The Effect of Early Family Conflict on Psychological and Biological Processes in Young Women

Sneha Venkatraman

Vanderbilt University
Introduction

Decades of psychological research have provided evidence that the family environment strongly impacts the social and emotional growth of an individual. This is in part because children begin to model behavior and feelings after the individuals they interact with during young ages, thus the early years are critical for development. Since children learn emotion regulation and management through modeling and observational learning, studies have shown that family practices affect future emotion regulation (Morris et al., 2009). Thus, it is important to have strong family support and low levels of conflict at a young age. Crick and Dodge (1993) explained that childhood events lead to the later development of mental structures that aid with processing social information.

However, prior research has shown that individuals who have experienced higher levels of family conflict in the past have had increased social and emotional difficulties. Davies and Cummings (1994) found that family variables such as elevated conflict and limited affection during childhood led to negative effects such as negative cognitive schemas, exaggerated threat appraisals, and poor emotional self-regulatory ability. They conjectured that prior events would have a negative impact on emotional and cognitive self-regulatory processes after childhood. Poor self-regulatory abilities and heightened emotional arousal often result from adverse, early environments (Repetti, Taylor, & Seeman 2002). However, though family context has a clear impact on the development of individuals, Darling and Steinberg (1993) reported that the mechanisms through which family conflict causes developmental effects have not been elucidated. Thus, further research is still necessary to understand how family history effects the emotional and social development of children and the effects of the development later in life.
From the observation that early family life has significant effects on the ability to regulate their emotions and may lead to consequences of having more negative cognitive schemas, it follows that individuals who have experienced higher levels of family conflict are at a higher risk for psychological disorders. Two of the most commonly experienced psychological disorders are major depressive disorder and generalized anxiety disorder. These two disorders have a high comorbidity rate and are found in higher rates in individuals who come from families with higher levels of conflict. Achenbach observed that in many cases, individuals showed several symptoms for anxiety and depression but were not diagnosed with generalized anxiety disorder or major depressive disorder because they did not exhibit enough of the symptoms. In addition, many of these individuals had several symptoms of both anxiety and depression. He created the Anxiety/Depressed scale to account for this combined manifestation of anxiety and depressive symptoms (Achenbach & Rescorla, 2003) since many studies showed comorbidity for these symptoms. For example, study focusing on young women who experienced childhood sexual abuse showed an association between childhood abuse and later symptoms of anxiety and depression (Yama, Tovey, & Fogas, 1993). Family conflict was found to be a moderating variable that increased current symptoms of depression. Family control and cohesiveness were moderating variables that decreased symptoms of depression. Another study by Herrenkohl (2009) et al. found that adults who experienced higher levels of family conflict earlier during adolescence as opposed to later had higher levels of depressive symptoms as adults. However, due to conflicting findings in research and inconsistent methods, Rapee (2011) found after conducting a recent review that considerably more research is necessary to show how certain family factors play key roles in the development of anxiety disorders.
Parental loss or divorce at a young age, a related topic with similar implications as childhood family conflict, has been hypothesized to have large effects on one’s development. For example, Luecken and Appelhans (2005) studied this topic since it has been hypothesized to increase risk for affective disorders such as depression, bipolarity, and other anxiety related disorders. One-hundred nine undergraduate students participated in this research study with ages ranging from 18 to 29 years. For the participants from bereaved and divorced families, the loss or divorce must have occurred before the age of 16 so that earlier development could be separated from effects that occur late in adolescent development (Luecken & Appelhans, 2005). Various questionnaires were administered to determine the effects of family cohesion or separation such as the Child Trauma Questionnaire (CTQ), Family Environmental Scale (FES), and the State-Trait Anxiety Inventory. Attention was also studied in the participants in the form of a dot-probe computer task. 48 social threat-neutral word pairs along with 48 loss related-neutral word pairs and 48 neutral-neutral word pairs were presented on the screen. The results showed that the participants from intact families avoided the social threat-neutral word pairs and the loss related-neutral word pairs by taking longer to respond to these word pairs. Those from divorced families paid more attention to the loss related-neutral word pairs, suggesting that they were looking for danger and hypersensitive to these words that may have bothered them during the experiment. Surprisingly, those from bereaved families did not avoid or respond more quickly to any of the word pairs, contrary to the hypothesis of the researchers. After controlling for group differences, the researchers concluded that the participants from divorced and bereaved families no longer had the protective bias against loss and social threat related words, which may result in higher rates of affective disorders.
Not only have psychological effects been documented among individuals who have experienced higher rates of family conflict, but biological effects have been noted as well. The first result stems directly from childhood abuse, a factor seen frequently in families with high levels of conflict. Due to early childhood abuse, many adults have experienced chronic health problems and have suffered from significantly more diagnoses of varying physical ailments (Walker et al., 1999). Another study conducted by Felitti and colleagues (1998) demonstrated that a strong relationship existed between the degree of abuse and household dysfunction during childhood and the risk for diseases such as ischemic heart disease, skeletal fractures, cancer, chronic lung disease, and liver disease.

These psychological and biological problems may have an underlying cause that relates to stress. Stress is defined as “a physical, chemical, or emotional factor that causes bodily or mental tension and may be a factor in disease causation” (Anderson, 2005). It is strongly linked to higher rates of depression and anxiety. In many studies, neuroendocrinal activity is measured through cortisol, which along with heart rate, is a biological measure often used to quantify stress. Cortisol, classified as a glucocorticoid of the hypothalamic-pituitary-adrenal axis, is a stress hormone that is produced by the adrenal glands in response to stressful situations. By periodically measuring cortisol levels before and after a stressful event, one can measure the peaks in the stress hormone and how long it may take for cortisol levels to return to baseline (MacMillan et al., 2009). The increase of cortisol in the bloodstream is a process needed to efficiently respond to a stressor. Since a supporting, positive experience from an early age is crucial for the development of physiological regulatory processes, this response can be disrupted through suffering from early psychological stress (Gunnar & Quevedo, 2007; cited in Luecken
This is often the case for individuals from families of high conflict. In recent literature, family conflict has shown to cause changes in normal cortisol reactivity.

High levels of stress have been linked to individuals who have experienced family conflict. A study by Luecken and Appelhans (2006) linked early family adversity to changes in neuroendocrine functioning that caused detrimental physical and psychological effects. Young adults who were recruited for this study reported prior experiences of abuse and family conflict in conjunction with parental loss or no parental loss. The participants were asked to deliver a speech designed to elicit physiological stress responses. Luecken and Appelhans found that individuals who had experienced a parental loss showed the relationship between higher levels of family conflict and higher levels of cortisol. Individuals in the control group who had not experienced parental loss did not show a relationship between higher levels of family conflict and stress responses. Thus, parental loss served as a moderator for family conflict and neuroendocrine activity. From this study, the relationship between parental loss and stress was seen, but no clear conclusion had been reached concerning stress and family conflict.

Another study conducted by Luecken, Kraft, and Hagan, (2009) demonstrated that early adverse relationship with the family resulted in higher physiological stress responses in young adulthood. The stress responses were measured through comparing salivary cortisol levels between groups who experience highly adverse relationship with their families and those who experienced positive relationships with their families during childhood. The participants in this study were asked to engage in a role-play task that served as the stressor to measure the peaks and flow of cortisol over a period of 40 minutes. The challenging task reflected a real-life situation, which often results in a wider range of emotional and behavioral responses than those that focus on mental arithmetic (Waldstein et al., 1998; cited in Luecken, Kraft, & Hagan 2009).
The participant was asked to role-play as a student who wanted to convince their neighbor to decrease the volume level of her music so that the participant could study for an important exam. The duration of the task was 10 minutes. To increase stress responses, the interaction between the participant and the same-sex research assistant was videotaped. The research assistant, who was given a set of scripted responses that demonstrated that she did not want to comply to the participant’s request, was asked to maintain a neutral expression throughout the task. Results from this procedure demonstrated that participants who came from families with higher levels of early conflict had significantly lower levels of salivary cortisol throughout the duration of the study. Thus, the relationship between the HPA axis and early family conflict is once again seen, but the results differ from other studies, including Luecken’s earlier study on parental loss and cortisol levels.

Both physiological and emotional stress responses are not only modified by prior conflict, but can also be modified by gender. Women are at a higher risk for affective disorders which are often exacerbated by stress, such as major depressive disorder and bipolar disorder (Kessler et al., 2005). Physiological responses to stress have also been found to vary by gender (Kirschbaum, Wust, & Hellhammer, 1992). However, little research has been conducted on the effects of chronic stress in women.

Age and stage in life can also severely impact levels of stress on an everyday basis. One study was completed by Ross, Niebling, and Heckert (1999) to find the main sources of stress for college students. This study found that daily hassles were reported more often than major life events. College students are often reported to have higher levels of stress in comparison to other age groups. This is often because of unknown factors in their lives, such as occupation opportunities after graduation or stress concerning finding a life partner. Feelings of loneliness,
nervousness, sleeplessness, and anxiety were reported in students with higher levels of stress. Along with academic stressors such as unclear expectations in the classroom or examinations with high levels of difficulty, social stressors were also found to be important in affecting the lives of college students. Relationships with friends and family often had negative effects on eating and sleeping habits and caused feelings of loneliness in some students (Wright, 1967; cited in Ross, Niebling, Heckert, 1999). This study demonstrated the importance of studying stress levels in college students. Many of the questionnaires used in this study are beneficial and relevant to the study of stress in college students, such as the CPIC (Children's Perception of Interparental Conflict), RSQ (Responses to Stress Questionnaire), and the PANAS (Positive and Negative Affect Schedule). Determining the effects of stress on emotional and physiological process in young adults can help psychologists develop programs specifically aimed at college students who have had stressful experiences in the past. Since young adults experience more stress at this age and less research has been conducted on physiological and emotional stress reactivity in women, young women in college are an important group to study.

Because of the conflicting theories on cortisol levels in response to stress, studying coping in individuals is important since it may illuminate the differences between those who have normal patterns of stress reactivity and those who show flattened curves of cortisol levels. In chronically-stressed populations, such as college students who have suffered from high levels of family conflict, using of emotion regulation and coping strategies can often increase the chance of psychological and health related outcomes. Many studies have shown that a three-factor model of coping effectively categorizes various coping methods in both adolescents and adults (Compas et al, 2006). The first is primary control engagement coping which involves efforts to act directly on the efforts to act directly on the source of stress or one’s emotions. This
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includes problem solving, emotional expression, and emotional modulation. Secondary control engagement coping strategies measure the efforts to adapt to the source of stress rather than alter the circumstances. These include cognitive restructuring, positive thinking, acceptance, and distraction. The third is disengagement coping which involves efforts to cognitively or behaviorally withdraw from the source of stress by engaging in behaviors such as wishful thinking, avoidance, and denial. Compas and colleagues designed and validated a coping measure called the Responses to Stress Questionaire (Connor-Smith et al., 2000). It includes a Primary Control Coping Scale, Secondary Control Coping Scale, and a Disengagement Coping Scale. In addition, a fourth scale measures involuntary engagement or stress reactivity with subscales on emotional and physiological reactivity. An updated version of this questionnaire called the Responses to Stress Questionaire-II (RSQ-II) which also includes items measuring volitional coping responses and involuntary responses to stress was created. Engagement and disengagement responses are included for volitional and involuntary responses, with the volitional engagement responses further divided into primary and secondary control coping.

Coping has been linked to fewer depressive symptoms and physiological arousal from stress (Compas et al., 2006). Since coping is closely tied to emotion regulation in response to stress, it is important to study to create a holistic view on how social and emotional regulation, psychopathology, physiology, and prior family conflict interact and affect one another. However, which coping strategies are linked to family conflict levels and how they affect the psychological health of those who have experienced high levels of conflict is not fully understood.

For these reasons, we hypothesize that young women with higher rates of conflict in their family background will demonstrate changes from normal levels of cortisol and total cortisol output after exposure to an acute stressor. Since more recent studies with exact procedural
methods such as the study by Luecken (2009) have shown the flattening effect for individuals who have experienced higher levels of family conflict, we predicted that those with high-family conflict will have significantly lower amounts of cortisol at baseline and throughout the experiment. Because prior family conflict and stress is also associated with psychological affective disorders such as depression and anxiety, we hypothesize that young women with higher rates of prior family conflict will have significantly higher rates for both depression and anxiety. In addition, because coping methods can often illuminate the differences in stress reactivity, we believe that those who have low levels of prior family conflict will demonstrate more effective and more positive coping methods such as secondary control engagement coping.

Methods

Participants

One-hundred and sixteen female participants were recruited for this study. All were degree-seeking undergraduate students from Vanderbilt University. The participants were recruited through an online subject pool management system called Sona Systems that allowed students to receive course credit for their participation. The mean age of the sample was 18.96 years ($SD = 1.13$; range = 18 - 22 years). The sample was 79.3% Caucasian, 8.6% African-American, 6.9% Asian-American, 4.3% Hispanic-American, and 0.9% reported mixed race/ethnicity.

Measures

Demographics. All participants completed a demographics questionnaire to collect information on family structure, annual family income, parent education level, and non-academic extracurricular or work activities.

Cortisol. Salivary samples were collected from the participants at five points during the study. The saliva samples reflect the unbound “free” levels of cortisol in the plasma. The
Salivette sampling device created by Sarstedt from Rommelsdorf, Germany were used. During the experiment, participants are asked to place the cotton swab inside their mouth without letting it touch their hands and lightly chew on the swab for two minutes. The swabs were frozen immediately and stored at \(-30^\circ\text{C}\) for one to three months. The samples were then sent to Dr. Kirschbaum’s laboratory in Dresden, Germany. Because cortisol fluctuates diurnally during the day, all participants will be run between 2:00 PM and 6:00 PM on weekdays. To eliminate effects of various substances and chemicals on cortisol levels, all participants were asked not to eat, drink, smoke, or exercise two hours prior to the start of the study. Analysis of cortisol will be conducted in duplicate and the mean level of the two tests will be used in all analyses.

*Family Conflict.* All participants completed the nine-item conflict subscale of the Family Environment Scale (Moos & Moos, 1981). These items were designed to measure openly expressed anger and conflict among family members. Each item is a statement that the participant responds to by selecting true or false. In addition, all participants completed the frequency (6 items) and intensity (7 items) subscales of the Children’s Perception of Interparental Conflict Scale (Grych, Seid, & Fincham, 1992). This scale is designed to measure various aspects of conflict occurring in the home from a child’s perspective. Each item is rated on a 3-point scale (true/sort of true/false). Each scale has demonstrated adequate validity and internal consistency in samples of older adolescents. Standard scores from the Family Environment Scale and Children’s Perception of Interparental Conflict Scale were combined to create an index of self-reported family conflict during development.

*Psychopathology.* Symptoms of depression and anxiety were assessed through the Adult Self Report (ASR), a widely used self-report measure assessing emotional problems, behavioral problems, and social competence. It has been normed on a nationally representative sample
(Achenbach & Rescorla, 2001). The ASR contains 113 items scored on a three-point scale indicating how descriptive the items are of the individual during the preceding six months. The measure includes DSM-oriented scales for depression and anxiety and the Anxious/Depressed Scale which combines symptoms of both depression and anxiety. It also includes both Borderline and Clinical cutoffs that can be used to describe an individual’s responses with respect to the normative sample, taking into account the participant’s gender. For the narrow-band scales (anxiety/depression, affective problems), the Borderline range includes T scores ranging from 65-69, and T scores of 70 (98th %ile) and above fall in the Clinical range. The measure maintains high test-retest reliabilities and internal consistency scores for all subscales in a nationally representative sample. The current analyses utilized the Affective Problems scale as an index of depressive symptoms (items include lack of enjoyment, sleep disruption, appetite disturbance, sadness, suicidal ideation, underactivity, feelings of worthlessness).

**Coping.** All participants completed the RSQ-II which effectively measures and compares coping methods (Connor-Smith et al., 2000). This study analyzed responses to secondary control engagement coping items, which include cognitive restructuring (e.g., I tell myself that things could be worse), positive thinking (e.g., I tell myself that everything will be alright), acceptance (e.g., I just take things the way they are, I go with the flow), and distraction (e.g., I imagine something really fun or exciting happening in my life). The six items assessing cognitive reappraisal from the Secondary Control Coping Scale were also included separately in this analysis.

**Stress Reactivity Task.** All participants took part in the Noisy Neighbor Task which served as the acute laboratory stressor that was used to measure psychological and biological stress reactivity (Luecken, 2009). The task began with the experimenter explaining that the
participant would engage in a role-play. The experimenter states, “You are trying to study for an important exam. You really need to do well on this exam, but you can’t concentrate because your neighbor is playing her music to loud. You decide to ask her to turn down her music so you can study.” A video camera is set up and begins recording before the research assistant playing the Noisy Neighbor enters the room. The participant stands approximately 30 inches away from the research assistant. The experimenter remains in the room for this task and pretends to take notes on the interaction between the participant and research assistant. The duration of the task is ten minutes, though the participant is never told when the task will end. The research assistant is instructed to speak in a clear and monotonous voice and to not show emotion through facial expressions or body language. The research assistant remains still and unresponsive with their hands at their sides. The research assistant always waits for five seconds after the participant has stopped talking to respond.

After the participant starts speaking, the research assistant responds to the participant with the following prompts, in order, to mimic a conversation:

“Why”

“Don’t you like this music?”

“I like it like this”

“I’ll think about it”

“I don’t think it’s too loud”

“It’s my apartment”

“No one else has a problem with it”38

“Hey, we’re having a party”

“I have my rights too”
“I’ve never asked you to turn down your music’

“I don’t know”

“I don’t want to”

“I don’t see why I should turn it down”

“This is my favorite song”

"It isn't that loud"

"It's still early"

"It hasn't been playing that long"

"You can study with it on"

"Come on, we won't be playing it that much longer, only a couple of hours"

"Get some ear plugs. I'll be glad to get you some cotton if you don't have any."

“Why does it matter if the music is loud?

If all of the prompts are delivered before 10 minutes have elapsed, the research assistant will start again at the top of the list and respond with the prompts in the same order. If the participant stops speaking, the experimenter urges the participant to try her best and to continue with the task.

Procedure

Prior to the study, all participants received an email requesting for the participant to avoid certain actions or substances, such as eating, drinking, engaging in heavy exercise, smoking, or caffeine, and over-the-counter medications. Upon arrival, the participant was welcomed into a room and completed the consent form and Participant Screener and Measurements form which confirmed that the participant avoided the actions and substances listed. The participant was then asked to rinse her mouth and to sit in the room for 10 minutes so that a baseline cortisol
sample could be collected. After 10 minutes, the experimenter re-entered the room and collected the first saliva sample. The experimenter then explained the stress reactivity task and the participant engaged in the Noisy Neighbor task. The second cortisol sample was taken after this task.

The participant then completed two dot-probe tasks on the computer. These tasks were used to find if attentional biases existed in those who had previously been exposed to chronic stress by measuring their reaction to social threat words in comparison to neutral words. This was important to determine since individuals from intact families showed avoidance to social threat words and those from divorced and bereaved families did not show avoidance or vigilance (Luecken & Appelhans, 2009).

After this task, which lasted for approximately 15 minutes, the third saliva sample was collected. The participant was then given a questionnaire packet which included the demographic, coping measures (RSQ), psychopathological measures (ASR), and family conflict measures (FES, CPIC). Fifteen minutes into starting the questionnaires, the experimenter collected the fourth saliva sample. The fifth saliva sample followed twenty minutes later, thirty five minutes after the start of the questionnaires. The participant then completed various cognitive tests, was debriefed, and thanked for their participation.

Data Analyses

Data analyses first focused on using descriptive statistics to analyze the demographics of the current sample. All data for a participant was removed from analyses if scores on either self-report or salivary cortisol measures were outside the range of three standard deviations from the mean. A total of two participants were found to have scores outside this range for salivary cortisol, and their data was dropped from all analyses. Pearson correlations were used to examine
the relations among measures of family conflict, salivary cortisol levels, symptoms of anxiety and depression, and coping strategies. Finally, hierarchical linear regression analyses were used to ascertain the individual and total contributions to variance in baseline and total cortisol output accounted for by family conflict, secondary control engagement coping, and DSM symptoms of anxiety and depression.

Results

Descriptive Statistics

The descriptive statistics for family conflict measured through the CPIC, salivary cortisol, DSM depression and anxiety, anxious depressed, and coping methods are presented in Table 1. The means, standard deviations, minimum values, and maximum values are given for the variables being studied. The $T$-scores for the DSM anxiety, DSM depression, and the Anxious/Depressed Scale on the ASR have normative values of a mean of 50 and a standard deviation of 10. Thus, the means in this population are slightly higher than the normative mean (approximately one-half to two-thirds of a standard deviation above the normative means), but the values of the participants are closer together since the standard deviation is less than 10.

Correlations

Pearson correlations for the variables being measured in this study are reported in Table 2. The first set of correlations in the upper left corner of the correlation matrix relate to family conflict and cortisol measures. Family conflict measured by the CPIC was significantly related to both baseline cortisol ($r = .192, p < .05$) and area-under-the-curve measurements for total cortisol output ($r = .190, p < .05$). The correlation between family conflict and cortisol levels did not change when the method of cortisol measurement changed (baseline levels of cortisol and area-under-the-curve cortisol output). Baseline cortisol and area-under-the-curve measurements
were significantly related ($r = .834$, $p < .01$).

The second set of correlations in the middle of the correlation matrix in Table 2 reflects the relationship between family conflict, cortisol values, and symptoms of anxiety and depression. The CPIC as a measure of family conflict significantly correlated with the DSM measure of anxiety ($r = .234$, $p < .05$). In addition, DSM symptoms of anxiety, DSM symptoms of depression, and the Anxious/Depressed Scale were all significantly correlated with each other. DSM anxiety and depression were significantly correlated ($r = .663$, $p < .01$). The Anxious/Depressed Scale significantly correlated with DSM depression ($r = .873$, $p < .01$) and with DSM anxiety ($r = .783$, $p < .01$).

The last set of correlations at the bottom of the correlation matrix in Table 2 related coping methods from the RSQ-II to family conflict, cortisol values, and symptoms of anxiety and depression. Family conflict was significantly negatively correlated with Secondary Control Coping scale of the RSQ-II ($r = -.209$, $p < .05$). The correlation between family conflict and involuntary engagement/stress reactivity from the RSQ-II was positive and significant ($r = .154$, $p < .05$).

Symptoms of depression and anxiety shared relationships with coping methods as well. DSM depressive symptoms significantly negatively correlated with both the Primary Control Coping ($r = -.327$, $p < .01$) and Secondary Control Coping ($r = -.354$, $p < .01$) scales. DSM depressive symptoms and the Disengagement Control Coping scale of the RSQ-II were significantly positively related ($r = .416$, $p < .05$). DSM anxiety symptoms significantly negatively correlated with the Primary Control Coping ($r = -.200$, $p < .05$) and Secondary Control Coping ($r = -.328$, $p < .01$) scales. DSM anxiety symptoms were significantly positively correlated with the Disengagement Coping Scale of the RSQ-II ($r = .297$, $p < .01$). The
Anxious/Depressed Scale was significantly negatively related to the Primary Control Coping (r = -0.356, p < .01) and Secondary Control Coping (r = -0.396, p < .01) scales. Anxious depressed was significantly positively correlated with the Disengagement Coping (r = 0.475, p < .01) and involuntary engagement/stress reactivity (r = 0.187, p < .05) scales of the RSQ-II.

Finally, coping methods were significantly correlated to one another. Primary Control Coping was significantly positively correlated to Secondary Control Coping (r = 0.259, p < .01) and significantly negatively correlated to Disengagement Coping (r = -0.271, p < .01) and involuntary engagement/stress reactivity (r = -0.535, p < .01). The Secondary Control Coping and involuntary engagement/stress reactivity scales from the RSQ-II were significantly negatively correlated (r = -0.410, p < .05).

**Linear Multiple Regression Analyses**

Hierarchical linear multiple regression analyses were conducted to predict baseline levels of cortisol and total cortisol output measured through area-under-the-curve analysis. These analyses were conducted using CPIC Scores, Secondary Control Coping Scores from the RSQ-II, DSM Anxiety Problems T-Scores, and DSM Depressive Symptoms T-Scores. We constructed regression models in which the CPIC family conflict score was added to the model before the RSQ-II Secondary Control Coping Score (step 2). Following step 2, the DSM Anxiety Problems T-Score was added to the model (step 3). The total contributions are reported in step 4 after the DSM Depressive T-Score has been added. This allows us to assess the unique and shared contribution of each measure in the prediction of baseline and area-under-the-curve cortisol levels.

As seen in Table 3, the CPIC score was significant in predicting both baseline and area-under-the-curve measures for cortisol. The CPIC score remained significant in both regression...
models after the RSQ-II Secondary Control Coping Scores, DSM Anxiety Problems T-Scores, and DSM Depressive Symptoms T-Scores were entered. Secondary control coping, depression, and anxiety were not significant predictors of baseline cortisol or area-under-the-curve cortisol output. The effect size measured by $sr^2$, or the unique variance accounted for by each predictor, was four times greater for family conflict measured through CPIC scores as for DSM depressive symptoms in the model predicting baseline cortisol levels. The effect size for family conflict was approximately eight times greater than the effects for secondary control coping, DSM anxiety problems, and DSM depressive symptoms. The slope of the line measured by $\beta$ showed that the regression model created from CPIC scores and baseline cortisol data was significant and positive ($\beta = .19, p < .05$) and remained significant with the addition of secondary control coping, anxiety symptoms, and depressive symptoms. The line for the area-under-the-curve regression model predicted from CPIC scores was significant with a positive slope ($\beta = .19, p < .05$) and remained significant with the addition of the same variables.

**Discussion**

The current study analyzed the association between family conflict, salivary cortisol levels, symptoms of anxiety and depression, and coping methods in young women. Different measures of cortisol levels, symptoms of anxiety and depression, and coping were also analyzed so that the measures of similar items could be compared to one another for internal validity and examine relationships between coping strategies. Evidence was found for the relationship between family conflict and baseline salivary cortisol levels and total cortisol output measured through area-under-the-curve analysis. Support was also found for the relationship between family conflict and DSM levels of anxiety.
In addition, higher levels of family conflict were also found to be directly related to lower scores on the Secondary Control Coping Scale of the RSQ-II as predicted. This indicates that the more that a participant was exposed to high levels of family conflict, the less they engaged in cognitive restructuring, positive thinking, acceptance, and distraction as means of coping. Higher scores on the RSQ-II scale measuring involuntary engagement which includes subscales of emotional and physiological reactivity was related to higher levels of family conflict. Lastly, DSM symptoms of anxiety and depression were negatively related to primary control coping and secondary control coping and positively related to disengagement coping. Higher scores on the Anxious/Depressed scale were negatively related to primary and secondary control coping and positively related to disengagement coping and involuntary engagement and stress reactivity. Thus, the more symptoms of anxiety and depression a participant experiences, the less they used primary control engagement coping strategies such as problem solving, emotional expression, and emotional modulation. More symptoms of anxiety and depression were correlated with more disengagement engagement coping strategies such as wishful thinking, avoidance, and denial.

Thus, the results from this study have many implications on current knowledge of stress reactivity and its relation to prior family conflict, psychopathology and coping. Through the regression analysis, we determined the impact of early family conflict, anxiety and depression, and secondary control coping on both baseline cortisol and area-under-the-curve total cortisol output. For both measures of cortisol, only family conflict had a significant impact on the cortisol levels. This suggests that DSM symptoms of depression and anxiety measured through the ASR and coping did not significantly impact cortisol levels, seen also through the correlational analyses. Thus, by knowing an individual’s family history, one could predict
baseline cortisol levels and total cortisol output. The fact that family conflict at an early age and not current symptoms of depression and anxiety or even an individual’s secondary control coping methods influences an individual’s baseline cortisol and cortisol output demonstrates how early life experiences significantly impacts current stress physiology.

Higher levels of early family conflict were linked to higher levels of baseline cortisol and total cortisol output measured through area-under-the-curve analysis. This finding is not consistent with the study by Luecken, Kraft, and Hagen (2009), which found that those who had experienced family conflict had lower cortisol levels throughout the experiment. However, our findings are consistent with an earlier study by Luecken and Appelhans (2006) which demonstrated that for the group that had experienced parental loss, those that had more conflict and abuse in their past had higher levels of cortisol throughout the study. Because the baseline cortisol levels of the participants who had experienced high family were elevated in comparison to those who had experienced less family conflict, this could be interpreted to mean that those with conflict in their past have persistently higher levels of cortisol even when under non-stressful circumstances. However, Luecken and Appelhans attributed the high baseline cortisol levels to stress from participation in the study. This is concordant with the allostatic load hypothesis which states that “physiological dysregulation at rest, during reactivity, and during recovery from stress contributes to long-term health status” (Luecken and Appelhans, 2006). Thus, the laboratory atmosphere and anticipation of the study and the stress reactivity task mentioned in the consent form may contribute to higher levels of stress in those participants who had experienced family conflict. Therefore, whether participants who experienced higher levels of family conflict always have higher levels of cortisol or react more rapidly to the anticipation of stress, they are experiencing long-term and prolonged levels of cortisol in their bloodstreams.
which can be measured through bodily secretions such as saliva. Chronic higher levels of cortisol have been linked to suppressed thyroid function, decreased bone density and muscle tissue, blood sugar imbalances, higher blood pressure, decreased immune function, increased abdominal fat, and impaired cognitive performance (Ebrecht et al., 2004). These serious health effects caused by high levels of cortisol put individuals who have experienced family conflict at high risk for these health issues. Many of these health problems are strongly related with the physical illnesses that those who had suffered from more abuse and household dysfunction are at higher risks for such as skeletal fractures, heart disease, and cancer (Felitti et al., 1998). This study further supports the relationship between family conflict and its physiological effects.

The results from this study demonstrate that family conflict is significantly correlated with higher levels of anxiety. This finding is consistent with prior research (Yama, Tovey, & Fogas, 1993; Rapee, 2011) that found that increased anxiety is related to family conflict and abuse. However, depressive symptoms were not significantly related with family conflict as demonstrated by several studies (Yama, Tovey, & Fogas, 1993; Herrenkohl et al., 2009). Our hypothesis that family conflict would result in increased symptoms of both depression and anxiety was not supported.

There were several strengths to this study which expanded on previous studies and utilized effective research methods that have been validated in the past to produce reliable and valid results. There was a strict protocol which all experimenters and research assistants abided by and followed while running the study. Only previously validated questionnaires were used to measure levels of family conflict, symptoms of anxiety and depression, and coping methods such as the CPIC, the ASR, and the RSQ-II. Methods such as that used in the stress reactivity task and cortisol collection had also been used before in previous studies and had provided valid,
significant results. The large number of participants included in this study increases the statistical power of the results. The effect sizes of the correlations were also large enough to produce statistically significant results which also contributed to more statistical power in this study.

A few weaknesses still existed in the study. The participant pool did not significantly differ from a normative pool of individuals in terms of exposure to family conflict. Because few participants had extremely high levels of family conflict and many had very low levels of family conflict, the analysis could not as effectively demonstrate differences between participants with high and low exposure to family conflict. If a large number of participants with specifically high levels of family conflict could have been recruited for the study and a control group with little to no exposure to family conflict could have been matched to this population, then perhaps the differences in cortisol levels, symptoms of depression and anxiety, and coping methods could have been better elucidated.

This study on the effects of family conflict on the psychological and biological processes of young adults lends itself to future research to further understand the repercussions of exposure to family conflict from a young age. Since a clear relationship has now been established between family conflict and salivary cortisol levels in young women, perhaps future research could study a population of both young men and women to determine if family conflict affects patterns of cortisol similarly or shows marked differences between genders.

The serious health effects caused by elevated levels of cortisol direct future research towards creating interventions for those who have experienced high family conflict. Because the results from this study demonstrated that young women who experienced family conflict use less secondary control engagement coping and more involuntary engagement and higher stress
reactivity, perhaps more interventions focusing on developing secondary control coping skills and managing stress reactivity could be implemented. Since both primary and secondary control engagement coping were related to fewer anxiety and depressive symptoms, training in these coping methods could also lead to lower rates of anxiety and depression. Perhaps from this study, future research could lead to the creation of these interventions. The difference in physiological stress reactivity immediate before, immediately after, and the long-term effects of these interventions on cortisol levels, symptoms of depression and anxiety, and could demonstrate positive findings for those who have previously experienced family conflict.


Table I. Descriptive Statistics for Family Conflict, Cortisol, Anxiety and Depression, and Coping Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean (SD)</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPIC</td>
<td>23.92 (7.38)</td>
<td>14.00</td>
<td>40.00</td>
</tr>
<tr>
<td>Cortisol Baseline</td>
<td>7.22 (4.22)</td>
<td>1.79</td>
<td>27.93</td>
</tr>
<tr>
<td>Cortisol Total (auc)</td>
<td>410.38 (227.85)</td>
<td>130.50</td>
<td>1194.15</td>
</tr>
<tr>
<td>DSM Depressive (T-Score)</td>
<td>56.08 (7.15)</td>
<td>50</td>
<td>76</td>
</tr>
<tr>
<td>DSM Anxiety Problems (T-Score)</td>
<td>55.53 (7.09)</td>
<td>50</td>
<td>77</td>
</tr>
<tr>
<td>Anxious/Depressed (T-Score)</td>
<td>57.87 (8.53)</td>
<td>50</td>
<td>83</td>
</tr>
<tr>
<td>RSQ-II Primary Control Coping</td>
<td>.40 (.06)</td>
<td>.07</td>
<td>.89</td>
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<tr>
<td>RSQ-II Secondary Control Coping</td>
<td>.17 (.04)</td>
<td>.03</td>
<td>.25</td>
</tr>
<tr>
<td>RSQ-II Disengagement Coping</td>
<td>.13 (.027)</td>
<td>.02</td>
<td>.18</td>
</tr>
<tr>
<td>RSQ-II Involuntary engagement/Stress Reactivity</td>
<td>.17 (.10)</td>
<td>.02</td>
<td>.85</td>
</tr>
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</table>
Table II. Correlations Among Measures of Family Conflict, Cortisol, Anxiety and Depression, and Coping

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
<th>6.</th>
<th>7.</th>
<th>8.</th>
<th>9.</th>
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</thead>
<tbody>
<tr>
<td>1. CPIC</td>
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<td>2. Baseline Cortisol</td>
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<tr>
<td>3. Total Cortisol Output (auc)</td>
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<td>.834**</td>
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<tr>
<td>4. DSM Depressive Raw Sum</td>
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<tr>
<td>5. DSM Anxiety Problems Raw Sum</td>
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<td></td>
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<tr>
<td>6. Anxious/Depressed Raw Sum</td>
<td>.142</td>
<td>.034</td>
<td>.074</td>
<td>.873**</td>
<td>.783**</td>
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<tr>
<td>7. RSQ-II Primary Control Coping</td>
<td>-.064</td>
<td>.009</td>
<td>.041</td>
<td>-.327**</td>
<td>-.200*</td>
<td>-.356**</td>
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<tr>
<td>8. RSQ-II Secondary Control Coping</td>
<td></td>
<td>-.209*</td>
<td>-.041</td>
<td>-.008</td>
<td>-.354**</td>
<td>-.328**</td>
<td>-.396**</td>
<td>.259**</td>
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<tr>
<td>9. RSQ-II Disengagement Coping</td>
<td>-.081</td>
<td>.013</td>
<td>.047</td>
<td>.416**</td>
<td>.297**</td>
<td>.475**</td>
<td>-.271**</td>
<td>-.042</td>
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<tr>
<td>10. RSQ-II Involuntary Engagement/Stress Reactivity</td>
<td>.154*</td>
<td>-.043</td>
<td>-.064</td>
<td>.096</td>
<td>.169</td>
<td>.187*</td>
<td>-.535**</td>
<td>-.410**</td>
<td>-.044</td>
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</tbody>
</table>

*p<.05, **p<.01, ***p<.001
Table III. Step-wise Regression Predicting Baseline and Area-Under-the-Curve Cortisol Levels with CPIC Scores, Secondary Control Coping Score from the RSQ-II, Anxiety Problems T-Score from DSM, and the Depressive Symptoms T-Score from DSM

<table>
<thead>
<tr>
<th>Block</th>
<th>R² Δ</th>
<th>β</th>
<th>sr²</th>
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<tbody>
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<td>CPIC</td>
<td>.19*</td>
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<tr>
<td>Block 2</td>
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<td>CPIC</td>
<td>.19*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RSQ-II Secondary Control Coping</td>
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<tr>
<td>Block 3</td>
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<td>CPIC</td>
<td>.19*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RSQ-II Secondary Control Coping</td>
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<tr>
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<td></td>
<td>DSM Anxiety Problems T-Score</td>
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<td>.20*</td>
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<tr>
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<td></td>
<td>RSQ-II Secondary Control Coping</td>
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<td>DSM Anxiety Problems T-Score</td>
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<td></td>
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<td>DSM Depressive T-Score</td>
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<tr>
<td>Block 5</td>
<td>.04*</td>
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</tbody>
</table>

Dependent Variable: Total Cortisol Output (Area-Under-the-Curve Analysis)

<table>
<thead>
<tr>
<th>Block</th>
<th>R² Δ</th>
<th>β</th>
<th>sr²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.04*</td>
<td>CPIC</td>
<td>.19*</td>
</tr>
<tr>
<td>Block 2</td>
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<td>CPIC</td>
<td>.20*</td>
</tr>
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<td></td>
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<tr>
<td>Block 3</td>
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<td>CPIC</td>
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</tr>
<tr>
<td></td>
<td>Block 4 $R^2 \Delta = .00$</td>
<td>Block 5 $R^2 \Delta = .04^*$</td>
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</tr>
<tr>
<td>--------------------------</td>
<td>-----------------------------</td>
<td>-----------------------------</td>
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<tr>
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<td>.00</td>
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<td>DSM Anxiety Problems T-Score</td>
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<td>.00</td>
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<tr>
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<td>.04</td>
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<tr>
<td>DSM Depressive T-Score</td>
<td>.07</td>
<td>.00</td>
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*p<.05, **p<.01, ***p<.001