother's

marital status, or mother's education level (see Table 6). RSA reactivity was not significantly associated with child sex, race, or mother's education level; on average, however, children of mothers not currently married scored higher on RSA reactivity than children of married mothers (see Table 7).

Duration of mother's depressive episodes was not significantly related to child sex, race, mother's education, or marital status (see Table 8). Likewise, duration of mother's depressive episodes was not significantly associated with child age or family income (see Table 4).

In all, we conducted 36 tests to assess for potential confounding effects of sociodemographic variables. Due to the high number of tests, the chance of observing at least one type 1 error in these analyses was 84%. This inflated type 1 error rate, coupled with a lack of theoretical or empirical bases for expecting these variables to play a significant role, led us to decide not to control for sociodemographic variables in subsequent hypothesis testing.

Hypothesis Testing

Maternal depression and mood repair. Contrary to the first hypothesis that children of depressed mothers would score lower on adaptive mood repair and higher on maladaptive mood repair than children of mothers without a history of depression, independent samples t-tests revealed that children of depressed mothers did not significantly differ from controls on adaptive mood repair or maladaptive mood repair (see Table 9).

Maternal depression and RSA. Contrary to the second hypothesis that children of depressed mothers would differ from controls regarding baseline RSA and RSA reactivity, independent samples t-tests revealed that children of depressed mothers did not significantly differ from control children regarding baseline RSA or RSA reactivity (see Table 9). As baseline RSA is known to influence RSA reactivity, we decided to test whether maternal depression

history (depressed or not in the child's lifetime) was significantly associated with RSA reactivity by using an analysis of covariance (ANCOVA). Our ANCOVA model included baseline RSA as a covariate and depression history status as a predictor of RSA reactivity. Prior to running the analysis, we examined the relationship between baseline RSA and maternal depression. As the two were not significantly associated, we concluded that there was no violation of the assumption of independence between the covariate and predictor. We also determined that the regression slopes were parallel. After running the analysis, results indicate that there was no significant relation between maternal depression and RSA reactivity, after accounting for baseline RSA (see Table 10).

Mood repair and RSA. Contrary to the third hypothesis, that adaptive and maladaptive mood repair scores would be significantly associated with baseline RSA and RSA reactivity, Pearson's correlations revealed that neither adaptive mood repair nor maladaptive mood repair were significantly associated with baseline RSA or RSA reactivity within the sample as a whole (see Table 4). Likewise, neither adaptive mood repair nor maladaptive mood repair were significantly associated with baseline RSA and RSA reactivity when the two groups were considered separately, i.e. in children of depressed mothers or in children of mothers without a lifetime history of depression (see Table 11).

Duration of maternal depressive episodes. To test the fourth hypothesis that, for the subset of children with depressed mothers, duration of maternal depressive episodes would be associated with mood repair and RSA scores, a series of Spearman's correlations was conducted. The results of the analyses indicated that duration of maternal depressive episodes was not significantly associated with adaptive or maladaptive mood repair scores, baseline RSA, or RSA reactivity (see Table 11). Likewise, duration of maternal depressive episodes during the child's

lifetime did not add significant predictive power to the relationship between baseline RSA and RSA reactivity (see Table 12).

Discussion

Evidence for a familial influence on risk for developing depression continues to grow as numerous studies highlight a heightened prevalence of the disorder among family members of depressed individuals (e.g.Williamson et al., 2004). Within this family context, recent findings have begun to shed light on the significant relationships between depression risk and regulation of negative emotions as indexed by mood repair (Bylsma et al., 2016; Joormann & Vanderlind, 2014) and RSA (Gentzler et al., 2009), as well as the detrimental effects of a more chronic (defined as two or more episodes) course of maternal depression on children's risk for depression and emotional difficulties compared to less persistent maternal depression (Bland et al., 1986; Zubenko et al., 2001). However, few studies to date have examined mood repair and RSA specifically among children of depressed mothers or among children in middle childhood. The current study aimed to clarify these relationships by examining whether children of depressed mothers in middle childhood differ from their low-risk peers in regards to mood repair and RSA, whether mood repair is significantly related to RSA during middle childhood, and whether duration of maternal depressive episodes is related to either measure of emotion regulation.

Contrary to our hypothesis that children of depressed mothers would score lower on adaptive mood repair and higher on maladaptive mood repair than children of mothers without a history of depression, we found that the two groups did not significantly differ on either mood repair scale. This may be due in part to methodological differences between past studies and the current study. Specifically, past studies have administered the parent-rated Feelings and My Child to capture mood repair (e.g. Gentzler et al., 2009), rather than directly asking the children

using the parallel form of the measure designed for children, the FAM-C. As depression and depressive tendencies are often shared between parents and their children, and studies have found that individuals prone to depression demonstrate selective attentional biases towards negative stimuli (Gotlib, Krasnoperova, Yue, & Joormann, 2004), it is possible that these biases may be reflected in the responses of parents with depression when asked to describe their children. Another consideration is that past studies on mood repair among high-risk youth have often focused on siblings of depressed adolescents (e.g. Bylsma et al., 2016) or children of parents with a childhood-onset mood disorder, including fathers and those with bipolar disorder (Gentzler et al., 2009). As our study focused on children of mothers who had at least one depressive episode during the child's lifetime, it is possible that these past findings of atypical mood repair among high-risk youths may be due in part to upbringing (shared between siblings) or do not generalize to children of mothers with depression, but not restricted to childhood-onset.

Despite past findings of lower baseline RSA (Shannon et al., 2007) and augmented RSA reactivity (Blandon et al., 2008) among children at high risk for depression compared to controls, we did not find significant group differences on either baseline RSA and RSA reactivity. However, it is worth noting that children who scored higher on baseline RSA, which is considered to be protective against negative stressors such as maternal melancholic symptoms (Shannon et al., 2007), tended to demonstrate RSA withdrawal in response to negative emotional stimuli, which is also considered to be normative (Yaroslavsky, Bylsma, et al., 2013). Consistent with our null findings, the same study by Yaroslavsky, Rottenberg, and Kovacs (2013), examined baseline RSA and RSA reactivity among depressed adolescents and healthy controls and also found no significant group differences. However, they did find that depressive symptoms were significantly related to RSA when considering baseline RSA and RSA reactivity

jointly (Yaroslavsky, Bylsma, et al., 2013). As their study did not include a non-depressed, high-risk group, we sought to extend the scope of this investigation by examining the relationship between both forms of RSA and maternal history of depression. We found that maternal depression did not account for significant variance in RSA reactivity scores when considering baseline RSA as a covariate of RSA reactivity. With discrepancies persisting in the field and a lack of studies which examine both baseline RSA and RSA reactivity in relation to depression risk and depressive tendencies, future studies are needed to shed light on the nature of these relationships.

With regards to the relationship between mood repair and RSA, we found no significant associations between adaptive or maladaptive mood repair and baseline RSA or RSA reactivity. These findings are inconsistent with past evidence of significant relationships between emotion regulation and baseline RSA (Beauchaine, 2015) as well as RSA reactivity (Gentzler et al., 2009; Rottenberg, 2007). In a recent study, researchers found that combining baseline RSA and RSA reactivity into one score as a way to index "patterns" was more effective in predicting maladaptive mood repair among depressed and non-depressed youths than using the two RSA scores as separate predictors (Yaroslavsky et al., 2016). As the combined RSA approach of Yaroslavsky et al (2016) is still relatively new and unexplored among non-depressed, high-risk children, future studies on the utility of such an approach may shed light on the nature of the relationship between baseline RSA, RSA reactivity, and adaptive and maladaptive mood repair within the context of depression and depression risk.

Despite evidence to suggest that children of mothers with chronic (high frequency) and severe depression experience greater difficulties with emotion regulation than children of mothers with milder, more infrequent depression (e.g. Ashman et al., 2008), we did not find

significant associations between duration of maternal depressive episodes and adaptive mood repair, maladaptive mood repair, baseline RSA, or RSA reactivity among our subset of children of depressed mothers. Likewise, duration of maternal depressive episodes during the child's lifetime did not add predictive power to the relationship between baseline RSA and RSA reactivity. These discrepancies may exist in part because our model did not account for severity of maternal depressive symptoms, while previous studies have used both severity and chronicity together to examine children's emotion regulation (Ashman et al., 2008). A future step would be to include severity of maternal depressive symptoms in the model to assess their relationship with children's regulation of emotions.

Unexpectedly, we found that adaptive and maladaptive mood repair were significantly positively associated for the sample as a whole and for the depressed and non-depressed subsets. While many studies involving mood repair do not report findings for a relationship between adaptive and maladaptive mood repair in children, those that did often focused on teenagers and adolescents and reported a negative or nonsignificant association (Bylsma et al., 2016; Gentzler et al., 2009; Yaroslavsky, Bylsma, et al., 2013). One exception to these past findings was a study that reported a modest, positive association between adaptive and maladaptive mood repair among children ages 5 to 13 years old (Tamás et al., 2007). However, it is important to consider that this study covered a wide age and found age to be a covariate for both forms of mood repair (Tamás et al., 2007). For these reasons, it is possible that past findings on the relationship between adaptive and maladaptive mood repair among adolescents and teenagers do not generalize to children in middle childhood. Future studies are needed to clarify this discrepancy and account for additional factors that may influence the relationship between adaptive and maladaptive mood repair among children in middle childhood.

There are certain limitations to consider when interpreting these findings. While ethnically diverse, this was a particularly well-educated sample with 74.2% of mothers having achieved a four-year college degree or higher. Thus, our findings may only be generalizable to highly-educated populations. Similarly, the sample as a whole was of high socioeconomic status, with a median annual income between $90,000 and $110,000. There is a substantial amount of literature linking lower socioeconomic status with more extensive depressive symptoms among mothers as well as children's greater emotional difficulties (e.g. Lupien, King, Meaney, & McEwen, 2000), so it is possible that our findings do not generalize to more resource-poor families afflicted with depression. It is also important to note that we did not account for mother's age of depressive onset in this study. While past studies have reported that family members of individuals with recurrent depressive episodes are more likely to develop depression themselves than family members of individuals with childhood-onset depression, having a relative with a childhood-onset mood disorder still places an individual at increased risk for depression over and above family history alone (Zubenko et al., 2001). Notably, the same study found that age of depression onset interacts with depressive episode chronicity to confer the highest familial risk (Zubenko et al., 2001). Regarding emotion regulation specifically, children's emotion regulation has been linked to family history of childhood-onset mood disorders (e.g. Bylsma et al., 2016; Gentzler et al., 2009). Given consistent findings for a relationship between age of onset, depression risk, and difficulties with emotion regulation, it is important to account for age of onset in future analyses. Additionally, while having at least one depressive episode during the child's lifetime was part of the inclusion criteria for this study, we did not account for the specific timing of maternal depressive episodes within the child's lifetime. As maternal depressive episodes may impact children's maturation and mastery of tasks

at particular stages of development, this is another important factor to consider in future analyses (Goodman & Gotlib, 1999). Furthermore, we did not account for depressive tendencies and symptomology among the children's fathers. As fathers likely moderate the relationship between maternal depression and child psychopathology (Goodman & Gotlib, 1999), this is an important factor to consider in the future when assessing children's risk for depression within a family context.

While our hypotheses were not ultimately supported and our study design had its limitations, our study does highlight future steps that can be taken to resolve discrepancies in the literature. Regarding risk for depression and mood repair, future studies should account for the role of negative cognitive bias that can arise when parents in high-risk families report their child's effective and ineffective emotion regulation strategies when they are feeling sad or upset. For example, future studies could consider parents' scores on the Feelings and My Child alongside children's self-reported Feelings and Me- Child Version scores, or even administer a similar mood repair questionnaire to children's teachers or other adults who are likely to observe children's use of mood repair strategies from an additional, alternative viewpoint. It is also worth further examining whether findings regarding emotion regulation of individuals with a childhood-onset mood disorder generalize to families where the relative's first depressive episode onset occurred during adulthood. Our study also found that children' RSA scores were not significantly related to maternal depression status or children's strategies for mood repair. These findings reflect disagreements in the literature surrounding RSA and its relation to depression risk and depressive tendencies. Further investigation is needed to clarify these relationships so that researchers can come to a consensus. Finally, the precise nature of how chronicity of maternal depression influences children's emotional development and regulation

has yet to be understood. Future studies might take depression chronicity and its relationship with maternal depressive symptom severity, maternal emotion regulation, and parenting behaviors into account when assessing children's emotional development and regulation. Overall, future directions such as these could inform intervention strategies aimed at identifying and tackling early risk factors for the development of depression.

References

Alkon, A., Goldstein, L. H., Smider, N., Essex, M. J., Kupfer, D. J., & Boyce, W. T. (2003). Developmental and contextual influences on autonomic reactivity in young children. *Developmental psychobiology, 42*(1), 64-78.

Ashman, S. B., Dawson, G., & Panagiotides, H. (2008). Trajectories of maternal depression over 7 years: Relations with child psychophysiology and behavior and role of contextual risks. *Development and psychopathology, 20*(1), 55-77.

Avenevoli, S., Swendsen, J., He, J.-P., Burstein, M., & Merikangas, K. R. (2015). Major depression in the National Comorbidity Survey–Adolescent Supplement: prevalence, correlates, and treatment. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(1), 37-44. e32.

Beauchaine, T. P. (2015). Respiratory sinus arrhythmia: A transdiagnostic biomarker of emotion dysregulation and psychopathology. *Current opinion in psychology, 3*, 43-47.

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology, 98*(2), 338-350.

Bland, R. C., Newman, S. C., & Orn, H. (1986). Recurrent and nonrecurrent depression: a family study. *Archives of General Psychiatry, 43*(11), 1085-1089.

Blandon, A. Y., Calkins, S. D., Keane, S. P., & O'brien, M. (2008). Individual differences in trajectories of emotion regulation processes: The effects of maternal depressive symptomatology and children's physiological regulation. *Developmental psychology, 44*(4), 1110.

Bylsma, L. M., Yaroslavsky, I., Rottenberg, J., Kiss, E., Kapornai, K., Halas, K., . . . Vetró, Á. (2015). Familiality of mood repair responses among youth with and without histories of depression. *Cognition and Emotion*, 1-10.

Bylsma, L. M., Yaroslavsky, I., Rottenberg, J., Kiss, E., Kapornai, K., Halas, K., . . . Vetró, Á. (2016). Familiality of mood repair responses among youth with and without histories of depression. *Cognition and Emotion, 30*(4), 807-816.

Cribbet, M. R., Williams, P. G., Gunn, H. E., & Rau, H. K. (2011). Effects of tonic and phasic respiratory sinus arrhythmia on affective stress responses. *Emotion, 11*(1), 188.

First, M. B., Gibbon, M., Spitzer, R. L., & Williams, J. B. (Writer) & N. Y. S. P. I. Biometrics Research Department (Director). (2002). SCID 101 for DSM-IV: Training Video for the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID).

Gentzler, A. L., Rottenberg, J., Kovacs, M., George, C. J., & Morey, J. N. (2012). Atypical development of resting respiratory sinus arrhythmia in children at high risk for depression. *Developmental psychobiology, 54*(5), 556-567.

Gentzler, A. L., Santucci, A. K., Kovacs, M., & Fox, N. A. (2009). Respiratory sinus arrhythmia reactivity predicts emotion regulation and depressive symptoms in at-risk and control children. *Biological psychology, 82*(2), 156-163.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological review, 106*(3), 458.

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: a meta-analytic review. *Clinical child and family psychology review, 14*(1), 1-27.

Gotlib, I. H., Krasnoperova, E., Yue, D. N., & Joormann, J. (2004). Attentional biases for negative interpersonal stimuli in clinical depression. *Journal of abnormal psychology, 113*(1), 127.

Greenberg, P. E., Fournier, A.-A., Sisitsky, T., Pike, C. T., & Kessler, R. C. (2015). The economic burden of adults with major depressive disorder in the United States (2005 and 2010). *The Journal of clinical psychiatry, 76*(2), 155-162.

Joormann, J., & Stanton, C. H. (2016). Examining emotion regulation in depression: a review and future directions. *Behaviour research and therapy, 86*, 35-49.

Joormann, J., & Vanderlind, W. M. (2014). Emotion regulation in depression: The role of biased cognition and reduced cognitive control. *Clinical Psychological Science, 2*(4), 402-421.

Kovacs, M. (2000). The "Feelings and Me" emotion regulatory strategy utilization questionnaires. *Pittsburgh, PA: University of Pittsburgh School of Medicine*.

Kovacs, M., Rottenberg, J., & George, C. (2009). Maladaptive mood repair responses distinguish young adults with early-onset depressive disorders and predict future depression outcomes. *Psychological Medicine, 39*(11), 1841-1854.

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. *Biological psychiatry, 48*(10), 976-980.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology, 32*(4), 301-318.

Porges, S. W. (2007). The polyvagal perspective. *Biological psychology, 74*(2), 116-143.

Rottenberg, J. (2007). Cardiac vagal control in depression: a critical analysis. *Biological psychology, 74*(2), 200-211.

Sarıtaş, D., Grusec, J. E., & Gençöz, T. (2013). Warm and harsh parenting as mediators of the relation between maternal and adolescent emotion regulation. *Journal of adolescence, 36*(6), 1093-1101.

Shannon, K. E., Beauchaine, T. P., Brenner, S. L., Neuhaus, E., & Gatzke-Kopp, L. (2007). Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. *Development and psychopathology, 19*(3), 701-727.

Tamás, Z., Kovacs, M., Gentzler, A. L., Tepper, P., Gádoros, J., Kiss, E., . . . Vetró, Á. (2007). The relations of temperament and emotion self-regulation with suicidal behaviors in a clinical sample of depressed children in Hungary. *Journal of Abnormal Child Psychology, 35*(4), 640-652.

Williamson, D. E., Birmaher, B., Axelson, D. A., Ryan, N. D., & Dahl, R. E. (2004). First episode of depression in children at low and high familial risk for depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 43*(3), 291-297.

Yaroslavsky, I., Bylsma, L. M., Rottenberg, J., & Kovacs, M. (2013). Combinations of resting RSA and RSA reactivity impact maladaptive mood repair and depression symptoms. *Biological psychology, 94*(2), 272-281.

Yaroslavsky, I., Rottenberg, J., Bylsma, L. M., Jennings, J. R., George, C., Baji, I., . . . Kapornai, K. (2016). Parasympathetic nervous system activity predicts mood repair use and its effectiveness among adolescents with and without histories of major depression. *Journal of abnormal psychology, 125*(3), 323.

Yaroslavsky, I., Rottenberg, J., & Kovacs, M. (2013). The utility of combining RSA indices in depression prediction. *Journal of abnormal psychology, 122*(2), 314.

Zubenko, G. S., Zubenko, W. N., Spiker, D. G., Giles, D. E., & Kaplan, B. B. (2001). Malignancy of recurrent, early-onset major depression: A family study. *American Journal of Medical Genetics Part A, 105*(8), 690-699.

Table 1

Associations between History of Maternal Depression and Dichotomous Sociodemographic Variables

	Group					
Variable	Depressed	Non-Depressed	*n*	*dfs*	X^2	OR
Child sex (% female)	56.9	50.0	131	1, 131	.63	1.32
Race (% Caucasian)	46.2	63.6	131	1, 131	4.04*	2.05
Marital status (% married)	70.3	89.2	129	1, 129	7.73*	3.46
Maternal education (% four years of college or more)	68.3	80.0	128	1, 128	2.31	1.88

Note. OR = odds ratio.

*$p < .05$

Table 2

Differences in Continuous Sociodemographic Characteristics between Children of Depressed Mothers and Children of Mothers without a Lifetime History of Depression

Variables	Depressed	Non-Depressed	*n*	*df*	*t*	*d*
Child Age, mean (SD)	9.26 (.82)	9.44 (.88)	131	129	-1.24	-.21
Family Income, mean (SD)	6.60 (3.92)	4.37 (3.49)	127	125	3.39*	.60

Note. A family income score of 6.60 corresponds to $80,000-$90,000 while a family income score of 4.37 corresponds to $100,000-$110,000.

*$p < .05$

Table 3

Mean Scores, Standard Deviations, t-tests, and Effect Sizes for the Differences between Dichotomous Sociodemographic Groups on Adaptive Feelings and Me, Child Version Scores

	n	Mean (SD)	95% CI	*t*	*df*	*d*
Child sex						
Male	61	16.98 (6.61)	-5.74, -.36	-2.24*	129	-.40
Female	70	20.03 (8.64)				
Child race						
Caucasian	72	17.69 (7.55)	-4.76, .69	-1.48	129	-.26
Not Caucasian	59	19.72 (8.19)				
Marital status						
Currently married	103	18.27 (7.61)	-5.26, 1.62	-1.05	127	-.22
Not currently married	26	20.10 (9.06)				
Maternal Education						
Less than bachelor's degree	33	19.15 (9.14)	-2.57, 3.79	.38	126	.07
Bachelor's degree or higher	95	18.54 (7.50)				

Note. $*p < .05$

Table 4

Correlations between Continuous Sociodemographic Variables, FAM-C, RSA, and Duration of Maternal Depressive Episodes for the Sample as a Whole

Variables	1.	2.	3.	4.	5.	6.	7.
1. FAM-C Adaptive	--						
2. FAM-C Maladaptive	.63*	--					
3. Baseline RSA	-.12	-.06	--				
4. RSA Reactivity	.03	.09	-.35*	--			
5. Duration of MDE[a]	-.05	.02	.02	.10	--		
6. Child Age	.13	.09	.07	-.04	.15	--	
7. Family Income	.11	.15	-.03	-.15	-.02	-.02	--

[a]Values in row 5 and column 5 use Spearman's ρ, all other values use Pearson's *r*

*$p < .05$

Table 5

Mean Scores, Standard Deviations, t-tests, and Effect Sizes for the Differences between Dichotomous Sociodemographic Groups on Maladaptive Feelings and Me, Child Version Scores

	n	Mean (SD)	95% CI	*t*	*df*	*d*
Child sex						
Male	61	14.64 (5.73)	-3.10, 1.26	-.83	129	-.15
Female	70	15.56 (6.75)				
Child race						
Caucasian	72	14.00 (5.63)	-4.65, -.35	-2.30*	129	-.40
Not Caucasian	59	16.50 (6.81)				
Marital status						
Currently married	103	14.85 (6.03)	-4.13, 1.30	-1.03	127	-.22
Not currently married	26	16.27 (7.05)				
Maternal education						
Less than bachelor's degree	33	14.91 (7.41)	-2.87, 2.16	-.28	126	-.05
Bachelor's degree or higher	95	15.26 (5.85)				

Note. *$p < .05$

Table 6

Mean Scores, Standard Deviations, t-tests, and Effect Sizes for the Differences between Dichotomous Sociodemographic Groups on Baseline Respiratory Sinus Arrhythmia Scores

	n	Mean (SD)	95% CI	*t*	*df*	*d*
Child sex						
Male	52	6.57 (.98)	-.38, .50	.28	114	.05
Female	64	6.51 (1.33)				
Child race						
Caucasian	63	6.41 (1.08)	-.71, .16	-1.26	114	-.20
Not Caucasian	53	6.69 (1.29)				
Marital status						
Currently married	92	6.56 (1.11)	-.56, .51	-.08	112	-.02
Not currently married	22	6.58 (1.24)				
Maternal education						
Less than bachelor's degree	28	6.50 (1.18)	-.60, .35	-.52	111	.10
Bachelor's degree or higher	85	6.63 (1.07)				

Table 7

Mean Scores, Standard Deviations, t-tests, and Effect Sizes for the Differences between Dichotomous Sociodemographic Groups on Respiratory Sinus Arrhythmia Reactivity Scores

	n	Mean (SD)	95% CI	*t*	*df*	*d*
Child sex						
Male	52	.22 (.51)	-.12, .23	.61	111	.13
Female	61	.16 (.44)				
Child ethnicity						
Caucasian	61	.21 (.44)	-.14, .22	.43	111	.08
Not Caucasian	52	.17 (.51)				
Marital status						
Currently married	89	.24 (.48)	.03, .47	2.27*	109	.59
Not currently married	22	-.02 (.39)				
Maternal Education						
Less than bachelor's degree	28	.16 (.52)	-.24, .18	-.28	108	-.06
Bachelor's degree or higher	.19	.46 (.05)				

Note. $*p < .05$

Table 8

Mean Scores, Standard Deviations, Mann-Whitney's U Statistics, and Effect Sizes for the Differences between Sociodemographic Groups on Duration of Maternal Depressive Episodes

	n	Median (SD)	95% CI	*U*	*df*	ES
Child sex						
Male	25	1.54 (.13)	-.53, .17	334.00	55	-.14
Female	32	1.72 (.11)				
Child race						
Caucasian	24	1.62 (.74)	-.38, .32	378.00	55	-.04
Not Caucasian	33	1.65 (.58)				
Marital status						
Currently married	39	1.64 (.64)	-.34, .41	322.00	54	-.02
Not currently married	17	1.60 (.67)				
Maternal education						
Less than bachelor's degree	17	1.52 (.62)	-.51, .24	291.50	53	-.08
Bachelor's degree or higher	38	1.66 (.66)				

Note. Following Field (2009), we calculated effect size by converting Mann-Whitney's *U* into a z-score, then dividing the score by the square root of *N*.

Table 9

Differences between Groups on Feelings and Me, Child Version (FAM-C) and Respiratory Sinus Arrhythmia (RSA) Scores

	Group							
	Not Depressed		Depressed					
Variable	M (SD)	n	M (SD)	n	95% CI	*df*	*t*	*d*
FAM-C								
Adaptive	18.96 (7.29)	66	18.25 (8.48)	65	-2.03, 3.44	129	.51	.09
Maladaptive	14.84 (6.03)	66	15.43 (6.58)	65	-2.77, 1.59	129	-.53	-.09
RSA								
Baseline	6.49 (1.17)	56	6.59 (1.20)	60	-.54, .34	114	-.45	-.08
Reactivity	.18 (.52)	54	.20 (.43)	59	-.19, .16	111	-.18	-.04

Note. FAM-C = Feelings and Me, Child Version; RSA = Respiratory Sinus Arrhythmia

Table 10

Analysis of Covariance for Respiratory Sinus Arrhythmia Reactivity between Maternal Depression Groups, with Baseline Respiratory Sinus Arrhythmia as a Covariate

Variable	MS_e	F	df	p	η_p^2
Maternal depression	.03	.14	1	.71	.00
Baseline RSA*	3.06	15.22	1	.00	.12

Note. RSA = Respiratory Sinus Arrhythmia

*$p < .05$

Table 11

Correlations between Feelings and Me, Child Version, Respiratory Sinus Arrhythmia, and Duration of Maternal Depressive Episodes for Children of Mothers with and without a History of Depression

Variables	1	2	3	4	5
1. FAM-C Adaptive	--	.51*	-.18	.01	--
2. FAM-C Maladaptive	.73*	--	-.02	.12	--
3. Baseline RSA	-.08	-.09	--	-.38*	--
4. RSA Reactivity	.05	.06	-.32*	--	--
5. Duration of MDE[a]	-.05	.02	.02	.10	--

Note. Upper diagonal represents associations among children of non-depressed mothers, while lower diagonal represents children of mothers with a history of depression in the child's lifetime.

FAM-C = Feelings and Me, Child Version; RSA = Respiratory Sinus Arrhythmia; MDE = Maternal Depressive Episode(s)

[a]We calculated values in row 5 as Spearman's ρ, and all other values as Pearson's *r*

*$p < .05$

Table 12

Predicting Respiratory Sinus Arrhythmia Reactivity Scores from Baseline Respiratory Sinus Arrhythmia and Duration of Maternal Depressive Episodes

Predictor	β	SE	*p*	95% CI
Baseline RSA	-.12*	.05	.02	-.21, -.02
Duration of MDE	.00	.00	.40	-.00, .00

Note. $\Delta R^2 = .03$

RSA = Respiratory Sinus Arrhythmia, MDE = Maternal Depressive Episode(s)

*$p < .05$

Appendix

Critical Incidents Protocol

Purpose: To ensure assessment and documentation for any cases of current serious distress, as evidenced by suicidal ideation over the past week or other suicidal behavior in the past 30 days. These procedures are designed to provide accurate, concise instructions and lead interviewers in the appropriate direction for guidance and assistance.

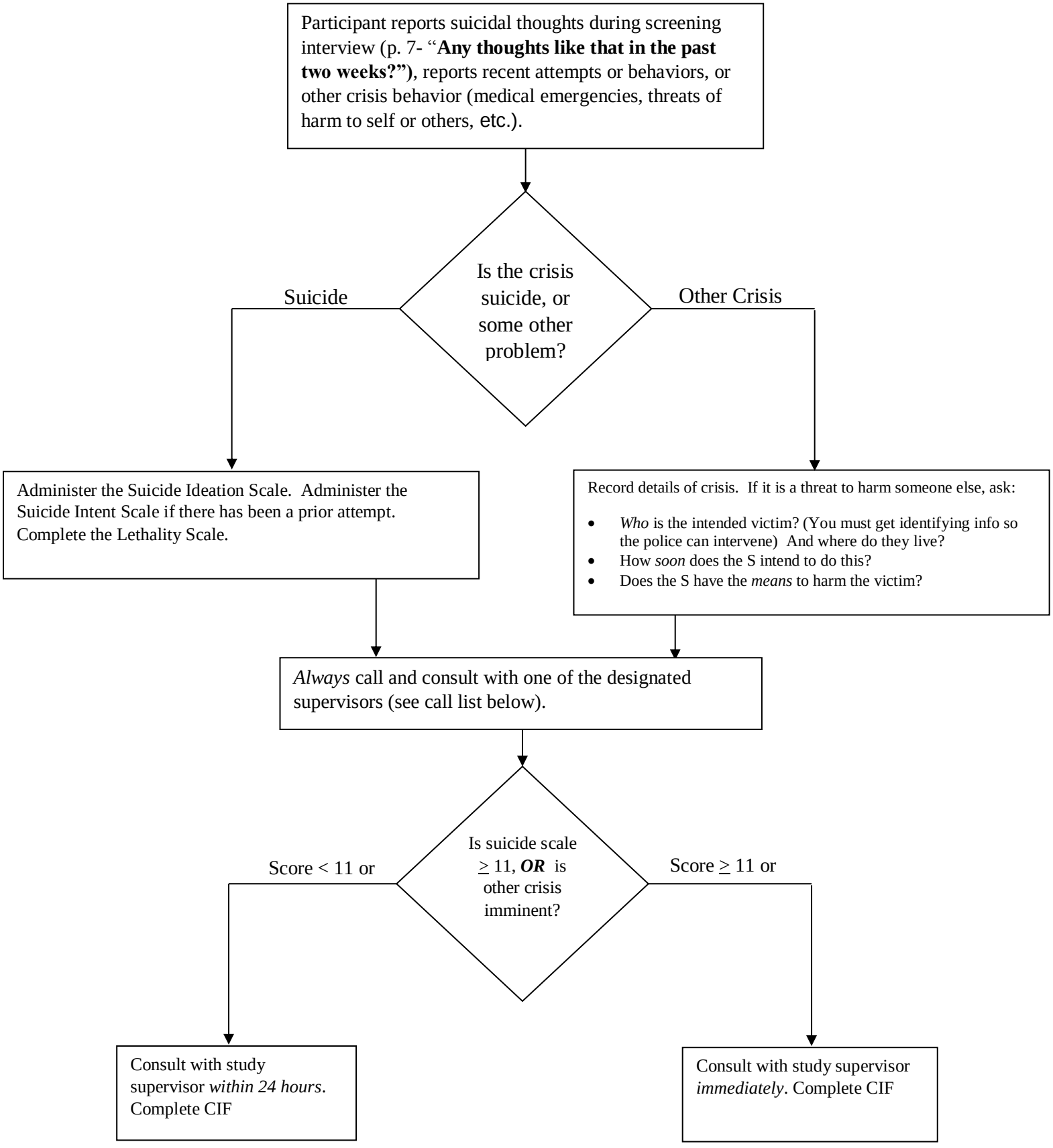
POLICIES:

- All documentation for a Critical Incident Form (CIF) must be completed immediately after the assessment and stored without identifying information in a secure, locked location.
- Interviewers are responsible for completing all appropriate documentation and making all required notifications and consultations regarding any participant with suicidal ideation. Interviewers will be held accountable for any failure to follow this procedure.
- All steps need to be documented within the CIF.
- If a participant volunteers information about suicide or another crisis during a conversation, the Suicide Ideation Scale, the Suicide Intent Scale (as appropriate), and the Critical Incident Form must be completed when the crisis is first detected.

FORMS:

- The Critical Incident Form: Summarizes the information collected, the nature and time period of the crisis, who was consulted, etc. See last page for instructions.
- The Suicide Ideation Scale: An interview guide consisting of 19 questions regarding recent ***thoughts*** ("ideation") about suicide plans. Record total score (sum of all item ratings) on the CIF.
- The Suicide Intent Scale: An interview guide consisting of 12 questions regarding the circumstances surrounding suicide ***attempt(s).*** To be used when a person has RECENTLY (past month) made an attempt on their own life. Record total score on the CIF.
- Suicide Lethality Ratings: This form is used when there has been a prior suicide attempt. Use information about the method (guns, hanging, ingestion, etc.) to rate the lethality of the most serious prior attempt on an 11 point scale. Record on the CIF whenever a suicide attempt, or near-attempt, is reported in the RECENT past.
- The Crisis Table: This table details steps to take for a variety of crises, including suicidal crises but also breaches of confidentiality, threats of harm to others, etc.
- The Crisis Flow Chart: This flow chart details steps to take, and scripts for key phrases, for dealing with an IMMINENT and SEVERE CRISIS (most often suicide threats or threats of harm to someone else that are likely to happen right away).

FLOWCHART OF CRITICAL INCIDENT ACTIVITIES



TASKS	Procedures
Detecting a Crisis	It is possible that a participant will report suicidal or other crises at any time. Whenever a suicidal or other crisis is reported, the experimenter should stop the regular assessment procedures and begin administering the suicidal ideation form(s), or other forms as necessary. Regardless of when the crisis is reported, follow these procedures. Crises include, but are not limited to: • Reports of suicidal thoughts, of recent attempts or behaviors, or of other crisis behavior (medical emergencies, threats of harm to self or others, etc.). • Suicidal plans and/or attempts • Medical emergencies (e.g., extreme psychotic behavior) • Threats of harm to someone else Call the appropriate study consultant (see below) if you are uncertain whether situation is a crisis.
Assessing Suicidal Reports	Before contacting the appropriate consultant, collect enough information to help judge the severity of the problem. If suicidal thoughts or attempts are reported, two instruments must be administered. For both of these forms, ask each item as an interview question. *Do not read the answers to the participant.* Record their answers as nearly verbatim as you can, writing on the form itself. Then rate the respondent's answer for each item as 0, 1 or 2 according to the criteria for each item (high numbers = more serious). If uncertain about how to rate a given item, save that item for your consultation (which should be immediate in this case). • The Suicide Ideation Scale: At a minimum, this form should *always* be used whenever suicidal thoughts are reported. • The Suicide Intent Scale: This form should be used whenever a suicide attempt, or near-attempt, is reported. • Lethality Scale: Complete this rating if there has been one or more past suicide attempts. The purpose is to rate how lethal the most serious suicide attempt was, on an 11-point scale. Using details collected about the method(s) employed via the Suicide Intent Scale, follow the scale instructions to make this rating.
Assessing other crises	Because crises other than suicide are unpredictable, and could consist of many different problems, we do not have a specific procedure or any specific instruments to administer. In general, please collect as much information as possible before calling the study consultant. For "Threats of Harm to others" please see below.
Assessing threats of harm to others	For threats of harm to other people, be certain to ask: • *Who* is the intended victim? • How *soon* does the participant intend to do this? • How close (or far away) does the intended victim live? • Does the participant have the *means* to harm the victim (e.g., if they are threatening to shoot someone, do they have a gun)?
When to Consult	Interviewers *must* contact a study consultant whenever there is a report of suicidal ideation, attempt, threat of harm to others, or other crisis. The issue is whether this consultation is *immediate*, or can be completed *within the next 24 hours*.

TASKS	Procedures
	Immediate: Contact study consultants whenever uncertain about a given crisis (suicide or otherwise). Interviewers are *required* to consult *immediately* whenever the participant scores 11 or higher on either the Suicide Ideation Scale or Suicide Intent Scale, or if you need to talk to someone immediately (e.g., imminent threats to harm someone else; imminent medical emergency; a suicide risk that concerns you, even if the Scale score is lower than 11). Also, if you are unable to rate certain items (as 0, 1, or 2) on either the Suicide Ideation or Intent Scales, consult immediately if that item would make the score reach 11 or higher. **Within the next 24 hours**: If neither the Suicide Ideation nor Intent scale has a total score greater than or equal to 11, then the consultation can be completed within the next 24 hours. Always complete the Critical Incident Form and notify someone on the consultant list within 24hrs.
Who to call to Consult (and in *what order*)	Follow each of these steps below in sequence until you reach a study investigator and have consulted with one of them. In each case, if you cannot reach the study investigator, leave a message including the subject ID, your name, how to reach you, and that this is a crisis. 1. Sherryl Goodman- 678-984-4477(c); 404-727-4134(w); 404-876-4441(h) 2. Meaghan McCallum-401-450-9134(c) 3. Cara Lusby-615-479-3439(c); 404 727 0249 4. 1-800-273-8255- National Suicide Prevention 5. If you cannot reach anyone, and the crisis is imminent, follow the procedures below for "What if you cannot reach the study staff to consult?"
What if you cannot reach the study staff to consult?	**Immediate or High Risk Crisis** If you cannot reach any of these study consultants, AND there is a suicidal crisis requiring *immediate* consultation, THEN follow the procedures on the suicide crisis flowchart (summarized here): • Call Respond (327-7000). Explain the situation and request that they talk to the subject to evaluate the extent of risk. • If this fails, and the crisis is imminent, call 911 and have the police escort participant to the ER. **If Crisis is not Imminent or High Risk** If you cannot reach any of the study investigators, AND the crisis does *not* require immediate consultation, continue trying to contact one of the consulting supervisors until you reach one (to be completed within 24 hrs).
Completing a Critical Incident Form (CIF)	See the next page for specific item by item instructions. • The CIF *must* be completed immediately • See the sample on the next page for specific item instructions. • Write clearly and express all relevant elements of the conversation with the participant, and subsequent consults. • On the template of the write-up, it is imperative to include that a critical incident form has been completed and give a 4-6 sentence concise information statement explaining the CI.

TASKS	Procedures
	Send e-mail to Sherryl Goodman, notifying them that a CI has been completed.