

A Prospective Study of Childhood Negative Events, Temperament, Adolescent
Coping, and Stress Reactivity in Young Adulthood

by

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A Dissertation Presented in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Approved April 2012 by the
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ARIZONA STATE UNIVERSITY

August 2013

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ABSTRACT

Accumulating evidence implicates exposure to adverse childhood experiences in the development of hypocortisolism in the long-term, and researchers are increasingly examining individual-level mechanisms that may underlie, exacerbate or attenuate this relation among at-risk populations. The current study takes a developmentally and theoretically informed approach to examining episodic childhood stressors, inherent and voluntary self-regulation, and physiological reactivity among a longitudinal sample of youth who experienced parental divorce. Participants were drawn from a larger randomized controlled trial of a preventive intervention for children of divorce between the ages of 9 and 12. The current sample included 159 young adults (mean age = 25.5 years; 53% male; 94% Caucasian) who participated in six waves of data collection, including a 15-year follow-up study. Participants reported on exposure to negative life events (four times over a 9-month period) during childhood, and mothers rated child temperament. Six years later, youth reported on the use of active and avoidant coping strategies, and 15 years later, they participated in a standardized psychosocial stress task and provided salivary cortisol samples prior to and following the task. Path analyses within a structural equation framework revealed that a multiple mediation model best fit the data. It was found that children with better mother-rated self-regulation (i.e. low impulsivity, low negative emotionality, and high attentional focus) exhibited lower total cortisol output 15 years later. In addition, greater self-regulation in childhood predicted greater use of active coping in adolescence, whereas a greater number of negative

life events predicted increased use of avoidant coping in adolescence. Finally, a greater number of negative events in childhood predicted marginally lower total cortisol output, and higher levels of active coping in adolescence were associated with greater total cortisol output in young adulthood. Findings suggest that children of divorce who exhibit better self-regulation evidence lower cortisol output during a standardized psychosocial stress task relative to those who have higher impulsivity, lower attentional focus, and/or higher negative emotionality. The conceptual significance of the current findings, including the lack of evidence for hypothesized relations, methodological issues that arose, and issues in need of future research are discussed.

ACKNOWLEDGMENTS

First and foremost, I am deeply grateful to my advisor, mentor, and dissertation chair, Linda Luecken for her unrelenting support, guidance, and investment in my training as a clinical scientist and my future academic career. Her mentorship has been invaluable, and I will be forever grateful for our many thought-provoking discussions. I would also like to especially thank Irwin Sandler and Sharlene Wolchik for their wisdom and dedication as well as the exceptional research training in prevention science they provided to me over the last several years. A particular thank you to my other committee members: Leah Doane for her thoughtful comments and questions and the integrity she brings to her work and mentoring of students, and David MacKinnon for his warm encouragement, statistical expertise and sense of humor. I feel truly fortunate to have had the opportunity to work under these individuals throughout my graduate career.

I would like to extend a very special thank you to my classmate, colleague and friend Catherine Purdom, without whom my productivity, sanity, and confidence would not have survived the journey from first year to internship. I am also especially thankful my family who have always believed in me and encouraged me in my pursuit of this degree. Finally, I am extremely grateful to my partner for providing me with love, support, and joy throughout this process.

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CHAPTER 1

Introduction

Exposure to stressful events during childhood is recognized as a critical risk factor for the development and/or maintenance of psychopathology and physiological dysregulation across the lifespan (Grant, Compas, Thurm, McMahon, & Gipson, 2004; Pollack, 2005; Trickett, Noll, Susman, Shenk, & Putnam, 2010). The integration of the psychosocial and biological sciences represents one of the most exciting venues for explicating the processes by which these relations unfold (Ganzel, Morris, & Wethington, 2010). Accumulating evidence implicates the potential for negative life events to result in alterations to one of the body's major stress response systems, the hypothalamic-pituitary adrenal (HPA) axis, and its end product, cortisol (Bremner & Vermetten, 2001). More specifically, exposure to negative events during childhood has been shown to predict attenuated physiological reactivity later on. However, little is known about the pathways by which this relation occurs. A child's inherent self-regulatory abilities and their subsequent style of coping with life stressors represent two plausible mechanisms.

Researchers are increasingly incorporating measures of coping processes, or volitional/purposeful self-regulation during stress, into studies of the psychobiological effects of stressful events. Many of these empirical investigations have been based on the broad premise that "stress is bad" and "coping is good." The complexity of the central and peripheral nervous systems and the interaction between biological, psychosocial and contextual factors in the

prediction of adaptation suggests the need to look beyond one-to-one linear relationships (Granger & Kivlighan, 2003). Temperament represents a influential person-level variable that is likely to impact relations between stress and the development of later coping style, which may have implications for physiological functioning, and many researchers have called for increased attention to temperament in the study of stressful events, coping, and health (e.g., Friedman, 2000; Skinner & Zimmer-Gembeck, 2007). Taking a developmentally and theoretically informed approach is critical for making meaningful predictions with regard to stress, inherent and voluntary self-regulation, and physiological reactivity.

I begin with a brief overview of the anatomy and functions of the HPA axis, which will lay the foundation for subsequent discussions of the measurement and interpretation of cortisol activity and the developmental course of HPA axis functioning. Next, I summarize the empirical evidence of the negative effects of adverse life events on HPA axis functioning in the short and long-term and the theoretical framework within which this relation has been studied (i.e. allostasis). Coping style is introduced as one mechanism by which negative events might influence the biological stress response in the long-term and evidence in support of this hypothesis is reviewed. Finally, I discuss the importance of individual differences in behavioral and biological responses to stress and the potential for temperament to moderate the impact of negative life events on coping and physiological reactivity.

Grounded in the theoretical and empirical evidence reviewed, the purpose of the current research is to explore relations between stressful experiences, temperament, coping and physiological activity over developmental time among children of divorce. Using a longitudinal design, the current study has the following aims: 1) to examine the impact of childhood negative life events on physiological functioning during emerging adulthood in a sample of youth who experienced parental divorce during childhood, 2) to test the hypothesis that this relation is mediated by maladaptive coping, such that childhood negative events increases the use of maladaptive coping in adolescence (i.e. greater use of avoidant coping and/or limited use of active coping), and this coping style predicts attenuated cortisol reactivity in young adulthood, and 3) to examine whether temperament moderates the cascade proposed in the prior aim, such that the relation between greater negative events, poor coping, and subsequent cortisol activity is strongest for those who exhibited a “difficult” temperament in childhood (i.e. high levels of negative emotionality and low levels of constraint-attentional control).

CHAPTER 2

Neuroendocrine Stress Response System

Overview of the stress response

The neuroendocrine stress response system in humans evolved to support adaptation by facilitating the preservation of the individual's physical and psychological integrity within the context of a dynamic and constantly changing environment (Tsigos & Chrousos, 2002). The two arms of the peripheral stress response system, the sympathetic-adrenomedullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis, have been the main focus of countless neurobiological studies of stress and coping, owing to the relative ease with which they can be assessed in the laboratory and under natural conditions (for reviews see Denson, Spanovic, & Miller, 2009; Dickerson & Kemeny, 2004; Ganzel et al., 2010; Miller, Chen, & Zhou, 2007). Immediately following the stressor, the sympathetic-adrenomedullary (SAM) axis of the autonomic system initiates rapid alterations in physiological states (i.e., the classic 'fight or flight' response) via the adrenal medulla (the inner structure of the adrenal gland), including increases in adrenaline and noradrenaline, elevations in heart rate and blood pressure, and energy mobilization. These responses then quickly wane through activation of the parasympathetic arm of the autonomic nervous system, which acts to oppose the sympathetic responses and maintain homeostasis.

Unlike the SAM axis, the HPA axis response to psychological stress takes several minutes to unfold and involves a hormonal cascade resulting in the production of glucocorticoids (cortisol in humans and corticosterone in animals).

Whereas the SAM axis can be activated in arousing situations that are not necessarily distressing but that require engagement and/or effort (e.g. playing of a videogame; Skosnik, Chatterton, Swisher, & Park, 2000; see also Dienstbier, 1989 for a review), activation of the HPA axis stress response is contingent upon the meaning of the stressor to the particular individual. In addition, the primary product of the HPA axis response to stress, the hormone cortisol, is unlike many other stress related hormones in that it crosses the blood-brain barrier and has direct effects on the neural activity associated with threat appraisal and self-regulation. Indeed, one of cortisol's functions is to "coordinate information processing in the limbic circuitry to promote emotion, cognition, and motivation" (de Kloet, 2010, p. 20). For these reasons, the current research focuses on the HPA axis and the release and regulation of cortisol.

The HPA axis is activated by both ascending (from the brainstem) and descending (from limbic structures) inputs (Herman, Mueller, Figueiredo, Cullinan, 2005; Ulrich-Lai & Herman, 2009). Input from multiple brain regions converge in the paraventricular nucleus of the hypothalamus, which results in the secretion of corticotropin-releasing factor (CRF); CRF travels through the hypophyseal portal system (the blood vessels that link the hypothalamus and the anterior pituitary) and stimulates the anterior pituitary to release adrenocorticotropin hormone (ACTH); ACTH is then carried via blood circulation to the adrenal cortex (the outside structure of the adrenal gland), which synthesizes and releases cortisol (Fulford & Harbuz, 2005). During stress, cortisol facilitates an increase in cardiovascular activity, alteration in cognitive and

sensory thresholds, increase in alertness, promotion of stress-induced analgesia, suppression of nonessential functions (e.g., growth, digestion, and reproduction) and the processing and consolidation of emotionally-laden memory (Susman, 2006; Davies, Sturge-Apple, Cicchetti, & Cummings, 2007). High levels of cortisol then trigger a negative feedback cycle in which the subsequent release of CRF and ACTH is inhibited, ultimately leading to a decrease in cortisol and a return to a pre-stress state (Heim & Nemeroff, 2001; Tsigos & Chrousos, 2002). Thus, a typical cortisol response to stress involves a period of reactivity (a rise in cortisol levels that are then sustained for an appropriate amount of time) and a period of recovery (a decline in cortisol levels back to baseline).

The HPA axis maintains a circadian rhythm in which ACTH is secreted in a large pulse in the morning, followed by smaller pulses throughout the day. In general, cortisol secretion mirrors these pulses of ACTH such that ultradian and circadian rhythms appear: pulsatile bursts of cortisol occur every hour and average levels of cortisol in body fluids peak upon awakening and then decrease throughout the day, with levels reaching their lowest points at the start of the sleep cycle (Young et al., 2004). The cortisol awakening response (CAR), which is the sharp increase in salivary cortisol levels approximately 30 minutes after morning waking, appears distinct from diurnal variations (Fries, Dettenborn, Kirschbaum, 2009; Pruessner et al., 1997). Both the diurnal slope of cortisol and the CAR are increasingly being examined in relation to acute and chronic stress and psychological states and traits (e.g., Adam, 2006; Chida & Steptoe, 2009; Doane & Adam, 2010; Gunnar & Quevedo, 2007; Turner-Cobb et al., 2010).

Cortisol as a measure of HPA axis functioning

The most common method of assessing HPA activity is by collecting saliva and measuring the amount of biologically active cortisol present (Miller et al., 2007). Although the current study will focus on the cortisol secretion during stress, different measures of cortisol *activity* (the term I will use when referencing all methods of assessing cortisol-related HPA functioning collectively) appear throughout the literature. As noted by Miller et al (2007), each method of assessing cortisol “provides a slightly different temporal window on cortisol activity” (p. 28); thus, any review of the literature benefits from an explication of the terms to be used when describing these measures. Adam and Kumari (2009) offer an excellent overview of cortisol parameters often examined in research, however descriptions and interpretations are briefly reviewed here.

Cortisol *reactivity* refers to the pattern of cortisol observed in response to a discrete stressor. It is generally assessed by collecting multiple samples of saliva before, during, and after a task designed to elicit a cortisol response, such as a public speech or an interpersonal interaction including a social-evaluative component (Dickerson & Kemeny, 2004). Activation of the HPA axis and cortisol release in response to stress occurs via a constellation of neural circuits that underlie the cognitive and emotional processes involved in coping, making stress-induced cortisol particularly relevant for the current study. To assess *diurnal* cortisol, researchers collect several salivary samples throughout the day and, ideally, on multiple days to account for potential day to day variability. Statistical analyses are then employed to investigate the slope of cortisol output throughout

the day, total daily output, and/or average level of cortisol throughout the day. Researchers interested in *waking* cortisol as an index of HPA axis functioning typically collect cortisol immediately upon waking and one to three samples post-awakening (15, 30, and/or 45 minutes later). Finally, *basal* cortisol is considered an index of non-stress cortisol that does not take into account the slope of cortisol across the day (unless time of day measured and controlled for in the study). Basal cortisol is commonly assessed with only one or two samples of salivary cortisol in the morning or afternoon.

Although offering the potential for a richer and more nuanced understanding of the human stress response system, the myriad ways with which cortisol can be measured and interpreted have led to significant confusion and seemingly conflicting evidence in the field. Researchers continue to struggle with identifying the particular patterns of cortisol reactivity, basal cortisol, and diurnal cortisol that reflect HPA axis dysregulation, and comparing findings across different measures of cortisol can result in inaccurate conclusions. For example, as will be reviewed, studies have shown that HPA axis functioning may be altered as a result of exposure to environmental stressors early in life; however, there are conflicting reports of when and why one might observe HPA hyper-activity versus hypo-activity. In spite of the confusion, much has been learned about HPA axis functioning as an index of stress and health across human development.

Developmental course of HPA axis functioning

As described in Gunnar and Quevedo (2007), the HPA axis undergoes development and eventual organization over the years of early childhood.

Newborns do not exhibit the typical diurnal patterns observed in adults. Although they can show cortisol responses to extreme stress, such as when undergoing significant medical procedures, this brief period of cortisol responsiveness is followed by a period of stress hypo-responsivity that continues throughout childhood. Beyond middle childhood, cortisol reactivity to stress increases with age. Basal levels of cortisol also appear to increase with age during late childhood, early adolescence, and throughout early adulthood (Trickett et al., 2010; Walker, Walder, & Reynolds, 2001). However, changes in cortisol levels during adolescence appear to depend on pubertal status (Kiess et al., 1995). Indeed, the processes associated with the production of sex steroids and the hormone cortisol are known to be interrelated (Shirtcliff, Zahn-Waxler, Klimes-Dougan, & Slattery, 2007), and there is evidence that early puberty increases stress sensitivity, especially in girls (Natsuaki et al., 2009). Importantly, animal models and preliminary research in humans indicates that these normative patterns of cortisol activity across development can be interrupted or altered as a result of exposure to early adverse experiences (Gunnar & Quevedo, 2007). Many types of childhood adversity have been implicated in alterations to the HPA axis. Before turning to this literature, it is helpful to review the primary theoretical framework within which this phenomenon has been studied.

CHAPTER 3

Allostatic Load and the HPA Axis

Theories of allostasis and allostatic load

Throughout the first half of the 20th century, physiological responses to stress were regarded within a homeostatic framework (e.g. Cannon, 1935; Selye, 1956). That is, each response was seen as a discrete attempt by the body to mobilize the resources necessary to meet the demand of the threat while also containing this mobilization in order to return the body to its baseline internal state. As evidence in the biological sciences accumulated that the larger central nervous system played a critical role, the homeostatic framework gave way to articulation of a more systems-oriented view, namely allostasis. Within an allostatic theoretical framework, exposure to a stressor perceived as threatening results in physiological accommodation such that parameters of an individual's internal milieu are varied appropriately to meet environment demand (Sterling & Eyer, 1988). Whereas homeostasis presumes a specific 'set point' that must be returned to, allostasis "provides for continuous re-evaluation of need and for continuous readjustment of all parameters toward new set points" (Sterling & Eyer, 1988, p. 637). Thus, the process of allostasis refers to the body's ability to maintain internal stability in the face of changing environments and challenges (McEwen & Wingfield, 2003). The neuroendocrine stress system is one of the primary mediators of this larger process of adaptation.

According to the allostatic load hypothesis proposed by McEwen (1998), chronic or prolonged stress responses can result in wear and tear on the organism

to such an extent that allostasis is no longer compensatory. That is, allostatic processes become harmful rather than protective, resulting in pathophysiology in the brain and body. Allostatic load has been used as the theoretical framework within which to interpret evidence that chronic and severe stress can cause alterations to HPA axis functioning, such that cortisol is no longer appropriately released and/or regulated in service of biological adaptation in the face of a stressful event. This may appear in cortisol responses to stress that continue when no longer needed, are not turned off efficiently, are not of sufficient magnitude to meet the demands of the situation, or do not habituate to the recurrence of the same stressor (McEwen, 2007).

Although cortisol is a hormone that is necessary for critical metabolic and cognitive processes during and outside of stress, it can have very negative effects on the brain over time, including neural degeneration and immune system suppression (McEwen, 2001). Moreover, when cortisol is not secreted at an optimal rate or amount, physical and mental health problems can result. For example, higher basal cortisol and greater cortisol reactivity have been linked to internalizing disorders, such as depression (see Lopez-Duran, Kovacs, & George, 2009 for a review). Conversely, there is some indication that low levels of basal cortisol are associated with externalizing symptoms (see Alink, van IJzendoorn, Bakermans-Kranenburg, Mesman, Juffer & Koot, 2008 for a review). Thus, understanding the mechanisms by which exposure to early adversity can lead to physiological dysregulation may be a critical step in an ongoing effort to prevent or intervene in the development of stress-induced mental health problems.

Stress, cortisol activity and the attenuation hypothesis

Historically, exaggerated cortisol responses and elevated levels of basal cortisol were regarded as the primary correlate to stress-induced physical ailments, whereas relatively lower levels of cortisol activity were interpreted as an index of positive adaptation. However, a growing body of evidence suggests that early adverse experiences may result in a deficiency in basal cortisol or attenuation of the cortisol stress response. *Attenuated* cortisol activity is evidenced by reduced cortisol secretion at some point during the circadian cycle, reduced cortisol reactivity to stress, or enhanced negative feedback inhibition of the HPA axis as indicated by a pharmaceutical challenge (Heim, Ehlert, & Hellhammer, 2000). Gunnar and Vazquez (2001) reviewed a number of studies that identified an association between adverse early life conditions and a flattening of daytime cortisol production among infants and toddlers. This association was found among infants and toddlers living in orphanages in Eastern Europe and family-reared children experiencing neglect or at high-risk of neglect in the United States. Negative childhood family environments, such as those characterized by marital conflict and/or low parental warmth and care, have also been associated with attenuated cortisol reactivity in children (Davies et al., 2007; Granger et al., 1998) and emerging adults (Luecken, Kraft, & Hagan, 2009). Prospective studies have found evidence that chronic stress during childhood results in diminished basal cortisol and cortisol reactivity to subsequent environmental stressors at later developmental periods. For example, among parentally bereaved youth, a greater number of post-bereavement negative events

in childhood predicted lower cortisol output during a parent-child conflict discussion task six years later when youth had reached adolescence (Hagan, Luecken, Sandler, & Tein, 2010).

Despite the evidence for a relation between early adversity and attenuated cortisol activity, there have also been reports of chronic and/or episodic stressors during childhood resulting in exaggerated cortisol activity (e.g., Bevans, Cerbone, & Overstreet, 2008; Cutuli, Wiik, Herbers, Gunnar, & Masten, 2010; Lupien, King, Meaney, & McEwen, 2000, 2001; Marin, Martin, Blackwell, Stetler, & Miller, 2007). It has been proposed that this seemingly inconsistent pattern of results is an artifact of the timing at which cortisol is measured relative to the stressful experiences (Miller et al., 2007). More specifically, it is believed that individuals exposed to major stressors will initially exhibit a sensitized HPA axis and exaggerated cortisol reactivity to subsequent stressors; however, this pattern will change as result of the powerful negative feedback system built into the HPA axis, in which higher cortisol concentrations act back upon glucocorticoid receptors and facilitate the down-regulation of the HPA axis. In this way, a chronically activated HPA axis could “mount a counter-regulatory response such that cortisol output *rebounds below normal* [italics added]” in the service of biological adaptation over developmental time (Miller et al., 2007, p. 26).

This “attenuation hypothesis” received support from a meta-analysis conducted by Miller and colleagues (2007), which indicated an initial positive association between stress onset and cortisol activity (e.g., elevated morning levels, total daily output, or cortisol response to pharmaceutical challenge)

followed by an inverse association between stressors and cortisol as time since the stressor increased. More recent retrospective studies provide additional evidence for this inverse relation. For example, in a sample of healthy older adults (ages 54 – 68), an increased number of stressful life events during childhood and adolescence (e.g., severe illness of self or significant other, negative socio-economic circumstances, relational stress, problem behavior of significant others, etc.) was associated with lower cortisol reactivity to a psychosocial stress task (Armbruster, Mueller, Strobel, Lesch, Brocke, & Kirschbaum, 2011). Similarly, young adults' cortisol response to the same kind of task was shown to be inversely related to the number of major negative life events that their mothers experienced while pregnant with them (Entringer, Kumsta, Hellhammer, Wadhwa, & Wust, 2009). Carpenter and colleagues (2009) also found an inverse relation between self-reported history of emotional abuse during childhood and a diminished cortisol response to a pharmaceutical challenge. Importantly, they found that the magnitude of the association increased with advancing age, offering indirect support of the development of attenuation over time.

The primarily cross-sectional nature of these studies prevents an examination of whether trajectories of cortisol activity within individuals across time “corresponded, in any systematic manner, to psychosocial demands and stressors of key developmental periods” (Trickett et al., 2010, p. 165). A few recent longitudinal studies, however, offer preliminary evidence. In a prospective study of the impact of early neglect and abuse on cortisol activity in adulthood, adoptive parents provided information on neglect and abuse prior to adoption, and

adoptees' diurnal cortisol was later assessed when they reached adulthood. Morning cortisol levels were significantly lower in those who had experienced severe neglect or abuse compared to those who did not experience neglect or abuse (van der Vegt, van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009). Although prospective, cortisol was not measured at the time of adoption nor was cortisol assessed at different developmental stages prior to adulthood. These limitations were addressed in a study conducted by Trickett and colleagues (2010). They examined the developmental course of non-stress morning cortisol among 173 females (84 of whom had confirmed familial childhood sexual abuse) across an 18-year period that spanned childhood, adolescence, and early adulthood. Interestingly, in comparison to females who had not experienced sexual abuse in childhood, those with a history of childhood sexual abuse exhibited higher morning cortisol in childhood, attenuation of basal cortisol across adolescence and significantly lower levels by early adulthood. Women who had not experienced sexual abuse in childhood showed increasing levels of non-stress cortisol across the 18-year period. This result offers compelling evidence in support of the attenuation hypothesis, suggesting that individuals who experience stressors during childhood may exhibit lower cortisol activity by the time they reach emerging adulthood.

Needless to say, these relations are highly complex and do not reflect simple causal pathways from stress to cortisol activity. Indeed, the process by which exposure to stressful life events leads to psychobiological dysregulation over time is not well understood. Theories abound regarding child-level (e.g., ego

resilience and ego control; Cicchetti & Rogosch, 2007), family-level (e.g., positive parenting; Hagan et al., 2011), and contextual (e.g., racial discrimination; Sellers, Copeland-Linder, Martin & Lewis, 2006) variables that may interact with exposure to adverse events to affect psychological and/or physiological functioning. In addition, much remains to be learned about the pathways by which adverse childhood events lead to particular patterns of physiological activity later in life and factors that might account for the individual variability seen in the biological effects of stressful events. There is a need to examine potential moderators and mediators of the relation between stressful events and cortisol activity. Such an examination could identify groups of individuals who are most vulnerable to the neurobiological effects of stress as well as more proximal predictors of physiological dysregulation, thereby offering additional ways to halt or mitigate the consequences of allostatic load.

CHAPTER 4

Coping Style as a Candidate Mechanism

Conceptualizations of coping

Over the last 15 years, there has been extensive research on how coping processes contribute to successful (or unsuccessful) adaptation in youth facing stressors such as family conflict, illness, and chronic pain (for a review see Compas, Conner-Smith, Saltzman, Thomsen, & Wadsworth, 2001). How individuals cope has been shown to play a role in the relation between stressful life events and mental health outcomes (Grant et al., 2006; Zautra, 2003), and coping is no less likely to play a similar role in the link between early adversity and later physiological functioning. Decades of studies of coping in humans exposed to psychosocial stress are rooted in a relational and process-oriented cognitive theory proposed by Lazarus and colleagues (Lazarus, 1966; Lazarus & Folkman, 1984). In their conceptualization, psychological stress is a particular interaction between an individual and his or her environment that is “appraised by the person as taxing or exceeding his or her resources” (Lazarus & Folkman, 1984, p. 19). Within this framework, coping is described as an individual’s “constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands” associated with the psychological stress (p. 141).

Although described within this cognitive framework as a situational process dependent upon a particular transaction (some refer to this as “contextual coping”; Moos & Holahan, 2003), coping can also be conceptualized as a style or trait (“dispositional coping”; Moos et al., 2003) that may influence new situations

(Carver & Scheier, 1994). Coping style refers to “a general and pervasive tendency of an individual to prefer a particular class of coping reactions regardless of the specific problem” (Kavšek & Seiffge-Krenke, 1996, p. 653). This conceptualization is particularly relevant to studies of coping within the context of adaptation across developmental time. Indeed, the multi-level systems perspective of coping articulated by Skinner and colleagues (Coping Consortium, 1998, 2001; Skinner & Zimmer-Gembeck, 2007, 2009) depicts coping as an adaptive transactional process in which coping and influential variables (e.g. appraisal processes, social factors, individual factors, etc.) interact to affect the management of stressors within different scales of time, including developmental time. The model is organized into three nested levels in which coping is 1) an interactional process on the scale of “real time”; 2) an episodic process that is shaped by previous interactions and includes the resources and liabilities that those stressful transactions have produced; and 3) an adaptive process, on the scale of developmental time, through which adversity has long-term effects on individual adjustment (Skinner & Zimmer-Gembeck, 2009). Within the current study, coping is viewed from this top-level, developmental perspective.

Although research on coping processes has largely focused on coping as an episodic process (Skinner & Zimmer-Gembeck, 2007), attempts to categorize and measure episodic coping are relevant to the study of coping at a more macro-level. Lazarus and Folkman (1984) identified two major classes of coping efforts: problem-focused and emotion-focused coping. Problem-focused coping includes responses that are directed toward the stressor and the relationship between

oneself and the environment. Emotion-focused coping includes efforts that manage the emotions that result from the stressor. According to Lazarus & Folkman (1984), coping efforts are neither inherently good nor bad; however, emotion-focused coping has often been regarded as an approach most associated with internalizing disorder (e.g. Compas et al., 2001). Unfortunately, the qualitative overlap between emotion-focused coping efforts and depressive symptoms (e.g., expression of emotion) has made it impossible to infer a causal relationship between the two.

Other researchers have suggested that an alternative higher-order model best captures coping efforts. Based on a confirmatory factor analysis of studies that measured various aspects of coping, Tobin and colleagues (1989) concluded that the over-arching constructs of engagement and disengagement provide a better structure for categorizing coping processes. Engagement coping includes efforts that are oriented toward the source of stress or toward one's own emotions and cognitions regarding the stressor, whereas disengagement coping includes efforts oriented away from the source of stress or one's own emotions and cognitions. Thus, Lazarus's problem-focused and emotion-focused coping could be characterized as either disengagement or engagement depending on whether the effort was oriented toward or away from the stressful experience. Similar conceptualizations of engagement and disengagement coping are common throughout the literature, including repression/sensitization, passive/ active, and avoidant/approach (Compas et al., 2001; Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000; Kavšek & Seiffge-Krenke, 1996).

Child and adolescent coping styles: stability, consistency, and outcome

Overall as children develop their coping repertoire and self regulatory abilities increase (although not necessarily in a linear fashion; see Losoya, Eisenberg, & Fabes, 1998). Studies have documented moderate stability in coping efforts during childhood and adolescence, including correlations ranging from .26 to .35 over a 9-month period (Compas, Malcarne & Fondacaro, 1988) and .29 to .34 over a 12-15 month period (Moos, 1993). Stability has also been noted in the transition from adolescence to young adulthood, with moderate relations between active and avoidant coping in early adolescence and in emerging adulthood reported (Hussong & Chassin, 2004). Despite these moderate correlations, significant variability has been found in the relative use of different coping styles across time. Whereas active coping appears to increase from ages 12-19, avoidant coping ceases to increase after early adolescence (Seiffge-Krenke, Aunola, & Nurmi, 2009).

Although moderate levels of consistency in adolescents' use of a particular coping style across different situations has been documented by some (Griffith, Dubow, & Ippolito, 2000; Jaser et al., 2007), others have found that developmental changes in coping are situation specific, with older adolescents using more active coping in school-related stressful events and greater avoidant coping in response to stressors that occur at home (Seiffge-Krenke et al., 2009; Zimmer-Gembeck & Locke, 2007). Developmental changes may also vary depending on the life histories of youth; whereas Losoya and colleagues (1998) found increased use in older adolescents compared to early adolescents in a

normative sample of youth, Hussong and Chassin (2004) reported decrements in active coping from early adolescence to emerging adulthood in a sample of children of alcoholics.

The impact of childhood experiences on adolescent coping

Although the better part of brain development occurs prior to age 5, the brain regions involved in coping undergo development throughout much of childhood and in some cases adolescence. Basic cognitive functions that depend on areas of the prefrontal cortex (PFC) develop rapidly during the school-age years (Diamond, 2002). As such, children develop more complex language and meta-cognitive capacities; they increasingly use mentalistic strategies to cope; and their responses to environmental stressors continue to become more differentiated (Eisenberg et al., 1997). These methods are likely to include more sophisticated problem-focused efforts, such as generating alternative solutions to solving problems and using self-reassuring statements to calm negative emotions (Aldwin, 2007; Compas et al., 2001; Moss, Gosselin, Rousseau, & Dumont, 1997). The development and employment of these stress management strategies depend a great deal on working memory and other aspects of executive functioning, processes that require strong neural connections between regions of the PFC and other areas of the brain (Compas, Campbell, Robinson, & Rodriguez, 2009). The PFC does not reach full maturity until young adulthood (Diamond, 2002), suggesting that stress experienced throughout childhood, if not managed, could alter the structure or function of PFC regions (Gunnar, Fisher, & Early Experience, Stress, & Prevention Network, 2006). These alterations, in turn, may

lead to deficits in processes critical to active coping efforts, such as decrements in the ability to hold information in mind and access that information to guide actions while inhibiting other potential responses.

As noted by Ganzel et al. (2010), the process of regulating one's cognitions, emotions, and physiological functioning likely "changes over time as a function of the life history of the individual" (p. 139), and prospective studies support this notion. Research findings suggest a risk-oriented pattern of relations such that increased exposure to negative events leads to more avoidant coping (and/or less active coping), which in turn predicts increases in mental health problems (for a review of increased stress predicting increased avoidant coping see, Seiffge-Krenke, 1998). In a longitudinal study of youth coping behaviors across early and late adolescence, Seiffge-Krenke and colleagues (2009) found that greater increases in stress in early adolescence predicted higher subsequent use of particular active coping strategies (e.g., acceptance, cognitive restructuring). A greater number of negative events also predicted increased use of cognitive restructuring and problem-solving strategies in a sample of children of divorce (Sandler, Tein, & West, 1994).

Findings from several investigations also suggest that coping may be an important mediator in the link between stressful events and mental health outcomes. In a sample of children who had recently experienced parental divorce, avoidant coping was found to mediate the relation between divorce-related stressors and depression, anxiety and conduct problems (Sandler et al., 1994). Avoidant coping has also found to mediate the relation between environmental

stress post-divorce (as indicated by high levels of maternal demoralization, lower family income, frequency of negative life events over the previous month and exposure interparental conflict) and internalizing symptoms (Sandler, Tein, Mehta, Wolchik, & Ayers, 2000). Studies of relations between negative events, coping and mental health among with children of divorce have largely been cross-sectional; however, investigations with other populations have demonstrated prospective relations as well. For example, using path analysis, Cheng & Lam (1997) found that the accumulation of negative life events among a sample of adolescents increased deficits in problem-focused coping (i.e. active coping) several months later, and these deficits were associated with lower self-esteem and greater dysphoria. Although partially cross-sectional, the temporal precedence of negative life events and coping/mental health allowed for statistical control of earlier dysphoria and coping efforts. Snow et al. (2003) found partial mediation in a sample of adult females such that increased work stressors were associated with avoidant coping, and this style of coping, in turn, predicted increased mental health symptoms four months later. Although not unequivocal (i.e., Grant & Compas, 1995 did not find evidence of coping as a mediator between family stress and psychological symptoms in female youth), investigations of coping as a mediator between stressors and mental health outcomes offer reason to believe that coping may be one mechanism by which stressful experiences lead to ongoing physiological dysregulation.

Empirical evidence for direct relations between coping & cortisol activity

Research implicating coping as a key player in the stress-mental health connection coupled with evidence that cortisol plays a role in the development of mental health problems suggests that coping may also be associated with cortisol activity. The neural underpinnings of reactivity and regulation in the face of stress offer reason to believe that adversity-induced alterations to an individual's capacity to cope could lead to physiological dysregulation in the long-term. For example, the activation of the hormonal cascade involved in cortisol production is dependent upon the integration of the cognitive and affective processes critical to adaptive coping efforts. Areas of the PFC participate in both the activation and regulation of the HPA axis, suggesting that higher order cognitive functioning plays a critical role in HPA axis reactivity (Sullivan & Gratton, 2002). Activation in the ventrolateral areas of the *left* PFC, regions that have been shown to facilitate the processing of approach-related goals (e.g. Davidson, 1994), has been associated with decreased cortisol reactivity to psychosocial stress (Wang et al., 2005). Conversely, activity in the lateral and ventral regions of the *right* prefrontal cortex has been associated with increased cortisol responses to psychosocial stress (Kern et al., 2008; Taylor et al., 2008; Wang et al., 2005). Excessive activity in these areas has been associated with withdrawal behaviors and negative felt emotions (Sullivan & Gratton, 2002), suggesting an association between avoidance behaviors, sadness, and contemporaneously high levels of cortisol.

Sub-cortical regions involved in the processing of stressful events also participate in the modulation of the HPA axis. For example, the amygdala is involved in processing threatening stimuli (for recent reviews, see Dedovic et al.,

2009; Ganzel et al., 2010). In humans, the amygdala has also been implicated in the fear-conditioning process, attention to emotionally-laden stimuli and other associative learning processes (Pessoa, 2008). It has been shown to play a role in adjusting attention thresholds and participating in the ongoing excitation of the HPA axis (Dedovic et al., 2009). This has implications for coping because if emotional and cognitive regulation in the context of stress fails, cortisol levels remain elevated and “promote processing of emotional information by recruiting molecules in the amygdala supporting this positive feedback action” (de Kloet, 2010, p. 20).

Almost thirty years ago, Lazarus and colleagues (1982) suggested that coping was also a key factor in understanding the physiological stress response system (as cited in Bohnen, Nicolson, Sulon, and Jolles, 1991). Although the research on coping and physiological reactivity has been primarily cross-sectional and results have been inconsistent across studies, a brief review of the major themes from research on relations between coping efforts and physiological functioning can guide investigations into the influence of coping on cortisol activity over the long-term. With some exceptions (e.g., Brandtstadter et al., 1991; Gunlicks-Stoessel & Powers, 2009; Lam, Dickerson, Zoccola, & Zaldivar, 2009), active coping strategies have been primarily associated with lower cortisol activity among non-clinical populations, including lower baseline cortisol during a psychosocial stress task in a young adult sample (Taylor, Lerner, Sherman, Sage, & McDowell, 2003), lower total cortisol output across the day among middle-aged adults caring for a relative with traumatic brain injury (Turner-Cobb

et al., 2010), and lower cortisol reactivity among young women participating in an anger-priming experiment (Matheson & Anisman, 2009). In addition, Spangler et al. (2002) found that students who utilized acceptance and adaptive cognitive appraisal strategies during an oral psychology exam exhibited attenuated cortisol reactivity to the exam compared to high anxiety students. Bohnen, Nicolson, Sulon and Jolles (1991) found a significant negative correlation between cortisol responses during four hours of continuous mental stress tasks (one of which was a videotaped speech task) and “comforting cognitions” (e.g. consideration of the problem in a relative way, use of self-encouragement, and positive reframing of the event). Similarly, healthy older adults who engaged in social support seeking and problem engagement during the day were found to exhibit lower total daily cortisol output (O’Donnell, Badrick, Kumar, & Steptoe, 2008).

Disengagement strategies, such as denial, avoidance, and wishful thinking, have been related to both higher and lower levels of cortisol activity. Rohrmann, Hennig, and Netter (2002) reported that individuals who used coping strategies characterized by avoiding perceptions of threat exhibited significantly higher cortisol concentrations following a public speaking task than those with a coping style characterized by approach and increased attention. Similarly, a study of the stress response in newly trained male firefighters found that those endorsing greater avoidance of threat information exhibited greater increases in cortisol reactivity in a similar stress task (Roy, 2004). In one of the few studies to explicitly examine coping responses and neuroendocrine activity in youth, children who were faced with hospitalization for a surgical procedure and who

used denial during their hospital stay had higher urinary cortisol compared to children who used intellectualization (Knight et al., 1979).

In contrast, at the advent of human psychoneuroendocrinology studies during the 1950's and 1960's, Mason and colleagues found a pattern across studies in which attenuated cortisol responses were seemingly induced by disengagement (see Mason et al., 2001). For example, immediately prior to a highly risky cardiac surgical procedure, individuals who "disengaged" (e.g. social and intellectual withdrawal) exhibited lower cortisol compared to those who "engaged" (e.g. "active emotional participation"; Mason et al., 2001, p. 388) in the stressfulness of pre-operational procedures. They found a similar pattern in a prospective study of parents of children with terminal illness: parents who typically used denial and avoidant coping strategies exhibited "surprisingly low mean cortisol" (Mason et al., 2001, p. 388), and levels of cortisol dropped further when parents were exposed to acute stress. Similarly, a longitudinal study of a special forces team in Vietnam (as cited in Mason et al., 2001) found that the men who used disengagement coping strategies had lower cortisol levels on days they expected an attack, whereas those who were engaged in meeting the threat showed elevated cortisol levels. Although these studies may be criticized for the questionable operationalization of disengagement coping (i.e. "active defensive or antiarousal intrapsychic mechanisms"; Mason et al., 2001, p. 388), an inverse relation between disengagement and cortisol has also been seen in young and middle-aged adults in experimental studies. For example, Hori and colleagues (2010) found that healthy adults with an avoidant coping style evidenced blunted

cortisol reactivity to a pharmaceutical challenge. Blackhart, Eckel and Tice (2007) reported that the relationship between peer rejection and cortisol activation was moderated by high repressive/defensive coping (a component of avoidant coping): rejected participants who exhibited repressive/defensive coping styles had significantly lower cortisol than less defensive rejected participants after peer rejection.

In sum, with some exceptions, active coping appears to be contemporaneously associated with relatively lower cortisol output, including lower baseline levels and attenuated reactivity to a lab task. Unfortunately, little is known about longitudinal or prospective relations between an active coping style and later cortisol activity. The evidence for associations between avoidant coping and cortisol is more mixed. Although the majority of cross-sectional studies revealed a positive relation between avoidant coping and cortisol activity, a few prospective, longitudinal studies found the opposite relation. Several limitations preclude drawing substantive conclusions from this literature. For example, the studies reviewed above vary in population sampled (e.g., age, clinical vs. non-clinical, etc.), methods of cortisol measurement (e.g., urinary vs. salivary, diurnal vs. basal vs. reactivity, etc.), and theoretical and methodological approach to coping style (e.g. psychoanalytic vs. cognitive-behavioral).

Most importantly, in the majority of studies attention was not given to individuals' life histories nor was the influence of individuals' inherent self-regulatory abilities (i.e. temperament) considered. In addition to impacting physiological activity in the long-term, exposure to adverse events early in life is

likely to play a critical role in how individuals cope with events later in life. However, it is also known that not everyone responds to an increase in negative life events with one particular style of coping. Factors not yet discussed but critical to examinations of relations between stress, coping and cortisol activity are the individual-level resources and liabilities that influence behavioral and physiological responses to stress in the short and long-term. As will be reviewed in the next section, child temperament has long been recognized as a significant influence on behavioral and physiological responses to stress; however, little research has been conducted examining all of these factors simultaneously and across different developmental stages.

CHAPTER 5

The Role of Child Temperament

As indicated by the variability seen in youth outcomes following stressful experiences, negative life events in childhood do not impact all youth in the same way. It is likely that not all youth respond to increases in negative life events by developing a coping style that is primarily avoidance-oriented. Indeed, some studies have found that increased stress leads to lower active coping (not necessarily higher avoidant coping), which in turn leads to symptoms, and other studies have found no evidence of coping as a mediator of the stress-mental health connection (e.g., Grant & Compas, 1995). Based on a review of research on the relations between stress and psychopathology in youth, Grant et al. (2003) emphasized the need to examine the influence of theory-based moderators on the relation between stressors and outcomes via a particular mediator in an effort to better explain variability in stress-related outcomes. Derryberry, Reed & Pilkenton-Taylor (2003) discuss the usefulness of an individual differences approach to studying children's coping, with an emphasis on temperament as a powerful influence on stress and coping. Owing to its biological basis and relative stability, temperament is particularly relevant to the long-term functioning of the stress response system. As a pre-existing characteristic, temperament may be the diathesis that stress acts upon, subsequently influencing the development of particular coping strategies and an overall style of responding to stress.

Temperament characterizes individual's emotional and behavioral style across different situations and settings; it is considered a heritable trait that is

biologically based, apparent very early in life, and relatively stable across development (Bates, 1987; Rothbart & Bates, 1998). Temperament influences how children appraise and encode stressful and non-stressful events, how they feel in relation to the events, and how they respond behaviorally and biologically (Rueda & Rothbart, 2009). It has long been considered a multidimensional construct, and starting with Thomas and Chess (1977), countless models have been proposed to capture its primary dimensions. Three broad dimensions appear consistently across models of temperament: regulation of attention and activity, negative affectivity, and extraversion/surgency (Rothbart, 2007). Regulation of attention and activity is often conceptualized as “effortful control of emotions and behaviors, self-regulation, task persistence, and attentional focus” (Compas, Connor-Smith, & Jaser, 2004, p. 23). Negative affectivity, or emotional reactivity, refers to sensitivity to threat and negative environmental stimuli; a tendency to feel discomfort, anger, fear or sadness; and a resistance to being soothed (Buss & Plomin, 1984; Rothbart, 2007). Traditionally, extraversion or surgency have been conceptualized as reflecting positive emotionality and tendency toward approach-related behavior (e.g., Buss & Plomin, 1984). The different dimensions described above can be traced back to the combination of these characteristics into three broad temperament types that were proposed by Chess and Thomas (1985): easy, difficult, and slow-to-warm up. An “easy” temperament reflects well-regulated approach behavior, adaptability, and overall positive mood. In contrast, a “difficult” temperament is characterized by biological, behavioral, and emotional reactivity and frequent negative moods. Finally, the “slow-to-warm-up” style

characterizes those children who exhibit a combination of negativity initially and adaptability over time.

Although temperament has been described as encompassing “primitive coping mechanisms” (Derryberry, Reed, & Pilkenton-Taylor, 2003, p. 1049), there is conceptual and empirical precedent for distinguishing between the two. Saarni et al. (2006) offer a useful metaphor for temperament and emotion that can be modified to distinguish between temperament and coping. Whereas temperament is like a season of the year, coping is more like the weather. As such, “the season provides constraints on the daily weather, just as temperament may provide some degree of limitation [on coping]” (p. 273). But to what degree is temperament related prospectively related to coping style? Clark, Watson, & Mineka (1994) offer alternative models for how personality relates to psychological functioning, and two of these models are particularly relevant when considering associations between temperament and coping: the vulnerability model and the pathoplasty model. Whereas the vulnerability model would predict that a certain temperament would predispose individuals to developing maladaptive coping styles, the pathoplasty model would predict that a particular temperament would interact with a stressful environment to predict maladaptive coping “without necessarily having a direct etiological role” (Clark et al., 1994, p. 103).

Some researchers have found a direct relation between negative emotionality and avoidant coping and links between constraint-attentional control and active coping (e.g., Lengua & Long, 2002; Rueda & Rothbart, 2009), thereby

offering support for the vulnerability model. For example, among children who have experienced parental divorce, negative emotionality has been found to indirectly predict avoidant coping via its influence on threat appraisal of stressful events (Lengua, Sandler, West, Wolchik, & Curran, 1999). However, others have shown interactions between temperament and environment that are indicative of the pathoplasty model with regard to overall adjustment (e.g., Kliewer et al., 2004; Kliewer, Reid-Quinones, Shields, & Foutz, 2009; Lengua, 2002; Lengua, Wolchik, Sandler, & West, 2000), however, less research has been conducted on the interaction between temperament and negative events on coping.

Muris & Ollendick (2005) note that “it is likely that both reactive and regulative temperament factors really come into play when the child is exposed to adverse or stressful circumstances,” (p. 284), and many other researchers have echoed this sentiment (e.g., Compas et al., 2004; Wachs & Kohnstamm, 2001). In an early review of the determinants of children’s coping within the context of medical issues Rudolph et al. (1995) suggested that children with temperaments characterized by deficits in self-regulation may respond to an increase in stressful events with greater mental distress and rely on avoidant strategies to manage this distress rather than active coping efforts oriented toward the stressor. More recently, Strelau (2001) articulated a compelling argument for temperament as a moderator of life stress and supported it with human and non-human studies that demonstrate health as a function of an interaction between stress, emotional reactivity and arousability. Consistent with this, it has been found that childhood exposure to stressors such as marital conflict and parental problem drinking

predicts mental and physical health outcomes only among those with low vagal tone, a biological index of self-regulation (see Eisenberg, Valiente, & Sulik, 2007 for a review).

The current research proposes a pattern of relations between negative events, temperament, coping and physiological activity that is in line with the pathoplasty model, whereby temperament is proposed to moderate the relation between negative life events in childhood and coping style in adolescence, which is proposed to relate to cortisol activity in young adulthood. Consistent with the predominant model of temperament as encompassing both reactivity and regulation (Rothbart & Rueda, 2009), temperament in the current study is operationalized as a composite of negative emotionality, constraint (e.g., low impulsivity) and attentional-control.

CHAPTER 6

The Proposed Study

The purpose of the current research is to explore relations between childhood negative life events, child temperament, coping style in adolescence, and cortisol activity in young adulthood among individuals whose parents divorced during childhood. As a vulnerable population, children of divorce are an ideal group in which to study the effects of stressful events on physiological functioning in the long-term. It is estimated that approximately 30% of children in the U.S. will experience parental divorce before reaching age 12 (Kennedy & Bumpass, 2008). A significant body of research demonstrates that parental divorce increases the risk for multiple problems throughout the lifespan, including clinical levels of mental health problems, mental health services use, psychiatric hospitalization, substance abuse and other risky health behaviors, and physical health problems (Amato, 2001; Chase-Lansdale, Cherlin, & Kiernan, 1995; Kessler et al., 1997; Maier & Lachman, 2000; Makikyro et al., 1998; Rodgers, Power, & Hope, 1997; Troxel & Matthews, 2004).

According to transitional events theory (Felner, Terre, & Rowlinson, 1988), it is the cascade of stressful life events that occur after the divorce rather than the event itself that impact children's long-term adjustment. Indeed, following divorce, affected family members experience an increased number of negative life events. As noted by Sandler, Kim-Bae and MacKinnon (2000), "post-divorce stressors present unique challenges because they often involve family changes that are beyond the child's direct control", such as change in

residence, parenting challenges, and decreased family income (p. 337). During the transition period that follows parental divorce, negative life events may be particularly influential in the development of youth's coping strategies (Skinner, 1995; Sandler et al., 2000). Given that many cross-sectional studies have found significant relations between coping behaviors and physiological reactivity, it may be that coping behaviors mediate relations between early negative events and later cortisol activity. Further, dimensions of child temperament have been found to impact the ways that children of divorce respond to stressful events (Lengua & Long, 2002), and many researchers have called for more research that incorporates both coping and temperament in studies of the neurobiology of stress (see Skinner & Zimmer-Gembeck, 2009, for an extensive discussion of this issue).

In an effort to meet these calls for additional research, the current research examines prospective relations between post-divorce negative events and later cortisol activity and tests a theoretically-based moderated mediation model in an effort to elucidate how such relations might unfold. More specifically, the current study will utilize a prospective, longitudinal design to test the following hypotheses among a sample of individuals who experienced parental divorce during late childhood and early adolescence (between ages 7 and 12 years of age):

Hypothesis 1 (H1): A greater number of negative life events during a discrete period of childhood (assessed at 4 time points over a 9-month period when youth were between the ages of 9 and 12 years old) will predict attenuated cortisol activity (as measured by total cortisol output during a task and cortisol

reactivity to a task) 15 years later, when youth have reached emerging adulthood (see Figure 1a).

Hypothesis 2 (H2): A greater number of negative life events will lead to greater reliance on the use of maladaptive coping (greater avoidance and less active coping) 6 years later when youth have reached adolescence; maladaptive coping, in turn, will predict attenuated cortisol reactivity in emerging adulthood (see Figure 1b).

Hypothesis 3 (H3): Given that temperament may strongly influence a child's response to stress, a third model will be examined. It is proposed that compared to youth with high self-regulatory abilities (low negative emotionality, low impulsivity, and high attentional focus, assessed by mother report at baseline), negative life events will lead to greater use of maladaptive coping in adolescence among youth with poor self-regulation (high negative emotionality, high impulsivity, and low attentional focus), and maladaptive coping, in turn, will be inversely related to cortisol activity in young adulthood (see Figure 1c).

CHAPTER 7

Methods

Participants

The sample in the current study was drawn from a larger randomized controlled trial that evaluated a preventive intervention, the *New Beginnings Program* (NBP), designed to reduce mental health problems in children from divorced families. The NBP was provided in late childhood, and six waves of data were collected. The current study includes all youth who participated and provided saliva samples in the 15-year follow up study. As described in previous publications (e.g., Wolchik et al., 2000, 2002), participants in the original controlled trial of the NBP were recruited via court records of over 1,800 divorce decrees (randomly selected) that were granted in Maricopa County. Families were eligible to participate in the study if (a) divorce occurred in the previous two years; (b) the mother was the primary residential parent; (c) at least one child between the ages of 9 and 12 lived with the mother (more than 50% of the time); (d) neither mother nor child were receiving mental health services at the time and the child was not in a special education program; (e) mother had no plans to remarry during the trial and custody arrangements were likely to stay stable throughout the trial; (f) mother and child could complete the assessment batteries in English; (g) the child was taking medication if diagnosed with attention-deficit/hyperactivity disorder, did not score above the 97th percentile on the Children's Depression Inventory (Kovacs, 1981) or the Externalizing subscale of the Child Behavior Checklist (Achenbach, 1991), and did not endorse items

related to suicidal ideation. Of those contacted by phone, 671 met eligibility criteria, and 240 were enrolled in the study. Enrollees were randomized to one of three conditions: 1) a program for custodial mothers (n=81), a dual-component program for custodial mother and child (n=83), or a self-study condition (n=76).

Of the 240 families originally enrolled in the controlled trial, 194 participated in the 15-year follow-up. The current study includes participants who provided saliva samples at the 15-year follow-up, regardless of group assignment. Of the 194 individuals who participated in the follow-up, seven people refused to provide a saliva sample and four were out of the country (they were interviewed via *Skype*), leaving 183 individuals for whom salivary cortisol samples were available. Participants were excluded if they met any of the follow criteria established *a priori*: cortisol concentration that was outside of normal physiological parameters, only one viable cortisol sample was available (thereby providing insufficient information to look at change across task), pregnant or breast feeding at the time of cortisol collection, current use of medication known to impact cortisol activity (e.g., steroids), and/or saliva samples were collected outside before 2pm and/or after 10pm (one hour pre and post the *a priori* collection time frame of 3pm – 9pm). The decision to exclude pregnant and breast-feeding women was based on evidence that cortisol responses to laboratory stress tasks (such as the one used in the current study) appear to be impacted by stage of pregnancy and lactation (Foley & Kirschbaum, 2010). For example, women in the second trimester of pregnancy have been found to exhibit increased responses to a standardized task whereas women who are breast-feeding show

blunted cortisol responses. Finally, the collection time frame of 3pm – 9pm was chosen in order to avoid the peak in cortisol that occurs after awakening. A one hour window on each side was deemed acceptable *a priori* so as to not exclude more individuals than was really necessary.

Of the 183 individuals who provided saliva samples, one person had a cortisol concentration that was outside normal physiological parameters, one person provided saliva samples outside of the designated timeframe, one person had only one viable cortisol sample, and nine women were pregnant or breast-feeding. In addition, seven individuals were taking thyroid medication, which artificially impacts gland function directly involved in the stress response. Thus, 19 individuals were excluded based on pre-set exclusion criteria. Although not specified *a priori*, it was decided that an individual with human immunodeficiency virus would also be excluded, based on the rationale that this individual's stress response may be affected by this rare condition. The 20 excluded individuals were compared to the remaining 163 individuals on all primary study variables, baseline mental health, and current mental health. Two of the 16 *t*-tests conducted showed marginal differences with a trend toward significance. Excluded participants exhibited higher average total cortisol compared to those included in the study (2.10 vs. 1.53; $t = 1.92$, $df = 17.97$, $p = .07$) and lower levels of adolescent avoidant coping compared to included participants (8.63 vs. 9.45; $t = -1.988$, $df = 22.17$, $p = .06$). Of the 163 individuals included in the current study, 86 were male (53%) and 153 were White Non-

Hispanic (94%). The average age at the 15-year follow-up was 25.5 years old (SD = 1.2; Range = 24 - 28).

Procedure

Participants were followed across a period of 15 years, with assessments conducted over six waves: baseline (prior to randomization; W1), approximately 3 months post-baseline (after the intervention groups received the intervention; W2), 6 months post-baseline (W3), 9 months post-baseline (W4), 6 years post-baseline (W5), and 15 years post-baseline (W6). All measures were administered in interview format. Children and mothers were interviewed separately by trained interviewers. At W1-W5, mothers signed consent forms and children signed assent forms (if under the age of 18). At W6, young adults signed consent forms. The ASU Institutional Review Board approved all measures and procedures.

At the 15-year follow-up (W6), young adults participated in a modified version of the Trier Social Stress Task (TSST), which consisted of two stressors: mental arithmetic and a videotaped speech task in which the participant discussed their strengths and weaknesses. Owing to the inclusion of social evaluation (one of the factors most consistently related to cortisol reactivity, see Dickerson & Kemeny, 2004), this task has been found to induce a stress response in healthy and non-healthy individuals (Foley & Kirschbaum, 2010), producing up to a three-fold rise in salivary cortisol among 70 - 85% of individuals who participate in the task (Kudielka et al., 2007). In the current study, the study design was such that participants were expected to provide four samples of cortisol throughout the task at baseline (T1), post-task (T2), 20 minutes later (T3) and 40 minutes later

(T4). Across the sample, the actual average time between tasks was 18 minutes (T1 to T2), 27 minutes (T2 to T3), and 18 minutes (T3 to T4). In addition, participants were instructed not to eat, drink, smoke, or exercise during the two hours prior to the first saliva sample, based on evidence that doing so may influence their cortisol reactivity to the task (Hansen, Garde, & Persson, 2008). Six individuals violated this protocol. Four people drank in the previous two hours, but their total cortisol output ($p = .73$) and cortisol reactivity ($p = .98$) were not significantly different from non-violators. Two individuals ate a meal in the previous two hours, but they were also not different from non-violators in terms of total cortisol output during the task ($p = .32$) and cortisol reactivity to the task ($p = .58$).

Measures

Primary variables.

Negative life events. Negative life events were assessed by child self-report on the Negative Life Events Scale (NLES), a measure derived from two questionnaires – the Divorce Events Schedule for Children (DESC; Sandler, Wolchik, Braver, & Fogas, 1986) and the General Life Events Schedule for Children (GLESC; Sandler, Ramirez, & Reynolds, 1986). The DESC includes 16 divorce-related items that had been classified by children (ages 8 - 15 years) as being undesirable and confirmed by a panel of expert adult judges to be undesirable (Sandler et al., 1986). The GLESC includes 22 items that have been rated by two teams of expert judges as being negative. Five of the items overlap on both scales; thus, in total, the NLES includes 33 items. For all items, youth

reported on whether each event had occurred in the past month. This measure was administered at five of the six waves (W1 - W5), but only the measures at W1-W4 are included in the current study. A childhood composite that represents the average number of negative life events occurring during 9-month period (i.e. count of past month negative life events conducted every 3 months, from W1 – W4) was created by computing the mean of the total events reported at each of the four waves.

Active and avoidant coping. Voluntary coping efforts were assessed at five of the six waves (W1 - W5) using a modified version of the Child Coping Strategies Checklist (CCSC-R1), a measure of dispositional coping. Youth were asked to report how often (*never, sometimes, often, or most of the time*) they used a list of coping strategies during the previous month. The original version contained four dimensions of coping (active, avoidance, distraction, and support-seeking; Ayers et al., 1996). However, it has been more commonly used as a measure of two broad dimensions of coping (e.g., Sandler et al., 2000; Sandler et al., 1994; Suter, 2000; Velez, Wolchik, Tein, & Sandler, 2011), with six 4-item subscales reflecting active coping (cognitive decision making, direct problem solving, seeking understanding, positive focus, optimism and control) and three 4-item subscales reflecting avoidant coping (suppression, wishful thinking, and avoidant actions). Using 8 of the 9 scales (the control subscale was excluded) in a study assessing coping in children of divorce (ages 9-12), Sandler and colleagues (2000) conducted a confirmatory factor analysis (CFA) and found the two-dimensional model to be an adequate fit (Sandler et al., 2000).

In order to get the most comprehensive assessment of the active coping factor as possible and to ensure that the two-dimensional model would be accurate at a later developmental stage than was examined in prior studies using this measure (15 – 18 years old), scores on all nine coping subscales at the 6-year follow-up for those in the current sample were subjected to a CFA using MPlus software (Version 5.2, Muthén & Muthén, 2008). Results of the CFA indicated that optimism loaded on both active and avoidant factors. Given that this subscale is not theorized to load on the avoidant coping factor, a revised model, in which this subscale was removed and the errors associated with the seeking understanding subscale (an active coping indicator) and the wishful thinking subscale (an avoidant coping indicator) were allowed to correlate, was subjected to another CFA. Overall, this model demonstrated an adequate fit to the data: $\chi^2 = 36.83$, $df = 18$, $p < .01$; CFI = .95; RMSEA = .08; SRMR = .06.

Given the complexity and number of models to be tested in the primary analyses, the five active coping subscales were standardized and averaged to form an active coping composite, and the remaining three subscales were standardized and averaged to form an avoidant coping composite. For data reduction purposes, this was done at each wave of data (W1 - W5). Coping efforts measured at W5, when youth were in late adolescence, is of primary interest in this study. In addition, for the purpose of controlling for the effects of past active and avoidant coping efforts (measured during childhood, at the same time as childhood negative life events, at W1 - W4), two composites were created by averaging the active coping and avoidant coping subscales (following the procedure above)

across the first four waves of data. Maladaptive coping is conceptualized in the current study as a reliance on avoidant coping and low use of active coping. Although a ratio of avoidant to active could provide a measure of the extent to which individuals rely (or do not rely) on avoidant coping as opposed to active coping, this ratio would not address the possibility that negative life events leads to decreased active coping regardless of avoidant coping levels. Therefore, avoidant and active coping will be analyzed separately, but in the same model.

Child Temperament. At baseline, mothers completed two measures that assess three different aspects of temperament. Negative emotionality was assessed using the emotionality subscale of the Emotionality, Activity, and Sociability scales (Buss & Plomin, 1975), and attentional focus and impulsivity were assessed using two dimensions from the Children's Behavior Questionnaire (Goldsmith & Rothbart, 1991). Using a combination of conceptual (expert ratings) and empirical (confirmatory factor analyses), Lengua et al. (1998) created a reduced version of the instrument, such that only items that were identified as being more related to temperament than they were to depression or conduct problems. These uncontaminated temperament subscales were created by removing 4 items from the negative emotionality subscale, 6 items from the impulsivity subscale, and 1 item from the attentional focusing subscale. It is important to note that despite these decontamination methods, these subscales continued to be related to mother report of depressive symptoms and conduct problems, suggesting that temperament is still very much related to psychopathology (Lengua et al., 1998). The uncontaminated scales, however,

were determined to have less conceptual overlap with symptomatology (Lengua et al., 1998), and therefore Lengua and colleagues' version was used in the current study.

Studies using data from the NBP trial and follow-ups have used these three subscales to assess child temperament, with the negative emotionality subscale reflecting one dimension of temperament and the impulsivity and attentional focusing subscales combined to reflect temperamental regulation (e.g., Lengua et al., 1999; Lengua et al., 2000). This two dimensional model of temperament was subjected to a CFA using MPlus software (Version 5.2, Muthén & Muthén, 2008), with negative emotionality and impulsivity reverse coded. The model fit the data poorly: $\chi^2 = 460.195$, $df = 208$, $p < .001$; CFI = .74; RMSEA = .09; SRMR = .08. Three items were found to have very low loadings on their respective factor. When these items were removed and the CFA was re-run, the fit improved only slightly: $\chi^2 = 308.367$, $df = 151$, $p < .001$; CFI = .82; RMSEA = .08; SRMR = .07. Moreover, the two factors correlated almost perfectly with each other ($r = .96$, $p < .001$), and modification indices suggested that several items in each factor be allowed to correlate with items representing the other factor. As such, another CFA was conducted with all items proposed to load on one factor (minus the three items above that were removed). The model fit the data well: $\chi^2 = 181.331$, $df = 142$, $p = .01$; CFI = .95; RMSEA = .04; SRMR = .05. Thus, a single composite representing temperament was created by averaging the remaining items, with higher scores reflecting an "easy" temperament (low negative emotionality, low impulsivity, high attentional-focusing) and lower

scores reflecting a more “difficult” temperament. Given the categorical connotation of “easy” and “difficult” temperament (Chess & Thomas, 1985), the continuous measure of temperament in the current study will be referred to as *self-regulation* throughout the remaining section of the Methods chapter and the entirety of the Results chapter. Higher scores can be interpreted as reflecting better self-regulation. See the Appendix for the items included in the uncontaminated measure in Lengua et al. (1998). Note that the three items excluded due to the results of the CFA conducted in the current study are marked with an X.

Cortisol activity. As described above, participants provided four salivary cortisol samples at the beginning of and following the TSST. Analyses evaluated total cortisol output across the role-play task as well as the magnitude of cortisol reactivity (change from T1 to T3), both of which are theoretically meaningful aspects of cortisol stress responses (Nicolson, 2008). Assessment of total cortisol output allows one to examine individual variability in overall level of cortisol during the task, which offers different information from cortisol change across the task. For example, individuals may start high and remain high throughout the four time points, whereas others may exhibit lower levels overall. These differences would not be detected if one only looked at the change in cortisol from baseline to peak task levels. In the current study, total cortisol output during the task was assessed by computing area under the curve with respect to ground (AUCG) with the trapezoidal formula (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003), a commonly employed parameter for summarizing total concentration of

cortisol across a given time period (Fekedulegn et al., 2007). The formula for AUCG is as follows, with t_i denoting the individual measurement of cortisol at that time point and m_{i-j} denoting number of minutes between t_i and t_j :

$$\text{AUCG} = [(t_2+t_1) / 2] * m_{1-2} + [(t_3+t_2) / 2] * m_{2-3} + [(t_4+t_3) / 2] * m_{3-4}.$$

AUCG was computed using raw cortisol values and then log-transformed to correct for deviations from normality. Assessment of cortisol reactivity, which allows one to investigate potential predictors of the change in cortisol concentration as the task proceeds, was also examined. For the evaluation of cortisol reactivity to the task, raw cortisol values were log-transformed, and a residualized change score was computed by regressing T3 cortisol (chosen because this was the average peak time for the sample) on the T1 cortisol; the standardized residuals were then used as the measure of cortisol of reactivity to the task. The residualized change score is used as an alternative to calculating difference scores because it adjusts for the baseline level but avoids some of the reliability concerns with difference scores (MacKinnon, 2008).

Potential covariates.

Mental health problems. Youth mental health problems have been shown to predict the occurrence of negative life events, particular coping behaviors, and patterns of cortisol activity (both total cortisol output and cortisol reactivity). Given the potential for childhood mental health to impact the relation between negative life events and later coping, baseline mental health problems (W1) was examined as a potential covariate. Importantly, however, the assessment of baseline mental health took place at the same time as assessment of negative life

events and self-regulation, thereby precluding determination of confounding of relations between negative events and coping or self-regulation and coping. Given that baseline mental health problems may have explained variance in negative life events at subsequent waves (e.g., W2 - 4) and coping efforts that occurred in adolescence, it was considered as a covariate. In addition, current mental health problems will also be considered as a covariate, based on mounting evidence that particular patterns of cortisol activity are related to various psychiatric disorders (e.g., Alink et al., 2008; Lopez-Duran et al., 2009). At W1, internalizing symptoms were measured by mother report on the internalizing subscales of the Children's Behavior Checklist (CBCL; Achenbach, 1991) and child-report on two self-report scales designed to assess depressive symptoms and anxiety in youth: the 27-item, multiple choice Children's Depression Inventory scale (CDI; Kovacs, 1981, 1985), which is a modification of the adult Beck Depression Inventory (BDI; Beck & Steer, 1993), and the Revised Children's Manifest Anxiety Scale (RCMAS; also known as the "What I Think and Feel Scale"; Reynolds & Richmond, 1978), which includes 28-items that assess a child's chronic state of anxiety. A single composite of internalizing symptoms at W1 was computed by averaging the standardized total scores on the three measures. Externalizing symptoms were measured at W1 by mother report on the externalizing subscales of the CBCL and by youth-report on the aggression and delinquency subscales of the Youth Self Report scale (Achenbach, 1991). Response options included *not true*, *sometimes true*, and *often true*. Items were summed and then an average of the standardized scores (mother-report and child-report) was computed. At W6,

internalizing and externalizing symptoms were measured by self-report only (given that the youth were now between ages 24 - 28) using the Adult Self Report Scale (Achenbach & Rescorla, 2003), with higher scores reflecting the young adult's report of externalizing and/or internalizing symptoms over the previous six months.

Variables known to influence cortisol activity. A number of between- and within-person health factors have been found to influence cortisol reactivity to social stressor tasks in different ways, including the use of alcohol, chronic smoking, daily caffeine use, body mass index, and certain medications (Hansen et al., 2008; Nicolson, 2008; Zimmerman et al., 2004). In the current study, young adults reported on their alcohol use ("On average, how many servings of alcohol do you consume in a week?"), nicotine intake ("How many cigarettes and cigars do you smoke in an average day?"), caffeine intake ("How many servings of caffeinated beverages do you drink in an average day?"), and medication use over the last 24 hours. Body mass index was calculated by dividing participant weight by the square of his or her height. In addition, to address the diurnal rhythm of cortisol, *time of day* was calculated by taking the number of minutes between midnight and the time at which the baseline cortisol sample was taken. Due to the variability in the time between each cortisol sample across individuals, the number of minutes between sample 1 and sample 4 was also considered as a covariate (hereafter this variable will be referred to as *task time*).

Data Analytic Plan

Plan for preliminary analyses. As noted earlier, the data in the current

study are drawn from a larger randomized controlled trial of a preventive intervention targeting children of divorce. The intervention was not found to have direct effects on negative life events, active or avoidant coping in adolescence, or total cortisol output. However, there was an indirect effect of the intervention on active coping in adolescence (measured similarly to the construct in the present study, but not identically), such that program-induced improvement in the mother-child relationship predicted greater use of active coping strategies (without a direct effect of program on coping present; Velez et al., 2011). In addition, there was a group by age effect on cortisol reactivity to the task (Luecken et al., 2012). For this reason, two approaches were taken. First, a Box's M test was conducted to ascertain whether the covariance structures of the variables of interest in the current study were significantly different in the two groups (intervention and control): if not significantly different, then combining the groups would be appropriate (Tabachnick & Fidell, 2006). Second, each model in the primary analyses was subsequently tested with group and/or the group x age interaction included; if results did not change, then model fit and parameter estimates were not reported for analyses including these as covariates.

In addition, the descriptive information for all variables of interest was examined, including childhood negative life events, self-regulation, adolescent avoidant and active coping, total cortisol output (AUCG), and cortisol reactivity (residualized change score). Inter-correlations between all variables were then inspected. Potential covariates were identified based on previous research and examining correlations between the covariate, the predictors and the outcome

measures (e.g., cortisol, active and avoidant coping in adolescence). A variable was included as a covariate in the analysis if 1) it appeared to be a potential confounder (i.e. it predicted both the independent and dependent variables), or 2) it related significantly ($p < .05$) to total cortisol output and/or cortisol reactivity. If two covariates were highly correlated with one another in addition to the variables of interest, the one most highly correlated with the outcome was chosen for inclusion as recommended by Tabachnick & Fidell (2006). As noted above, the following variables were examined and considered for inclusion as covariates in the main analyses: past and current internalizing and externalizing symptoms, nicotine intake, caffeine intake, body mass index (BMI), medication use in the 24 hours prior to task, alcohol use, and task time. Gender and age were also considered as potential covariates given that these characteristics have been shown in some studies to play a role in exposure to negative events, choice of coping strategies, and cortisol activity. In addition, all variables were screened for outliers and subjected to tests of the assumption of normality. If necessary, variables were transformed to correct for deviations from normality according to standard procedures. Cases that were statistical outliers (i.e. they were $>$ than at least 3 standard deviations on the variable of interest) and unduly and meaningfully (i.e. someone who scores extremely high on the predictor may mask relations between the predictor and outcome) impacted analyses were removed. Finally, patterns of missingness among the variables were examined and potential auxiliary variables were identified (variables that are related to missingness and if included in analyses can increase precision of modeling missing data).

Plan for primary analyses. For each hypothesis, two separate models were estimated and tested: one with total cortisol output (AUCG) as the outcome and the other with cortisol reactivity (residualized change score) as the outcome. All analyses were conducted using MPlus software (Version 5.2, Muthén & Muthén, 2008), which addresses potential bias resulting from missing data by using all available data, with bootstrap resampling (appropriate for non-normal data and the recommended procedure for testing indirect mediation effects; MacKinnon, 2008). All predictors were mean centered. For H1, a simple multiple regression model was estimated and tested, with the cortisol outcome regressed on average number of childhood negative events and relevant covariates. For H2, a multiple mediation model was estimated and tested, which included the predictor (negative life events), two putative mediators (adolescent active and avoidant coping), the cortisol outcome variable, and relevant covariates. Based on the indirect effects of the group on active coping (see Velez et al., 2011) and the interactive effect of group* age on reactivity, group was initially included in the multiple mediation model (specifically, in the prediction of adolescent active coping from negative life events) and group, age, and group*age were included as covariates in models predicting reactivity. If their effects were non-significant, these variables were removed and the results without group and/or group, age, and group*age is reported. For H3, a moderated mediation model was tested by adding an interaction term that was created using the centered first order variables negative life events and self-regulation in the prediction of active and avoidant coping.

CHAPTER 8

Results

Preliminary Analyses

Test of homogeneity of within-class covariance matrices. To examine whether it would be appropriate to combine individuals in the intervention (n=117) and control groups (n=46) for the main analyses, a Box's M test was conducted to test the assumption of homogeneity of within-class covariance matrices in the two groups. This assumption was important to examine because relations among the variables of interest could have been affected by intervention participation (in which case, it would be inappropriate to combine the two groups for the current analysis). Rejection of this assumption requires a significance level below .01 (given the sensitivity of the test; Tabachnick & Fidell, 2006). Results from the Box's M test revealed that this assumption of homogeneity could not be rejected in the case of either total cortisol ($\chi^2 = 10.847, df = 15, p = .76$) or cortisol reactivity ($\chi^2 = 11.84, df = 15, p = .69$), indicating that it was appropriate to combine the intervention and control groups.

Descriptive statistics, tests of normality, and outlier screening. Means, standard deviations, ranges, skewness and kurtosis of the study variables and potential covariates are included in Tables 1 and 2, and frequencies of categorical variables are included in Table 3. The average level of salivary cortisol across the four time points was: T1 = .093 ug/dl ($SD = .061$; Range .023-.441), T2 = .089 ug/dl ($SD = .062$, Range = .012-.40), T3 = .094 ug/dl ($SD = .075$; Range = .012-.395), and T4 = .077 ug/dl ($SD = .053$; Range = .013-.310). As is standard in the

field, cortisol was log-transformed to correct for deviations from normality (all samples were positively skewed and kurtotic). Inspection of QQ plots revealed that childhood negative events also deviated from a normal distribution; however, exposure to negative events is not expected to be normal in the population. Therefore, following the suggestion of Tabachnick and Fidell (2006), an estimation method that addresses non-normality was used rather than transforming this variable.

All variables, including the primary study variables and potential covariates, were screened for univariate outliers using SAS UNIVARIATE and SPSS EXPLORE, and multivariate outliers were identified by inspecting regression diagnostics available through the SAS REGRESSION procedure (e.g., DFFITS, DFBetas, Cook's). Several cases were flagged as univariate outliers and appeared to bias results (for a discussion of the problems associated with outliers, see Cohen, Cohen, West, & Aiken, 2003). Two individuals were identified as consuming more than 4 times the at-risk cut-off for average weekly alcohol servings (a male who consumed an average of 60 drinks per week and a female who consumed an average of 30 drinks per week). One individual reported a significantly greater average of negative life events than other participants (>4 standard deviations above the mean), and one individual was found to be morbidly obese (BMI=49; >3 standard deviations above the mean), a condition related to particular levels of cortisol. Their removal did not significantly impact intercorrelations among the majority of study variables; however, when the primary analyses were run with and without the outliers, parameters associated

with several variables in many of the models changed substantially and models demonstrated a better fit (according to fit indices) without the outliers included. Thus, a conservative approach was taken such that all analyses were run without these outliers, bringing the sample size to 159. In addition to the four univariate outliers, multivariate diagnostic analyses revealed a high number of potential multivariate outliers (>20), indicating the need to use robust statistical procedures that can handle multivariate abnormalities (e.g., bootstrap methods).

Intercorrelations and identification of covariates. Intercorrelations between the main study variables were examined. As shown in Table 4, there was a trend for self-regulation to be negatively correlated with total cortisol output. Higher self-regulation was related to fewer negative life events in childhood. In addition, self-regulation was significantly positively correlated with adolescent active coping, as expected, but was not related to avoidant coping. Average childhood negative life events were positively correlated adolescent avoidant coping, which was in turn negatively correlated with cortisol reactivity in young adulthood. Finally, active and avoidant coping during adolescence in the present sample were not correlated at all ($p = .88$), despite a significant correlation between the two coping styles in childhood ($r = .47, p < .001$; not shown in table).

Correlations between potential covariates and the main study variables were also examined. As shown in Table 5, females exhibited significantly lower cortisol reactivity at W6 and higher self-regulation at W1 compared to males. Participant ethnicity was not related to any of the study variables. Consistent with what has been found in other populations of youth (e.g., Compas et al., 1988),

there was moderate stability in the use of active and avoidant coping across childhood and adolescence. Surprisingly, childhood active and avoidant coping were positively and significantly related to cortisol reactivity 15 years later. Given that these relations were unexpected and not hypothesized, one can only speculate as to whether these correlations represent true relations in the population or artifacts of a third variable.

Although mental health problems at W1 did not correlate with total cortisol output or cortisol reactivity, a greater number of externalizing problems at W1 were related to significantly lower self-regulation, greater childhood negative life events, and lower adolescent active coping. Based on the qualitative, temporal, and statistical overlap between W1 externalizing problems and self-regulation, it was anticipated that inclusion of W1 externalizing as a covariate in models that also include self-regulation in the prediction of active coping might result in poor fitting models and/or mask effects of self-regulation. Examination of the partial correlations between self-regulation and active coping, controlling for W1 externalizing confirmed the problematic overlap (the two constructs “canceled each other out” – that is, neither was significant in predicting adolescent active coping when both taken into consideration; not shown). Therefore, W1 externalizing was initially included as a covariate in analyses that tested mediation by adolescent active coping, but was dropped if it was non-significant. W1 internalizing symptoms was positively correlated with negative life events and negatively correlated with both self-regulation and adolescent active coping. Given that W1 internalizing was highly correlated with W1

externalizing ($r = .52, p < .0001$; not shown in table), W1 internalizing was not included as an additional covariate per recommendations of Tabachnick & Fidell (2006).

As shown in Table 6, average number of alcohol servings consumed in a week was positively correlated with total cortisol output and time of day was marginally related to lower total cortisol output. Although the positive correlation between task time and total cortisol output did not quite reach significance, preliminary analyses showed that it was significantly related to total cortisol when alcohol use and time of day were also controlled for (not shown). Thus, alcohol use, time of day, and task time were included as covariates in all models that predicted total cortisol output (AUCG).

As shown in Table 6, there were significant positive correlations between avoidant coping, time of day at which the baseline cortisol sample was taken, and duration of the task. This was unexpected, but not completely surprising given that participants were responsible for choosing the time of day; those with an avoidant coping style could have been more likely to delay the appointment until later in the day and to take a longer time completing questionnaires in between saliva samples. Finally, in addition to the correlation with gender, cortisol reactivity was positively correlated with average weekly alcohol use and negatively correlated with W6 externalizing problems (which is consistent with the literature, see Alink et al., 2008). Thus, all analyses that predicted cortisol reactivity included participant gender, alcohol use, and W6 externalizing symptoms as covariates.

Missing data analysis. SPSS Missing Value Analysis was used to identify potential correlates of missingness. The analysis included theoretically plausible variables (age, gender, ethnicity, treatment group, current medical conditions, and risky behaviors such as smoking, alcohol use, and caffeine) and other variables of interest in the current study (negative life events, childhood active and avoidant coping, total cortisol, self-regulation, and adolescent active and avoidant coping) that might be related to missingness. All of these variables were evaluated for possible associations with measures that were missing greater than 5% of the cases. Complete data was available for average number of childhood negative life events and childhood active and avoidant coping. There were seven cases missing data points on self-regulation, but missingness was not related to any of the other variables examined. Fourteen cases were missing data points for W5 avoidant and active coping, and missingness on these composites was related to average number of childhood negative life events and participant age: Cases missing W5 active and avoidant coping were one year older on average ($t = 2.2, df = 16.1, p = .04$) and reported fewer negative life events during childhood ($t = -2.2, df = 15.6, p = .04$).

Six participants were missing at least one of the four cortisol samples (T1 – T4): one case had no T1 sample, three cases were missing T2, one case was missing T4, and one case was missing both T3 and T4. Missingness of cortisol was not related to any other variable examined. Multiple imputation was considered and then abandoned when it resulted in biologically implausible estimates for the missing values. Other methods were also considered (e.g.,

imputing a mean or calculating AUCG using the samples available) but it was decided that doing so was unlikely to produce a valid approximation of what the cortisol concentration of a particular sample would have been. Therefore, AUCG (total cortisol output) was not calculated for these participants and cortisol reactivity was not computed for the two participants missing T1 or T3.

Little's MCAR test of whether data are missing completely at random was computed across all variables included in the Missing Value Analysis. The result suggested that data were missing completely at random: $\chi^2 = 105.56$, $df = 115$, $p = .73$; that is, the probability of missing data on the outcome variables (cortisol activity) is unrelated to the other measured variables and is unrelated to itself. As noted earlier, MPlus software (Version 5.2, Muthén & Muthén, 2008) was used to estimate all models with the bootstrap resampling approach (which addresses the non-normal distribution in the data); thus, data from all participants, minus the four substantive outliers, was used and the effective sample size for all primary analyses was 159.

Primary Analyses

Hypothesis 1 (H1). First, *total cortisol output* (AUCG) was regressed on average number of childhood negative life events, time of day, task time, and alcohol use at W6. As shown in Table 7, the effect of negative life events on total cortisol was non-significant ($p = .19$). Next, *cortisol reactivity* (the residualized change score) was regressed on time of day, participant gender, alcohol use, externalizing symptoms at W6, and childhood negative life events. There was no relation between childhood negative life events and cortisol reactivity 15 years

later ($p = .30$; see Table 7). The analysis was re-run including group, age, and group*age as covariates (due to the impact of the intervention on cortisol reactivity in older participants; Luecken et al., 2012). Results did not change.

Hypothesis 2 (H2). A multiple mediation model was tested using *total cortisol output* as the dependent variable, negative life events and self-regulation as the predictor variables, and adolescent active and avoidant coping as the mediating variables. In addition to controlling for time of day, task time, and alcohol use at W6, the effects of childhood active and avoidant coping (W1 - W4) and W1 externalizing were included in the model, specifically in the prediction of the mediators. The model fit the data well: $\chi^2 = 19.80$, $df = 21$, $p = .53$; RMSEA = .00, 95% CI [0, .06]; CFI = 1.00; SRMR = .04. As shown in Table 8, there was a significant positive relation between negative life events and adolescent active coping ($p = .02$) and a significant positive effect of negative events on adolescent avoidant coping ($p = .001$); however, negative life events ($p = .18$), adolescent active coping ($p = .17$), and adolescent avoidant coping ($p = .83$) did not predict total cortisol output. When group assignment (intervention vs. control) was included as an additional covariate in the prediction of active coping from negative life events, results did not change and the parameter associated with group assignment was not significant ($p = .89$). Given this, group assignment was not included in the prediction of coping in subsequent models.

Next a multiple mediation model was tested using *cortisol reactivity* as the dependent variable, and negative life events as the predictor variable, adolescent active and avoidant coping as the mediating variables, and the relevant covariates.

With the exception of the CFI, which was .88, the model fit the data well: $\chi^2 = 30.55$, $df = 21$, $p = .08$, RMSEA = .05, SRMR = .05. As shown in Table 8 and consistent with the previous model, negative life events predicted higher levels of active and avoidant coping in adolescence. Active coping, in turn, was related to marginally lower levels of cortisol reactivity and avoidant coping was marginally related to greater reactivity; however, these relations did not reach significance ($p = .10$ and $.12$, respectively). Negative life events did not predict cortisol reactivity ($p = .48$). The model was re-run with group, age, and group*age included as additional covariates. Results did not change.

Hypothesis 3 (H3). A moderated multiple mediation model was tested with *total cortisol output* as the dependent variable, negative life events, self-regulation, and negative events*self-regulation (the interaction term) as the predictor variables, and adolescent active and avoidant coping as the mediating variables. In addition to controlling for the covariates above, the effects of childhood active and avoidant coping and W1 externalizing were included in the model. As anticipated (due to the conceptual and statistical overlap between W1 externalizing and self-regulation), the model fit was inadequate. W1 externalizing was not significantly related to active coping and was dropped; the final model fit remained inadequate: $\chi^2 = 103.89$, $df = 36$, $p < .001$, RMSEA = .11, CFI = .47, SRMR = .09. In addition, the interaction term was not significant for any of its pathways. Next, a moderated multiple mediation model was tested with *cortisol reactivity* as the dependent variable, negative life events, self-regulation, and negative events*self-regulation (the interaction term) as the predictor variables,

adolescent active and avoidant coping as the mediating variables, and the relevant covariates (alcohol use, gender, time of day, W6 externalizing). Consistent with the previous model, the fit was inadequate: $\chi^2 = 56.23$, $df = 30$, $p = .002$, RMSEA = .07, CFI = .69, SRMR = .06. The fit improved only slightly when group, age, and age*group controlled for, and the interaction term in all pathways was non-significant. Owing to the poor fitting models, details of parameter estimates, standard errors, and p values are not reported for these moderated mediation models.

Post hoc exploratory analyses. In the H1 analysis, negative life events in childhood exhibited a non-significant negative relation with total cortisol output. Correlations and subsequent analyses showed that self-regulation was also negatively related to cortisol output and negatively related to negative life events. One might ask whether a stronger association between negative events and cortisol was being suppressed by the unaccounted for relations between level of self-regulatory ability, exposure to negative events, and total cortisol output. To examine this possibility, total cortisol output was regressed on time of day, task time, alcohol use, self-regulation, and negative life events. Holding self-regulation constant, there was a stronger negative relationship between negative events and total cortisol compared to the regression that did not control for self-regulation, but it still did not quite reach significance, $B = -.04$, $SE = .02$, $t = -1.64$, $p = .10$. Interestingly, self-regulation exhibited a significant inverse relation with total cortisol, such that higher self-regulatory abilities in childhood predicted lower total cortisol output 15 years later, $B = -.16$, $SE = .07$, $t = -2.45$, $p = .01$.

Relatedly, based on the findings that negative life events marginally and self-regulation significantly predicts total cortisol, one might ask whether a better fitting mediation model (as was done for H2) could be estimated, such that the effect of self-regulation was mediated by active coping, whereas the potential relation between negative life events and cortisol was mediated by avoidant and/or active coping. This model fit the data well: $\chi^2 = 22.79$, $df = 21$, $p = .36$; RMSEA = .02, CFI = .97, SRMR, = .05. As shown in Figure 2, there was a significant positive relation between self-regulation and active coping ($p = .01$) and a significant positive relation between negative life events and avoidant coping ($p = .001$). Higher self-regulation predicted lower total cortisol output ($p = .01$), with an absolute value effect size of .19 (based on the standardized path coefficient for c' ; see Figure 2), controlling for negative life events, the coping variables, and the covariates. According to Cohen (1992), this effect size represents a small effect. Similarly, negative life events exhibited a negative association with total cortisol, with a small effect size of .15 (based on the standardized path coefficient of c' ; see Figure 2), but this relation did not reach significance ($p = .07$). Finally, active coping was positively related to total cortisol output but this relation also did not reach significance ($p = .07$). Interestingly, there was evidence of a small indirect effect of self-regulation on total cortisol output via adolescent active coping at the $p = .10$ level, $B = .03$, 90% CI [.001, .085].

CHAPTER 9

Discussion

Several researchers have noted the importance of using longitudinal, prospective research designs to examine the relation between early negative life events and later physiological functioning (e.g. Adam, Klimes-Dougan, & Gunnar, 2007), and many have called for more research that incorporates both coping and temperament in studies of the neurobiology of stress (see Skinner & Zimmer-Gembeck, 2009, for an extensive discussion of this issue). In an effort to meet these calls for additional research, the current study utilized a prospective, longitudinal design to examine relations between exposure to negative life events in childhood, child temperament, adolescent coping style, and physiological activity in young adulthood among a population of individuals whose parents divorced during their childhood. It was hypothesized that negative life events would predict lower cortisol activity (indexed by total cortisol output and cortisol reactivity in the context of a psychosocial stress task); this relation would be mediated by maladaptive coping in adolescence (lower active coping and higher avoidant coping), such that negative events led to higher adolescent maladaptive coping, which in turn predicted lower cortisol activity in young adulthood; and this cascade would be strongest for those with a more “difficult” childhood temperament (high impulsivity, low attentional focus, and high negative emotionality).

Contrary to hypotheses, a moderated mediation model in which temperament interacted with childhood negative events to predict adolescent

coping behavior and subsequent cortisol activity was a poor fit to the data. Rather, the best fitting model was one that estimated direct and indirect relations between negative life events, self-regulation, coping behaviors, and cortisol activity (Figure 2). This model demonstrated a trend for negative life events in childhood to predict lower total cortisol output 15 years later and a significant relation between self-regulation and total cortisol output 15 years later such that higher self-regulation (lower impulsivity, lower negative emotionality, and high attentional focus; an “easier” child temperament) predicted lower total cortisol output in young adulthood (and, conversely, that a more difficult child temperament was related to higher total cortisol output).

In addition, childhood negative life events predicted significantly greater use of avoidant coping in adolescence, whereas higher levels of self-regulation predicted increased active coping in adolescence, both controlling for earlier levels of active and avoidant coping in childhood. Finally, there was a trend for partial mediation of the effect of self-regulation on total cortisol by adolescent active coping, such that self-regulation increased active coping in adolescence, which in turn marginally predicted higher cortisol output in young adulthood (indicating statistically inconsistent mediation; see MacKinnon, Fairchild, & Fritz, 2007). The conceptual significance of the current findings, including the lack of evidence for hypothesized relations, methodological issues that arose, strengths and limitations of the current study, clinical implications, and issues in need of future research are discussed below.

Direct Relations Between Negative Events and Cortisol Activity

Total cortisol output. Multiple regression analyses did not find a significant direct effect of childhood negative life events on total cortisol output, thus Hypothesis 1 was technically not supported. However, the estimate of the relation was in the expected direction (i.e. negative), and when relations between temperament, active coping, and cortisol output were also estimated in the model, the magnitude of this inverse relation between negative life events and total cortisol output increased and approached significance (Figure 2). The increase in magnitude of the relation between negative events and total cortisol output due to the inclusion of self-regulation in the model indicates the presence of suppression (Cohen, Cohen, West, & Aiken, 2003). That is, the relationship between negative events and cortisol appears to have been suppressed due the significant correlation between negative events and self-regulation. One might speculate that if there were truly no relation between negative events and total cortisol output, the magnitude of the association would decrease rather than increase when a factor (in this case, temperament) that explains significant variability in total cortisol output is controlled for. Indeed, as Cohen and colleagues (2003) noted, the zero-order effect between the independent and dependent variables (i.e. the estimate of the relation between negative events and cortisol without controlling for self-regulation) is “misleading” in the case of suppression, whereas the coefficients in the regression that includes the suppressor “may be considered to reflect appropriately the causal effects” (p. 78).

A relation between negative events and lower total cortisol output is consistent with previous research that found significant negative associations between childhood adverse events and basal cortisol assessed in the morning (Bevans, Cerbone, & Overstreet, 2008; Gerritsen et al., 2010; Suglia et al., 2010; Trickett et al., 2010; van der Vegt et al., 2009) and between early adversity and dampened cortisol responses to stress among healthy adults (Carpenter, Shattuck, Tyrka, Geraciotti, & Price, 2011; Carpenter et al., 2009). Importantly, previous research found significant relations between early adversity and various measures of cortisol activity despite sample sizes that were smaller than or similar to the current study (e.g., $n = 68$, Bevans et al., 2008; $n = 110$, Carpenter et al., 2011; $n = 132$, Suglia et al., 2010; $n = 173$, Trickett et al., 2010), suggesting that the effect sizes were larger in these studies than in the current investigation (effect sizes for almost all of these studies were not available). Unfortunately, it is difficult to compare effect sizes across studies given the different measures of cortisol (e.g., serum cortisol vs. salivary cortisol), the timing of the cortisol assessment (e.g., morning vs. late afternoon/evening), and the different types of adverse events studied (i.e., cumulative emotional abuse is not necessarily a similar index to stress as number of negative events experienced after parental divorce). However, in a study that found early childhood maltreatment was associated with a dampened salivary cortisol response to a psychosocial stress task in adulthood, a “large” unstandardized effect size was reported (Carpenter et al., 2011, p. 371), whereas the current study found a small marginal effect.

Several speculations can be made about the more marginal relation found in the current study compared to others. One possibility is that a stronger association would have been found if, instead of measuring cortisol in the afternoon/evening, cortisol activity was measured in the morning as was done with a number of studies investigating childhood adversity and adult outcomes (e.g., Trickett et al., 2010). Effects may be more or less extreme in the morning when cortisol is at its peak compared to evening when cortisol is decreasing toward its lowest levels. Alternatively, it may be that a relation between childhood *post-divorce negative events* and cortisol activity 15 years later truly is small, regardless of time of sampling. The two longitudinal studies that found significant evidence of a prospective inverse relation between early adversity and later cortisol activity focused on stressors that may be objectively assessed as being more severe and/or traumatic (compared to post-divorce stressful life changes), including sexual maltreatment (e.g., Trickett et al., 2010) and extreme early neglect and institutionalization (e.g., van der Vegt et al., 2009). Individuals in the current study reported on a range of negative events, many of which may be considered more common than physical abuse and neglect. Yet another possibility is the existence of a “critical window” in which exposure to negative events following divorce is most strongly related to later physiological activity. In the current study, childhood negative life events were averaged over a period of nine months and doing so may have masked an effect of negative events that occurred most closely to the divorce (e.g., W1 only or prior to W1). Moreover, an assessment of exposure to negative events that occurred prior to or during the

divorce was not available, a time that may include greater and/or different types of stressful life changes than those experienced more than a year later.

Should the small, marginal association bear itself out in the larger population of children of divorce, what is the clinical meaning of this relation? There is no agreed upon range of cortisol that would indicate problematically attenuated or exaggerated cortisol activity in individuals who otherwise evidence cortisol levels that fall within normal physiological parameters (i.e. those do not have a physical health condition that causes abnormally low or high cortisol levels, such as Addison's Disease or Cushing's Syndrome). One might argue that the cortisol concentration range in the current study is not significantly different from that identified in other investigations of healthy young adults. For example, in a community sample of young adults (ages 20-30), it was shown that average salivary cortisol concentrations at 10pm ranged from .04 ug/dl in women to .08 ug/dl in men (Aardel & Holm, 1995). Ninety percent of the participants in the current study provided their first cortisol sample by 7pm (three hours earlier than the study above) and the average cortisol concentration was .09 ug/dl (with 75% of participants exhibiting a concentration between .02 ug/dl and .11 ug/dl). Given that cortisol typically decreases further into the night, it may be that by 10pm the participants' cortisol concentrations reached a level comparable to that reported by Aardal and Holm (1995) in their sample of healthy young adults. However, such a conclusion would be very misleading. The fact that the cortisol concentrations in the current study *may be* comparable to those reported in 46 Swedish young adults is far from conclusive, especially given that little is known

about those young adults (perhaps they all came from a population at higher risk of experiencing childhood negative life events).

Several researchers who have spent the last few decades studying the impact of adversity on the HPA axis have noted the complexities in interpreting cortisol levels as either adaptive or maladaptive (e.g., Cicchetti & Rogosch, 2007; Gunnar & Donzella, 2002). For example, among non-maltreated children, lower morning cortisol was related to higher resilience (e.g., ego control and ego resiliency), whereas higher morning cortisol was related to higher resilience in physically abused children. As such, it has been suggested that other indices of pathology (i.e. mental health disorders) and/or adaptive functioning be assessed to help understand whether particular physiological patterns in a sample population are indicative of risk versus resilience (e.g., Gunnar & Donzella, 2002). In the current sample, both concurrent internalizing and externalizing problems were related to lower cortisol reactivity; however, there were no relations between past or current mental health problems and total cortisol output. Indeed, as will be discussed, having better self-regulation as a child (low impulsivity, high attentional focus, and low negative emotionality), which is generally considered a protective factor, also predicted lower total cortisol output. This does not preclude the possibility that an inverse relation between negative events and cortisol is problematic (two different pathways could exist from negative life events and self-regulation to cortisol), however it underscores the need for further investigation into whether risk or resilient cortisol profiles can be identified in children of divorce.

Cortisol reactivity. It is interesting that no association was found between cortisol reactivity to the task and negative events. This is contrary to what has been found in other studies that have found a significant relation between greater adversity and diminished reactivity to stress (Armbruster et al., 2011; Carpenter et al., 2007, 2009; Elzinga et al., 2008). Although there are several differences between the studies cited above and the current examination (including older samples and the retrospective assessment of negative events), these factors alone are unlikely to account for the differences in findings. One very real possibility is the lack of attention to gender in the current study. Elzinga and colleagues (2008), for example, found that male subjects primarily drove the relation between adversity and reactivity. Although males exhibited higher cortisol reactivity in the current study, relations among independent variables and reactivity were not examined within each gender. Participant sex was controlled for, but this may not adequately address the influence of gender on relations between post-divorce negative events and cortisol reactivity to the task.

Alternatively, it may be that post-divorce events are not related to *reactivity* specifically. Various measures of cortisol have been differentially related to stressors (Hagan et al., 2011) and mental health outcomes (Alink et al., 2008), with some studies finding effects for one measure (e.g., basal or baseline cortisol) and not the other (e.g. reactivity) or vice versa. The non-relation between negative events and reactivity in the current study may reflect that post-divorce negative life events in childhood impact HPA axis on a more macro level than is evident in a finer grained analysis of stress reactivity, especially when the average

magnitude of reactivity is very small (e.g., Gunnar, Frenn, Wewerka, & Van Ryzin, 2009), as it was in the present study.

Adolescent coping as a mediator of negative events and cortisol

Path analyses within a structural equation modeling framework found no evidence of an indirect effect of negative life events on total cortisol output or cortisol reactivity by way of adolescent active or avoidant coping. Thus, hypothesis 2 was not supported. The lack of an indirect effect of negative events on cortisol reactivity via avoidant coping is surprising given the strong positive relation between negative events and adolescent avoidant coping and the significant, albeit modest, negative correlation between adolescent avoidant coping and cortisol reactivity ($r = -.17$). Based on the non-significant relation between avoidant coping and reactivity in the overall multiple mediator model (Table 8), current externalizing symptoms, current alcohol use, gender appear to be more influential in their effects on cortisol reactivity than adolescent avoidant coping in this sample of children of divorce. The relations between cortisol reactivity and gender, externalizing, and alcohol use is consistent with several studies of at-risk populations. For example, among those who experienced higher lifetime adverse events, it was found that males exhibited higher reactivity to this psychosocial stress task compared to females (Elzinga et al, 2008). In addition, associations between higher externalizing problems and lower cortisol activity have been found among adolescents who had experienced family disruption (Luecken et al., 2010; Hagan et al., 2010), and alcohol use has been found to

predict higher cortisol among youth at-risk of later substance use disorders (Zimmerman et al., 2004).

The main effect of negative life events in childhood on adolescent coping and the lack of correlation between active and avoidant coping in adolescence are worthy of note. In the current study, *childhood* active and avoidant coping were significantly positively correlated. Indeed, it has been noted that active and avoidant coping in childhood post-divorce can be expected to be correlated in this way given that increased stress often translates to increased coping in general, with one approach facilitating the other at younger ages (Sandler et al., 1994). Interestingly, however, the two coping styles appeared to diverge as children aged into adolescence, as they were not correlated at all in the current study. Although some have found moderate stability in coping style in childhood and adolescence (e.g., Compas et al., 1988) as well as from adolescence to emerging adulthood (e.g., Hussong & Chassin, 2004), the relative use of different styles appears to change across time (Hussong & Chassin, 2004; Seiffge-Krenke et al., 2009). Given the advancement of cognitive abilities and new developmental tasks that come with the stage of adolescence, youth are likely to become more discriminating in their use of different coping strategies (see Kavšek & Seiffge, 1996 for empirical evidence of this), which would result in a reduction in covariation between styles. Indeed, studies have found that avoidant coping may decrease with age (Amirkhan & Auyeung, 2007), whereas problem-focused coping may increase or remain stable (Hampel & Petermann, 2005).

Although exposure to negative life events uniquely predicted greater use of avoidant and active coping six years later, the relation between negative events and active coping became non-significant when self-regulation was included in the model. Greater exposure to negative events in childhood predicted greater use of avoidant coping in adolescence, however, above and beyond earlier levels of childhood coping and baseline levels of mental health problems. The positive relation between negative events and avoidant coping is consistent with other studies of children of divorce (e.g., Sandler et al., 1994; Sandler et al., 2000), but the current finding is notable for the 6-year time period between the assessments of the two constructs. Previous studies finding associations between stressful life events and avoidant coping have been primarily cross-sectional (Sandler et al., 1994; Sandler et al., 2000) or over a short time period (Cheng & Lam, 1997; Snow et al., 2003). This suggests that among children of divorce, negative event exposure in childhood increases greater long-term use of a coping strategy that is often ineffective. Although the following is just speculation (as this current study did not measure the contexts within which adolescents used particular coping strategies), greater use of avoidant coping in the context of romantic relationships may be particularly problematic for this population. Children of divorce are more likely to have poorer marital quality and have greater likelihood of experiencing divorce (Amato & Booth, 1991; Webster & Herzog, 1995), and maladaptive coping has been found to mediate relations between exposure to interparental conflict and lower quality romantic relationships later on (Rodriguez & Kitzmann, 2007).

Contrary to what was hypothesized, there was no evidence of an interaction between negative life events and temperament on either active or avoidant coping in adolescence. As such, hypothesis 3 was also not supported. A number of speculations can be made regarding this lack of an interactive effect on adolescent coping style and subsequent cortisol activity. First, there was about a 6-year time lag between measurement of self-regulation/negative life events and coping behaviors. Research has shown that although pre-adolescence (ages 8 – 12, which was the age of the current sample of participants at baseline) is a time of rapid development of coping abilities, stabilization of coping style isn't likely to occur until late adolescence (Skinner & Zimmer-Gembeck, 2007). It may be that temperamental traits interact with stressful life event exposure post-divorce to predict coping in the shorter term (i.e., throughout childhood), which, in turn, predicts coping at a later developmental stage (i.e. adolescence). If the relation between negative events, temperament and adolescent coping is fully mediated by changes seen in coping during childhood, there could appear to be a non-significant *direct* relation between negative events, temperament and coping in adolescence (see Mackinnon, 2008). Interaction effects on coping behaviors in the short-term (during childhood and closer to the divorce) may be particularly likely given the need for children to cope with a high frequency of post-divorce negative events. As noted earlier, the increase in negative life events after divorce is has been associated with greater concurrent use of all coping strategies (e.g., Sandler et al., 1994).

Second, the current study utilized a composite that reflected multiple aspects of temperament rather than examining different aspects separately. It may be that particular aspects of temperament interact with stressful events whereas others may not. Lengua et al. (2000) found that among children of divorce impulsivity interacted with exposure to inconsistent discipline to predict externalizing and internalizing problems, whereas only a direct effect was found for negative emotionality on adjustment. It has also been found that emotionality is more likely to predict avoidant coping and effortful control to predict active coping (Lengua & Long, 2002). Although these latter relations were regardless of level of stress, the differential prediction depending on the type of temperamental trait offers further justification for looking at aspects of temperament separately in the future. Relatedly, it has been found that different aspects of temperament interact with one another to moderate relations between stress and outcomes. Muris and Ollendick (2008) suggest that stressors may interact with negative emotionality to predict poor coping levels only if levels of effortful control are low, whereas negative emotionality may have no effect if self-control is high enough to “regulate” emotionality.

Wachs and Kohnstamm (2001) noted the inherent difficulty in finding individuals of different temperaments existing in similar circumstances and suggested “temperament-environment covariation may act to mask temperament-by-context interactions” (p. 213). Consistent with their observation, temperament was significantly related to the average number of negative life events measured over a 9-month period, with higher self-regulation related to fewer negative life

events. The overlap of assessments (i.e., although negative life events exposure was assessed at four time points, the baseline assessment overlapped with timing of the assessment of temperament) precludes causal interpretation, but the moderate correlation between these two constructs speaks to the difficulty in detecting moderation.

Alternatively, it may be that other individual-level or contextual factors play a more prominent role in influencing relations between childhood negative life events and later coping style among children of divorce. For example, individual differences in appraisal of threat, control beliefs, and cognitive errors have been found to interact with stressful events following divorce in the prediction of psychological problems (e.g., Mazur, Wolchik, & Sandler, 1992; Mazur, Wolchik, Virdin, Sandler, & West, 1999; Sandler, Kim-Bae, & Mackinnon, 2000). Parenting and quality of parent-child relations have also been shown to either exacerbate or attenuate relations between stress and physiological outcomes among children who have experienced family disruption, such as the death of a parent (Hagan et al., 2011).

Childhood Temperament and Total Cortisol Output in Young Adulthood

The significant prospective relation between higher levels of self-regulation in childhood and lower total cortisol output during a standardized stress task 15 years later when subjects were in young adulthood is remarkable given the time span. With very few exceptions (e.g., Spinrad et al., 2009), this finding is consistent with a large body of evidence supporting an association between “difficult” temperament and greater cortisol activity in the short-term among very

young children. For example, pre-school aged children who were rated by mothers as being high in both inhibition and approach (a combination that might be characterized as higher emotionality overall) exhibited higher baseline cortisol prior to a lab-based task. (Blair, Peters, and Granger, 2004). Low levels of self-control have also been found to predict higher cortisol across the day among toddlers attending day care (Dettling, Gunnar, & Donzella, 1999). A prospective association between aspects of a “difficult” temperament and cortisol activity has also been found: temperamental distress to novelty in infancy predicted greater cortisol reactivity in toddlerhood (Blair et al., 2008).

A review of the literature revealed no prospective investigations into the relation between child temperament and physiological activity later in life among children of divorce (nor among other at-risk populations). Studies that have examined concurrent relations between personality (e.g., neuroticism) or temperament (e.g., negative emotionality) and cortisol activity among adolescents and adults have produced mixed results. For example, negative emotionality and neuroticism has been found to predict flattened patterns of cortisol across the day in samples of adolescents (Hauner et al., 2008) and adults (Doane et al., 2011), but only among males. Others have found relations between neuroticism and higher diurnal (Nater, Hoppmann, & Klumb, 2010) and enhanced morning cortisol (Portella, Harmer, Flint, Cowen, & Goodwin, 2005) regardless of gender. Still others have found no relation between neuroticism and average basal or cortisol responses to stress (Kirschbaum, Bartussek, & Strasburger, 1992; Schommer, Kudielka, Hellhammer, & Kirschbaum, 1999).

There is a theoretical basis for the likelihood of relations between aspects of temperament and particular patterns of cortisol activity. Temperamental aspects of impulsivity and negative emotionality, both of which were included in the temperament construct in the current study, are related to sensitivity to stimuli, behavioral activation, and high levels of negative affect. In children of divorce, in particular, greater negative emotionality has been associated with increased threat appraisal and increased depressive symptoms (e.g., Lengua et al., 1999), both of which are often related to high cortisol output during stress (Denson et al., 2009; Dickerson & Kemeny, 2004; Lopez-Duran et al., 2009). In the current study, an “easy” temperamental style in childhood (i.e. low impulsivity, high attentional focus, and low negative emotionality) was related to lower levels of total cortisol output during a psychosocial stress task in young adulthood. It may be that temperamental self-regulation predicts appraisal of psychosocial stress as less threatening. Negative affect (e.g., anger, irritability, sadness, loneliness) was measured before and after the task in the current study; however, there appeared to be no relation between child temperament and change in negative affect across the task (results not reported here). Unfortunately, no measures were taken of the stressfulness or threat appraisal in regard to the task, so the potential for child temperament to influence cortisol activity via threat appraisal is only speculation. It may also be that childhood temperament operates on long-term physiological activity via trait levels of negative affect and/or depressive symptoms. Indeed, Doane and colleagues (2011) found this to be the case in a cross-sectional study of middle-aged men.

It is interesting that none of the studies of temperament/personality and cortisol activity among adults reviewed above focused on individuals who had experienced adverse events in childhood. The finding in the current study suggests that temperament may play a role in physiological regulation in the long-term among children of divorce. As noted earlier in the discussion, however, it is unclear whether lower total cortisol output in the current study is an index of “regulation” per se. If future research found that this relation was part of a constellation of indicators of adaptive functioning, then it might be that high levels of childhood self-regulation represents one pathway by which children of divorce remain resilient to alterations in HPA axis functioning.

One compelling speculation is the potential relations between self-regulation, alcohol use, and cortisol activity. Illustratively, children of divorce are at a higher risk of developing alcohol problems in adulthood (Wolchik, Schenk, & Sandler, 2009) and alcohol was significantly related to higher levels of total cortisol output and cortisol reactivity in the current study. This is consistent with studies that have found that youth who drink more on average exhibit higher stress sensitivity (i.e. greater cortisol reactivity to a psychosocial stress task; Zimmerman et al., 2004). Interestingly, use of alcohol has dampening effects on the stress response system immediately following ingestion (Zimmerman et al., 2004). It follows then that children of divorce who also have low self-regulatory abilities may be particularly likely to initiate use of alcohol, and given alcohol’s stress-dampening effects, may be more likely to experience ongoing addiction (e.g., Haddad, 2004). This was not systematically examined in the current study

and the correlation between self-regulation and average weekly alcohol use 15 years later was non-significant (Table 6) and close to zero, therefore this speculation is tenuous.

Temperament, Adolescent Active Coping, and Cortisol

Contrary to what was hypothesized, the *pathoplasty model*, in which difficult temperament interacts with stressors to increase use of maladaptive coping but does not itself play a causal role, was not supported. The main effect of self-regulation on both adolescent active coping and cortisol activity suggests that the *vulnerability model* is at play within this sample of children of divorce (i.e., temperament directly contributed to the development of particular patterns of coping and physiological functioning). It is critical to recognize that as a sample of children of divorce, the risk of problematic outcomes, including maladaptive coping behaviors, is already heightened. Thus, if one were to compare this sample to a population of youth from intact families, it may be that the *pathoplasty model* is operating at a higher level of context (divorce status). On the other hand, researchers have made the point that although the *pathoplasty model* seems more likely, the evidence overall is in favor of temperament acting as a vulnerability factor (Muris & Ollendick, 2005).

The direct positive relation between self-regulation and active coping is consistent with cross-sectional research that has shown a relation between negative emotionality and less use of active coping (e.g., Eisenberg et al., 1993; Fikova, 2001; Lengua et al., 1999). For example, in a sample of older adolescents, Fikova (2001) found that negative emotionality (as indexed by levels of

neuroticism) predicted less use of positive reinterpretation (e.g., reappraisal), and attentional control (as indexed by a measure of conscientiousness) predicted preference for problem-focused strategies. Among children of divorce, Lengua and colleagues (1999) found that negative emotionality was related to greater avoidant and less active coping indirectly via greater threat appraisal. In addition, they found that higher impulsivity directly predicted less use of active coping. Interestingly, Lengua et al. (1999) reported that these relations were apparent for child-report measures of the constructs but not for parent-report measures of child temperament and coping. The current study found that mother-rated child temperament was associated with youth-rated coping behaviors in adolescence, suggesting that this prospective association is not method specific. Little, if any, research has looked at temperament and coping prospectively across different developmental stages, and even cross-sectionally, researchers have noted that the number of studies is fairly thin (see Skinner & Zimmer-Gembeck, 2007).

In addition to the significant relationship between temperament and active coping, adolescent active coping was in turn marginally related to greater total cortisol output during a standardized stress task in young adulthood. Moreover, this effect partially mediated the impact of child temperament on cortisol activity 15 years later. Although many studies have found concurrent associations between active coping and *lower* cortisol, no studies to date have examined longitudinal relations between active coping and later physiological activity. The trend for adolescent active coping to predict *greater* cortisol output during a standardized task in the present investigation is consistent with another study that

found that trait reappraisal (i.e. tendency to engage in reappraisal strategies when under stress, representing an active coping style) predicted exaggerated cortisol reactivity to a speech task (Lam et al., 2009). However, it stands in stark contrast to the number of studies that have found concurrent relations between different aspects of active coping and various measures of *lower* cortisol activity (Bohnen et al., 1991; Matheson & Anisman, 2009; O'Donnell et al., 2008; Spangler et al., 2002; Taylor et al., 2003; Turner-Cobb et al., 2010).

The marginal positive relation found in the current study must be taken with extreme caution. Although also in the positive direction, the correlation between active coping and total cortisol was non-significant ($p = .25$), and a post hoc multiple regression analysis controlling for temperament and relevant covariates found that active coping was not even marginally related to cortisol output (results not reported here). Additionally, the mediation of the effect of temperament on total cortisol output by active coping was only partial in addition to being marginal (i.e. significant at the $p = .10$ level). There was an increase in the magnitude of the estimate of the effect of temperament on total cortisol when active coping was included in the model. Given this and the fact that active coping and total cortisol output were only marginally related (i.e. $p = .07$), the question arises as to whether this is evidence of a purely suppressive effect of active coping. If this is the case, it might be prudent to avoid over-interpreting the positive association between active coping and cortisol and the marginal, partial mediation of the effect of temperament on cortisol by adolescent active coping.

On the other hand, given that the correlation between active coping and cortisol was also in the positive direction (consistent with the path between active coping and cortisol in the final model), the possibility of a positive relation between adolescent active coping and cortisol activity in young adulthood should not be totally discounted. Moreover, examples of inconsistent mediation have been detected in other intervention studies, suggesting that, although not hypothesized, this marginal finding of inconsistent mediation should also not be completely discounted. For example, in addition to a number of beneficial effects, a prevention program designed to reduce adolescent use of anabolic steroids was found to increase the number of reasons for using steroids, which in turn increased intentions to use steroids (MacKinnon et al., 2001). The authors noted that this effect was not surprising given that the program included discussion of the benefits (as well as the limitations) of steroid use.

Two speculations can be made about the meaning of the positive association between active coping and cortisol and, relatedly, the evidence of inconsistent mediation should these relations be found in the population. First, individuals who exhibit a predominately active coping style respond to stressful situations by directing attention toward problem-solving efforts and cognitive restructuring. Lam and colleagues (2009) suggest that reappraisal may require effortful processing and control, which in turn might increase activation of the stress response system. Brain imaging studies do not support this theory, however. For example, approach coping is related to greater activity in the right ventromedial prefrontal cortex (Kern et al., 2008) and dampened activity in the

amygdala (Dedovic et al., 2009), and this neural activity, in turn, is related to decreased cortisol reactivity (Taylor et al., 2008). Importantly, Taylor and colleagues found these relations for individual high in coping resources, suggesting that these associations are not just evident for coping in “real time” but are also trait-based. Second, it may be that the marginal relationship between active coping and total cortisol output is actually the result of a third variable. Among children of divorce, active coping has been found to predict lower levels of externalizing behaviors (e.g., Sandler et al., 1994). Externalizing behaviors, in turn, have been most consistently related to lower levels of basal cortisol (Alink et al., 2008). It may be that lower levels of externalizing problems may account for the marginal positive association between active coping and total cortisol output. This is unlikely in the current data set, as externalizing problems are not related to total cortisol output, but are significantly related to lower cortisol reactivity (see Table 6).

In sum, in contrast to the plausibility of inconsistent mediation in MacKinnon et al. (2001), the positive relation between active coping and total cortisol output in the current study was unanticipated and surprising. Further, the finding stands in contrast to several studies that have found an opposite relation. Finally, as outlined above, there is little evidence to support the speculations of why this relation might exist, suggesting that the finding should not be interpreted as meaningful until a study design theorizing and testing this relation can be conducted.

Strengths and Limitations

As noted throughout the discussion, the study had a number of strengths including the focus on a sample of youth known to be at higher risk of negative mental and physical health problems (i.e. children of divorce), the longitudinal design, and the application of advanced, robust statistical methods. In addition, the current research involved prospective examination of negative events, coping style, and physiological activity at different stages of development (childhood, adolescence, and young adulthood, respectively) within the same sample and used different reporters for measures of temperament (mother-rated) and coping behaviors (youth self-rated), thereby circumventing the potential for relations to be due solely to method effects (e.g., Lengua et al., 1999). Further, the present investigation utilized a measure of negative life events that included subscales developed specifically for the population of interest: children of divorce. For example, several items on the stressful life events measure were derived from reports from parents and children who had experienced divorce as well as professionals (e.g., lawyers, psychologists) who worked with divorced families (Sandler et al., 1986). Further, events included on the final measure were those that were deemed as being beyond the child's control and uncontaminated by children's mental health problems, decreasing the likelihood that the events were the result of particular child personalities and increasing the objectivity of the measure (Mazur et al., 1999).

There are several conceptual and methodological limitations that must be taken into account. First, the lack of significant interactive and mediational effects

may be the result of limited statistical power to detect small effects, rather than evidence that no such associations exist. For example, the effect size for the non-significant mediation effects of negative life events on total cortisol output by active and avoidant coping in the current study was .01 and .001, respectively. If this effect size is an accurate representation of the effects one would find in the larger population, there is a less than 5% chance of detecting it with a sample of 160 subjects. That said, even if one had 1,000 subjects, power would still not exceed 5% for detecting mediation by avoidant coping. Moreover, even if the effect size in the population were double what was found in the current study, 160 subjects would still result in inadequate power to detect mediation effects. Although it is unlikely that a larger sample would have increased power to find mediation by avoidant coping, a much larger sample ($N > 500$) would have provided adequate power ($> .80$) for detecting mediation by active coping.

The sample size was also small for detecting higher order interactions. This is important to note because gender had an impact on cortisol reactivity in the current study and may be one reason why no associations were found in regard to cortisol reactivity. Gender was not examined as a moderator in any of the analyses due to the number of analyses already planned and the relatively small sample size for three-way interactions (about 70 individuals of each gender). Another limitation is the extent of physiological reactivity that occurred among participants: there was likely less opportunity to detect effects on cortisol reactivity (e.g., increase from baseline and decrease from peak) given that the

psychosocial stress task resulted in a very small average increase overall and did not induce reactivity in many subjects.

Other potential limitations include the particular make-up of the current sample of young adults who experienced parental divorce in childhood and the lack of observational measures of childhood temperament. First, the sample was predominately Caucasian, represented a very narrow age range (24 – 28 years old), and excluded pregnant women and individuals taking medications that might impact the HPA axis. The present findings, therefore, may not generalize to a larger, more diverse population of young adults who experienced parental divorce. As discussed earlier, the ability to detect interactive effects between two related constructs is statistically challenging. The likelihood of detecting effects lessens even more if the measures of the constructs are not highly reliable (e.g., Aiken & West, 1991). Although reliability of the assessment of temperament was adequate ($\alpha = .86$), the measure of temperament in the current study included mother-report only; the addition of well-designed, expertly coded observational measures may have contributed to a more comprehensive and potentially more reliable assessment of temperament.

Finally, one of the goals of the current study was to examine relations across developmental time. A stronger investigation would have included assessment of cortisol at multiple time points, such as was done by Trickett and colleagues (2010). Moreover, earlier and more frequent assessments of negative life events would have allowed a more developmental view of negative event exposure prior to, during, and following divorce. The current study can only

speaking to the post-divorce events that happened after parental separation and formal divorce proceedings and prior to entry into adolescence. As mentioned earlier, there may be a critical window or an effect of accumulation of events over developmental time that was not captured in the current study. The measure of negative events in the present investigation, although wholly consistent with what is most often done to assess episodic stressors, may not have effectively captured subtler, chronic events (e.g., daily caregiver distress) more readily assessed by interview (e.g., Badanes, Watamura, & Hankin, 2011). For example, Marin and colleagues (2007) used in-depth interviews to determine exposure to episodic and chronic stressors among adolescents and found that the relation between episodic stressors and total daily cortisol output was moderated by level of chronic stress.

Future Directions.

The current study offers a number of directions for future research. Only some of these possibilities will be highlighted here. First, future studies should look at interactions between negative life events and different aspects of temperament separately. Negative emotionality, for example, may have more influence on the relation between stress, coping and cortisol than other aspects of temperament (e.g., Lengua et al., 2000), or different aspects of temperament may interact with one another to impact how one responds to stress over the long-term (Muris & Ollendick, 2005). Second, a large body of research implicates gender as a critical variable influencing the relation between stress, coping and different measures of cortisol activity (e.g., Bento, Goodin, Fabian, Page, Quinn, & McGuire, 2010; Gunlicks-Stoessel & Powers, 2009; Schmeelk-Cone,

Zimmerman, & Abelson, 2003) as well as relations between temperament and cortisol reactivity (e.g., Dettling et al., 2009) and diurnal cortisol (e.g., Hauner et al., 2008). Kliewer and colleagues (2009), for example, found that the presence of multiple demographic and psychosocial risk factors predicted lower basal cortisol levels six months later, but the direction of association varied across levels of self-regulatory skills and gender. Interventions targeting aspects of self-regulation or coping in the hopes of preventing physiological dysregulation later on would do well to understand how relations differ between females and males. Third, relations between negative events and later physiological functioning may be strongest when cumulative risk is examined. That is, future studies should include measures of exposure to negative events over a greater time period than one year and/or in combination with factors known to be related to higher likelihood of negative event exposure (e.g., Kliewer et al., 2009; Lengua, 2002). Fourth, given that the association between negative events and later active coping appeared to be a result of confounding by temperament, future longitudinal investigations of relations between stress and later use of problem-focused strategies to cope with stress should consider the role of temperament.

Conclusions and Clinical Implications.

The current research examined relations between childhood negative life events, child temperament, adolescent coping behaviors, and cortisol activity during a standardized psychosocial stress task administered in young adulthood among individuals who had experienced parental divorce in childhood. Notable results from this investigation include a significant prospective relationship

between greater negative events in childhood and greater reliance on avoidant coping in adolescence, a strong association between an “easy” childhood temperament and active coping in adolescence, a marginal relation between number of negative life events and cortisol output 15 years later, and a significant inverse relation between greater childhood self-regulation (i.e. an “easy” temperament) and cortisol output 15 years later.

Children of divorce are at an increased risk of developing mental and physical health problems across the lifespan. It is widely accepted that the cascade of negative life events that occur in the wake of parental divorce further exacerbates this risk. Accumulating evidence points to the stress response system as a mediating variable between childhood adversity and later health. However, no study prior to the current investigation has examined whether post-divorce events contribute to stress response system dysregulation. The current findings suggest that although post-divorce events may play a small role in physiological functioning in the long-term, children of divorce who exhibit high levels of impulsivity and negative emotionality as well as low levels of attentional focus may be most at-risk of experiencing alterations in cortisol activity as long as 15 years after the divorce.

In terms of implications for interventions, the current findings suggest that prevention programs targeting children of divorce may be increasingly effective in nurturing adaptive coping behaviors and physiological regulation over the long-term if they are designed to capitalize on the opportunities (and/or to minimize the vulnerabilities) afforded by a child’s temperament. For example, interventions

might best serve those children who have fewer self-regulatory skills by focusing on increasing use of active coping strategies, rather than focusing on minimizing use of avoidant strategies. In addition, professionals working with children who have experienced family disruption may want to target basic self-regulation skills, such as focusing attention, regulating one's negative affect, and applying restraint when needed, in addition to the more manifest coping behaviors (changing negative appraisals, engaging in distraction, seeking support) that are often targets of intervention.

Table 1

Descriptive information for primary study variables.

Variable	N	M	SD	Mdn	Min	Max	Skew	Kurtosis
NLE	159	3.14	2.15	2.75	0	11.00	0.91	0.67
Raw AUCG	154	5.56	3.73	4.51	1.02	22.11	1.88	4.55
Reactivity	158	-0.01	1.00	-0.22	-2.90	4.07	1.07	2.06
Active (A)	145	11.68	2.01	11.60	6.80	15.80	0.06	-0.65
Active (C)	159	10.45	1.71	10.35	4.85	15.35	0.07	0.25
Avoidant (A)	145	9.46	1.79	9.33	4.33	14.33	-0.02	0.13
Avoidant (C)	159	9.73	1.54	9.67	5.33	14.42	-0.02	0.16
SR	152	3.31	0.62	3.37	1.95	4.58	-0.12	-0.51

Note. Total cortisol calculated as area under the curve with respect to ground using raw cortisol values. Cortisol reactivity was computed by taking the standardized residuals of the regression of log-transformed cortisol at T3 on the T1 log-transformed cortisol. A=Adolescent; C=Childhood.

Table 2

Descriptive information for potential covariates.

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	<i>Mdn</i>	Min	Max	Skew	Kurtosis
Time of day	159	1075	70	1088	858	1228	-0.84	0.99
Task time	155	62	9	61	45	93	0.92	0.81
BMI	159	25.94	5.67	25.02	16.64	44.29	1.13	1.29
Alcohol Use	159	4.33	5.86	3.00	0	30	2.50	7.05
Caffeine	159	2.11	2.08	2.00	0	12	1.97	5.08
Nicotine	159	2.65	5.74	0	0	20	2.24	3.75
Intern (W6)	159	4.54	3.25	4.00	0	17	0.77	0.58
Extern (W6)	159	10.61	8.28	9.00	0	38	1.00	-0.09

Note. Time of day refers to number of minutes past midnight at the time the first cortisol sample was taken. Task time refers to the number of minutes between the first and final cortisol samples; time of day and task time are rounded to the nearest whole number (minutes). Alcohol use is the average number of alcoholic beverages consumed per week. Caffeine intake is measured as the average number of caffeinated beverages per day.

Table 3

Frequency information for potential covariates

Variable	N	Yes (%)	No (%)
Medicine Use Past 24hr	159	46 (29)	113 (71)
Current Smoker	159	48 (30)	111 (70)
Oral Contraception (females only)	74	25 (34)	52 (66)

Note. “Yes” responses coded 1 and “No” responses code

Table 4

Zero-order correlations among primary study variables

Variable		1	2	3	4	5	6
1. Log AUCG		1.0					
2. Raw AUCG	<i>r</i>	.92**	1.0				
	<i>N</i>	154					
3. Cortisol Reactivity	<i>r</i>	.43**	.43**	1.0			
	<i>N</i>	154	154				
4. Slf-reg	<i>r</i>	-.15 [†]	-.08	-.01	1.0		
	<i>N</i>	147	147	151			
5. NLE	<i>r</i>	-.07	-.08	-.06	-.16*	1.0	
	<i>N</i>	154	154	158	152		
6. Active Coping	<i>r</i>	.06	.09	.14	.21**	.09	1.0
	<i>N</i>	140	140	144	139	145	
7. Avoidant Coping	<i>r</i>	-.07	-.03	-.17*	-.02	.27**	-.01
	<i>N</i>	140	140	144	139	145	145

Note. AUCG = Area under the curve with respect to ground. Slf-reg = Self-regulation; NLE = Negative life events.

[†]*p* ≤ .10. **p* ≤ .05. ***p* ≤ .01.

Table 5

Correlations among main variables and potential covariates measured in childhood and adolescence.

Variable		Log AUCG	Cortisol React.	Slf-Reg	NLE	Active Coping	Avoidant Coping
Age	<i>r</i>	-.07	-.08	-.01	-.06	.14 [†]	.09
	<i>N</i>	154	158	152	159	145	145
Gender	<i>r</i>	-.11	-.27**	.21**	-.11	-.02	.07
	<i>N</i>	154	158	152	159	145	145
Race	<i>r</i>	.08	.05	-.09	.07	.05	-.11
	<i>N</i>	154	158	152	159	145	145
Active Coping (C)	<i>r</i>	.02	.23**	.03	.02	.22**	-.18*
	<i>N</i>	154	158	152	159	145	145
Avoidant Coping (C)	<i>r</i>	.02	.17*	-.05	.17*	.13	.20*
	<i>N</i>	154	158	152	159	145	145
W1 Int.	<i>r</i>	.01	-.10	-.31**	.31**	-.19*	.16 [†]
	<i>N</i>	154	158	152	159	145	145
W1 Ext.	<i>r</i>	.02	-.01	-.49**	.39**	-.20*	.13
	<i>N</i>	154	158	152	159	145	145

Note. AUCG = Area under the curve with respect to ground; Cortisol React. = Standardized residualized change score; Slf-Reg = Self-regulation; NLE = Negative life events; W1 Int. = Composite of mother and child report of internalizing problems at W1; W1 Ext. = Composite of mother and child report of externalizing problems at W1. Gender is coded 1 (male) and 2 (female).
[†]*p* <= .10. **p* <= .05. ***p* <= .01.

Table 6

Correlations between main variables and potential covariates measured in young adulthood.

Variable		Log AUCG	Cortisol React	Slf-reg	NLE	Active Coping	Avoidant Coping
Alcohol Use	<i>r</i>	.25**	.23**	-.01	.05	-.11	-.07
	<i>N</i>	154	158	152	159	145	145
BMI	<i>r</i>	.05	-.06	-.22**	.05	-.12	.07
	<i>N</i>	154	158	152	159	145	145
Caffeine	<i>r</i>	.08	.03	-.22**	.01	-.10	-.03
	<i>N</i>	154	158	152	159	145	145
Nicotine	<i>r</i>	-.02	-.08	-.30**	.04	-.08	.03
	<i>N</i>	154	158	152	159	145	145
Time of Day	<i>r</i>	-.15 [†]	.11	-.03	-.11	.08	.19*
	<i>N</i>	154	158	152	159	145	145
Task Time	<i>r</i>	.12	-.03	-.09	.07	-.01	.18*
	<i>N</i>	150	154	148	155	142	142
24hr Med*	<i>r</i>	-.04	-.05	.07	-.08	-.08	.04
	<i>N</i>	154	158	152	159	145	145
W6 Ext.*	<i>r</i>	-.10	-.21**	-.09	.01	-.15 [†]	.11
	<i>N</i>	154	158	152	159	145	145
W6 Int.*	<i>r</i>	-.06	-0.15 [†]	-.21**	.11	-.17*	.10
	<i>N</i>	154	158	152	159	145	145

*Note. AUCG = Area under the curve with respect to ground; Slf-reg. = Self-regulation; NLE = Negative life events; 24hr Med = Prescription or over the counter medication use in the past 24 hours (1=Yes, 0= No); W1 Ext. = Externalizing problems at W6; W6 Int. = Internalizing problems at W6.

[†]*p* <= .10. **p* <= .05. ***p* <= .01

Table 7

The regression of negative life events on cortisol activity (total cortisol and reactivity to the task)

	<i>Total Cortisol (AUCG)</i>				<i>Cortisol Reactivity</i>			
	B	SE	Est/SE	p	B	SE	Est/SE	p
<i>Time of Day</i>	-.001*	.001	-2.015	.032	.002*	.001	1.83	.067
<i>Task Time</i>	.01*	.004	2.132	.032				
<i>Alcohol Use</i>	.03*	.009	2.932	.002	.04*	.016	2.202	.028
<i>Gender</i>					-.44*	.147	-2.970	.003
<i>W6 Externalizing</i>					-.02*	.009	-2.728	.006
<i>Negative Events</i>	-.03	.024	-1.303	.192	-.04	.036	-.320	.302

Note. † $p \leq .10$. * $p \leq .05$. ** $p \leq .01$.

Table 8.
Parameter estimates and indirect effects for multiple mediation of childhood negative life events on cortisol activity.

Outcome	Mediator	<i>a</i>			<i>b</i>			<i>c'</i>			<i>ab</i>		
		β	B (SE)	<i>p</i>	β	B (SE)	<i>p</i>	β	B (SE)	<i>p</i>	β	B(SE)	<i>p</i>
AUCG	Active Coping	.17	.16(.01)	.02	.12	.04 (.03)	.17	-.11	-.03 (.02)	.18	.02	.01(.01)	.29
		.21	.17(.05)	.001	-.02	-.01(.03)	.83	-.003	-.001(.01)	.84			
	Avoidant Coping	.17	.16(.07)	.02	.13	.06(.04)	.10	-.05	.02 (.03)	.48	.02	.01(.01)	.23
		.21	.18(.06)	.001	.14	-.07 (.05)	.12	-.03	-.01(.01)	.17			
Reactivity	Active Coping												
	Avoidant Coping												

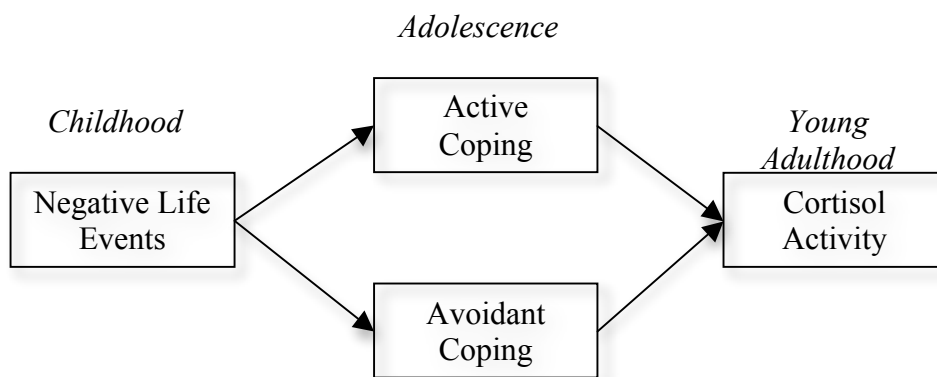
Note: Coefficients in the model predicting AUCG control for childhood active coping; childhood avoidant coping, W1 externalizing problems time of day, task time, and alcohol use. Coefficients in the model predicting Cortisol Reactivity control for childhood active coping, childhood avoidant coping, W6 externalizing problems, time of day, alcohol use, and participant gender.

Figure 1.

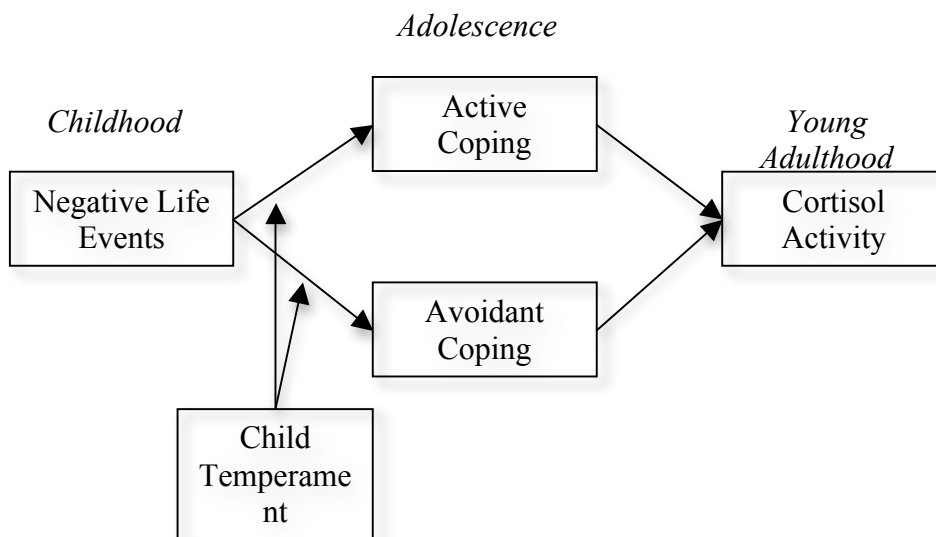
Conceptual models of hypothesized relations.



a) Direct pathway



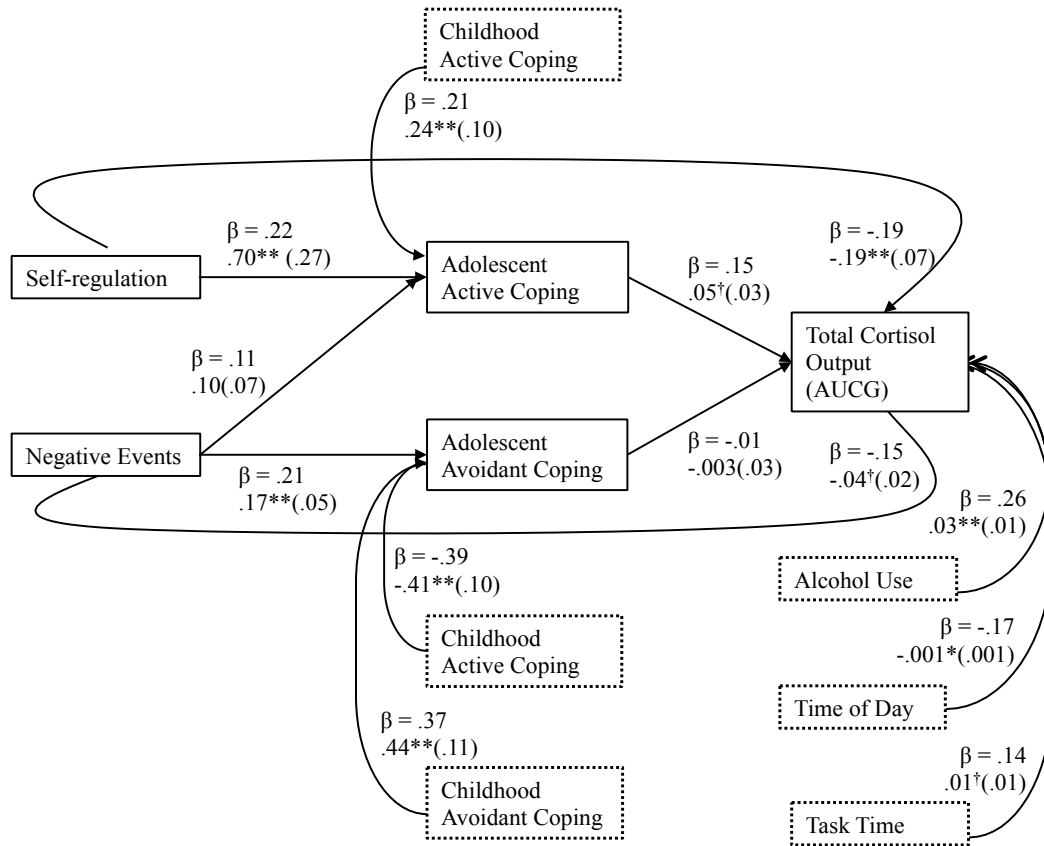
b) Multiple indirect pathways



c) Multiple moderated indirect pathways

Figure 2.

Final multiple mediation model (self-regulation and negative life events predicting total cortisol output via active and avoidant coping).



Note. Standardized coefficients are followed by unstandardized regression coefficients (standard errors). Adolescent avoidant coping and time of day were allowed to covary.

$^{\dagger}p \leq .10$. $*p \leq .05$. $**p \leq .01$.

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APPENDIX A
MEASURES

**Negative Life Events Scale
Child Report**

Items
Your brother or sister had serious trouble (such as trouble with the law, school, drugs).
Your close friend had SERIOUS troubles, problems, illness, or injury.
You suffered from a SERIOUS physical illness, injury, or extreme pain (something that required rest of one week in bed, hospitalization, or surgery).
Your brother or sister suffered from SERIOUS physical illness, injury, or extreme pain (something that required rest for one week in bed, hospitalization, or surgery).
One of your brothers or sisters was very angry or upset.
Your parents physically hit each other or hurt each other.
People in your family other than your parents (such as your brothers or sisters) physically hit each other hard or hurt each other.
Your mom or dad suffered from serious illness, injury, or extreme pain, something that required rest for one week in bed, hospitalization, or surgery.
Your mom or dad talked about having SERIOUS money troubles (being worried about bills for ordinary things).
Your relatives such as aunts, uncles, grandparents said bad things about your mom or dad.
Mom or dad fought or argued with your relatives such as aunts, uncles, grandparents.
People in your neighborhood said bad things about your mom or dad.
Your mom or dad acted badly in front of your friends (did things like yelled at them or criticized them).
You saw your mom or dad drunk.

Your mom or dad forgot to do important things for you that they promised they would do, such as take you on a trip, take you to nice places, or come to your school or athletic activities.
Your mom or dad was arrested or sent to jail.
Your mom or dad lost their job.
A close family member died such as a parent, close uncle, grandparent, or some other relative.
A close friend of yours died.
A close friend of yours moved away.
Mom and dad differed in how they want you to be (such as activities they want you to do or how you should think about things).
Dad acted very worried, upset, or sad, not because of something you did.
Your friends teased you or were mean to you.
Mom told you she doesn't like you spending time with dad.
Mom asked you questions about dad's private life.
Dad said bad things about mom.
Mom said bad things about dad.
Mom got mad at you or told you that you are bad.
Mom and dad argued in front of you.
Dad asked you questions about mom's private life.
Your dad missed scheduled visits.
Dad told you not to tell some things to your mom.
Dad told you that he doesn't like you spending time with mom.
Dad got mad at you or told you that you are bad.

Mom told you not to tell some things to your dad.
Mom acted very worried, upset, or sad, not because of something you did.
You had to give up pets, toys, or other things that you like.
Mom or dad made you follow different rules while you were at their house.
Your mother's boyfriend or husband told you to do things.
Your father's girlfriend or wife told you to do things.
Dad started to go on dates.
Dad remarried or had a girlfriend come live with him.
Dad or mom told you that the divorce was because of you.
You changed schools.
Mom had a boyfriend come live with her.
Dad got a steady girlfriend.
Mom got a steady boyfriend.
Your dad moved out of town.
Your brother or sister moved to a different house.
You had to talk to a lawyer or judge.

Answer Set:

- (1) Happened in the last 30 days
- (2) Did not happen in the last 30 days

**Child Coping Strategies Checklist
Adolescent Report**

<i>Subscale</i>	<i>Items</i>
Cognitive Decision Making	During the past month, when you had problems you thought about what you could do before you did something.
Control	You told yourself that you could handle this problem.
Repression	You tried to ignore it.
Direct Problem Solving	You did something to make things better.
Wishful Thinking	You wished that things were better.
Avoidant Actions	You tried to stay away from the problem.
Seeking Understanding	You thought about why it happened.
Positivity	You tried to notice or think about the only good things in your life.
Cognitive Decision Making	You considered consequences before you decided what to do.
Control	You told yourself you have taken care of things like this before.
Direct Problem Solving	You tried to make things better by changing what you did.
Wishful Thinking	You daydreamed that everything was okay.
Seeking Understanding	You tried to understand it better by thinking more about it.
Positivity	You reminded yourself that you are better off than a lot of other young adults.
Avoidant Actions	You avoided the people who made you feel bad.
Cognitive Decision Making	You thought about which things are best to do to handle the problem.
Repression	You tried to put it out of your mind.
Control	You told yourself you could handle whatever happens.
Direct Problem Solving	You did something to solve the problem.
Wishful Thinking	You imagined how you'd like things to be.

Avoidant Actions	You tried to stay away from things that upset you.
Seeking Understanding	You thought about what you could learn from the problem.
Positivity	You reminded yourself that overall things are pretty good for you.
Repression	You just forgot about it.
Cognitive Decision Making	You thought about what you needed to know so you could solve the problem.
Control	You reminded yourself that you knew what to do.
Direct Problem Solving	You did something in order to get the most you could out of the situation.
Wishful Thinking	You wished that bad things wouldn't happen.
Repression	You didn't think about it.
Seeking Understanding	You tried to figure out why things like this happen.
Avoidant Actions	You avoided problems by going to your room.
Positivity	You reminded yourself about all the things you have going for you.

Answer Set:

- (1) Never
- (2) Sometimes
- (3) Often
- (4) Most of the time
- (5) DK/NR

**Child Temperament
Mother Report**

Subscale	Items Excluded	Items
		Please complete this questionnaire by circling the one number for the answer that best represents how you think or feel. This first series of questions asks for some information about your child who is in our study.
Attentional Focusing		Your child usually keeps at the task until it's done.
Impulsivity		Your child usually rushes into an activity without thinking about it.
Impulsivity	X	Your child sometimes interrupts others when they are speaking.
Attentional Focusing		▼ When practicing an activity, your child has a hard time keeping his/her mind on it.
Negative Emotionality		Your child often feels frustrated.
Attentional Focusing	X	▼ Your child will move from one task to another without completing any of them.
Attentional Focusing		When doing detailed work, your child concentrates strongly.
Negative Emotionality		Your child gets troubled by everyday events.
Negative Emotionality		▼ Your child has fewer fears than others his/her age.
Impulsivity		▼ Your child usually stops and thinks things over before deciding to do something.
Impulsivity		▼ Your child is slow and unhurried in deciding what to do next.
Attentional Focusing		Your child has difficulty leaving a project he/she has begun.
Impulsivity		Your child tends to say the first thing that comes to mind, without stopping to think about it.
Attentional Focusing		▼ Your child is easily distracted when listening to a story.
Negative Emotionality		Your child gets annoyed by many things.
Impulsivity		When your child is eager to go outside, sometimes he/she rushes out without everything he/she needs.

Attentional Focusing		▼ Your child has trouble concentrating on an activity when there are distracting noises.
Attentional Focusing		▼ When watching TV, your child is easily distracted by other noises or movements.
Attentional Focusing	X	▼ Your child is distracted from projects when someone enters the room.
Negative Emotionality		▼ It takes a lot to get your child mad.
Attentional Focusing		▼ Your child often shifts rapidly from one activity to another.
Impulsivity		When your child sees something he/she wants, your child is eager to have it right then.

▼ = reverse code for scoring

X = Item excluded based on CFA (see Methods section)

Answer Set:

- (1) Very unlike your child
- (2) Somewhat unlike your child
- (3) Neither like or unlike your child
- (4) Somewhat like your child
- (5) Very like your child
- (6) DK/NA

