THESIS

EXPLORING THE PATHWAY BETWEEN FAMILY CHAOS, STRESS REACTIVITY, AND EMOTION REGULATION

Submitted by

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ABSTRACT

EXPLORING THE PATHWAY BETWEEN FAMILY CHAOS, STRESS REACTIVITY, AND EMOTION REGULATION

Family chaos is the cumulative exposure to disorienting environments (Fiese & Winter, 2010), and is associated with poor academic outcomes, health problems, and adjustment problems (e.g., Evans & Kim, 2013). Two distinguishable elements of family chaos are a) instability, or unpredictable events that disrupt continuity of the household, and b) disorganization, or enduring experiences that contribute to overwhelming disorder (Garrett-Peters et al., 2019). Although a relatively new distinction, it appears critical, in that instability is a stronger predictor of executive functioning than disorganization (Andrews et al., 2021). Our goal was to provide an investigation of this model to other key outcomes: stress reactivity and emotion regulation. A total of 153 adolescents (10-17yrs) completed the stress test and reported emotion regulation (Zeman et al., 2001; Garnefski et al., 2001). Family instability was significantly but weakly associated with disorganization, $r = .16$, $r^2 = .03$, $p < .05$. Generalized Estimating Equations controlling for age, income, and race revealed that family instability, but not disorganization, significantly negatively predicted cortisol reactivity (b= -4.65, SE= 4.17, p<.05). The distinction of family chaos into instability and disorganization requires further research to elucidate the relationship between family chaos and poor developmental outcomes.

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INTRODUCTION

Family chaos, or the cumulative exposure to extremely disruptive and disorienting environments (Fiese & Winter, 2010), includes the moving households, household cleanliness, noise level, household violence, and household density (Fiese & Winter, 2010; Evans & Kim, 2013). Family chaos is associated with increased chronic stress, dysregulated stress reactivity, poor emotion regulation, and a host of chronic illnesses (Evans & Marcynyszyn, 2004; Fiese & Winter, 2010; Evans & Kim, 2013). The chronic stress model (Evans & Kim, 2013) explains that chronic stress mediates the relationship of family chaos with acute responses to stress (e.g. physiological reactivity and emotional reactivity) and general patterns of responding to stress (e.g emotion regulation). Deficits in acute responses and general responses both contribute to poor distal outcomes, including increased externalizing and internalizing problems, greater drinking problems and conduct disorders, and higher levels of chronic diseases like heart disease, diabetes, and obesity (Evans & Kim, 2013; Fiese & Winter, 2010; Masarik & Conger, 2017).

Recently, Andrews et al. (2021) suggested that the measures of family chaos have two important subdimensions —instability and disorganization—and found that, relative to disorganization, instability was a stronger predictor of executive function. Although current research has shown family chaos, stress reactivity, and emotion regulation to be related, this association has not explored the differentiation of family chaos into instability and disorganization. The present study attempted to replicate and extend the findings of Andrews et al. (2021) in relation to executive function to stress reactivity and emotion regulation, specifically whether instability is more strongly correlated with stress reactivity and emotion regulation than disorganization. In addition, I explored if stress reactivity mediates the association between family chaos and emotion regulation, as suggested by the chronic stress model (Evans & Kim, 2013).

Acute Responses to Stress

Acute responses to stress include the physiological stress response and the emotional stress response. The emotional stress response (e.g., an increase in negative affect and/or a decrease in positive affect) involves coordination between the amygdala, which processes fear and threat response, hippocampus, which processes memory, and the prefrontal cortex, which houses executive control (National Scientific Council on the Developing Child, 2020). The physiological stress response incorporates several interrelated systems of the body to dynamically adapt to environmental stimuli. The physiological stress response involves two arms, the autonomic nervous system and the neuroendocrine system (National Scientific Council on the Developing Child, 2020). When activated by an acute stressor, the autonomic nervous system increases heart rate and breathing to provide oxygen-rich blood to the brain and muscles (National Scientific Council on the Developing Child, 2020). The neuroendocrine system regulates hormone levels, like cortisol, metabolism, and immune responses (National Scientific Council on the Developing Child, 2020). These biological systems are thoroughly enmeshed, providing and accepting feedback from the others. These systems forward signals to the brain, which in turn can alter brain chemistry and even architecture, most notably in the amygdala (National Scientific Council on the Developing Child, 2020). The physiological stress response ensures survival in the presence of a stressor; however, when stressors become chronic and overwhelm the system, the physiological stress response causes permanent physiological impairment.

Under chronic stress, the systems of the physiological stress response become overworked and cause severe, lasting damage, especially among young children and adolescents. Chronic stress causes prolonged activation of the immune system manifesting as chronic inflammation (National Scientific Council on the Developing Child, 2020). This persistent state of alert exposes the body's organs and tissues to potent inflammatory chemicals, meant to kill harmful bacteria, which can inflict damage on them (National Scientific Council on the Developing Child, 2020). The recurrent activation of the immune system during chronic stress weakens it, making the body more susceptible to infection and lifelong chronic inflammatory diseases, like cardiovascular disease, depression, diabetes, asthma, arthritis, autoimmune diseases, cancer, and dementia (National Scientific Council on the Developing Child, 2020). Unbridled levels of stress hormones produced by an overactive neuroendocrine system compounds chronic inflammation to result in insulin resistance, leading to metabolic syndrome, obesity, diabetes, cardiovascular disease, and cognitive impairment (National Scientific Council on the Developing Child, 2020). These changes to stress reactivity are exacerbated in young children experiencing chronic stress until the overworked system collapses.

Chronic stress in children often creates hyper-reactivity, or a greater sensitivity of the physiological system to stressors (Evans & Kim, 2013; National Scientific Council on the Developing Child, 2020); however, eventually chronic stress can create a collapse in stress systems, which can result in hypo-reactivity, or a reduced sensitivity of the physiological system to stressors (Lucas-Thompson, 2012; Susman, 2006). For example, low-income children, who often experience higher levels of chronic stress, have increased sympathetic nervous activity, higher HPA axis activity, and greater inflammation (Evans & Kim, 2013), indicating hyper-reactive physiological stress responses. These children also have hyper-reactive emotional stress responses (Evans & Kim, 2013; Lucas-Thompson, 2012). This reaction is a biologically adaptive response to harsh environments (Giudice et al., 2011). Although these physiological and emotional changes to stress reactivity are highly functional in the short term, they can, with time, lead to damage of these systems (Brody et al., 2014). For children and adolescents exposed to long term chronic stressors, like systemic racism and discrimination, physiological stress responses are eventually hardened, leading to less flexible reactions to stress (Lucas-Thompson, 2012; Susman, 2006; Brody et al., 2014). For instance, after exposure to long-term chronic stress, the HPA-axis becomes less resilient to stressors and less able to up- and down-regulate (Susman, 2006). However, seems to only hold true for physiological systems, creating poor coordination between physiological and emotional stress responses. Adolescents and children experiencing long-term chronic stressors have greater and more sensitive emotional reactivity (Cummings et al., 1991; Cummings et al., 2007; Lucas-Thompson, 2012). As a result, these individuals have a disruption in the coordination of their physiological and emotional stress reactions, displaying dampened stress responses and hyper-active emotional responses (Lucas-Thompson, 2012). The poor coordination of physiological and emotional systems disrupts control of emotional responses (Evans & Kim, 2013), and can lead to poor academic achievement and behavioral issues (Gumora & Arsenio, 2002; Hong et al., 2021). Targeting indices of disorganization and instability of family chaos can help mitigate the damage caused by chronic stress and reverse this disruption between physiological and emotional systems (Andrews et al., 2021; Evans & Kim, 2013).

General Patterns of Responding to Stress

An important indicator of general patterns of responding to stressors is emotion regulation (Evans & Kim, 2013). Emotion regulation is an individual's oversight of their emotions and emotional expressions that allow for appropriate functioning and optimal engagement (Evans & Kim, 2013; Hong et al., 2021; Toria et al., 2020). Developing emotion regulation in children is crucial to building academic performance and reducing emotional and behavioral issues (Gumora & Arsenio, 2002; Hong et al., 2021). Emotion regulation deficits have been well-documented in children in high chaotic environments (Evans & Kim, 2013; Hong et al., 2021; Toria et al., 2020). Deficits in emotion regulation due to chronic stress and family chaos lead to poor academic performance and internalizing and externalizing issues creating a downward cascade that perpetuates systems of poverty (National Scientific Council on the Developing Child, 2020).

Historically, this relationship has been explained solely with parenting style and actions, overlooking social processes. Punitive, harsher parenting styles, like those frequently found in chaotic environments, are also associated with poor social-emotional competence and emotion regulation (Eisenberg & Fabes, 1994; Jones et al., 2002; Morris et al., 2007). Previous research has not considered the mediating path of chronic stress from family chaos to emotion regulation deficits (Evans & Kim, 2013). Unlike the stress system that eventually deflects long term chronic stressors (Lucas-Thompson, 2012), the emotion reaction system becomes hyper-sensitive in its dysregulation (Cummings et al., 1991; Cummings et al., 2007; Lucas-Thompson, 2012). This emotional sensitivity appears to worsen emotion regulation abilities (Evans & Kim, 2013). As discussed above, chronic stress can lead to dysregulation of physiological stress responses (Cummings et al., 1991; Cummings et al., 2007; Lucas-Thompson, 2012), which in turn can alter brain structure (National Scientific Council on the Developing Child, 2020). The amygdala, which provides circuitry for fear processing and threat assessment, and the prefrontal cortex, which develops attention, impulse control, and decision making, are both affected by chronic exposure to a dysregulated stress system (National Scientific Council on the Developing Child, 2020). The development of the amygdala and prefrontal cortex are essential to the development of emotion regulation (Evans & Kim, 2013; National Scientific Council on the Developing Child, 2020). Under frequent exposure to a dysregulated stress system, these neural structures can get stuck in a state of high alert (National Scientific Council on the Developing Child, 2020). The continual threat activation of the amygdala and prefrontal cortex, as documented in hyper-active emotional responses, keep the body on high alert and delay the growth and development of these systems (Evans & Kim, 2013; National Scientific Council on the Developing Child, 2020). Furthermore, children experiencing high chronic stress have disrupted connectivity between the prefrontal cortex and the amygdala (Evans & Kim, 2013). This means that the impulse control and emotion regulation structures of children and adolescents experiencing long term chronic stress simply cannot communicate with each other. I explored the distinctions in the pathway from family chaos to chronic stress and dysregulation to ameliorate acute and general patterns of responding to stress.

Family Chaos

Family chaos is the cumulative exposure to extremely disruptive and disorienting environments (Fiese & Winter, 2010). The Chronic Stress Model (CSM) offers foundational insight into the pathway between family chaos and poor developmental outcomes and opportunities for prevention. The processes underlying the relationship from family chaos to poor outcomes and the factors contributing to stressful and harsh environments are key points to consider for intervention efforts and preventing the long-term, harmful effects of stress dysregulation.

The CSM articulates that chronic stress mediates the relationship between family chaos and acute stress responses and general patterns of poor developmental outcomes (Evans & Kim, 2013). The CSM explains that the social environment associated with family chaos dramatically increases risk of chronic stress which in turn leads to dysregulation of stress responses in the immediate and poor emotion regulation and executive function in the future (Evans & Kim, 2013). Measures of family chaos like familial conflict, rates of family dissolution, maternal depression, exposure to violence, and parental harshness strengthens rates of pediatric chronic stress (Evans & Kim, 2013). Similarly, household crowding and structural instability, neighborhood noise and exposure to violence all raise chronic stress in children (Evans & Kim, 2013). Exposure to chronic stress encourages an adaptive change to stress reactivity which, in the long-term, raises the risk of chronic diseases, like cardiovascular disease, diabetes, and obesity (Evans & Kim, 2013). Chronic stress also leads to hyper-sensitive emotional reactivity and poor emotion regulation through the disconnect in the prefrontal cortex and the amygdala (Evans & Kim, 2013; Lucas-Thompson, 2012).

Family chaos is a multi-dimensional measure and historically consists of household cleanliness, noise, density, and violence, number of times moving households, family routines, neighborhood conditions, and sensory overload (Fiese & Winter, 2010; Evans et al., 2005; Matheny et al., 1995; Andrews et al., 2021). Furthermore, measures of family chaos disproportionately affect low-income, marginalized populations and are often comorbid in that qualifications in one domain of family chaos translate to increased risk in another. For instance, low income families, out of necessity, often have reduced organized time for children's needs (Roy et al., 2004) and often live in households with higher noise, more crowding, more chaos, toxins, and allergens (Evans & Kim, 2013).

These diverse measures of family chaos are associated with a multitude of poor developmental outcomes, including dysregulated stress reactivity and poor emotion regulation. Family chaos is related to high levels of pediatric chronic stress (Evans & Kim, 2013; Evans & Marcynyszyn, 2004; Fiese & Winter, 2010). Family chaos in early childhood predicts reduced glycaemic reactivity (Chae et al., 2016), increased free cortisol reactivity in middle childhood (Doom et al., 2018), and systemic inflammation (Schreier et al., 2014). Family chaos is also linked to poor physical health, increased externalizing and internalizing problems, rise in adolescent drinking problems and conduct disorders, and higher levels of chronic diseases like heart disease, diabetes, and obesity (Evans & Kim, 2013; Fiese & Winter, 2010; Masarik & Conger, 2017). As early as infancy, family chaos has a dysregulatory impact, affecting infant and parent sleep (Whitesell et al., 2018). Higher family chaos is also predictive of lower positive family emotional context and lower emotion regulation scores (Toria et al., 2020). This study further examined this mediation pathway from family chaos to chronic stress to dysregulation, and differentiate family chaos into instability and disorganization.

Instability and Disorganization as Measures of Family Chaos

Family chaos is a multidimensional measure (Fiese & Winter, 2010). Two important subdimensions of family chaos, instability and disorganization, are often considered (Vernon-Feagans et al., 2012; Garrett-Peters et al., 2019; Andrews et al., 2021). Family instability encapsulates unpredictable, intermittent events that disrupt continuity of the household and threaten the family system (Garrett-Peters et al., 2019; Garrett-Peters et al., 2016; Matheny et al., 1995; Sameroff, 2010). Family disorganization captures enduring, daily experiences that contribute to overwhelming, general disorder (Garrett-Peters et al., 2019; Garrett-Peters et al., 2016; Matheny et al., 1995; Sameroff, 2010; Fiese & Winter, 2010). Although exposure to disorganization may result in habituation to circumstances and stress, the unpredictability of indices of instability minimizes a child's sense of control and instills helplessness, and is challenging to adapt or habituate to (Evans & Stecker, 2004). It is hypothesized that without proper support this unpredictability could result in deficits in affect and behavior management (Andrews et al., 2021). This distinction, therefore, provides essential information for interventions targeting the plethora of poor developmental outcomes stemming from acute stress responses and general patterns of responses to chronic stress.

These dimensions of family chaos include a wide range of family indicators. Indices of instability include frequent changes in residence or caregivers, changes in job or income, and an absence of or unpredictable routines (Vernon-Feagans et al., 2012; Andrews et al., 2021). Similarly, measures of instability also examine the number of times a child moved to another residence, changes in the primary and/or secondary caregiver, the number of different people in the household, the number of household members moved into or out of the household, and a lack of or unpredictable routines (Andrews et al. 2021). In contrast, indices of disorganization referred to clutter, ambient noise, crowding, renovations, ongoing home repair, and lack of structure (Andrews et al., 2021). Evaluations of disorganization survey household density, household cleanliness, neighborhood noise, and hours spent watching TV per day (Andrews et al. 2021). Although instability and disorganization are distinct measures of family chaos, the conceptualization of these factors is broad and not completely mutually exclusive (Andrews et al., 2021).

For the current study, instability and disorganization were measured according to the empirically-supported strategy to assess instability and disorganization of Vernon-Feagans et al. (2012). More specifically, they created 10 indicators of family chaos from data collected over

36-months (Vernon-Feagans et al., 2012). Vernon-Feagans et al. (2012) selected these 10 chaotic indicators based on previous research. After an exploratory factor analysis forcing 2 factors, they found five indicators mapped onto instability—including number of times a child moved, changes to the primary caregiver, and changes to secondary caregivers—and five indicators mapped onto disorganization—number of hours the TV is on, household density, and household cleanliness (see Table 1 for the full list).

Although many studies including Vernon-Feagans et al. (2012) found mixed effects of instability and disorganization across various outcomes, a meta-analysis of 35 studies concluded that both family chaos dimensions are significantly associated with child executive functions; however, instability is a stronger correlate than disorganization (Andrews et al., 2021). Nonetheless, the effect sizes of both instability ($r^2 = .029$) and disorganization ($r^2 = .004$) on executive functions were very small. Andrews et al. (2021) also speculated that chronic stress mediated this relationship. They asserted that instability was more strongly correlated to executive functions because unpredictability drove higher levels of perceived chronic stress (Andrews et al., 2021). These high levels of chronic stress then drove higher levels of dysregulatory stress responses (Andrews et al., 2021), and then dysregulation contributed to lasting neurological changes resulting in lower levels of executive functioning (Andrews et al., 2021). The distinction between instability and disorganization allows interventions to better target the detrimental components of family chaos and thus provide more effective, more precise, and more efficient programs. The present study examined the hypothesized relationship proposed by Andrews et al. (2021) and the chronic stress model (Evans & Kim, 2013), and extend their findings to stress reactivity and emotion regulation.

The Current Study

Family chaos is linked to higher chronic stress (Evans & Kim, 2013; Evans & Marcynyszyn, 2004; Fiese & Winter, 2010), maladaptive stress reactivity (Doom et al., 2018; Schreier et al., 2014), and poor emotion regulation (Toria et al., 2020). Interventions to prevent the development of chronic diseases and promote resilience, healthy stress responses, and adaptive emotion regulation greatly benefits low-income families, where the risk for deficits in these are highest. Although a substantial body of work exists on the links between family chaos and stress reactivity and emotion regulation, there is a gap in the literature examining the effect of differentiating family chaos into instability and disorganization on this association and its underlying processes. Thus, this distinction is in effort to clarify the mediating pathways from which family chaos affects stress reactivity and emotion regulation. This study investigated this divergence in the pathway from family chaos to dysregulation in adolescents. I seek to analyze the effect of instability and disorganization, as measures of family chaos, on sibling pairs' stress responses and emotion regulation. I hypothesize the following:

H_1: The strength of the association of instability and stress reactivity is stronger than the association between disorganization and stress reactivity.

H_2: The strength of the association of instability and emotion regulation is stronger than the association between disorganization and emotion regulation.

H_3: Stress reactivity mediates the relationship between both indices of instability and indices of disorganization and emotion regulation.

METHOD

Participants

My sample consists of 153 adolescents aged 10 to 17 from 98 families who participated in a larger study on family relationships and health (Lucas-Thompson & Granger, 2014). The sample is made up of approximately equal males and females (52% female) and had an average age of 12.97 (SD = 2.17). Adolescents are 49% non-Hispanic White, 26% other or mixed ethnicities, 17% African American, 6% Asian American, 1% American Indian, and 1% Hispanic. Mothers are 61% non-Hispanic White, 20% African American, 8% Asian American, 7% other or mixed ethnicities, 2% American Indian, and 2% Hispanic. Fathers are 56% non-Hispanic White, 23% African American, 13% other or mixed ethnicities, 4% Asian American, 3% Hispanic, and 1% American Indian. Participants' are diverse in their socioeconomic status. Family's annual income ranged from $3375 to $450,000 (*Median* = $67750, *SD* = $63879.39). On average, both caregivers completed an Associate's Degree or vocational training after high school.

Procedure

Only procedures relevant to the current study are discussed here. All visits required the presence of a child and two caregivers and occurred at a small, private, liberal arts college in the Midwest. Visits primarily were scheduled in the afternoon to control for diurnal patterns in cortisol. Children and adolescents were asked to refrain from alcohol the night before, cold medicine the day of, and caffeine, eating, smoking, and exercise for two hours prior to the visit (for assessment of stress physiology). Caregivers and children were taken to separate, quiet rooms after giving informed consent.

The researchers accompanied the child to a quiet, private room and attached the materials necessary to record heart rate and blood pressure. These measurements continued through the remainder of the visit. Children gave a 'practice' cortisol sample to familiarize participants with the cortisol collection procedure. Then children filled out the Positive and Negative Affect Schedule (PANAS; Laurent et al., 1999) and sat quietly for 10 minutes. The second, baseline cortisol sample was collected. Participants were then exposed to the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), to induce general social-evaluative threat response. The TSST is a standardized psychological stressor that creates mild to moderate stress and corresponding cortisol (Dickerson & Kemeny, 2004) and cardiovascular reactions in 70-80% of participants (Kirschbaum et al., 1993). A modified TSST that is appropriate for younger children and adolescents (Yim, Quas, Cahill, & Hayakawa, 2010) was used. This stressor was chosen because it is a robust social-evaluative stressor which consistently produces physiological responses across systems where other stressors often do not (Kudielka & Kirschbaum, 2007). During the modified TSST, participants gave a speech about personal characteristics and then completed out-loud mental arithmetic. A female evaluator who remains neutral observes these activities. Participants were told the evaluator examined their speech, posture, and tone of voice. The task was videotaped and children were told that the tape will be evaluated by experts. Samples of cortisol were collected immediately after, as well as 10, 20, and 30 minutes after the TSST. Next, children finished their questionnaires using an Audio Computer Assisted Self Interview (ACASI) software that had the option to read the questions and answers to participants. This software allowed sensitive information to be collected confidentially, while accommodating for the fact that not all participants would be at the same reading level. Finally, all participants were debriefed.

Measures

Instability and Disorganization

To measure indices of instability and disorganization, I sought to fulfill the 10 indicators of family chaos established in Vernon-Feagans et al. (2012) and Garrett-Peters et al. (2019). Vernon-Feagans et al. (2012) conducted a confirmatory factor analysis with their 10 indicators, mapping five indicators onto instability and five indicators onto disorganization. Their five indicators of instability were (a) the number of times the child moved physically to another residence, (b) the number of changes in the primary caregiver, (c) the number of changes in the secondary caregiver (either primary caregiver partner or child's grandmother), (d) the total number of different people in the household, and (e) the number of times household members moved into or out of the household (Vernon-Feagans et al., 2012). They also mapped five indicators onto disorganization: (f) the average number of hours that the TV was on each day, (g) average household density, (h) home visit preparation, (i) household cleanliness, and (j) neighborhood noise (Vernon-Feagans et al., 2012). To approximate these indices of instability and disorganization, I used items from the Home Conditions Scale (Matheny et al., 1995), the Neighborhood Conditions Scale (Sooman & Macintrye, 1995), the Stressful Life Events scale (revised from the Social Readjustment Scale; Hobson & Delunas, 2001), and participant addresses (see Table 1). The Stressful Life Events scale asks parents to answer about specific stressors over the past year. Parents rated the severity of each stressor on a scale from 1 (the event did not occur, or occurred), 2 (somewhat severe), 3 (moderately severe), 4 (extremely severe), but scores were dichotomized as present (answers of 2-4) or absent (answer of 1). All other items except the participant addresses were dichotomous and summed after appropriate reverse-scoring to make continuous count variables representing each indicator above.

Participant addresses were searched on Zillow and through apartment websites to determine the number of bedrooms (relative to parent reports of the number of people in the household) to measure household density. Indicator c, the total number of changes in the secondary caregiver, was altered to be the total number of changes to the family system. This was partly due to availability of items in my dataset and partly to be more inclusive to diverse family structures. Finally, indicator h, home visit preparation, was not able to be approximated as this study did not conduct home visits. After being standardized, items of instability and disorganization were summed to create total variables in which high scores indicated high levels of instability or disorganization.

Emotional Stress Reactivity

The Positive and Negative Affect Scales (PANAS) were used to assess emotional stress reactivity. The PANAS are one 20 item instrument which measure positive affect, or the extent to which an individual feels enthusiastic, active, and alert (Watson et al., 1988), and negative affect, or the extent to which an individual feels subjective distress and unpleasurable engagement (Watson et al., 1988). The scale asks for a rating of one word emotions like "sad" and "proud" (Watson et al., 1988, p.1070). Items are rated on a 5-point Likert scale from 1 (very slightly or not at all) to 5 (extremely) (Watson et al., 1988). After separating out positive and negative scales, high scores mean higher affect in those categories. The PANAS (Positive Affect and Negative Affect) scales have strong internal consistency (α=0.86 and α= .87, respectively) and high test-retest reliability (r=0.79 and r=0.81, respectively) (Watson et al., 1988). The difference scores pre- and post-TSST of both total scores of the positive and negative affect scales were used to examine emotional stress reactivity.

Physiological Stress Reactivity

Cortisol, blood pressure (systolic and diastolic), and heart rate were used to measure participants' physiological stress reactivity. Blood pressure was monitored every three minutes throughout the TSST and the first 5 minutes of the recovery period. For the rest of the visit, blood pressue was measured every 5 minutes. Blood pressure and heart rate were averaged for three timepoints: TSST baseline (5 and 10 minutes prior to the TSST), TSST (0, 3, 6, and 9 minutes post-TSST onset), and TSST recovery (0 and 3 minutes post-TSST). Finally, difference scores were calculated comparing reactivity to baseline.

To measure cortisol and sAA levels during the stressor, as advocated by Pruessner et al. (2003), I calculated the area under the curve with respect to increase (AUCi). As the total area under the curve, AUCi depicts change over time. AUCi may be regarded as whether sAA/cortisol levels changed over the course of the visit. AUCg and AUCi were calculated using the guidelines in Pruessner et al. (2003).

Emotion Regulation

To assess emotion regulation, I used the Children's Emotional Management Scale (CEMS) and the Cognitive Emotion Regulation Questionnaire (CERQ). CEMS assesses a child's ability to regulate and manage negative emotions in a 12-item scale (Zeman et al., 2001). The CEMS (Zeman et al., 2001) has children rate from 'hardly ever' to 'often' when they engage in suppression of emotional expression (e.g., "I get sad inside but I don't show it"), dysregulated or inappropriate emotional expression (e.g., "I say mean things to others when I'm mad"), and emotion regulation coping (e.g., "When I am feeling sad, I do something totally different until I calm down"). After reverse-coding, a summary score, with high scores meaning better regulation, had moderate-high internal consistency (α=0.70) and has been shown to be reliable and valid (Zeman et al., 2001).

CERQ examines children's cognitive coping strategies and thoughts after negative life events (Garnefski et al., 2001). The 32-item CERQ is used for children 12 and older and the 36-item CERQ-k is used for children 11 and younger (Garnefski et al., 2007). The CERQ and the CERQ-k have been shown to be reliable and valid (Garnefski et al., 2007). There are nine subscales rated from "almost) never" to "(almost) always." There are nine subscales: a) refocus on planning, b) rumination, c) putting into perspective, d) catastrophizing, e) positive refocusing, f) positive reappraisal, g) acceptance, h) self-blame, and, i) other blame. Each item (e.g. "I feel that I am the one to blame for it") is rated from "(almost) never" to "(almost) always." High scores in a subscale indicate high levels of each cognitive coping strategy. The CERQ had a very high internal consistency of (α=1.00). The total scores of the CEMS and CERQ were used as indices of emotion regulation.

Proposed Data Analysis

I controlled for the following variables' effect on the association: age, income, and race. I controlled for age as age is a developmentally salient factor in the relationship of family chaos with stress reactivity (Doom et al., 2018) and emotion regulation (Hong et al., 2021). In addition, there is a robust literature documenting that family chaos is associated with income (Evans et al., 2005; Matheny et al., 1995) and as such is a potential confounding variable that should be controlled for in analyses such as these. Similarly, I controlled for race as it is also a well-established confound of family chaos and developmental outcomes (Fiese & Winter, 2010). Race was dichotomous and coded as white or non-white.

To test the hypothesis that instability is a stronger predictor of stress reactivity than disorganization, and that instability is a stronger predictor of emotion regulation than disorganization, I used Generalized Estimating Equation (GEE) analyses to examine the relationships of instability and disorganization with physiological reactivity, emotional reactivity, and emotion regulation, controlling for age, income, and race. This GEE approach is regression-based and non-parametric and was used to adjust for clustering within families, as my data includes some sibling pairs. For each outcome variable, I performed three models, one with: instability alone (Model 1), disorganization alone (Model 2), and instability and disorganization (Model 3). I used three different models to inspect the separate contributions of instability and disorganization and their unique effects controlling for each other. To test whether instability is a stronger predictor of outcomes than is disorganization I compared the significance of instability and disorganization across models for each outcome variable (see Table 4). To test the hypothesis that chronic stress mediates the relationship between family chaos and poor emotion regulation, I used a path analysis to examine a partial mediation model and the extent to which the association between family chaos and emotion regulation is mediated by stress reactivity.

RESULTS

Measures of Instability and Disorganization

Instability and disorganization were weakly and non-significantly, positively associated with each other, $r = .12$, $p > 0.05$. The five measures of instability were mostly significantly, moderately, positively associated with each other, except for the number of people in the household which was largely non-significantly, positively associated with the other instability indicators (see Table 2). Other than a moderate, positive association between household commotion and household cleanliness and a small, positive association between household commotion and household noise, the four measures of disorganization were not significantly associated with each other (see Table 3). Instability had a positive skew (skew = 1.78, kurtosis = 2.9; see Figure 1) and disorganization was normally distributed (see Figure 2).

Physiological Stress Reactivity

When controlling for age, race, and income, but without controlling for disorganization, instability significantly, negatively predicted cortisol reactivity (see Table 4; Model 1). In contrast, instability did not significantly predict systolic blood pressure, diastolic blood pressure, or heart rate difference scores, when controlling for age, race, and income. In addition, disorganization was not significantly associated with cortisol reactivity, systolic blood pressure, diastolic blood pressure, or heart rate difference scores, when controlling for age, race, and income (Model 2). When both disorganization and instability were included as predictors, instability only negatively predicted cortisol reactivity at trend levels of significance (Model 3). Neither instability nor disorganization predicted systolic blood pressure, diastolic blood pressure, or heart rate difference scores, when controlling for each other and control variables (Model 3).

Emotional Stress Reactivity and Emotion Regulation

There were no significant associations between instability and disorganization in any of the models predicting differences scores of PANAS positive or PANAS negative scales, when controlling for age, race, and income (see Table 5). Finally, instability and disorganization were not significant predictors of CERQ or CEMS total scores in any of the models, when controlling for age, race, and income (see Table 5).

Mediation

As no significant direct effects were not found between family chaos and emotion regulation, true mediation was not possible but we proceeded to test indirect effects. A path analysis was performed to test an indirect effect of family chaos on emotion regulation through stress reactivity in adolescents. Due to the significant association between instability and cortisol reactivity (but not other physiological outcomes), AUCi was used in the path analysis as the indirect stress reactivity variable (as the only possible variable through which there was an indirect effect). Similarly, in a model with instability, disorganization, and AUCi, stress reactivity significantly positively predicted CEMS scores (b = .003, SE = .002, p < 0.05) but not CERQ scores (b = -0.014, SE = .041, p > 0.05). Accordingly, due to AUCi's significant association with CEMS, CEMS was used in the path analysis as the measure of emotion regulation. Together, instability, disorganization, and cortisol reactivity explained 4.5% of the variance in the child emotion regulation questionnaire. The total effect, c1, of instability on emotion regulation was non-significant (see Table 6). The total effect, c2, of disorganization on emotion regulation was trending significant and positive. The direct effect, c1', of instability on emotion regulation was non-significant. The direct effect, c2', of disorganization on emotion regulation was trending-significant and positive. The indirect effect, a1b, of instability on emotion regulation (through stress reactivity) was non-significant, small, and negative. The indirect effect, a2b, of disorganization on emotion regulation (through stress reactivity) was non-significant, small, and negative.

DISCUSSION

In the current study, I sought to extend the findings on the effects of instability and disorganization, as two components of family chaos, on executive function (Andrews et al., 2021) to other critical developmental outcomes, particularly stress reactivity and emotion regulation; I also aimed to test possible mediation from family chaos to stress reactivity and then to emotion regulation. Results indicated that instability, but not disorganization, significantly and negatively predicted cortisol reactivity, but this effect was reduced to trend levels when both family chaos variables were included in the model. In other words, as instability increased, cortisol reactivity decreased, reflecting a potential habituation to stressors in adolescents with high instability. However, neither instability nor disorganization predicted emotional reactivity or emotion regulation. Finally, there was no evidence that there was an indirect effect from family chaos to emotion regulation through physiological reactivity. These findings contribute to the growing body of literature on the importance of instability as a measure of family chaos and suggest a key area for intervention to reduce the long-term effects of chronic stress.

Past literature has suggested that instability and disorganization are both significantly related to executive functioning, but that instability is a stronger predictor of executive function than is disorganization (Andrews et al., 2021). Similarly, in this study, instability seemed to be a stronger predictor than disorganization of stress reactivity. In past research, greater instability predicted reduced executive functions (Andrews et al., 2021); in the current study, greater instability predicted reduced cortisol reactivity. Although this negative association was reduced to trend levels of significance when disorganization was added as a predictor to the model, this pattern is consistent with literature emphasizing the strength of the effect of family instability over disorganization (Andrews et al., 2021). This finding is also consistent with the HPA axis attenuation model of Susman's (2006) that early life stress predicts a less flexible HPA axis and, as a result, attenuated cortisol reactivity. It is also consistent with previous findings that family chaos in early life predicted dampened cortisol reactivity in middle childhood (Doom et al., 2018). This dampened cortisol reactivity has life-long effects and is associated with behavioral issues, conduct disorders, and lowered academic performance (Susman, 2006; Evans & Kim, 2013). This association with an attenuated stress response differs from proposed theories hypothesizing instability to be more challenging to adapt to and therefore leads to greater sensitivity (Evans & Stecker, 2004; Andrews et al., 2021). The findings above call into question the long-term effects from family instability and the theory underlying these effects. Future research should seek to further elucidate the theoretical connections between instability and stress reactivity and examine buffering protective factors. Parent-child relationships, for instance, buffer the effects of interparental conflict on stress reactivity (Lucas-Thompson & Granger, 2014). Subsequent research examining the effect of strong, supportive relationships on instability and stress reactivity are likely to shed light on avenues for targeted prevention.

Although past research found links of instability and disorganization with executive function (including measures of emotional control), in this study, I found that neither instability nor disorganization significantly predicted measures of emotional reactivity or emotion regulation. Previous literature has found *chronic* stress to be associated with hyper-reactive emotional stress responses (Evans & Kim, 2013; Lucas-Thompson, 2012). This study may not have mirrored these findings due to the operationalization of instability which emphasized more recent events (within a year) than past studies (Andrews et al., 2021; Vernon-Feagans et al., 2012); more recent stress may not have had time to wear down emotional stress systems. The discrepancy between these findings with past work on emotional control (Andrews et al., 2021) also could be due to the difference in age or size of our samples. More specifically, the sample used in Andrews et al. (2021) had a mean age of 6.42 whereas my sample had a mean age of 12.97. This difference is in line with Hong et al. (2021) which indicated a sensitive period at age 6 where family chaos moderated non-supportive parental responses and emotional regulation but by age 9 this effect dissipated. Similarly in this study, by adolescence, the link from family chaos to emotion regulation was not significant. Furthermore, Andrews et al. (2021) had high power to detect small associations, with a very large sample of 16,480 children. This is reflected in their very small effect sizes for instability and disorganization of $r^2 = .029$ and $r^2 = .004$, respectively (Andrews et al., 2021). Therefore, this study was underpowered with 153 participants to find similar effects.

Finally, the discrepancy of findings could also have originated in operationalization differences of emotion regulation. Andrews et al. (2021) included a multitude of different scales, consisting of direct assessments and informant-completed questionnaires, to measure executive functions in their meta-analysis. To measure emotion regulation in this study, the CEMS and CERQ were used, neither of which were utilized by Andrews et al. (2021). Despite their wide usage, the CEMS and CERQ have been critiqued for their internal consistency (Freitag et al., 2023). In a systematic review of 510 studies, the CERQ scored Cronbach's alphas as low as 0.32 and the CEMS had nearly 45% of studies reporting below 0.70 (Freitag et al., 2023). Further, following a distinction proposed in Freitag et al. (2021), the measures of emotion regulation in Andrews et al. (2021) measured temperament and behavioral manifestations of emotion regulation; however, the CEMS and CERQ measure the processes engaged in regulating emotion (Freitag et al., 2023). This distinction may be critical to examining the effect of instability and disorganization on emotion regulation. Children and adolescents experiencing chronic stress, like those with high levels of family chaos, have altered prefrontal cortex and amygdala structures (Evans & Kim, 2013; National Scientific Council on the Developing Child, 2020). Changes in these structures result in disrupted communication between impulse control and emotion regulation systems in the brain. Children experiencing these neurological changes are likely to then manifest poor impulse control and behavioral issues. Therefore, the measures examining behavioral manifestations of emotion regulation, like those utilized in Andrews et al. (2021), might better capture the effects of family chaos on emotion regulation.

Nevertheless, systemic factors might also contribute to the association of family chaos with behavioral issues and temperament but not emotional regulation processes. A large number of the emotion regulation scales used in Andrews et al. (2021) were informant-completed questionnaires, meaning that assessments were completed by individuals reporting on the child. In fact, the association between family chaos and executive functions using informant-completed questionnaires was significantly stronger than using direct assessment (Andrews et al., 2021). However, this measurement strategy could be biased as family chaos is associated with ethnic and racial minority status (Fiese & Winter, 2010) and children of color are more likely to be singled out for behavioral issues (Amemiya et al., 2020). Therefore, children of color experiencing family chaos could be disproportionately perceived as having more behavioral issues in Andrews et al. (2021). Future research should explore the connections between instability and disorganization and distinct measures of emotion regulation: behavioral and temperamental, as measured in Andrews et al. (2021), and emotion regulation processes, as measured here.

Past literature has also found disorganization to be a weaker but significant predictor of executive functions (Andrews et al., 2021), whereas this study found no significant association between disorganization and any of the studied outcomes. The measures of disorganization captured in this study also might not reflect cultural differences and individual appraisals of disorganized environments. Higher number of family members living in the home is more culturally normative for some cultures and not others (Taylor et al., 2011). Additionally, disorganized environments do not necessarily reflect unclean environments. Individuals' appraisals of the effect of aspects of disorganization on their own life might better consider an individual in their unique context and culture. Aside from methodological considerations, it also may be that family chaos is simply a less robust predictor of stress reactivity than of executive functions. Executive functioning and stress reactivity both are affected by family chaos factors like noise level, household commotion, and household density (National Scientific Council on the Developing Child, 2020; Andrews et al., 2021). However, the effects of family chaos on executive functioning may compound because of the effects of family chaos on systemic factors that then themselves reduce executive function. For example, high household commotion decreases the time available to spend playing with a child or helping to complete homework, both of which have repercussions on executive functions (Evans et al., 2005; Evans & Wachs, 2010). Whereas family chaos does impact stress reactivity, those factors may not interact in the same way and could result in a weaker association.

Lastly, this study did not support chronic stress as a mediator from family chaos to emotion regulation. Theories like the Chronic Stress Model enumerate this connection from family chaos to chronic stress and finally poor developmental outcomes (Evans & Kim, 2013). This model provides a unique framework emphasizing systemic factors, their effects on physiological systems, and a downward cascade leading to poor outcomes. This study found no significant indirect effects between instability and disorganization to stress reactivity and then emotion regulation. This failure to replicate support for the Chronic Stress Model offers insight into the specific contexts this theory applies. For example, the poor developmental outcomes specified in the Chronic Stress Model may not be captured by the CEMS and CERQ. Alternatively, the Chronic Stress Model's application to emotion regulation might be consistent with the distinction proposed in Freitag et al. (2023). That is to say, the Chronic Stress Model may apply to behavioral or temperament emotion regulation measures but not to measures of emotion regulation processes. Therefore, alternative measures of emotion regulation, like those utilized in Andrews et al. (2021) should be considered to test indirect effects of stress reactivity on family chaos and emotion regulation and clarify the applicable contexts of the Chronic Stress Model (Evans & Kim, 2013).

My study did find significant influences from family chaos on some outcomes of stress reactivity, our mediator, but could not demonstrate evidence for indirect effects without influences from family chaos to emotion regulation. Therefore, alternative measures of emotion regulation, like those utilized in Andrews et al. (2021) should be considered to test indirect effects of stress reactivity on family chaos and emotion regulation and demonstrate support for the Chronic Stress Model (Evans & Kim, 2013).

Although this study makes important contributions to the literature on family chaos and developmental outcomes, there are several limitations to note. First, the sample was relatively small, resulting in limited power. As a number of the analyses were non-significant and my risk for Type II error high, my confidence in these null findings is relatively low. Future research utilizing larger samples is needed to elucidate the relationships between instability, disorganization, stress reactivity, and emotion regulation.

Second, this study's operationalization of instability differed from Andrews et al. (2021). Andrews et al. (2021) predominantly utilized the Confusion, Hubbub, and Order scale (CHAOS; Matheny et al., 1982) to measure disorganization and the Family Instability Questionnaire (FIQ; Ackerman et al., 1999) to measure instability. This study assessed instability from items exclusively from the Stressful Life Events Scale (Hobson & Delunas, 2001), which looked at the impact of stressful events that occurred in the last year. The Family Instability Questionnaire assesses changes in family residence, changes to primary and secondary caregivers, romantic relationship changes, income loss, and death or illness of close family member (Ackerman et al., 1999; Forman & Davies, 2003). The conceptual constructs of the measure of instability used in this study were incredibly similar to the Family Instability. Building each of these conceptual constructs was difficult and could only be approximated as I was limited by available questions in the dataset. However, one key difference between the instability measure of this study and the instability measures of Andrews et al. (2021), is that the instability variable measured here captures recent instability, or unstable events occurring in the last year. Longitudinal studies have shown that timescale matters on the effect of family chaos on stress reactivity (Doom et al., 2018). Family chaos in early childhood is known to have more detrimental effects on cortisol reactivity in middle childhood (Doom et al., 2018). As such, recent instability in the lives of an older, less developmentally sensitive sample as measured in this study might be less likely to show effects. And yet, recent instability measured here still predicted dampened cortisol reactivity. These findings then suggest a potential potency of instability, such that even recent occurrences of family instability in the last year still predicted cortisol reactivity. The potency of family instability and its effects on stress reactivity over short time frames could lend itself as an early warning system and an indicator for intervention. Further research must examine time since stressors to better understand the extent to which time moderates the effect of stressful life events on dysregulated stress reactivity.

Third, this study could not fully test mediation. In order to fully test mediation, data must be collected from at least three time points (MacKinnon et al., 2023). The cross-sectional design employed here does not control for temporal precedence or eliminate alternative model configurations exploring the relationship between family chaos, stress reactivity, and emotion regulation. Future research should utilize longitudinal designs to test a mediating effect. Lastly, I was unable to statistically compare the magnitude of coefficients for instability and disorganization to cortisol reactivity and thus had to rely solely on differences in statistical significance. Moving forward, researchers should seek to examine not only significant differences but also the strength of coefficient differences between instability and disorganization.

CONCLUSION

In this study, I sought to extend past research on instability and disorganization to other important outcomes, specifically examining their effects on stress reactivity and emotion regulation. I found that instability, but not disorganization, predicted dampened cortisol reactivity, although this effect was reduced to trend levels of significance when disorganization was controlled for in the model. My findings support previous literature about the importance of instability and its effects on developmental outcomes (Andrews et. al, 2021). My findings also suggest that instability seems to be more potent than disorganization and predicts stress reactivity as early as a year after exposure. This study has important implications for future research on instability and stress reactivity and prevention interventions that target family instability.

TABLES

Table 1

Indicators of Chaos and Corresponding Variables

10 Indicators of Household Chaos (Vernon-Feagus et al., 2019)	My Variables	Frequency (Percentage)
	Instability	
(a) number of times the child moved physically to another residence	"Change in Residence" [A]	45 (29%)
	"Does your teen live outside your home with another parent for any part of the year?"	3 (2%)
	"Foreclosure on loan/mortgage" [A]	25 (16%)
(b) number of changes in the primary caregiver	"Parental separation or divorce" [A]	15 (10%)
	"Separation or reconciliation with spouse/mate" [A]	29 (19%)
	"Getting married/remarried (self)" [A]	18 (12%)
	"Becoming a single parent" [A]	12 (8%)
	"Death of spouse/mate" [A]	15 (10%)
(c) number of changes in the secondary caregiver (changed to family system)	"Death of close family member" [A]	71 (46%)
	"Adult child moving in with parent/parent moving in with adult child" [A]	17 (11%)
(d) number of different people in the household	Number of adults supported by family income	M= 4.86
	Number of children supported by family income	SD = 1.13

(e) number of times household members moved into or out of the household	"Gaining a new family member" [A]	36 (24%)
	"Pregnancy of spouse/mate/self" [A]	19 (12%)
	"Dealing with infertility/miscarriage" [A]	21 (14%)
	"Child leaving home" [A]	18 (12%)
	"Getting married/remarried (self)" [A]	18 (12%)
	"Becoming a single parent" [A]	12 (8%)
	"Death of spouse/mate" [A]	15 (10%)
	Disorganization	
(f) average number of hours that the TV was on each day	"There is very little commotion in our home" [B]	40 (26%)
	[D]	26 (17%)
	"You can't hear yourself think in our home" [B]	36 (23%)
	"The telephone takes up a lot of our time at home" [B]	93 (61%)
	"The atmosphere in our home is calm" [B D]	
(g) household density	Number of people divided by the number of bedrooms (taken from Zillow, apartment websites, etc.)	M= .74 SD = .30
(h) home visit preparation	N/A	N/A
(i) household cleanliness	"It's a real zoo in our home" [B]	61 (39%)
	"We can usually find things when we need them"[B D]	99 (65%)
(j) neighborhood noise	"Are any of the following a problem in your neighborhood?"	
	"Speeding Traffic" [C]	105 (67%)
	"Nuisance from dogs" [C]	63 (41%)
	"Disturbance by children or teens" [C]	84 (55%)
	"How crowded do you feel that your neighborhood is?" [C]	111 (73%)

[A] from the Stressful Life Events Scale, parent-report (Hobson & Delunas, 2001)
[B] from the Home Conditions Scale (Matheny et al., 1995)
[C] from the Neighborhood Conditions Scale (Sooman & Macintrye, 1995)

[D] reverse scored

Table 2

Unstandardized Descriptive Statistics and Correlations between Indices of Instability

	1	2	3	4	5
1. Change in Residence	X				
2. Change in Primary Caregiver	0.70***	X			
3. Change in Secondary Caregiver	0.43***	0.62***	X		
4. Number of Members in the Household	0.28***	0.11	0.14	X	
5. Number of times members have moved into or out of the household	0.69***	0.90***	0.62***	0.16	X
M	0.49	0.60	0.59	4.86	0.93
SD	0.67	1.40	0.63	1.13	1.81

* $p < .05$ ** $p < .01$ *** $p < .001$ [a] 0=No; 1=Yes

Table 3

Unstandardized Descriptive Statistics and Correlations between Indices of Disorganization

	1	2	3	4
1. Household Commotion	X			
2. Household Density	-0.02	X		
3. Household Cleanliness	0.40***	-0.10	X	
4. Household Noise	0.24**	-0.02	0.01	X
M	1.58	0.74	0.77	2.44
SD	0.96	0.30	0.81	1.15

* $p < .05$ ** $p < .01$ *** $p < .001$ [a] 0=No; 1=Yes

Table 4

Generalized Estimating Equation Models Predicting Physiological Stress Responses. Presented are unstandardized coefficients (with standard errors in parentheses).

	AUCi		SBP		DBP		HR	
	b(SE)	*p*	*b(SE)*	*p*	*b(SE)*	*p*	*b(SE)*	*p*
Model 1								
Instability	-4.65(4.17)	0.03*	-0.04(0.28)	0.90	-0.10(0.18)	0.69	-0.27(0.24)	0.14
Age	-1.72(7.61)	0.82	-0.09(0.41)	0.77	0.15(0.31)	0.66	0.21(0.42)	0.61
Income	0.00(0.00)	0.36	0.00(0.00)	0.13	0.00(0.00)	0.36	0.00(0.00)	0.09+
White (0=Non-White, 1=White)	42.96(31.34)	0.17	7.29(2.35)	0.00**	0.79(1.41)	0.60	4.19(1.85)	0.02*
Model 2								
Disorganization	-6.48(7.34)	0.21	-0.09(0.54)	0.83	0.19(0.33)	0.55	-0.42(0.43)	0.29
Age	0.41(7.34)	0.96	0.17(0.42)	0.62	0.29(0.32)	0.37	0.61(0.44)	0.11
Income	0.00(8.49)	0.37	0.00(0.00)	0.14	0.00(0.00)	0.52	0.00(0.00)	0.04*
White (0=Non-White, 1=White)	54.16(0.00)	0.09+	7.69	0.00**	0.77(1.49)	0.61	4.11(1.93)	0.03*
Model 3								
Instability	-4.33(4.60)	0.06+	-0.12(0.30)	0.73	-0.04(0.19)	0.90	-0.15(0.26)	0.43
Disorganization	-5.49(7.38)	0.27	-0.06(0.55)	0.88	0.19(0.34)	0.55	-0.39(0.44)	0.33
Age	0.24(8.05)	0.98	0.15(0.42)	0.64	0.28(0.32)	0.37	0.60(0.44)	0.12
Income	0.00 (0.00)	0.43	0.00(0.00)	0.13	0.00(0.00)	0.53	0.00(0.00)	0.05+
White (0=Non-White, 1=White)	49.92(33.39)	0.11	7.56(2.53)	0.00***	0.73(1.51)	0.67	3.95(1.96)	0.04*

+ p < .10 * p < .05 ** p < .01 *** p < .001

Table 5

Generalized Estimating Equation Models Predicting Emotional Stress Responses and Emotion Regulation. Presented are unstandardized coefficients (with standard errors in parentheses).

	PANAS (positive)		PANAS (negative)		CERQ		CEMS	
	b(SE)	*p*	*b(SE)*	*p*	*b(SE)*	*p*	*b(SE)*	*p*
Model 1								
Instability	0.00(0.02)	0.81	0.03(0.02)	0.25	0.03(0.02)	0.25	-0.11(0.14)	0.44
Age	0.05(0.04)	0.21	-0.01(0.03)	0.71	-0.01(0.03)	0.71	0.25(0.25)	0.17
Income	0.00(0.00)	0.20	-0.00(0.00)	0.80	-0.00(0.00)	0.80	-0.00(0.00)	0.43
White (0=Non-White, 1=White)	0.13(0.14)	0.43	-0.08(0.19)	0.65	-0.08(0.19)	0.65	-2.19(1.02)	0.06
Model 2								
Disorganization	0.00(0.03)	0.77	-0.01(0.04)	0.86	-0.01(0.04)	0.86	0.21(0.17)	0.34
Age	0.06(0.04)	0.12	-0.01(0.03)	0.73	-0.01(0.03)	0.73	0.30(0.18)	0.12
Income	0.00(0.00)	0.51	-0.00(0.00)	0.76	-0.00(0.00)	0.76	0.00(0.00)	0.69
White (0=Non-White, 1=White)	0.10(0.16)	0.50	-0.11(0.19)	0.52	-0.11(0.19)	0.52	-0.77(0.73)	0.27
Model 3								
Instability	-0.02(0.02)	0.37	0.02(0.02)	0.37	0.02(0.02)	0.37	-0.03(0.10)	0.74
Disorganization	0.03(0.03)	0.33	-0.01(0.04)	0.76	-0.01(0.04)	0.76	0.22(0.17)	0.30
Age	0.08(0.04)	0.08+	-0.01(0.03)	0.75	-0.01(0.03)	0.75	0.30(0.18)	0.11
Income	0.00(0.00)	0.51	-0.00(0.00)	0.78	-0.00(0.00)	0.78	0.00(0.00)	0.71
White (0=Non-White, 1=White)	0.06(0.16)	0.69	-0.10(0.20)	0.69	-0.10(0.20)	0.69	-0.80(0.74)	0.24

+ $p < .10$ * $p < .05$ ** $p < .01$ *** $p < .001$

Table 6

Path analysis Coefficients, Standard Errors, and p-values

		Consequent						
		M (AUCi)				Y (CEMS)		
Antecedent		Coeff.	*SE*	*p*		Coeff.	*SE*	*p*
X1 (Instability)	*a1*	-5.25	4.42	0.24	*c1'*	-0.03	0.10	0.76
X2 (Disorganization)	*a2*	-4.74	7.05	0.50	*c2'*	0.28	0.15	0.07+
M (AUCi)		____	____	____	*b*	0.00	0.00	0.13
	Total Effect		*c1*	-0.05	0.10	0.64		
	Total Effect		*c2*	0.27	0.16	0.09+		
	Indirect Effect		*a1b*	-0.02	0.02	0.35		
	Indirect Effect		*a2b*	-0.01	0.02	0.54		

+ p < .10 * p < .05 ** p < .01 *** p < .001

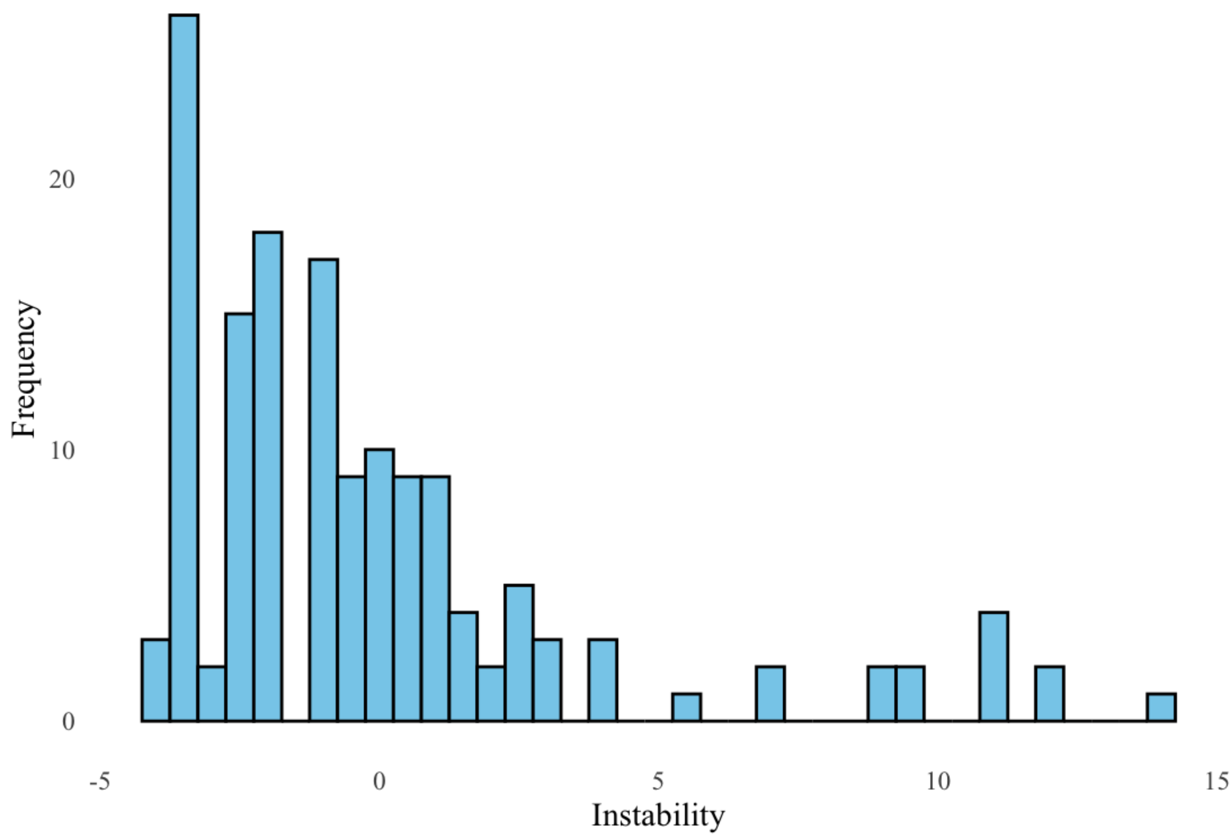

Figure 1

Distribution of Instability

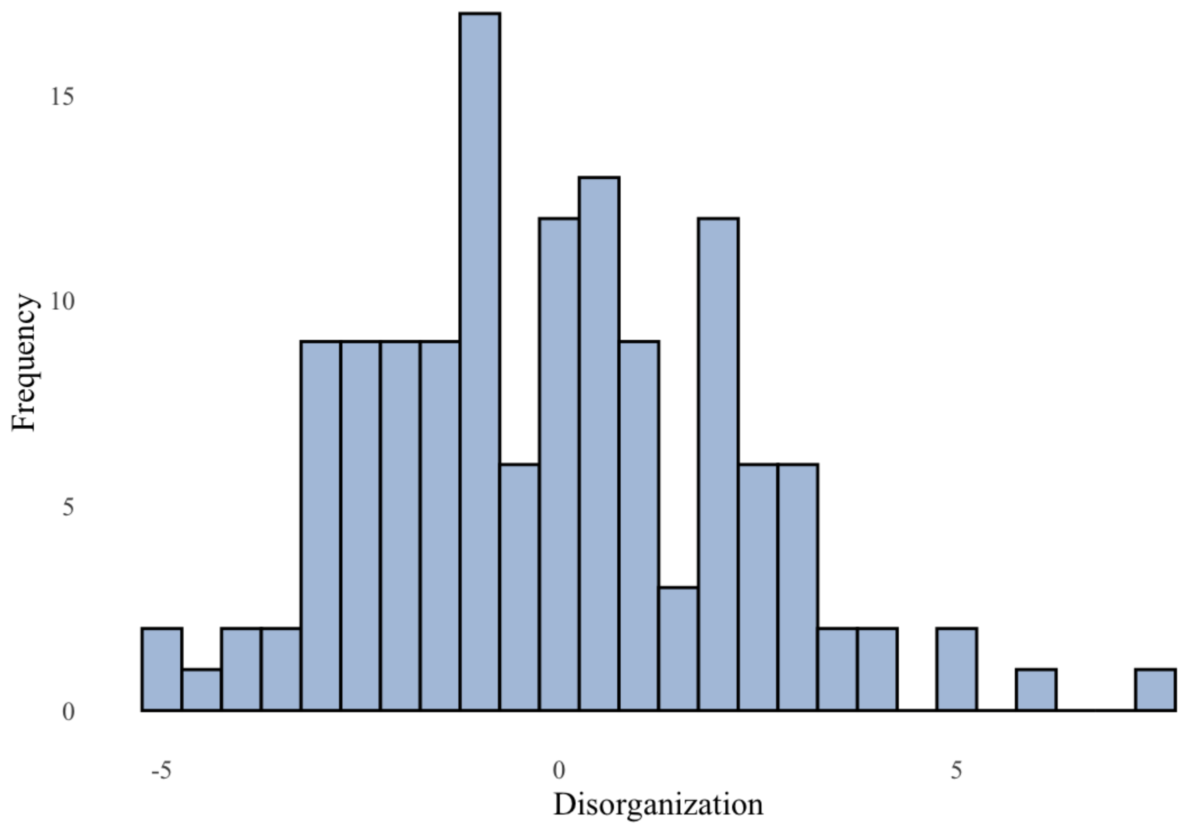

Figure 2

Distribution of Disorganization

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