

Cognitive-Affective Strategies and Early Adversity as Modulators
of Psychosocial Stress Reactivity in Children and Adolescents

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Dedication

To my grandmother, Beryl Gunderson,
whose kindness, humor, and love of children are my inspiration.

Abstract

The transition to adolescence is a key period in the reshaping of systems central to emotion and stress, including maturation of neural networks involved in cognitive-affective regulation and neuroendocrine changes driven by pubertal hormones. Adolescents experience an increased prevalence of everyday stressful events and seem to exhibit increased biological stress reactivity in response to psychosocial stressors. However, there is limited developmental evidence regarding what strategies adolescents use to regulate responses to stressors and even less evidence regarding how these regulatory strategies impact physiological stress reactivity. The purpose of this dissertation was to explore cognitive-affective strategies and early life experiences as predictors of physiological reactivity to a social stressor before and after the pubertal transition. The first study examined associations between cognitive-affective strategies and cortisol reactivity to the Trier Social Stress Test for Children in typically developing children and adolescents. Across age and gender, higher trait levels of cognitive reappraisal of emotion predicted higher cortisol reactivity. The second study extended these findings by testing the impact of early life stress on the development of cognitive-affective and stress regulatory systems before and after the pubertal transition. In contrast to findings within the typically developing youth, cognitive-affective strategies did not predict cortisol reactivity in post-institutionalized internationally adopted youth. Findings are discussed in terms of future research directions and implications for the development of intervention efforts to promote self-regulation during the transition to adolescence.

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Chapter 1: Introduction

Adolescence is a unique developmental period, marked by growth and opportunity, as well as risk and vulnerability. During adolescence, individuals experience numerous transitions across individual and contextual domains, from pubertal hormones and neural plasticity to new school contexts and romantic relationships. Through changes attributable to neural, physical, and cognitive-affective development, as well as socially and culturally defined opportunities, adolescents face these transitions with new levels of independence and responsibility (Steinberg, 2005). Therefore, it is not surprising that this developmental window is a transition period marked with both developmental vulnerability and potential. Indeed, the interplay between systems of regulation may “tip the balance” toward maladaptive or adaptive pathways (Dahl & Gunnar, 2009).

During this period of adaptation, diverse contextual transitions work in concert with neural and hormonal changes, which influence cognitive, affective, and behavioral processes (Giedd et al., 2006; Walker, Sabuwalla, & Huot, 2004). In adolescence, adaptation is coupled with heightened plasticity within neurobiological systems. Pubertal changes involve the restructuring of biological systems – including neuroendocrine balance, synaptic pruning, and cortical connectivity – which has broad ranging implications across social, cognitive, and affective systems. The combination of adaptive challenge and heightened plasticity in adolescence drives the increasingly prevalent suggestion that this transition, like early childhood, is a sensitive period for the

reorganization of regulatory systems (e.g., Dahl, 2008; Dahl & Gunnar, 2009; Steinberg, 2005).

Adolescents encounter adaptive challenges across all domains of development. Socially, adolescents face changes at school and at home through new and changing relationships with peers and family members. The transition to middle school is associated with both positive and negative peer relations (e.g., social competence, Proctor & Choi, 1994; poor adjustment and deviant peer relations, Berndt, Hawkins, & Jiao, 1999). With puberty, adolescents experience new social interests and motivations, ranging from increased risk-taking propensity (e.g., Martin et al., 2002; Spear, 2000; Steinberg et al., 2009) and social reward responsivity (e.g., Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009), to new romantic interests (e.g., Neeman, Hubbard, & Masten, 1995) and changing peer relationships (e.g., Larson & Richards, 1991). Further, adolescence is characterized by the complex balance between increased cognitive responsibility (e.g., independent decision making, Steinberg, 2005) and affective reactivity (e.g., stress and emotion reactivity, Dahl & Gunnar, 2009). Finally, regarding physical development, adolescents must adapt to pubertal changes, including socially and emotionally relevant experiences surrounding pubertal timing and tempo (e.g., Ge, Brody, Conger, Simons, & Murray, 2002; Waylen & Wolke, 2004).

In recent years, research has begun to uncover unique characteristics of the adolescent brain and physiology. Evidence continues to accumulate to indicate that the adolescent brain is unique compared to that of the child and the adult in terms of both

morphology and function across brain structures and systems (Steinberg, 2010). The most commonly cited characteristic of the adolescent brain is the decrease in grey matter density (largely, but not entirely, driven by synaptic pruning) and increase in white matter density and connectivity in the prefrontal cortex, an area of the brain central to cognitive control processes (e.g., Luna, Padmanabhan, & O'Hearn, 2010; Schmithorst & Yuan, 2010). Heightened dopaminergic activity in networks linking prefrontal, striatal, and limbic regions near the pubertal transition is also a characteristic of the “adolescent brain” (Wahlstrom, Collins, White, & Luciana, 2010). These changes are frequently discussed in terms of the balance between rapidly increasing activity in reward and risk-taking systems and prolonged maturation of prefrontal regulatory systems. Importantly, in addition to structural and functional changes, new evidence points to considerable neural plasticity during adolescence (e.g., Gogtay & Thompson, 2010). Research in developmental neuroscience indicates that patterns of synaptic growth and pruning are grounded in experience and the resulting neural reorganization has lasting impacts on the developing brain (Cicchetti, 2002).

As a potential sensitive period, the adolescent transition is frequently viewed as a period of vulnerability for the emergence of psychological disorders. Following this view and in response to the increased prevalence of psychopathology during the adolescent years, research has largely focused on elucidating pathways toward adolescent pathology, with the hope to strengthen intervention and prevention efforts during this critical period (Masten, 2006; Walker et al., 2004). Although developmentally informed examinations

of psychopathology contribute to models of typical development (Sroufe, 1997), there is a paucity of basic research on normative developmental processes across the pubertal transition and into the adolescent period (Steinberg, 2005). Notably, despite Hall's (1904) assertion that adolescence is characterized by disorder and deviance, accumulating evidence asserts that the majority of youth weather adolescent "storm and stress" without experiencing significant difficulties (Steinberg & Morris, 2001). Over the last decade, the field has begun to highlight the need for developmental systems models and holistic theories in the study of normative adolescent development. An integrative approach will help to delineate unique predictors of adaptation and maladaptation, and such individual difference factors may be particularly salient and even accentuated during this sensitive period (Caspi & Moffitt, 1991).

Stress Reactivity in Adolescence

Individuals are at risk for experiencing traumatic events or acute stressors at any stage of development. However, due to normative changes, challenges, and developmental tasks of this life transition, adolescents are faced with an increased prevalence of stressful events in their everyday life (e.g., Caspi & Moffitt, 1991; Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin, Mermelstein, & Roesch, 2007). Typical, everyday stressors in the adolescent period include changes in social relationships, increased family conflict, identity concerns, adjustments to new roles and responsibilities, among others (Seiffge-Krenke, Aunola, & Nurmi, 2009). Stressors related to changes in interpersonal relationships, such as increased conflict with parents,

close friends, and romantic partners, are the most common source of stressful events named by adolescents (Compas & Phares, 1991; Seiffge-Krenke, 2006). Further, the normative experience of stressors changes throughout the adolescent period, as adolescents are faced with new challenges, such as social experiences characteristic of the middle school context, involvement in romantic relationships, increased academic demands, and preparation for educational and career goals (Seiffge-Krenke et al., 2009). Namely, early adolescents report more relationship stressors and social pressures (Petersen, Sarigiani, & Kennedy, 1991; Seiffge-Krenke, 2006) while achievement and school-related stressors peak later in adolescence (Seiffge-Krenke, 1995; Wagner & Compas, 1990).

Despite potential evolutionary benefits of increased stress reactivity during adolescence, an overly reactive behavioral and physiological system comes with both short- and long-term consequences. Behaviorally, heightened reactivity might overwhelm poorly organized cognitive-affective systems, leading to overly reactive responses in situations in which reflective means of self-regulation would be more immediately beneficial (Sontag, Graber, Brooks-Gunn, & Warren, 2008; Spear, 2000).

Physiologically, decades of research demonstrate that repeated or prolonged activation of biological stress responses leads to dysregulation within these systems (e.g., Charmandari, Tsigos, & Chrousos, 2005; McEwen, 1998) as well as affiliated pathways such as prefrontal cortical networks central to higher-order cognitive abilities (e.g., Arnsten, 2009). Physiological dysregulation following chronic stress has been linked to a

range of maladaptive outcomes including depressive psychopathology (Matthews, Nelesen, & Dimsdale, 2005), deficits in learning and memory (e.g., Sapolsky, 2003), and cardiovascular disease (e.g., Seeman et al., 2010). Although the majority of this research has been carried out within adult models and samples, recent research has demonstrated similar effects within animal and human models of adolescence (e.g., Dorn & Chrousos, 1997; McCormick & Mathews, 2010; Shea, Walsh, Macmillan, & Steiner, 2005). Further, increased plasticity in stress-related circuits of the adolescent brain may make individuals particularly sensitive to the effects of stressful experiences during this developmental period (e.g., Perlman, Webster, Herman, Kleinman, & Weickert, 2007; Romeo, 2010a). Notably, recent evidence from rodent models of adolescence suggests that repeated activation of stress response systems in this sensitive developmental period may drive reorganization within cortical-limbic systems, resulting in changes that last into adulthood (McCormick & Mathews, 2010; Romeo, 2010a).

Research regarding increased vulnerability to repeated and prolonged reactivity in physiological stress systems, as well as heightened neural plasticity and risk of long-term consequences during adolescence, highlight the need for further research on predictors and consequences of stress reactivity during this period. The first wave of research in this area has largely been driven by the rapid increase in prevalence of depression in adolescence, and evidence that depression is associated with dysregulation of the HPA axis among adults (Andersen & Teicher, 2008; Spear, 2000; Stroud et al., 2009). The vast

majority of these studies have focused on how puberty, pubertal timing, and associated sex differences predict stress system regulation and, in turn, psychopathology.

Despite these advances, there remains to be limited normative developmental evidence regarding how adolescents regulate novel domains and stress levels and even less evidence for the effect of regulatory strategies on physiological stress reactivity. Affective neuroscience points to a significant overlap between the neural circuitries of cognition and emotion (Davidson, 2000; Ochsner & Gross, 2007) and psychoneuroendocrinology highlights these networks as primary integrators of the stress response (Herman, Ostrander, Mueller, & Figueiredo, 2005). The presence and prevalence of these connections suggest that cognitive-affective self-regulatory systems may play a particularly important role in stress regulation. Indeed, understanding typically developing adolescents' psychological responses to stressors – that is, individual differences in stress reactivity – is a critical step toward understanding adaptive and maladaptive processes of self-regulation during this sensitive transitional period of development (e.g., Oldehinkel & Bouma, 2011).

The HPA Axis

Physiological responses to stressors involve two key regulatory systems: the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathetic-adrenomedullary (SAM) system. These systems work together via cortico-limbic pathways and physiological cascades, incorporating neuroendocrine, autonomic, immune, and metabolic systems, to organize a fine-tuned “neuro-symphony of stress” (Joëls & Baram,

2009; Lupien, McEwen, Gunnar, & Heim, 2009), highlighting the importance of a systems approach to self-regulation within the physiological stress response alone. The SAM system is the fast-acting energy-releasing arm of the autonomic nervous system and works in coordination with the energy-conserving parasympathetic nervous system. The major outputs of the SAM system, catecholamines epinephrine (adrenaline) and norepinephrine (noradrenaline), work in concert to mobilize energy and organize fight and flight behaviors in response to a threat.

In contrast to the relatively rapid and direct action of the SAM system, the HPA axis is characterized by complex slow-acting, but widespread effects in response to stressors. When a threat is detected, the HPA axis activates a chain of reactions composed of neuroendocrine cascades, which lead to the secretion of glucocorticoid hormone (cortisol in humans). A simple description of the cascade includes initial secretion of corticotrophin-releasing hormone and arginine vasopressin from the paraventricular nuclei of the hypothalamus, leading to release of adrenocorticotrophic hormone (ACTH) from the pituitary, which in turn, binds to receptors in the adrenal cortex to trigger release of glucocorticoids (GCs; Gunnar & Vazquez, 2006; Herman & Cullinan, 1997). The circulating GCs bind to glucocorticoid and mineralocorticoid receptors throughout the body and brain, through which they regulate the transcription of genes, leading to protein synthesis and a cascade of physiological effects, including increased cardiovascular drive, mobilization of energy, stimulation of immune responses, sharpened cognition, and decreased appetite (Sapolsky, Romero, & Munck, 2000). Critically, the GCs also regulate

the dynamics of the stress response by exerting negative feedback inhibition at multiple levels of the HPA axis and associated cortical and limbic regions, such as the prefrontal cortex, hippocampus, and amygdala (Oitzl, Champagne, van der Veen, & de Kloet, 2010). Along that line, the HPA axis is often referred to as a the limbic-hypothalamic-pituitary-adrenal-axis, due to close connections between functioning of the HPA and the limbic system, particularly in the activation and inhibition of the stress response.

Psychobiological research questions involving the HPA axis address both basal cortisol profiles and cortisol reactivity. Cortisol is not simply released in response to threats, and instead follows a diurnal circadian rhythm that is characterized by high cortisol levels in the morning with decreasing levels of cortisol production through the day and the lowest levels overnight (e.g., Edwards, Evans, Hucklebridge, & Clow, 2001; Oskis, Loveday, Hucklebridge, Thorn, & Clow, 2009). This basal pattern is critical for the mobilization of energy and motivation. Both basal cortisol levels and cortisol reactivity can be estimated in humans via salivary assays (Kirschbaum & Hellhammer, 1989). Developmental research has demonstrated that both basal and reactivity cortisol profiles follow age- and experience-related changes throughout infancy, childhood, and adolescence (Gunnar & Donzella, 2002; Gunnar & Quevedo, 2007; Hostinar & Gunnar, 2013). Although cortisol *reactivity* is the focus of this dissertation, it is noteworthy that this reactivity occurs in relation to the basal cortisol system.

The vast majority of research on adolescent stress reactivity has focused on the ways in which puberty, sex differences, and psychopathology are related to adolescents'

cortisol reactivity to psychosocial stressors. The HPA and hypothalamic-pituitary-gonadal (HPG) axes interact bidirectionally across regulatory systems from the brain to the periphery, but the specific impact of gonadal hormones on the HPA axis is largely unknown (Romeo, 2010b). The puberty-HPA stress hypothesis posits that gonadal hormones drive changes in the HPA axis during the pubertal transition and these changes increase adolescents' vulnerability for psychiatric disorder (e.g., Chrousos, Torpy, & Gold, 1998; Spear, 2000). This vulnerability is most commonly discussed in terms of depression, but has also been applied to conduct disorder and other types of psychopathology (e.g., Spear, 2000; Walker, Walder, & Reynolds, 2001).

Indeed, research has demonstrated a clear increase in basal cortisol levels related to pubertal maturation across both cross-sectional and longitudinal studies (for review see Gunnar & Vazquez, 2006; Gunnar, Wewerka, Frenn, Long, & Griggs, 2009b). Over the last decade, evidence for puberty-related increases in cortisol reactivity has begun to accumulate, although these results are less clear. Generally, findings indicate that cortisol reactivity to psychosocial stressors increases near the pubertal transition (Gunnar et al., 2009b; Stroud et al., 2009; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010; Westenberg et al., 2009), and findings regarding pubertal change in SAM reactivity are somewhat mixed (Gunnar et al., 2009b; Stroud et al., 2009).

Further, results from both animal and human studies suggest that gonadal hormones influence the HPA axis in a sexually dimorphic way; specifically, female sex hormones (i.e., estrogen and progesterone) might exert a stronger influence on the

physiology of the HPA axis than male sex hormones (e.g., Kirschbaum et al., 1996; McCormick & Mathews, 2007). Although this finding suggests that sex differences in cortisol reactivity should be prevalent after the pubertal transition, sex-specific findings on psychosocial laboratory stressor tasks have yielded mixed results and seem to partly depend on the specific nature of the stressor (Gunnar et al., 2009b; Stroud et al., 2009; Stroud, Salovey, & Epel, 2002). Further, due to interactions among multiple systems of regulation, sex differences in cortisol reactivity to a psychosocial stressor will likely depend on a host of additional factors such as pubertal timing, interpersonal stress, and cognitive-affective responses (Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Natsuaki et al., 2009).

Building on puberty- and sex-related differences in HPA reactivity, several studies have investigated associations between cortisol reactivity and psychopathology during the adolescent transition. Several studies have recently investigated the link between cortisol reactivity in response to social-evaluative or parent-child conflict stressors and internalizing or externalizing symptoms among typically developing and at-risk adolescents (e.g., Klimes-Dougan et al., 2001; Natsuaki et al., 2009; Spies, Margolin, Susman, & Gordis, 2011). Others have focused on individual differences in cortisol reactivity as potential moderators between risk and psychopathology. For example, Rudolph, Troop-Gordon, and Granger (2011) identified an interaction between peer victimization and anticipatory cortisol (before a psychosocial stressor task that involved conflict interactions with an unfamiliar peer) that predicted rumination and depressive

symptoms. Specifically, victimization predicted internalizing symptoms among those children who demonstrated heightened anticipatory cortisol levels, but among children who experienced low victimization, increased anticipatory cortisol predicted decreased internalizing symptoms. These types of studies highlight the value of cortisol reactivity as associated with adaptive and maladaptive outcomes in adolescence and demonstrate the importance of individual differences in understanding the development of internalizing symptomatology.

In line with Rudolph and colleagues' (2011) results, normative developmental changes may have adaptive or maladaptive effects based on the individuals' vulnerability upon entry to the pubertal transition. Caspi and Moffitt (1991) argued that individual differences are accentuated during life transitions. According to personal-accentuation and contextual-amplification models of the transition to adolescence, personal and contextual risks moderate individuals' ability to negotiate this developmental stage. Namely, personal and contextual characteristics may help predict and explain individual differences in adaptive versus maladaptive development during early adolescence (Rudolph & Troop-Gordon, 2010).

These models support the accentuation of individual differences during the sensitive adolescent period and highlight the importance of identifying individual predictors for adaptive and maladaptive trajectories. Because the developing brain undergoes reorganization and experiences increased plasticity during this life transition, there is opportunity for intervention and training of adaptive self-regulatory systems.

Indeed, consistent with a systems model of self-regulation, responding adaptively to unpredictable and challenging experiences is largely dependent on one's ability to regulate reactivity and arousal, and these cognitive-affective factors have been linked to exposure to negative events and subsequent development of depression in adolescence (e.g., Cole et al., 2008; LaGrange et al., 2008; Lengua, Bush, Long, Kovacs, & Trancik, 2008). The ability to regulate thoughts and emotions is also likely important for the regulation of stress reactivity (e.g., Lam, Dickerson, Zoccola, & Zaldivar, 2009), which may prevent the development of associated risk factors and maladaptive outcomes during the adolescent transition.

Self-Regulation in Adolescence

Self-regulation is a multidimensional construct that involves processes and strategies that allow the individual to willfully control thoughts, emotions, and behavior (Calkins & Howse, 2004; Rothbart, Sheese, Rueda, & Posner, 2011). These adaptive capacities improve throughout development and can be learned and trained (Buckner, Mezzacappa, & Beardslee, 2009). Self-regulatory systems are particularly salient during transition periods, such as early childhood (e.g., Calkins & Howse, 2004; Kopp, 1989) and adolescence (e.g., Dahl, 2008; Steinberg, 2010), when individuals are faced with adaptive challenges (Sokol & Müller, 2007), and self-regulation is a protective factor for positive adaptation during periods of challenge and adversity (e.g., Buckner, Mezzacappa, & Beardslee, 2003; Lengua et al., 2008). For example, following a risk-buffering model, adolescents faced with family adversity who demonstrate greater self-

regulatory skills engaged in more flexible and adaptive behaviors and had fewer mental health problems (Bakker, Ormel, Verhulst, & Oldehinkel, 2011; Eisenberg & Spinrad, 2004). Because these adaptive processes and strategies seem to promote positive outcomes and buffer risk, it is important to understand developmental systems of self-regulation to identify targets for prevention and intervention programs aimed at promoting positive development and resilience (Lengua et al., 2008).

As individuals approach the pubertal transition, rapid changes in dopaminergic systems increase appetitive motivation and affective reactivity, while prefrontal networks central to self-regulation mature more gradually across the adolescent years (for review see Steinberg, 2005). Because the changes in arousal and motivation precede increases in regulatory competence, adolescents might well be faced with regulatory challenges akin to starting an engine without a skilled driver behind the wheel (Dahl, 2008). Further challenging the goals of self-regulation, these cognitive-affective changes occur within the context of new social challenges and decreased monitoring and supervision from parents. In this way, youth are challenged to learn flexible solutions to self-control that will allow them to responsibly address challenges across a wide range of circumstances. Adolescents with higher levels of self-regulation may be able to manage increased reactivity and adaptive challenges, while those with lower levels of regulatory capabilities and less complex strategies may experience difficulty responding flexibly to changing demands (e.g., Morris, Silk, Steinberg, Sessa, Avenevoli, & Essex, 2002; Zelazo & Cunningham, 2007).

Cognitive-Affective Relations

Self-regulation is sometimes discussed in terms of two branches: cognitive control and social-emotional regulation (Bodrova & Leong, 2006; Steinberg, 2007). However, in recent years, the dichotomy between cognitive and affective processes has become increasingly blurred and research has demonstrated bidirectional interactions between cognition and emotion. As Davidson (2000) asserted in a commentary highlighting the connections between the fields of cognitive and affective neuroscience, “Cognition would be rudderless without the accompaniment of emotion, just as emotion would be primitive without the participation of cognition” (p. 91). Contemporary definitions of emotion within the field of psychology emphasize the highly functional value of affective processes. According to Cole, Martin, and Dennis’ (2004) developmental model, emotions are biologically endowed tools by which we appraise experience and prepare to act on situations. Similarly, Gross and Thompson (2007) stated that emotions are multifaceted, malleable, whole-body phenomena that arise when individuals attend to situations relevant to their goals. Emotions are involved in dynamic appraisal-action processes, which allow the individual to effectively evaluate and respond to favorable and unfavorable situations, and the regulation of these emotional experiences involves transactions between cognitive and affective systems.

Conceptually, emotions provide motivational aspects of cognition, organizing one’s thinking, learning and action, while cognitions impact the regulation of these emotions (e.g., Carlson & Wang, 2007; Liebermann, Giesbrecht, & Müller, 2007; Zelazo &

Cunningham, 2007). Physiologically, new evidence from affective neuroscience provides evidence against the dichotomous, segregated view of cognitive and affective processes, demonstrating significant overlap between the neural circuitries of cognition and emotion (e.g., Davidson, 2000; Ochsner & Gross, 2007; Olsson & Ochsner, 2008). A hierarchy of cognitive planning systems of the prefrontal cortex collects real-time input from the limbic system regarding emotional experience, and in turn, processes affective value and regulatory goals to complete a response that will regulate emotional arousal (Davidson, 2000; Zelazo & Cunningham, 2007).

Understanding the interplay between cognitive and affective systems is particularly important for modeling the maturation of self-regulation in the adolescent period. Adolescent cognitive regulation is characterized by gradual, yet marked improvements in executive control of mental processes and associated behavior. This increasingly self-directed and self-regulating mind develops gradually across the adolescent years, bringing skills important to cognitive control into an integrated system of executive processes (Steinberg, 2005). The conscious goal-directed control of thoughts and actions, known as executive function, involves a set of higher cognitive processes, including working memory, inhibitory control, planning, set shifting, and error detection (Zelazo, Carlson, & Kesek, 2008). The mechanisms of executive function form a complex, hierarchical framework that incorporates problem representation, planning, execution, evaluation, and feedback to form an effective response to the problem at hand (Zelazo, Gao, & Todd, 2007). The organization of this cognitive hierarchy is dependent on the

development of the prefrontal cortex and its diverse subdivisions.

Although executive function research often focuses on rapid changes in executive function development during early childhood, researchers studying adolescent brain development point to adolescence as an important period for maturation of the highest levels of executive function processing (e.g., Casey, Getz, & Galvan, 2008). In addition to the significant synaptic pruning within prefrontal regions central to executive functioning, connections between the prefrontal cortex and other brain regions are built, strengthened, and streamlined throughout adolescence (Giedd, 2008; Sowell, Trauner, Gamst, & Jernigan, 2002). The expansion of reciprocal linkages extending from the prefrontal “control center” of the brain might allow the adolescent to better integrate basic cognitive information with information about emotion, arousal, motivation, and context to best reflect on the situation, represent the problem, plan a response, and execute and evaluate the effectiveness of the appropriate action. Indeed, from preadolescence to late adolescence, individuals demonstrate advances in executive control, including improved strategic planning (e.g., Albert & Steinberg, 2011; Huizenga, Dolan, & van der Molen, 2006) and impulse control (e.g., Steinberg, 2008), and a reduction in perseveration and distraction errors on executive attention tasks (e.g., Crone, Donohue, Honomichl, Wendelken, & Bunge, 2006). Notably, improvements in various domains of cognitive control occur at different rates, which is important for a developmental understanding of self-regulation during adolescence. For example, improvements in inhibitory control seem to occur early in adolescence (Klimkeit,

Mattingley, Sheppard, Farrow, & Bradshaw, 2004; Luna et al., 2004), while set-shifting and strategic planning continue to improve through late adolescence (Conklin, Luciana, Hooper, & Yarger, 2007; Luna et al., 2004).

Many of the developing neural networks involved in cognitive control are also critical for a second integral part of self-regulation – the regulation of emotion. Emotion regulation includes a variety of extrinsic and intrinsic psychological mechanisms involved in the control of affective processing to enable adaptive functioning within emotionally arousing situations, including the monitoring, evaluation, and modification of emotional processes (Eisenberg, Spinrad, & Eggum, 2010; Thompson, 1994). Emotion regulation is commonly divided into two types: emotion as regulating and emotion as regulated (e.g., Cole et al., 2004; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). Zelazo & Cunningham (2007) provided a model of emotion regulation in the context of higher cognitive processes and defined emotion regulation as the modulation of the many manifestations of motivated cognition, highlighting an undeniable connection between emotion and cognition.

The achievement of effective emotion regulation has proven to be a central construct in the development of normal socioemotional and cognitive functioning across development, from positive peer interactions to decreased risk for behavioral and emotional disorders (e.g. Carlson & Wang, 2007; Denham et al., 2003; Garnefski, Rieffe, Jellesma, Terwogt, & Kraaij, 2007). Because adolescence is characterized by increased emotional reactivity and instability, as well as increased manifestation of anxiety and

depression symptoms, emotion regulation is a particularly critical process for adaptive functioning in this transition period. Indeed, regulatory systems adapt to balance both self-expression and socially acceptable behavior within specific contexts. Disturbances in this balance or the inflexibility to respond to contextual changes contribute to maladaptive regulatory responses and atypical behavior (Posner & Rothbart, 2000). Importantly, more regulation is not necessarily better regulation; the goal of emotion regulation is not to simply minimize emotion, but instead to best modulate affective experience and expression to fit the current circumstance (Bridges, Denham, & Ganiban, 2004). Further research is necessary to assess emotion regulation within the context of other individual and contextual factors to avoid mislabeling emotion *dys*regulation and to more accurately identify typical versus atypical emotion regulation across development.

Different emotion regulation strategies may be more or less effective across contexts and individuals, making the achievement of effective emotion regulation a challenging developmental process. Research on emotion regulation behaviors during adolescence has largely focused on predictors for psychopathology or groups at risk for development of internalizing disorder (e.g., high anxiety, behavioral inhibition). Only in recent years have researchers begun to address the developmental gap between children and adults' emotion regulation strategies and behaviors. For example, studies using self-report methods indicate that adolescents' emotion regulatory competency is related to engagement in risky behavior (e.g., drug use, sexual activity, behavioral adjustment, Hessler & Katz, 2010) and preadolescents' regulatory strategies in response to negative

life events (i.e., positive refocusing, positive reappraisal, self-blame, catastrophizing, and rumination) are related to symptoms of depression, worry, and fearfulness in expected ways (Garnefski et al., 2007).

Notably, the brain systems involved in willful emotion regulation continue to develop throughout adolescence (e.g., Blakemore & Choudhury, 2006; Dahl, 2004; Steinberg, 2005). In adults, emotion regulation involves activation of limbic and prefrontal systems, regions that undergo reorganization in terms of both structure and connectivity during adolescence. For example, Ray and colleagues (2005) asked adults to cognitively increase negative affect, decrease negative affect, or respond naturally in response to negative or neutral photographs while measuring neural activation via functional MRI. They demonstrated a correlation between level of self-reported rumination and unique patterns of activity in the amygdala, ventral prefrontal cortex, medial prefrontal cortex, and anterior cingulate cortex, which are neural networks involved in the representation and encoding of affective salience and self-referential thought. Further, Schaefer and colleagues (2002), among others (e.g., Ochsner, Bunge, Gross, & Gabrieli, 2002), reported greater activation in the amygdala during the presentation of negative visual stimuli and prolonged increases in this activation when participants cognitively maintained emotional responses to these stimuli. Testing specific emotion regulation strategies, Goldin, McRae, Ramel, and Gross (2008), found that voluntary reappraisal of emotional experience produced earlier prefrontal cortex responses and decreased amygdala and insular activation, while suppression of emotional

expression produced the opposite effects. These results suggest that deliberately diminishing psychological engagement with negative affect through cognitive reappraisal likely exerts a top-down inhibitory influence through synaptic projections from regions of the prefrontal cortex to the amygdala.

Together, the findings from varied domains of research on self-regulation highlight the interactions among cognitive and affective systems. Further, the temporal imbalance between the rapid increase in affective and motivational drive and the gradual maturation of cognitive-affective regulatory networks in adolescence provides a clear justification for the need to approach cognition and emotion as a system of regulation during this developmental period. Cognitive and affective systems exert transactional effects via reciprocal networks, including both “top-down” and “bottom-up” influences.

Importantly, the prefrontal-limbic network central to processes involved in the regulation of cognition and emotion interacts with and stress-response systems, including the HPA axis (Egloff, Schmukle, Burns, & Schwerdtfeger, 2006; Gross & Levenson, 1993; Root et al., 2009). The relation between cognitive-affective regulation and stress reactivity before and after the pubertal transition will be the focus of this dissertation.

The Current Dissertation

This dissertation study examines cognitive-affective strategies and early life experiences as predictors of physiological reactivity to a social stressor during childhood and adolescence. The primary aim of this research is to better understand the development of cognitive-affective and physiological stress regulatory systems. The

long-term goal is to inform the development of interventions that promote adaptive self-regulation during childhood and adolescence.

There is limited developmental evidence regarding how children and adolescents regulate novel domains and levels of stress and even less evidence for the effect of such regulatory strategies on physiological stress reactivity. Understanding individual differences in cognitive-affective processes is central to a comprehensive understanding of stress regulation. Affective neuroscience points to a significant overlap between processes involved in cognition and emotion (Davidson, 2000; Ochsner & Gross, 2007) and psychoneuroendocrinology highlights these systems as primary integrators of the stress response (Herman et al., 2003). Further, human neuroimaging studies have identified a neural circuit involving prefrontal and limbic regions that is associated with cortisol stress reactivity (Cunningham-Bussell et al., 2009; Putnam, Pizzagalli, Gooding, Kalin, & Davidson, 2008; Thomason, Hamilton, & Gotlib, 2011). Chapter 2 will examine associations between developing cognitive-affective processes and stress responsivity before and after the pubertal transition, specifically emotion regulation and coping strategies as predictors of cortisol reactivity to a social-evaluative stressor.

In addition to current regulatory and reactivity patterns, individual differences in experiences before the transition to adolescence likely play a critical role in the development of regulatory systems during this period. Chronic stress early in life is a particularly potent experience that alters neural structure, connectivity, and functioning within prefrontal and limbic regions associated with cognitive control and emotion

regulation (e.g., Liu, Diorio, Day, Francis, & Meaney, 2000; Meaney & Szyf, 2005; Tottenham et al., 2010) and biological stress systems (e.g., Gunnar & Quevedo, 2007; Hostinar & Gunnar, 2013). Further, long-term effects of early exposure to chronic stress may not become evident until adolescence (Lupien et al., 2009). Chapter 3 will build on the findings regarding typical development described in Chapter 2 and examine how the early experiences of adversity affect the long-term development of cognitive-affective and stress regulatory systems.

Overall, this dissertation will address a critical gap in the literature and will provide basic empirical information regarding the role of cognitive-affective regulation and early experiences in the development and regulation of stress response systems. Importantly, this line of research will inform the development of interventions that may work to bolster adaptive functioning within regulatory systems and strategies before the pubertal transition, potentially highlighting this sensitive period as a window of opportunity for positive developmental reorganization and growth.

Chapter 2: Cognitive-Affective Strategies and Stress Reactivity

in Typically Developing Children and Adolescents

The transition to adolescence is a key period in the reshaping of systems central to emotion and stress, including maturation of neural networks involved in cognitive-affective regulation (Giedd, 2004, 2008) and neuroendocrine changes driven by pubertal hormones (McCormick & Mathews, 2007). Further, compared to children, adolescents experience an increased prevalence of everyday stressful events (e.g., Hankin, Mermelstein, & Roesch, 2007) and seem to exhibit increased biological stress reactivity in response to psychosocial stressors (Gunnar et al., 2009b; Stroud et al., 2009). Despite potential evolutionary benefits of increased stress reactivity during adolescence (e.g., Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011), an overly reactive system has both behavioral and physiological consequences (e.g., McEwen, 1998; Sontag et al., 2008; Spear, 2000). Indeed, chronic overactivation of stress systems has been linked to a wide range of negative outcomes including depressive psychopathology, deficits in learning and memory, and cardiovascular disease (e.g., Sapolsky, 2003; Seeman et al., 2010).

Increased reactivity within stress and affective networks might promote adaptive responses to the dramatic changes in social and emotional domains that occur during adolescence, as more reactive and less reflective patterns of behavior are likely adaptive in environments in which predictability is low and conflict is high (e.g., Ellis et al., 2011; McCormick & Mathews, 2010). Although the specific impact of gonadal hormones on the HPA axis is largely unknown, research indicates that the HPA and hypothalamic-

pituitary-gonadal (HPG) axes interact bidirectionally across regulatory systems from the brain to the periphery (Romeo, 2010b). The puberty-HPA stress hypothesis posits that gonadal hormones drive changes in the HPA axis during the pubertal transition and these changes increase adolescents' vulnerability for psychiatric disorder (e.g., Chrousos, Torpy, & Gold, 1998; Spear, 2000). Indeed, research has demonstrated a clear increase in basal cortisol levels related to pubertal maturation across both cross-sectional and longitudinal studies (for review see Gunnar & Vazquez, 2006; Gunnar et al., 2009b). Over the last decade, evidence for puberty-related increases in cortisol reactivity has begun to accumulate, although these results are less clear. Generally, findings indicate that cortisol reactivity to psychosocial stressors increases near the pubertal transition (Gunnar et al., 2009b; Stroud et al., 2009; Sumter et al., 2010; Westenberg et al., 2009), and findings regarding pubertal change in SAM reactivity are somewhat mixed (Gunnar et al., 2009b; Stroud et al., 2009).

Understanding typically developing adolescents' individual differences in stress reactivity is a critical step toward understanding adaptive and maladaptive processes of self-regulation during this sensitive transitional period of development (e.g., Oldehinkel & Bouma, 2011). There is little developmental work examining how children and adolescents regulate stress and even less evidence for the effect of such regulatory strategies on physiological stress reactivity. Understanding individual differences in cognitive-affective processes is central to a comprehensive understanding of stress regulation (Denson, Spanovic, & Miller, 2009). Responding adaptively to unpredictable

and challenging experiences is largely dependent on one's ability to regulate reactivity and arousal, and these cognitive-affective factors have been linked to exposure to negative events and subsequent development of depression in adolescence (e.g., Cole et al., 2008; LaGrange et al., 2008; Lengua et al., 2008). The ability to regulate thoughts and emotions is also likely important for the regulation of stress reactivity (e.g., Lam et al., 2009), which may prevent the development of associated risk factors and maladaptive outcomes during the adolescent transition. The current study will assess developing cognitive-affective processes associated with affect and stress regulation, specifically, coping strategies, and emotion regulation (Gross, 1998).

Cognitive-Affective Regulation and Stress Reactivity

Processes involved in cognitive control and emotion regulation interact with stress systems. In response to a growing interest in neural substrates of emotion regulation and stress reactivity, recent human functional MRI studies have identified a neural circuit that is associated with stress reactivity and cortisol level (Cunningham-Bussel et al., 2009; Putnam et al., 2008). This network involves aspects of the prefrontal and limbic systems, most prominently the medial prefrontal cortex, hippocampus, and amygdala. Further, a recent resting state MRI study in young adolescents demonstrated that functional neural connectivity between the salience network and the anterior cingulate cortex was associated with cortisol responsivity during a social stress test (Thomason, Hamilton, & Gotlib, 2011). These regions are also central to cognitive and emotion regulation, which

together with previous findings among adults suggests that prefrontal and limbic networks play a role in the regulation of physiological stress response systems.

Although pioneering endocrinologist and father of stress research, Hans Selye, described physiological responses to physical stressors in terms of a universal stress response (Selye, 1936), research throughout the last several decades has highlighted the psychosocial and emotional characteristics of stressors that activate physiological stress responses. Indeed, stress systems respond to varying degrees depending on the nature of the stressor (Ulrich-Lai & Herman, 2009). However, Dickerson and Kemeny (2004) identified common characteristics of psychosocial stressors that elicit significant cortisol responses; namely, experiences that threaten the social self. If those experiences are also uncontrollable and/or unpredictable this amplifies their stress-inducing effects. According to models of stress and coping, these stress responses are a product of the subjective experience of stress and the emotional consequences following cognitive appraisal that the stressor involves demands beyond the resources at hand (Lazarus & Folkman, 1984; Lupien et al., 2006).

The social self is defined as one's social value, esteem and status (Dickerson & Kemeny, 2004) and the type of conditions that threaten the social self are those that involve evaluation of competence, capability, and/or worth. The Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) is considered the epitome of such a social-evaluative situation. In this laboratory task, participants complete an impromptu speech and math task in front of a panel of judges. The TSST contains all elements of

stress (i.e., unpredictability, uncontrollability, and threats to the social self) that stimulate elevations in cortisol to laboratory stressors in human adults. Dickerson and Kemeny's (2004) meta-analysis of acute psychological laboratory stressors demonstrated that the TSST reliably evoked significant elevations in cortisol. This task, modified for children and adolescents (TSST-C; Buske-Kirschbaum et al., 1997), was used in the present study.

Anticipatory cognitive appraisal of a future stressor is associated with cortisol stress response in adults, explaining up to 35% of the variance of the salivary cortisol response to the TSST (Gaab, Rohleder, Nater, & Ehlert, 2005). Sumter and colleagues' (2010) recent study among adolescents found that anticipatory cortisol levels were most strongly related to age and puberty (increasing with maturation). Because anticipatory responses are closely tied with cognitive-affective processes, these results further support cognitive-affective regulation as a key aspect of developmental stress reactivity and regulation (Herman et al., 2005). The emotional experience and physiological response to stress involves a set of cognitive processes within a motivationally significant context. The primary cognitive-affective processes involved in stress reactivity include cognitive appraisal of stressors, coping strategies, and emotion regulation (Gross, 1998).

Emotion regulation. In the current study, emotion regulation represents general extrinsic and intrinsic management of emotions and involves the modulation of both positive and negative emotions (Eisenberg & Spinrad, 2004; John & Gross, 2007). The prefrontal-limbic network central to processes involved in the regulation of emotional experience and expression – including the regulation of cognition, attention, behavior,

and physiology – interacts with fear- and stress-response systems, including the HPA axis (Egloff et al., 2006; Gross & Levenson, 1993; Root et al., 2009). In addition to the role of emotion regulation in the regulation of stress reactivity, the experience of a psychosocial stressor elicits the need to regulate emotions, as these stressors often instigate the experience of self-conscious emotions, such as shame, embarrassment, and pride.

Recent studies in adults have begun to test associations between emotion regulatory strategies – suppression and reappraisal – and reactivity to psychosocial stressors. Emotion suppression is a response-focused emotion regulation strategy that aims at inhibiting emotion-expressive behavior, while cognitive reappraisal is an antecedent-focused strategy that aims at changing the cognitive perception of a situation to modify the resulting emotional reaction (Gross, 2002). The majority of empirical studies of emotion regulation and stress reactivity have examined the effect of an experimental cognitive-affective manipulation (e.g., instructing participants to engage in cognitive reappraisal or emotion suppression) on stress reactivity (e.g., Gross & Levenson, 1993) or the influence of individual differences in emotion regulatory strategies on autonomic nervous system indices of stress reactivity (e.g., Egloff et al., 2006).

Lam and colleagues (2009) were the first to specifically test whether self-reported trait forms of the emotion regulatory strategies of suppression and reappraisal (Emotion Regulation Questionnaire; ERQ, Gross & John, 2003) predict individuals' stress reactivity to a social-evaluative stressor like the TSST. In line with hypotheses, trait

suppression was associated with exaggerated cortisol reactivity to a speech task. However, in contrast to hypotheses, trait cognitive reappraisal was also positively associated with increased cortisol reactivity. Although there was a significant positive association between suppression and reappraisal (a tendency for people to either use or to not use emotion regulation strategies), controlling for the effects of each strategy indicated that both reappraisal and suppression had independent effects on cortisol reactivity. Trait differences in anxiety also did not explain the observed results.

Also examining emotion regulation strategies and cortisol reactivity, de Veld, Riksen-Walraven, and de Weerth (2012) examined associations between emotion regulation strategies and cortisol reactivity during an adapted and extended version of the TSST-C in 10-year-old children. However, instead of measuring individual differences in trait forms of emotion regulation, they adapted the ERQ to measure participant's self-reported spontaneous use of reappraisal and suppression strategies during the laboratory stress task. In girls, more use of suppression was associated with lower cortisol reactivity. They found no association between reappraisal and cortisol responses to the task. As the authors discussed, the state measure of reappraisal may have incidentally indexed response-focused reappraisal rather than antecedent-focused reappraisal, because questions asked if participants used reappraisal during the stressor task (i.e., while they were experiencing physiological reactivity, not before the stressor began). This study was also limited by the use of difference scores (highest minus lowest point) as the measure

of cortisol reactivity rather than examining the magnitude of the cortisol response via an area under the curve analysis or its shape using growth curve modeling.

Coping strategies. In contrast to the more general regulatory processes involved in emotion regulation (e.g., reappraisal or suppression of positive or negative emotions), coping involves the utilization of specific behavioral and cognitive strategies to achieve goals in the context of a perceived stressor (Connor-Smith, Compas, Wadsworth, Harding Thomsen, & Saltzman, 2000; Lazarus & Folkman, 1984). Several theoretical models identify dimensions of coping, such as emotion- versus problem-focused coping (Carver, 1997) and engagement versus disengagement (Connor-Smith et al., 2000). From a developmental perspective, coping involves individuals' responses to challenges (influenced by their appraisal of the potential stressor) over the lifespan (Lerner & Castellino, 2002; Seiffge-Krenke et al., 2009). Because adolescents experience a broad range of stressful situations, the ability to select and utilize adaptive coping strategies is a key component of self-regulation during this period. For example, Rudolph and Troop-Gordon (2010) demonstrated an association between earlier pubertal maturation and heightened levels of depression; however, this association only held among youth who engaged in effortful disengagement coping (e.g., denial, avoidance) and involuntary, dysregulated responses (e.g., rumination, arousal, inaction). Those adolescents who utilized goal-directed coping (e.g., problem solving, effortful emotion regulation) in response to peer stressors were somewhat buffered from the negative consequences of early pubertal maturation.

Seiffge-Krenke and colleagues (2009) demonstrated that the use of active coping strategies predicted decreased perceived stress when presented with a similar situation four years later, while internal coping had the opposite effect, leading to increased perceived stress. Further, research suggests that coping strategies span systems of self-regulation in adolescence, from behavioral problems to stress reactivity. In line with a multisystem model of self-regulation, Valiente, Lemery-Chalfant, and Swanson (2009) examined whether 7- to 12-year-old children's coping strategies in response to stress mediated the relation between either children's effortful control or parents' responses to children's emotions and children's behavioral and academic adjustment. They identified significant correlations across outcomes, including children's coping styles, children's effortful control, parents' affective responses, and children's adjustment, suggesting that these processes may function as part of a self-regulatory system. Youth's coping strategies (specifically, engagement coping and involuntary stress responses) mediated the relation between effortful control and child adjustment and the relation between parenting and adjustment, further highlighting the importance of a multidimensional model of self-regulation. Additionally, Sontag and colleagues (2008) found that young adolescent girls with increased vulnerability (i.e., early pubertal maturation or higher levels of peer stress) demonstrated higher levels of internalizing problems and aggression. However, this association was fully mediated by the use of ineffective responses to stress (i.e., more disengagement, fewer primary control strategies, more

involuntary engagement). Further, utilization of involuntary engagement coping strategies was associated with increased cortisol reactivity during a challenge task.

The Current Study

This study examines cognitive-affective strategies as predictors of physiological reactivity to a social stressor before and after the pubertal transition. The primary aim of this research is to better understand the development of cognitive-affective and physiological stress regulatory systems during a period marked by development and plasticity within cognitive, emotion, and stress systems.

To examine this question, the current study will address three specific aims. First, this study will compare physiological stress reactivity to a psychosocial stressor in children and adolescents. Participants will include children (9 – 10-year-olds) and adolescents (15 – 16-year-olds) to best capture the developmental periods immediately before and after the pubertal transition. Physiological stress reactivity will be assessed by changes in salivary cortisol level in response to a social-evaluative laboratory paradigm. Gender differences will also be assessed, as gender differences in cortisol reactivity have been reported to emerge with puberty (e.g., Gunnar et al., 2009b; Vigil, Geary, Granger, & Flinn, 2010). It is hypothesized that adolescents will exhibit both heightened basal cortisol and increased cortisol reactivity in response to the psychosocial stressor compared to children (e.g., Dahl & Gunnar, 2009; Gunnar et al., 2009b; Stroud et al., 2009).

Second, this study will examine developmental differences in children and adolescents' emotion regulation and coping strategies. Participants will complete self-report measures of trait emotion regulation and coping strategies. Based on recent behavioral work among typically developing children and adolescents, it is expected that adolescents will report more mature and adaptive emotion regulation (i.e., cognitive reappraisal) and coping strategies (i.e., active engagement) compared to children, and that children will report more immature emotion regulation (i.e., emotion suppression) and coping strategies (i.e., disengagement, involuntary responses) (e.g., Gullone, Hughes, King, & Tonge, 2010; Seiffge-Krenke et al., 2009).

Based on previous research in adults (e.g., Gross & John, 2003) and children (Gullone et al., 2010), it is expected that boys will report higher levels of trait suppression compared to girls, and boys and girls will not differ in level of reappraisal. Regarding gender differences in coping strategies, the most consistent finding in research with both children and adults is that females seek more social support in response to challenges compared to males (e.g., Eschenbeck, Kohlmann, & Lohaus, 2007; Tamres, Janicki, & Helgeson, 2002; Taylor et al., 2006). However, the coping measure used in the current study does not include a specific measure of social coping behaviors, and results regarding gender differences in engagement and disengagement and voluntary and involuntary responses to stress are mixed (e.g., Calvete et al., 2011; Compas et al., 2001; Connor-Smith et al., 2000; Hampel & Petermann, 2005; Sontag & Graber, 2010).

Therefore, no specific directional hypotheses will be tested for gender differences in coping strategies.

Finally, the third aim will test the relation between cognitive-affective strategies and physiological stress reactivity before and after the pubertal transition. First, for emotion regulation factors, it is expected that reappraisal and suppression will predict decreased and increased cortisol reactivity, respectively (e.g., Butler et al., 2003; Egloff et al., 2006; Gross & Levenson, 1993). Second, for coping, it is expected that engagement coping will predict decreased reactivity, while disengagement coping and involuntary responses to stress will predict increased reactivity (e.g., Sontag et al., 2008). Third, I will test the hypothesis that the association between markers of adaptive cognitive-affective regulation and cortisol reactivity is stronger in adolescents than children. Because emotion and stress reactivity are at a developmental peak and cognitive-affective regulatory systems undergo substantial maturation during adolescence, adaptive cognitive-affective strategies might be increasingly strong predictors of stress system regulation in response to a psychosocial challenge in adolescents compared to children.

In summary, this study was designed to test the following specific hypotheses: 1) adolescents will exhibit both heightened basal cortisol and increased cortisol reactivity in response to the psychosocial stressor compared to children; 2) adolescents will report higher trait levels of reappraisal of emotion and engagement coping strategies compared to children, and children will report more suppression, disengagement, and involuntary responses compared to adolescents; 3) boys will report higher trait levels of emotion

suppression compared to girls; 4) reappraisal and suppression will predict decreased and increased cortisol reactivity, respectively; 5) engagement coping will predict decreased cortisol reactivity, while disengagement coping and involuntary responses to stress will predict increased reactivity; 6) associations between cognitive-affective strategies and cortisol reactivity will be stronger in adolescents compared to children. Gender differences in basal cortisol and cortisol reactivity to the TSST-C were tested, but there were no directional hypotheses.

Methods

Participants

Following a cross-sectional developmental design to compare individuals before and after the pubertal transition, participants included 40 9- and 10-year-old children (20 females, M age = 9.97, SD = .08) and 41 15- and 16-year-old adolescents (20 females, M age = 16.05, SD = .06). Nearly all participants (95%) lived in two-parent homes and 92.5% of families had at least one parent with a four-year college degree. The median family income for this sample was \$75,000 – \$125,000.

Participants were recruited from registries of families interested in being contacted about research studies and received a gift card as compensation for their participation. Exclusion criteria included diagnosis of Autism, Pervasive Developmental Disorder, or Fetal Alcohol Syndrome, or current use of steroid medication.

Procedure

Participants and one parent attended two laboratory sessions, scheduled within one

week of one another. At session 1, all participants completed a modified version of the Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum et al., 1997; Yim, Quas, Cahill, & Hayakawa, 2010). The TSST is a well-validated and widely recognized social-evaluative stressor paradigm that contains the elements necessary to stimulate cortisol hormone elevations in a laboratory setting, namely uncontrollability and social-evaluative threat to the self (Dickerson & Kemeny, 2004). At session 2, participants completed questionnaires and provided two saliva samples that served as baseline cortisol. Before each session, participants were instructed to refrain from eating dairy, caffeinated products, or high-protein meals within a few hours of the session, as these foods influence salivary cortisol levels.

Session 1. Participants and their parent arrived to the laboratory between 3:30 and 4:30 pm for a one-hour and 45-minute session. It is important to control the time of day because cortisol levels vary according to a distinct diurnal rhythm. A timeline of session one is depicted in Figure 1.

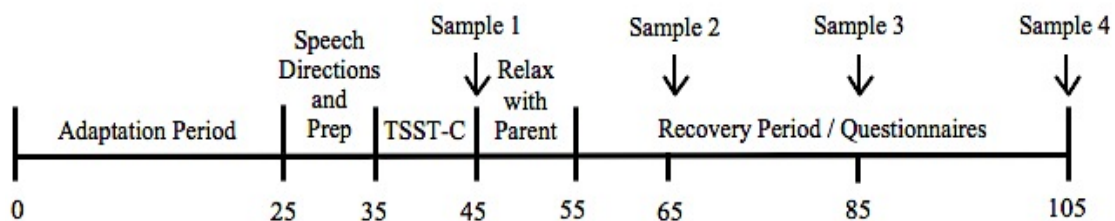


Figure 1. Timeline of events and salivary cortisol samples during session 1. Time scale reflects minutes after arrival to the laboratory.

During consent, the participants were instructed that they will be asked to give a five-minute speech and do a five-minute math problem out loud. They were told that two teachers will be observe and evaluate their speech and math, and their performance will also be videotaped and shown to a classroom of students who will rate how well they did compared to other participants in the study. Following consent, participants were instructed to read leisurely for 25 minutes (an adaptation period), after which the experimenter moved the participant to a new room and described the speech prompt. Participants were then given five minutes to prepare the speech. Participants were randomly assigned to one of two speech preparation conditions: half of the participants prepared the speech with the help of their parent (social support condition), while the other half of the participants prepared the speech with the experimenter (stranger support condition). Speech preparation condition was not a primary aim of this analysis and was entered as a covariate in all analyses (see Hostinar, 2013 for findings regarding the social support manipulation).

After five minutes of speech preparation, the participant moved to a new room unaccompanied by the parent to complete the speech (5 min.) and mental arithmetic (5 min.) tasks. For the speech, participants were to imagine that they were in a new class with about 20 other students and their teacher asked them to stand in front of the class and introduce themselves. They were instructed to talk about themselves, their personality, and why they would be liked by other students in the class. They were told to talk about at least one good thing and one bad thing about themselves (TSST-M; Yim et

al., 2010). The mental arithmetic was a continuous subtraction task; children were asked to subtract 3 consecutively from 304 and adolescents were told to subtract 7 from 758 as quickly and accurately as possible. After a mistake, the experimenter instructed the participant to start over from the beginning. The participant performed the 10-minute social stress task in front of a one-way mirror and a conspicuously placed video camera. They were told that two teachers and the experimenter were watching the performance from the other side of the mirror. Via pre-recorded voice recordings, the two teachers introduced themselves and explained that they will grade the speech and the math and that the video recording will be rated by a classroom of students. Replacing the live judges with a mirror has succeeded in elevating cortisol in 9-year-olds (Jansen et al., 2000). The experimenter administered the instructions that are typically carried out by judges during the TSST-C.

After completion of the TSST-C, participants provided a saliva sample and returned to the waiting room where they had a 10-minute break to talk with their parent. After the break they completed a battery of questionnaires and provided a saliva sample every 20 minutes for the remaining 50 minutes of the session. Participants were debriefed and given positive feedback before leaving the laboratory.

Session 2. Participants and their parents returned to the laboratory within one week of completing the first session. At this visit, they completed a set of questionnaires, which took approximately one hour to complete. They provided two saliva samples (45 and 65 minutes after arrival) as a measure of basal cortisol level.

Measures

Demographics and daily diary. Parents reported basic demographic information about their family, including income, education, and family structure. Participants and their parents completed a questionnaire at each session regarding behaviors that could influence cortisol levels, including sleeping patterns and medication use. Time since waking was included as a covariate in all cortisol analyses to control for differences in timing of the session within individuals' diurnal rhythm. Research indicates that some medications impact measurement validity of salivary cortisol (see Granger, Hibel, Fortunato, & Kapelewski, 2009 for review). Prevalence of medication use in this sample was extremely low: only five participants reported taking a type of medication that could influence salivary cortisol levels. Cortisol concentrations for these individuals were within normal range. Due to the low prevalence and typical cortisol concentrations, medication was not included as a covariate in the analysis for this study.

Pubertal status. Participants reported their current pubertal development using the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). Four questions regarding physical growth, skin changes, pubic hair, and breast/voice changes were averaged, with possible scores ranging from 1 (has not begun) to 4 (is complete). A 2 (Age) by 2 (Gender) ANOVA with mean Petersen score indicated a significant effect of age, $F(1, 77) = 399.8, p < .001$ and no main effect of or interactions with gender. As expected, adolescents reported mid to late pubertal status (M Petersen = 3.40, $SD = 0.38$) and children reported pre to early pubertal status (M Petersen = 1.62, $SD = 0.43$).

Stress appraisal. To measure appraisal of stress during session 1, participants completed the 5-question Lang Self-Assessment Manikin (Lang, 1980) at the end of the session. They reported their level of perceived stress at five points in the laboratory visit: arrival to the laboratory, while preparing the speech, during the speech, during the math task, and during the recovery period (5-point Likert scale ranging from Calm to Very Stressed).

Salivary cortisol. Salivary cortisol was collected at four times during session 1 and two times during session 2. At session 1, saliva was obtained 45, 65, 85, and 105 minutes after arrival to the laboratory (see Figure 1). The first sample assessed anticipatory baseline cortisol levels. The second sample was collected 20 minutes after the beginning of the speech, and assessed reactivity to the stressor task. The third and fourth samples were taken 40 and 60 minutes after the beginning of the TSST-C task, as measures of initial and full recovery from cortisol response. At session 2, saliva was collected 45 and 65 minutes after arrival to correspond to the adaptation period and reactivity samples from session 1.

Participants used a straw to expel the saliva into a prelabeled vial. The samples were stored in the refrigerator until all of the samples had been collected and then were stored at 220°C in the laboratory until mailed to the Biochemical Laboratory at the University of Trier for analysis. Prior research has shown that conditions experienced during mailing do not influence salivary cortisol concentrations (Clements & Parker, 1998). The samples were assayed for cortisol using a time-resolved fluorescence

immunoassay (DELFLIA). All of the samples from each participant were included in the same assay batch, and the assay batches were balanced by age group and gender. The samples were assayed in duplicate and were averaged. Duplicates varying by more than 15% were reassayed. The intraassay and interassay coefficients of variance were less than 7% and less than 10%, respectively.

Raw cortisol values that exceeded three standard deviations were winsorized at 99.7%. The two cortisol samples collected during session 2 were highly correlated, $r(79) = .86, p < .001$, and were averaged for a measure of basal cortisol level. To assess the shape of the cortisol response, the four cortisol samples collected during session 1 were modeled via linear and quadratic growth curves. All cortisol variables were not normally distributed and thus were log-transformed prior to analysis.

Emotion regulation. In contrast to instructing participants to recruit particular coping or emotion regulation techniques, the self-report design allows for participants to use their naturally occurring cognitive-affective regulatory strategies. The assessment of trait forms of these strategies provides information regarding the physiological correlates of everyday affect regulation. For this study, emotion regulation represents one's general regulation of both positive and negative emotions, while coping is considered to be a problem-specific response to a stressful or otherwise negative situation, which is why the questionnaires are distributed among categories as follows (John & Gross, 2007).

Participants completed the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003), a well-established measure of trait emotion regulation strategies. The ERQ

assesses both cognitive reappraisal and emotion suppression strategies. The ERQ consists of six reappraisal questions (e.g., “When I want to feel less negative emotion (such as sadness or anger), I change what I’m thinking about.”) and four suppression questions (e.g., “I control my emotions by not expressing them.”). Participants answered according to 7-point Likert scale ranging from Strongly Disagree to Strongly Agree and answers were averaged into composite measures of Reappraisal and Suppression. Reliability was acceptable for both the reappraisal (Cronbach’s alpha = .72) and suppression (Cronbach’s alpha = .65) subscales.

Coping strategies. To measure coping strategies, participants completed the Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000), a measure of how often an individual responds in various ways to a specific type of stressor. Due to the social nature of the TSST-C stressor, the peer stressors version of the RSQ was selected for this study. Also, participants were told that their speech and math performance would be videotaped and showed to a classroom of students who would evaluate how well they did compared to other participants in the study. The specific type of stressor for the selected version of the RSQ is problems getting along with peers (e.g., having problems with a friend, feeling pressured to do something, being around someone who is rude). At the beginning of the scale, participants report the frequency and severity of different types of problems with peers. Next, the 57 items in the questionnaire ask how often the participant does a given response when faced with “Problems getting along with other kids” (4-point Likert scale ranging from Never to Almost Always).

The RSQ contains three primary subscales: volitional engagement coping (e.g., Primary control: “I try to think of different ways to change the problem or fix the situation;” Secondary control: “I tell myself that everything will be alright”), volitional disengagement coping (e.g., Primary control: “I try to stay away from people and things that make me feel upset or remind me of the problem;” Secondary control: “I deal with the problem by wishing it would just go away, that everything would work itself out.”), and involuntary stress responses (e.g., Engagement: “I can’t control what I say or do; Disengagement: “My mind goes blank when I have problems with other kids, I can’t think at all.”). To control for differences in the base-rate endorsement of responses to stress, proportion scores were calculated as the total score for each factor divided by the total score on the RSQ (Compas et al., 2001). Scores for each subscale ranged from 0 to 1; higher scores reflect higher levels of each type of response to stress. Reliability was acceptable for all three subscales: Engagement Coping [Cronbach’s alpha = .86], Disengagement Coping [Cronbach’s alpha = .78], and Involuntary Responses [Cronbach’s alpha = .91]. Five participants had incomplete RSQ data and were excluded from analyses testing coping strategies.

Statistical Analysis

Preliminary analyses were conducted to identify outliers in cortisol concentrations, check normality of distributions, and calculate internal consistency of questionnaire measures. A 2 (Age) x 2 (Gender) repeated-measures ANOVA with five ratings of stress appraisal was examined as a manipulation check for the TSST-C

paradigm. Two 2 (Age) by 2 (Gender) MANOVAs with Emotion Regulation subscales and Coping subscales tested age and gender differences and interactions in trait emotion regulation and coping strategies.

The cortisol response curve was analyzed via hierarchical linear modeling using SAS 9.3 PROC MIXED procedure (Singer, 1998), a model that is ideal for examining change over time and maximizing statistical power. The Level 1 model represents individual change in the cortisol response as a function of linear and quadratic terms, Time and Time². Time represents cortisol increase in response to the stressor (positive slope) and Time² models the decrease in cortisol following the stressor (negative slope). The Level 2 model represents between-subjects differences in the cortisol response based on several independent variables: Age, Gender, and continuous measures of emotion regulation (Reappraisal, Suppression) and coping (Engagement, Disengagement, Involuntary Responses) that were entered as level two predictors in separate models.

All two-way interactions were included in the original models and non-significant interactions were removed from final models. Age and gender were dummy coded with female and adolescents as the reference groups. Time since wake-up to the laboratory session and speech preparation support condition were entered as covariates in all cortisol analyses. Although all sessions were scheduled in the late afternoon to reduce effects of the diurnal cortisol rhythm on cortisol reactivity, time since wake-up varied widely across participants. Time since wake-up was included as a covariate on the intercept term, because cortisol set point varies with diurnal rhythm. Speech preparation condition

(parent versus stranger support) was expected to impact cortisol response (see Hostinar, 2013) and was included as a covariate on intercept, linear, and quadratic terms. The mixed model was fit using restricted maximum likelihood estimation (REML) and degrees of freedom were computed using the Kenward and Roger (1997) method. Type 3 F tests of fixed effects are reported in the text, and estimated parameters are reported in the tables. Figures depict observed data, not estimated values.

Results

Preliminary Analyses

As expected, a RM-ANOVA with stress appraisal ratings demonstrated that ratings of stress appraisal varied significantly across the five time points in session 1, $F(3.55, 280.57) = 110.21, p < .001$ (M Arrival = 2.08, M Speech Preparation = 2.91, M Speech = 3.64, M Math = 3.84, M Recovery = 1.44), indicating that the participants perceived the TSST-C to be stressful. When the between-subjects factors of Age and Gender were included in the model, main effects of Age [$F(1, 76) = 6.77, p < .05$] and Gender [$F(1, 76) = 5.56, p < .05$] were observed. Across the session, children ($M = 2.94; SD = 0.51$) rated their perceived stress as higher than adolescents ($M = 2.62; SD = 0.62$) and females ($M = 2.93; SD = 0.58$) reported higher levels of perceived stress than males ($M = 2.64; SD = 0.56$). However, age and gender did not interact with time points of stress appraisal ratings, so these effects reflect ratings of general laboratory stress and not differences in perception of stress during the speech and math portion specifically.

Across all participants, the growth curve model demonstrated a significant linear [Time: $F(1, 233) = 8.21, p < .01$] and curvilinear [Time²: $F(1,233) = 20.44, p < .0001$] cortisol response, indicating that the TSST-C elicited expected increases in cortisol level. Further, paired-samples t-tests demonstrated that cortisol levels were significantly different at each time point as expected, namely time 1 levels ($M = .14, SD = .09$) were lower than time 2 levels ($M = .18, SD = .13$) [$t(79) = -2.02, p < .05$], time 2 levels were higher than time 3 levels ($M = .13, SD = .10$) [$t(79) = 6.28, p < .001$], and time 3 levels were higher than time 4 levels ($M = .10, SD = .06$) [$t(79) = 8.08, p < .001$].

Cortisol Response

Descriptive statistics for cortisol samples are listed by age and gender in Table 1.

Table 1

Means (SD) of Cortisol (ug/dl) Variables by Age and Gender

	Children		Adolescents	
	Males	Females	Males	Females
Session 1				
Adaptation (S1, T1)	.11 (.08)	.13 (.10)	.19 (.08)	.15 (.09)
Reactivity (S1, T2)	.10 (.08)	.18 (.15)	.23 (.12)	.20 (.15)
Recovery 1 (S1, T3)	.09 (.05)	.14 (.13)	.16 (.07)	.14 (.13)
Recovery 2 (S1, T4)	.07 (.05)	.10 (.07)	.12 (.04)	.10 (.06)
Session 2				
Basal 1 (S2, T1)	.09 (.05)	.10 (.08)	.18 (.08)	.12 (.07)
Basal 2 (S2, T2)	.09 (.06)	.09 (.06)	.17 (.07)	.12 (.08)

Note: For descriptive purposes the non-transformed values are shown.

First, a 2 (Age) by 2 (Gender) ANOVA with mean session 2 cortisol was conducted to test age and gender differences in basal cortisol level. As expected, adolescents had higher basal cortisol levels than children, $F(1, 76) = 19.20, p < .001$ (M adolescents = .15, $SD = .07$; M children = .09, $SD = .06$). There was also a trend level gender effect in which males had higher basal cortisol compared to females, $F(1, 76) = 3.83, p = .054$ (M males = .14, $SD = .07$; M children = .11, $SD = .07$). In the growth curve model, a main effect of age was observed on the intercept, $F(1, 112) = 10.55, p < .01$, with higher intercept cortisol levels in adolescents compared to children (see Table 2).

To examine age and gender differences in cortisol reactivity, cortisol was modeled via HLM as a function of Time and Time², with Age, Gender, and their interactions as between-subjects factors. A significant age by gender interaction was observed on the linear, Time $F(1, 233) = 4.51, p < .05$, and quadratic terms, Time² $F(1, 233) = 4.67, p < .05$ (see Table 2). Male children did not demonstrate a significant cortisol response to the stressor, while male adolescents showed a cortisol response that was similar to that of female children and adolescents (see Figure 2).

Table 2
Parameter Estimates for Growth Curve Model of Cortisol Reactivity with Age and Gender

Effect	Estimate	SE	df	<i>t</i> value	<i>P</i>
Intercept	.70	.14	115	5.11	<.0001*
Time	.08	.06	232	1.42	.16
Time ²	-.05	.02	232	-2.67	.01*
Age	-.11	.08	115	-1.26	.21

Age x Time	.10	.07	232	1.48	.14
Age x Time ²	-.03	.02	232	-1.20	.23
Gender	.11	.08	115	1.28	.20
Gender x Time	.04	.07	232	.64	.52
Gender x Time ²	-.01	.02	232	-.62	.53
Age x Gender	-.18	.12	115	-1.47	.14
Age x Gender x Time	-.21	.10	232	-2.12	.03*
Age x Gender x Time ²	.07	.03	232	2.16	.03*

* $p < 0.05$

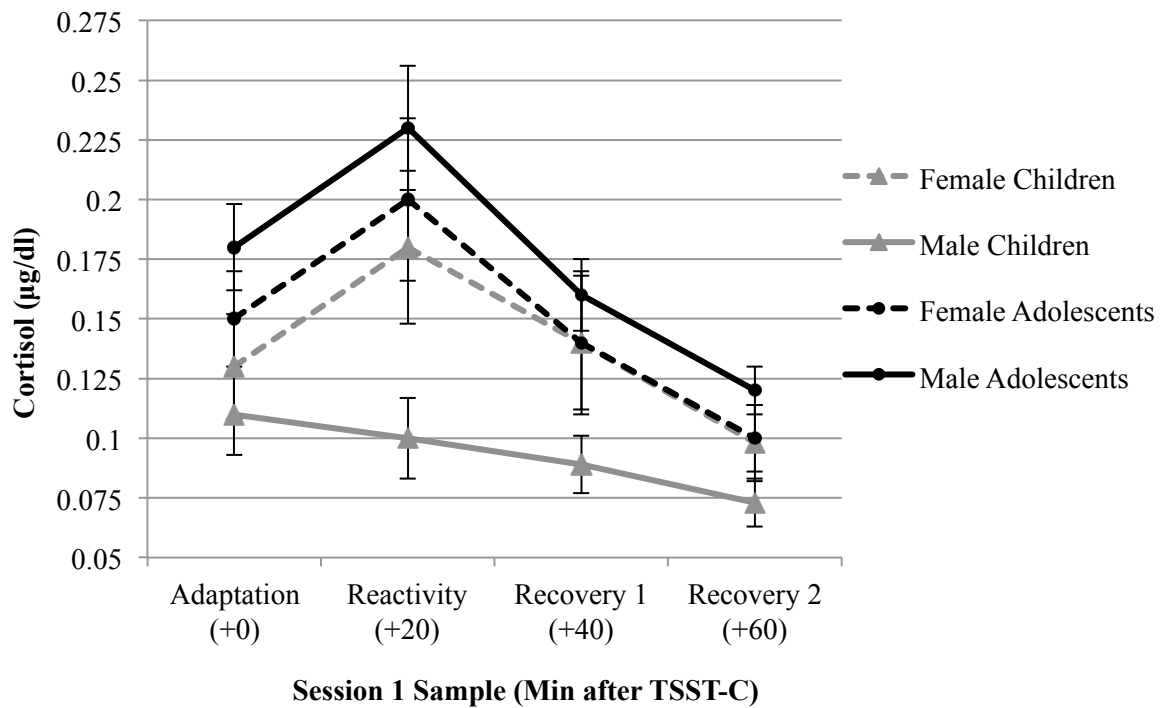


Figure 2. Mean cortisol response to the TSST-C for children and adolescents, split by gender. Error bars represent \pm SEM.

Cognitive-Affective Strategies

Descriptive statistics for emotion regulation and coping variables are listed by age and gender in Table 3. Correlations among variables are listed in Table 4.

Table 3

Means (SD) of Cognitive-Affective Strategies by Age and Gender

	Children		Adolescents	
	Males	Females	Males	Females
Emotion Regulation				
Reappraisal	4.80 (1.01)	4.80 (1.03)	4.98 (.85)	4.89 (.91)
Suppression	4.14 (1.37)	3.75 (.93)	3.62 (.98)	3.35 (1.01)
Coping				
Engagement	.36 (.05)	.37 (.05)	.40 (.06)	.40 (.07)
Disengagement	.23 (.03)	.22 (.03)	.20 (.03)	.21 (.02)
Involuntary Responses	.42 (.06)	.41 (.06)	.40 (.05)	.38 (.06)

Table 4

Correlations Between Emotion Regulation and Coping Strategies

	1.	2.	3.	4.	5.
1. Reappraisal	1	-.02	.11	.11	-.16
2. Suppression		1	-.32**	.12	.25*
3. Engagement			1	-.19	-.88**
4. Disengagement				1	-.30**
5. Involuntary Responses					1

* $p < .05$, ** $p < .01$

Emotion regulation. A 2 (Age) by 2 (Gender) MANOVA with Reappraisal and Suppression as dependent variables revealed no significant multivariate effects. Self-reported trait reappraisal and suppression did not differ by age and there were no main effects or interactions with gender (see Table 3).

Coping. A 2 (Age) by 2 (Gender) MANOVA with Engagement Coping, Disengagement Coping, and Involuntary Responses as dependent variables identified a significant multivariate effect for age, Wilks' $\lambda = .83$, $F(2, 71) = 7.45$, $p < .001$. Univariate tests revealed that for Engagement Coping, there was a main effect of age, $F(1, 72) = 9.48$, $p < .01$, with higher levels of engagement in adolescents ($M = .40$, $SD = .06$) compared to children ($M = .36$, $SD = .05$). For Disengagement Coping, there was a main effect of age, $F(1, 72) = 6.97$, $p < .05$, and an age x gender interaction, $F(1, 72) = 5.17$, $p < .05$, in which male children reported higher levels of disengagement than male adolescents (see Table 3). Reports of involuntary responses did not differ by age or gender (see Table 3).

Associations Between Cognitive-Affective Strategies and Cortisol Reactivity

Emotion regulation. Results from the growth curve model with Reappraisal, Age, and Gender indicated that trait reappraisal predicted cortisol reactivity to the TSST-C. There was a significant effect of reappraisal on the linear, Time $F(1, 231) = 6.27$, $p < .05$, and the quadratic terms, Time² $F(1, 231) = 7.86$, $p < .01$ (see Table 5). In contrast to expectations, greater trait cognitive reappraisal predicted increased cortisol reactivity to the TSST-C (see Figure 3). There was also a main effect of reappraisal on the intercept,

$F(1, 113) = 5.71, p < .05$; higher reappraisal predicted higher intercept cortisol level. The effect of reappraisal did not interact significantly with age or gender on the intercept, linear, or quadratic term. Suppression did not predict cortisol reactivity and there were no significant interactions with age or gender (see Table 6).

Table 5

Parameter Estimates for Growth Curve Model of Cortisol Reactivity with Age, Gender, and Reappraisal

Effect	Estimate	SE	df	<i>t</i> value	<i>p</i>
Intercept	.68	.13	82	5.31	<.0001*
Time	.08	.05	231	1.44	.16
Time ²	-.05	.02	231	-2.70	.01*
Age	-.10	.08	115	-1.24	.21
Age x Time	.11	.07	231	1.58	.12
Age x Time ²	-.03	.02	231	-1.32	.19
Gender	.10	.08	115	1.29	.20
Gender x Time	.04	.07	231	.57	.57
Gender x Time ²	-.01	.02	231	-.54	.60
Age x Gender	-.17	.11	115	-1.51	.13
Age x Gender x Time	-.20	.10	231	-2.09	.04*
Age x Gender x Time ²	.07	.03	231	2.12	.03*
Reappraisal	.07	.02	116	2.39	.02*
Reappraisal x Time	.07	.02	231	2.50	.01*
Reappraisal x Time ²	-.02	.01	231	-2.80	.01*

* $p < 0.05$

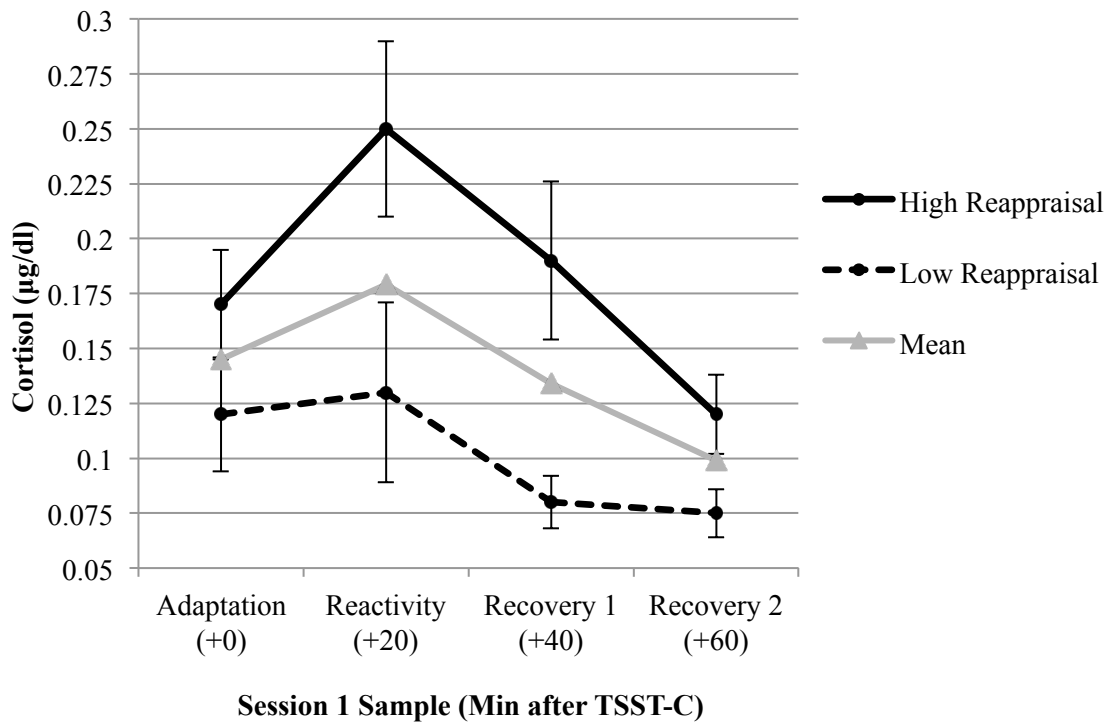


Figure 3. Mean cortisol response to the TSST-C for youth high in reappraisal versus youth low in reappraisal. High and low reappraisal are depicted as 1 SD above and below the mean, respectively; however, reappraisal was analyzed as a continuous variable. Error bars represent \pm SEM.

Coping. None of the RSQ coping subscales (i.e., Engagement, Disengagement, Involuntary Responses) predicted cortisol reactivity and there were no significant interactions with age or gender (see Table 6).

Table 6

Type 3 Tests of Fixed Effects for Suppression and Coping Predictors of Cortisol Reactivity

Effect	Type 3 Test of Fixed Effects
Suppression	$F(1, 112) = 3.15, p = .08$
Suppression x Time	$F(1, 227) = 1.94, p = .16$
Suppression x Time ²	$F(1, 231) = 2.81, p = .10$
Engagement	$F(1, 99.8) = 2.27, p = .13$
Engagement x Time	$F(1, 212) = 0.28, p = .60$
Engagement x Time ²	$F(1, 212) = 0.01, p = .91$
Disengagement	$F(1, 99.7) = 1.39, p = .24$
Disengagement x Time	$F(1, 212) = 0.33, p = .57$
Disengagement x Time ²	$F(1, 212) = 0.03, p = .87$
Involuntary Responses	$F(1, 100) = 0.63, p = .43$
Involuntary Responses x Time	$F(1, 212) = 0.00, p = .96$
Involuntary Responses x Time ²	$F(1, 212) = 0.02, p = .90$

Note: Suppression, Engagement, Disengagement, and Involuntary Responses were tested in separate HLM growth curve models with Age, Gender, and Group in each model (as depicted for Reappraisal in Table 5). The statistics reported here are the Type 3 Tests of Fixed Effects from each separate growth curve model.

Discussion

The purpose of this study was to examine trait emotion regulation and coping strategies as predictors of cortisol reactivity to a social stressor before and after the pubertal transition. Overall, the TSST-C elicited an increase in both perceived stress and the cortisol stress response. Consistent with previous research, adolescents exhibited

higher basal cortisol compared to children. Also, males tended to have higher basal cortisol levels than females. In general, adolescents did not demonstrate heightened reactivity to the stressor compared to children. Instead, an age by gender interaction was observed, in which male children demonstrated blunted cortisol reactivity to the TSST-C. Adolescents reported more frequent use of engagement coping. Among males, children reported more frequent use of disengagement coping compared to adolescents. Age and gender groups did not differ in trait reappraisal, trait suppression, or engagement in involuntary responses to stress. Regarding the primary research aim to examine the association between trait cognitive-affective strategies and physiological reactivity, higher trait levels of reappraisal predicted higher cortisol reactivity across age and gender. Suppression and coping subscales (i.e., engagement, disengagement, involuntary responses) were not related to cortisol response to the TSST-C.

The findings regarding developmental changes in HPA activity were partly consistent with previous research. The observed increase in basal cortisol levels after the pubertal transition for both males and females supports a now well-documented developmental pattern (e.g., Adam, 2006; Gunnar et al., 2009b; Netherton et al., 2004; Stroud et al., 2009; Walker et al., 2001). In the current study, the increased set point in cortisol during adolescence was demonstrated by basal measures at session 2 as well as an intercept effect at session 1, as the cortisol response curve was higher for adolescents throughout the TSST-C session (see Figure 2).

Moving to HPA reactivity to the stressor task, developmental changes in cortisol reactivity to the TSST-C were only observed among males. Specifically, 9- and 10-year-old boys failed to mount a stress response to the TSST-C, while significant cortisol reactions were observed among male adolescents and females in both age groups. Although several studies have not identified gender by development interaction effects in this age group (e.g., Sumter et al., 2010; Westenberg et al., 2009), some gender differences have been observed. In a recent study of 10-year-olds' physiological reactivity to a modified version of the TSST-C, de Veld and colleagues (2012) observed higher cortisol reactivity in girls compared to boys. In a previous study in our lab, both male and female 9-year-olds and 15-year-olds responded to the TSST-C, while neither boys nor girls demonstrated a significant cortisol response at age 11 years, and among 13-year-olds only girls responded to the stressor (Gunnar et al., 2009b).

One explanation for the current results could be that male children did not find the task particularly stressful. However, both male and female children rated their perceived level of stress throughout the session as higher than adolescents. Notably, ratings of stress appraisal were not significantly correlated with cortisol reactivity in the current study, and many studies have cited weak associations between perceived stress and physiological stress responses (e.g., Cohen et al., 2000). Perceived stress ratings and physiological responses represent reflective and reactive branches of the regulatory system, respectively. It is possible that although children appraise the laboratory experience to be highly stressful – perhaps because the task of giving a speech in front of

an audience is more novel at that age – their physiological reactivity to the experience is less pronounced, particularly in males. On the other hand, stressors that most reliably activate HPA reactivity, such as the social-evaluative TSST-C, include an element of threat to the self, which is likely more salient in adolescence; both behavioral and neurobiological studies indicate that individuals are particularly concerned about social evaluation and the social rewards associated with positive peer opinions during this developmental period (Guyer et al., 2009).

A second explanation for the gender by age interaction is a gender difference in pubertal status. In Gunnar and colleagues' (2009) study, hypo-reactivity among boys in early adolescence was explained by gender differences in pubertal status. Indeed, the pubertal development of girls is typically more advanced than their age-matched male peers at this age. Pubertal development undoubtedly influences physiological stress reactivity at many levels within the self-regulatory system, including hormonal, emotional, motivational, and psychosocial. In the current study, males and females did not differ in self-reported pubertal stage, although it is very plausible that changes in the HPG axis may interact with the HPA axis differently for boys and girls. More research is needed to test gender differences in stress reactivity before the pubertal transition and during early puberty. Due to interactions among multiple systems of regulation, sex differences in cortisol reactivity to a psychosocial stressor will likely depend on a host of additional factors such as pubertal timing, interpersonal stress, and cognitive-affective responses (Klimes-Dougan et al., 2001; Natsuaki et al., 2009). It is also unclear here

whether the more noteworthy effect is low reactivity in boys or marked reactivity in girls prior to the pubertal transition. Increased cortisol reactivity in girls compared to boys before the puberty transition might be associated with increased prevalence of depression among girls in early adolescence (e.g., Natsuaki et al., 2009).

Consistent with expectations, adolescents reported more use of engagement coping in response to social stressors than children. These results support previous work demonstrating that during adolescence, individuals' cognitive development allows them to approach challenges in a more reflective way, such as considering multiple perspectives and formulating a plan to flexibly respond to a specific circumstance (Kavsek & Seiffge-Krenke, 1996; Seiffge-Krenke et al., 2009). Among males, children reported more frequent use of disengagement coping than adolescents. Although this gender by development interaction was not expected, it is possible that during late childhood, boys are less mature than girls, which may contribute to the expected developmental difference (i.e., higher disengagement coping in childhood) in boys but not in girls. Contrary to expectations, adolescents and children did not differ in their level of cognitive reappraisal or suppression of emotions. A recent longitudinal study among 1,128 youth aged 9 to 15 years tested use of self-reported suppression and reappraisal studies each year for three years (Gullone et al., 2010). This study identified decreases in suppression with age, but no clear developmental pattern for reappraisal.

To further understand the results of the current study, parent emotion socialization practices such as emotion coaching and family emotion expressivity should be

considered. Parents play an important role in scaffolding emotion regulation throughout childhood and adolescence (e.g., Eisenberg, Cumberland, & Spinrad, 1998; Katz & Hunter, 2007). Families in the current sample are highly educated and of medium to high socioeconomic status. It is possible that within advantaged samples, parents scaffold children's use of more mature emotion regulation strategies at younger ages, possibly accounting for the lack of developmental effects between late childhood and mid-adolescence. Lastly, age groups did not differ in involuntary responses, suggesting that the tendency to engage in involuntary responses to stress might vary on an individual level or that the current measure was not sensitive enough to detect developmental differences in this type of coping strategy.

Regarding the association between cognitive-affective strategies and cortisol reactivity, higher trait levels of reappraisal predicted higher cortisol reactivity to the TSST-C in males and females, before and after the pubertal transition. This finding supports the hypothesis that the use of cognitive reappraisal as an emotion regulation strategy could be an important individual difference factor in understanding stress reactivity in late childhood and adolescence. However, the directionality of the association was not as predicted. Cognitive reappraisal of emotions did not decrease physiological reactivity to a psychosocial challenge, but instead, in this sample, higher cognitive reappraisal predicted *increased* stress reactivity. Further, those who reported using low levels of reappraisal did not show a significant cortisol response to the TSST-C (see Figure 3).

Although in contrast to predictions based on a top-down regulatory model, the current results support the one published study to date that has examined associations among trait emotion regulation strategies and cortisol reactivity to a psychosocial stressor. In a sample of undergraduates, Lam and colleagues (2009) found higher trait reappraisal as measured by the ERQ to be associated with greater cortisol reactivity to the TSST. In their study, trait suppression was also associated with greater cortisol reactivity. Notably, the effects were not explained by differences in trait anxiety level.

According to top-down models of self-regulation, reflection and reappraisal of experiences work to regulate physiological responses to stressors (e.g., Jamieson, Mendes, & Nock, 2013). However, the processes underlying these regulatory studies are not well understood. Regarding the current results, it is possible that cognitive reappraisal increases cognitive effort, attentional resources, and emotional engagement with the situation at hand, leading to increased activation in cortical-limbic systems and in turn increases activation of the HPA axis. Indeed, the prefrontal-limbic network central to the regulation of emotional experience and expression interacts with fear- and stress-response systems, including the HPA axis (Gross & Levenson, 1993; Root et al., 2009). However, if this were the case, experiments that instruct participants to engage in cognitive reappraisal during a stressor task would also demonstrate increased cortisol reactivity. Instead, these experimental studies tend to identify a negative association between instructed reappraisal and physiological reactivity (e.g., Gaab et al., 2003; Giuliani, McRae, & Gross, 2008; Goldin et al., 2008; Jamieson, Nock, & Mendes, 2012).

The developmental processes underlying interactions among cortico-limbic and stress axes involved in emotion and stress regulation are largely unknown. Understanding the development of this neurobiological regulatory system will be critical to developing a systems model of self-regulation in adolescence. For example, in a study examining emotion regulation strategies, preadolescent girls recruited more prefrontal networks to complete a voluntary reappraisal task than did adult women, possibly illustrating immaturity of the connections between prefrontal and limbic regions central to conscious emotion regulation in childhood and early adolescence (Levesque et al., 2004).

In the current study, self-reported trait reappraisal of emotions predicted increased cortisol reactivity to the TSST-C in both children and adolescents. This study is unable to tease apart the directionality of this association; use of reappraisal could increase cortisol reactivity, or higher trait cortisol reactivity could increase trait levels of reappraisal. This finding raises questions regarding characteristics of individuals who are high in trait cognitive reappraisal. The questions regarding cognitive reappraisal on the ERQ ask how often you “change what you’re thinking about” when you are feeling either positive or negative emotions. The questions are not specific to reappraising negative emotions in response to stress. The general tendency to rethink emotions could be related to a ruminative response style. Rumination would be particularly relevant to the current study, because rumination is associated with greater vulnerability to depression, which increases in prevalence during the adolescent transition (e.g., Nolen-Hoeksema, 1991; Sontag & Graber, 2010). Although rumination is typically conceptualized as an involuntary

response to stress, the RSQ subscale for involuntary responses would likely not be sensitive enough to detect this specific effect. Future studies will benefit from investigating rumination as well as other psychological characteristics that could help explain the association between cognitive reappraisal and cortisol reactivity in late childhood and adolescence.

Additionally, individuals who reported low trait levels of reappraisal had atypically blunted responses to the TSST-C. Again, the current design cannot differentiate whether low use of reappraisal impacts cortisol stress reactivity, or if individuals who tend to be hypo-reactive to social evaluative stressors do not develop a need for reappraisal as an emotion regulation strategy. One explanation could be that for individuals who avoid or disengage from their feelings (do not think about positive or negative feelings) also disengage from stressful situations, as demonstrated by blunted cortisol reactivity. Reappraisal was not correlated with suppression or disengagement in this study; however, it is not surprising that low reappraisal would be distinct from active suppression or disengagement responses.

Importantly, the relationship between cognitive reappraisal of emotion and physiological HPA axis reactivity will differ among children, adolescents, and adults. This is the first study to examine trait forms of emotion regulation in relation to cortisol stress reactivity in children and adolescents. Most similar in design to the current study, De Veld and colleagues (2012) examined associations between participant's self-reported spontaneous use of reappraisal and suppression strategies and cortisol reactivity to a

modified version of the TSST-C in 10-year-old children. They found no association between reappraisal and cortisol responses to the task. Further developmental research is needed to better understand developmental processes underlying associations between cognitive reappraisal, cortisol reactivity, and other branches of the self-regulatory system.

Contrary to expectations, none of the other cognitive-affective constructs (i.e., emotional suppression, engagement coping, disengagement coping, and involuntary responses to stress) predicted cortisol reactivity to the TSST-C. Also, associations between cognitive-affective strategies and cortisol reactivity did not differ by age group. More research is needed to understand how these cognitive-affective strategies contribute to the self-regulatory system in the context of a psychosocial stressor. Future studies that combine trait, state, and instructed measures of cognitive-affective strategies, along with assessments of other individual characteristics, such as rumination, anxiety, self-esteem, and executive function, as predictors of cortisol reactivity will help to elucidate both these null findings and the observed positive association between cognitive reappraisal and stress reactivity.

The current study has several limitations. First, as discussed above, although the results demonstrated a clear association between trait reappraisal and cortisol reactivity, the directionality of and the processes underlying this relationship remain unclear. Future studies should include both trait and state measures of cognitive-affective strategies to better differentiate individuals' general tendency to use strategies and specific strategies used during the laboratory stressor. It is likely that both individual dispositional factors

and situational demands will influence cognitive-affective strategy use (Egloff et al., 2006). De Veld and colleagues (2012) demonstrated that 10-year-olds are able to reliably report emotion regulation strategy use during the TSST-C. Further, inclusion of an experimental manipulation of cognitive-affective strategy use in combination with trait measures would help elucidate the directionality of these associations (i.e., reactivity predicting regulatory style or regulatory style predicting reactivity). Future research should also explore other factors that may contribute to the relationship between cognitive-affective strategies and cortisol reactivity, including individual differences such as executive function, emotional reactivity, and self-concept, as well as social factors like family socialization of emotion, parental emotion coaching, and peer status.

Second, regarding the measure of stress reactivity, the current study focused on salivary cortisol as a marker of HPA axis reactivity, which only captures one branch of the physiological stress system. The study would be strengthened by the inclusion of salivary alpha amylase and cardiovascular measures of sympathetic and parasympathetic system activity. Within the HPA axis alone, salivary cortisol is not able to differentiate which arm of the axis is driving the observed findings. Further, because all sessions were scheduled during the late afternoon to control for diurnal fluctuations in cortisol level and to capture a time during which the HPA axis should be most sensitive to stimulation, the current results cannot be generalized to other times of the day.

Third, the current study is unable to tease apart the effects of age and puberty, as these two measures were highly correlated in the present design comparing youth before

and after the pubertal transition. Future research should include participants across a greater range of pubertal status within, rather than between, age groups to specifically test the effect of puberty. This study is further limited by the cross-sectional design. However, longitudinal research on stress reactivity is difficult to carry out due to habituation effects of the stressor paradigms. Lastly, the current study focused on normative developmental changes and the findings cannot be generalized to higher risk populations. Additionally, the participants in this study were primarily upper middle class and Caucasian. Future research should examine these questions in a more socioeconomically and ethnically diverse sample.

Despite these limitations, the present study makes several important contributions. This is the first known study to examine associations among trait cognitive-affective strategies and cortisol reactivity to a social-evaluative stressor in children or adolescents. In addition to being the first to test these associations among youth, the current study included two age groups to directly test developmental differences in these associations. Further, extending previous research in adults (Lam et al., 2009), this study examined coping strategies in addition to trait emotion regulation strategies. This design allowed for the testing how two different types of cognitive-affective strategies (i.e., emotion regulation and coping with stress) predicted cortisol reactivity in two different age groups (i.e., before and after the pubertal transition). Developmental research on individual differences in these self-regulatory strategies has important implications for understanding variability in individuals' responses to circumstances that challenge the

regulatory system. The current study demonstrated that individual differences in how children and adolescents regulate their emotions predict physiological reactivity to and recovery from psychosocial stressors. Additionally, due to a large sample size, this study was able to test gender differences in cortisol reactivity, cognitive-affective strategies, and associations between these constructs. This proved to be an important contribution, as gender differences were identified for both cortisol and cognitive-affective outcomes.

In conclusion, the current study is the first to examine associations between trait cognitive-affective strategies and physiological reactivity to a psychosocial stressor in children and adolescents. The results illustrate increased basal cortisol after the pubertal transition in both males and females and blunted cortisol reactivity to a psychosocial stressor before the pubertal transition in males. Adolescents reported more frequent engagement coping strategies. In males, children reported more use of disengagement coping. Across age and gender, higher trait levels of cognitive reappraisal of emotions predicted higher cortisol reactivity. Overall, these results highlight the importance of examining relations among cognitive, emotional, and physiological processes to better understand the development of the self-regulatory system during the transition to adolescence.

Chapter 3: Cognitive-Affective Strategies and Stress Reactivity in Post-Institutionalized Children and Adolescents

The transition to adolescence is characterized by dramatic changes in physiological, emotional, and cognitive regulatory systems (Blakemore & Choudhury, 2006; Dahl & Gunnar, 2009). Self-regulation is particularly important during this transition period, as individuals are faced with adaptive challenges across individual and contextual domains as well as new levels of independence and responsibility (Steinberg, 2005). The normative changes, challenges, and developmental tasks of early adolescence are associated with increased prevalence of daily stressors (e.g., Caspi & Moffitt, 1991; Ge et al., 1994; Hankin et al., 2007). Further, compared to children, research suggests that adolescents exhibit increased biological stress reactivity in response to psychosocial stressors (Dahl & Gunnar, 2009; Gunnar et al., 2009b; Stroud et al., 2009). Despite these advances, there remains to be limited developmental evidence regarding how adolescents manage new experiences and stress levels and even less evidence for the effect of regulatory strategies on physiological stress reactivity.

The purpose of Chapter 2 was to test cognitive-affective correlates of psychosocial stress reactivity in typically developing children and adolescents. Individual differences in emotion regulation strategies were associated with cortisol reactivity to the TSST-C in both age groups. Specifically, increased trait level of cognitive reappraisal of emotions predicted higher cortisol reactivity to this social-evaluative stressor. In addition to current regulatory and reactivity patterns, individual differences in experiences during

childhood likely play a critical role in the development of cognitive-affective and stress regulatory systems during this period. The goal of Chapter 3 is to examine the impact of early life stress on cortisol reactivity and related cognitive-affective regulatory strategies before and after the pubertal transition.

In infancy and early childhood, self-regulatory systems develop within the caregiver-child relationship as sensitive and responsive caregivers modulate and scaffold young children's arousal and regulation (e.g., Carlson, Jacobvitz, & Sroufe, 1995; Cole et al., 2004; Gunnar & Donzella, 2002). Disruptions to this early caregiving environment have the potential to change developmental trajectories of physiological and cortico-lymbic systems involved in reactivity and regulation. Indeed, developmental research highlights deficits in self-regulatory behavior (e.g., Blair, 2010; Evans & Kim, 2013), changes in neural structure, connectivity, and functioning within prefrontal and limbic regions associated with cognitive control and emotion regulation (e.g., Liu et al., 2000; Meaney & Szyf, 2005; Teicher et al., 2003; Tottenham et al., 2010;), and dysregulation of stress physiology (e.g., Bosch et al., 2012; Gunnar & Quevedo, 2007, Gunnar, Frenn, Wewerka, & Van Ryzin, 2009a) among children and adolescents who have experienced deprived environments in early childhood, especially when caregiving relationships were disrupted. However, the majority of studies that examine effects of early life stress on developmental outcomes are unable to tease apart the impact of early deprivation versus the impact of ongoing adversity associated with high-risk contexts, such as poverty or

maltreatment (e.g., Evans & Kim, 1997; Gunnar & Donzella, 2002; Gunnar & Quevedo, 2007; Kaufman et al., 1997).

The current study will focus on post-institutionalized children and adolescents who were adopted internationally from institutional (e.g., orphanage) care as young children. This population provides a natural experiment wherein the period of early life stress is circumscribed to the time before adoption. Institutionalized children often experience multifaceted deprivation, which may include inadequate nutrition and medical care, inadequate physical and cognitive stimulation, and for nearly all, a lack of consistent and supportive caregiving relationships. Adoption acts as a dramatic natural intervention that marks the shift from a deprived to a supportive caregiving environment (van Ijzendoorn & Juffer, 2006). It is well known that early adversity in the form of institutional rearing impacts brain-behavior networks central to emotion and stress systems (Gunnar & Quevedo, 2007; Hostinar & Gunnar, 2013), children reared in institutions are at increased risk of attention and behavior regulatory problems (e.g., Kreppner, O'Connor, & Rutter, 2001; Tizard & Hodges, 1978), and these risks increase with the length of time spent in institutional care (e.g., Gunnar & van Dulmen, 2007; Kumsta et al., 2010; Wiik et al., 2011).

Only recently have researchers begun to examine the impact of early institutionalization on adolescent development (Colvert et al., 2008; Goff et al., 2012; Quevedo, Johnson, Loman, Laffavor, & Gunnar, 2012; Sonuga-Barke, Schlotz, & Kreppner, 2010). Importantly the adolescent transition is marked by reshaping of systems

central to stress and emotion (Andersen & Teicher, 2008; McCormick & Matthews, 2010; Romeo, 2010a; Romeo, 2010b) and may be a potential sensitive period for development within the HPA axis and other stress- and emotion-mediating systems.

This study will build on questions regarding typical development and examine the impact of early life stress on the long-term development of cognitive-affective and stress regulatory systems. Specifically, the purpose of this study is to examine associations between cognitive-affective strategies and psychological reactivity to a social stressor in post-institutionalized (PI) youth before and after the pubertal transition. To examine the impact of early life stress, PI children and adolescents will be compared to the children and adolescents described in Chapter 2. The participants included in Chapter 2 will serve as the non-adopted (NA) comparison group; all of these youth were born and raised in their birth families. Previous research indicates that the families who are recruited from the current registry tend to be highly educated and have medium to high incomes, similar to Minnesotan families who adopt internationally (see Participants section below).

The current study has three specific aims. The first aim is to test the impact of early life stress on cortisol reactivity to a psychosocial stressor before and after the pubertal transition. A large body of research in animal models has demonstrated that early life stress affects the development of the HPA axis and associated stress responses (Chrousos & Gold, 1992; Levine, 2005). However, the directionality of those effects is not clear. Comparison of maternal separation (severe stress) and early handling (moderate stress) paradigms in rodent models suggests that severe or overwhelming early life stress

leads to stress vulnerability (i.e., hyper-reactivity of the HPA axis; Sanchez et al., 2001) while stress that is mild or moderate in severity leads to stress resilience (i.e., hypo-reactivity of the HPA axis; Denenberg, 1999). In humans, most studies that identify increased HPA activity following early life stress are conducted with individuals who have psychological disorders or risk for psychological disorders (e.g., Heim et al., 2002), while studies in psychiatrically healthy individuals often reveal hypo-reactivity of the HPA axis (Carpenter et al., 2007; Cicchetti & Rogosch, 2001; Elzinga et al., 2008; Heim et al., 2001, 2002; MacMillan et al., 2009).

In a recent study of 10 – 12-year-old internationally adopted children, Gunnar and colleagues (2009a) tested the impact of mild/moderate versus severe early life stress on cortisol reactivity to the TSST-C. The study tested the stress-inoculation hypothesis, that posits that exposure to mild/moderate stressors may help the individual resist stressful experiences later in development (i.e., stress resilience), while stressors that are overwhelming may increase individuals' vulnerability and reactivity to later stressors (Garmezy, 1991). Indeed, the adopted children who experienced mild/moderate early life stress (adopted early primarily from foster care) showed lower cortisol reactivity to the TSST-C compared to children who experienced severe early life stress and non-adopted children. Surprisingly, the children who experienced severe early life stress did not differ in cortisol reactivity from the non-adopted group. Although the PI and NA groups did not differ, marked individual differences in cortisol reactivity were observed within the PI group. Individual child factors associated with severity of deprivation (physical growth

stunting at adoption) predicted cortisol reactivity. Chronic activation of stress-mediating systems inhibits physical growth, specifically height for age, and serves as an index of allostatic load in children who have experienced chronic early stress (Johnson, Bruce, Tarullo, & Gunnar, 2011; Sonuga-Barke et al., 2010). Physical growth stunting has now been related to cortisol activity in several studies with PI children (Gunnar et al., 2009a; Johnson et al., 2011; Kertes, Gunnar, Madsen, & Long, 2008).

This is the first study to test cortisol reactivity in PI adolescents. The pubertal transition could serve as a period of increased vulnerability or a window of opportunity for PI adolescents. Developmental changes during this period, such as increased prevalence of social stressors and increased reactivity in emotion and stress systems could overwhelm PI youth's regulatory systems and tip the balance toward increased risk. For example, Colvert and colleagues (2008) found that children adopted from Romania did not have heightened internalizing problems before adolescence, but after the pubertal transition, the adopted adolescents had higher rates of clinical emotional problems than comparison children. Similarly, a recent study comparing depressive symptoms in PI children and adolescents found increased parent-reported depression in PI adolescents, but not in PI children (Goff et al., 2012).

On the other hand, recent evidence in animal models suggests that the pubertal transition is a period of heightened plasticity and a sensitive period for not only increased vulnerability but for positive change within HPA axis and other stress-mediating systems (Romeo, 2010). Increased plasticity may allow the long-term effects of early deprivation

to be reorganized during the transition to adolescence given the dramatic contrast in environment between institutional care and supportive, advantaged families post-adoption. For PI children who are generally living under low stress conditions in supportive homes, the pubertal transition could be an opportunity for the HPA axis to recalibrate to a low stress context. Indeed, our findings from a recent study of PI youth during the pubertal transition support the pubertal recalibration hypothesis (Quevedo et al., 2012). We found that early life stress was associated with a blunted cortisol awakening response, but this effect was moderated by pubertal status: pre/early pubertal PI youth had a blunted cortisol awakening response, but mid/late pubertal PI youth did not differ from comparison groups. This study supports the hypothesis that the pubertal transition may provide an opportunity for reorganization of stress-mediating systems in the context of a low stress environment.

First, testing the pubertal recalibration hypothesis, I predict that PI and NA youth will differ in cortisol reactivity before, but not after the pubertal transition. It is not clear based on previous research whether early life stress will predict hyper- or hypo-reactivity in these age groups. Notably, the current design does not allow for the direct testing of pubertal recalibration, because pubertal status and age are confounded, comparing youth before and after the pubertal transition. Results will be discussed in terms of preliminary conclusions in the context of recalibration with adolescence and directions for future research specifically testing pubertal effects. Second, drawing from the stress-inoculation hypothesis, I predict that indices of severity of early life stress (i.e., age at adoption,

duration of institutionalization, physical growth stunting) will predict cortisol reactivity within the PI group. Specifically, greater severity of early life stress will be associated with greater HPA reactivity, while moderate early life stress will be associated with lower HPA reactivity. Notably, because the PI participants were sampled as a moderate to high early life stress group, there may not be enough variability in severity of early experience to adequately test this hypothesis.

The second aim is to test the impact of early life stress on emotion regulation and coping strategies before and after the pubertal transition. Research in both animal models and human samples indicates that early deprivation impacts brain-behavior networks central to affective processing (McEwen, 2007), including increased fear and anxiety behaviors (Sanchez et al., 2005) and heightened risk for affective disorders (Heim & Nemeroff, 2001). Research in post-institutionalized children indicates that institutional care is associated with alterations in amygdala functioning and structure, specifically larger amygdala volume and increased anxiety symptoms (Tottenham et al., 2010) and decreased glucose metabolism in limbic regions (Chugani et al., 2001) years after adoption. Behaviorally, PI children show deficits or delays in emotion understanding and emotion identification that are predicted by time in institutional care (Fries & Pollak, 2004). In addition to increased anxiety and larger amygdala volume, Tottenham and colleagues (2010) demonstrated that PI children who spent more time in institutional care prior to adoption made more errors on blocks of an emotional Go-Nogo task that contained negative/threat faces, indicative of problems with emotion regulation. Also in

one study, PI adolescents had higher levels of parent-reported depression, but PI children did not differ from the comparison group (Goff et al., 2012).

This is the first known study to examine trait emotion regulation strategies or coping strategies in PI children or adolescents. Based on previous research indicating emotional deficits in PI children, I hypothesize that PI children will demonstrate more immature emotion regulation (i.e., more suppression) and coping strategies (i.e., more disengagement and involuntary responses) compared to NA children. However, whether the expected group differences in emotional processes during childhood will continue through the pubertal transition into adolescence is largely unknown, as these questions have not yet been tested in adolescent samples. Adaptive challenges experienced during the transition to adolescence may increase vulnerability for emotional problems in PI youth (Colvert et al., 2008; Goff et al., 2012). On the other hand, developmental changes in cortico-limbic systems during the pubertal transition might provide an opportunity for positive development in the context of supportive adoptive homes, leading to fewer emotional deficits in PI youth after the pubertal transition.

The third and primary aim of this study is to examine the impact of early life stress on the relation between cognitive-affective strategies and cortisol reactivity before and after the pubertal transition. Although many studies in PI youth have tested the impact of early adversity on stress physiology, no published studies to date have examined how PI children manage their stress responses. Additionally, no published studies to date have assessed stress reactivity in PI youth during the transition to

adolescence. The current study addresses both gaps by examining cognitive-affective predictors of cortisol reactivity in PI youth in late childhood and adolescence, developmental periods in which one's ability to effectively regulate stress responses is particularly salient in the context of increased prevalence of daily stressors. It is possible that increased plasticity during the pubertal transition would allow the development of more typical connections between cortico-limbic and stress-mediated systems. Therefore, particularly if PI youth experience positive developmental changes in HPA activity during the pubertal transition, synchrony between cognitive-affective and stress response systems may also undergo change. On an individual level, it is possible that cognitive-affective strategies will be particularly important for the regulation of stress within this group; PI individuals who utilize adaptive emotion regulation and coping strategies may be more likely to mount typical physiological reactivity to a stressor. Due to the paucity of previous research in this area, this aim is largely exploratory.

In summary, this study was designed to extend the findings described in Chapter 2 to examine cognitive-affective strategies and stress reactivity in PI youth. The current study uses the same sample and the same data as described in Chapter 2 for the NA group. No hypotheses will be made regarding this group, because these findings have already been discussed. Regarding the post-institutionalized group, this study was designed to test the following specific hypotheses: 1) PI and NA children will differ in cortisol reactivity (it is not clear based on previous research whether early life stress will predict hyper- or hypo-reactivity), but groups will not differ in adolescence; 2) in the PI

group, greater severity of early life stress will be associated with greater cortisol reactivity and moderate early life stress will be associated with lower cortisol reactivity; 3) PI youth will demonstrate more suppression, disengagement coping, and involuntary responses, and less reappraisal and engagement coping compared to NA youth both before and after the pubertal transition; 4) reappraisal and engagement coping will be associated with more typical cortisol response patterns in PI children and adolescents (i.e., increased reactivity if PI youth demonstrate hypo-reactivity as a group and decreased reactivity if PI youth demonstrate hyper-reactivity); 5) the association between cognitive-affective strategies and cortisol reactivity will be stronger in PI adolescents compared to PI children. Gender effects and interactions will be examined, but no specific directional hypotheses will be tested.

Methods

Participants

The participants included 162 9- and 10-year-old children and 15- and 16-year-old adolescents divided into two groups based on early care experiences. The PI youth had been internationally adopted after 11 months of age after spending the majority of their life in institutional care (e.g., orphanage or hospital). The NA youth were born and raised in their birth families in the United States. The PI children were recruited through the Minnesota International Adoption Project (MnIAP) registry of families who have adopted children internationally and are interested in participating in research (Hellerstedt et al., 2008). The NA children were recruited from a similar registry of families interested

in participating in developmental research. Exclusion criteria included diagnosis of Autism, Pervasive Developmental Disorder, or Fetal Alcohol Syndrome, or current use of steroid medication. Three of the 162 participants were excluded from this analysis: two were excluded because they were taking corticosteroid medications that strongly impact salivary cortisol levels and one was excluded due to a Fetal Alcohol Syndrome diagnosis.

The final sample included 78 PI and 81 NA youth (see participant descriptive characteristics in Table 7). Groups did not differ in education or income. In both groups, over 92% of families had at least one parent with a four-year college degree and median family income of \$75,000 – \$125,000. PI youth (18%) were significantly more likely than NA youth (4%) to live in a single parent home, $\chi^2(1, N = 158) = 11.20, p < .01$.

Table 7

Participant Descriptive Statistics by Group and Age

	Post-Institutionalized <i>n</i> = 78 <i>M</i> (<i>SD</i>)	Non-Adopted <i>n</i> = 81 <i>M</i> (<i>SD</i>)
Children		
Females/Males (<i>n</i>)	20/18	20/20
Age at session (yrs)	9.7 (0.6)	10.0 (0.5)
Age at adoption (mos)	24.3 (14.3)	-
Time in institution (mos)	19.7 (9.7)	-
Region of origin (<i>n</i>)		
Eastern Europe	20	-
Asia	17	-
Central & South America	1	-
Africa	0	-

Adolescents		
Females/Males (<i>n</i>)	22/18	20/21
Age at session (yrs)	15.6 (0.7)	16.0 (0.4)
Age at adoption (mos)	27.3 (16.4)	-
Time in institution (mos)	22.3 (15.1)	-
Region of origin (<i>n</i>)		
Eastern Europe	19	-
Asia	18	-
Central & South America	2	-
Africa	1	-

Procedure

Participants and one parent attended two laboratory sessions, scheduled within one week of one another. At session 1, all participants completed a modified version of the Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum et al., 1997; Yim et al., 2010). The TSST is a well-validated and widely recognized social-evaluative stressor paradigm that contains the elements necessary to stimulate cortisol hormone elevations in a laboratory setting, namely uncontrollability and social-evaluative threat to the self (Dickerson & Kemeny, 2004). Participants were randomly assigned to prepare their speech with either their parent or the experimenter. Speech preparation condition was not a primary aim of this analysis and was entered as a covariate in all analyses (see Hostinar, 2013 for findings regarding the social support manipulation). At session 2, participants completed questionnaires and provided two saliva samples as a measure of basal cortisol. Before each session, participants were instructed to refrain from eating dairy, caffeinated

products, or high-protein meals within a few hours of the session, as these foods influence salivary cortisol levels. See Chapter 2 for detailed description of procedure and Figure 1 for timeline of session 1.

Measures

Demographics and daily diary. Parents reported basic demographic information about their family, including income, education, and family structure. Parents of adopted children provided information about their child's pre-adoptive experience, age at adoption, and height, weight, and head circumference at adoption. Participants and their parents completed a questionnaire at each session regarding behaviors that could influence cortisol levels, including sleeping patterns and medication use. Time since waking was included as a covariate in all cortisol analyses to control for differences in timing of the session within individuals' diurnal rhythm.

Research indicates that some medications impact measurement validity of salivary cortisol (see Granger et al., 2009 for review). As discussed in Chapter 2, prevalence of medication use in the NA group was extremely low, with only five NA participants taking a type of medication that could influence salivary cortisol levels. However, the prevalence of medication use was higher in the PI group: 24 PI youth were taking a medication that has some potential to influence salivary cortisol. Medication use was coded by type and count per Granger and colleagues' (2009) guidelines so that all participants received a medication code ranging from 0 (no medication use) to 7. Medication was included as a covariate in all analyses reported in the Results section.

Additionally, all analyses were replicated excluding for participants who were taking psychotropic medications (most likely to impact measurement validity of cortisol). Eight children and 12 adolescents were excluded in the PI group and 1 adolescent was excluded in the NA group. When excluding these individuals, the trend level effect of Group on linear ($p = .08$) and quadratic ($p = .08$) terms was no longer at trend level [Time $F(1, 402) = 2.68, p = .10$; Time² $F(1, 402) = 2.51, p = .11$]. Results regarding cognitive-affective strategies and associations between cognitive-affective strategies and cortisol did not change. The change in the cortisol reactivity group effect could be due to a direct influence of medication, but this effect could also be caused by real differences in cortisol reactivity within this subgroup, or alternatively, a reduction in statistical power. Because removing individuals who are taking psychotropic medications biases the sample by removing the majority of individuals who have a psychiatric diagnosis, all participants were included in the reported results and medication code was included as a covariate in all cortisol analyses.

Pubertal status. Participants reported their current pubertal development using the Pubertal Development Scale (Petersen et al., 1988). Four questions regarding physical growth, skin changes, pubic hair, and breast/voice changes were averaged, with possible scores ranging from 1 (has not begun) to 4 (is complete). As expected, adolescents reported mid to late pubertal status (M Petersen = 3.29, $SD = 0.53$) and children reported pre to early pubertal status (M Petersen = 1.52, $SD = 0.40$). Groups did not differ in pubertal status, $F(1, 152) = 1.47, ns$.

Stress appraisal. To measure appraisal of stress during session 1, participants completed the 5-question Lang Self-Assessment Manikin (Lang, 1980) at the end of the session. They reported their level of perceived stress at five points in the laboratory visit: arrival to the laboratory, while preparing the speech, during the speech, during the math task, and during the recovery period (5-point Likert scale ranging from Calm to Very Stressed).

Salivary cortisol. Salivary cortisol was collected at four times during session 1 and two times during session 2. At session 1, saliva was obtained 45, 65, 85, and 105 minutes after arrival to the laboratory (see Figure 1). The first sample assessed anticipatory baseline cortisol levels. The second sample was collected 20 minutes after the beginning of the speech, and assessed reactivity to the stressor task. The third and fourth samples were taken 40 and 60 minutes after the beginning of the TSST-C task, as measures of initial and full recovery from cortisol response. At session 2, saliva was collected 45 and 65 minutes after arrival to correspond to the baseline and reactivity samples from session 1. Saliva samples were collected and assayed as described in Chapter 2. The intraassay and interassay coefficients of variance were less than 7% and less than 10%, respectively.

Raw cortisol values that exceeded three standard deviations were winsorized at 99.7%. The two cortisol samples collected during session 2 were highly positively correlated, $r(156) = .86, p < .001$, were averaged for a measure of basal cortisol level. For cortisol reactivity, the four cortisol samples collected during session 1 were modeled via linear and quadratic growth curves. All cortisol variables were not normally distributed

and thus were log-transformed prior to analysis.

Emotion regulation. As in Chapter 2, participants reported trait forms of emotion regulation and coping. For this study, emotion regulation represents one's general regulation of both positive and negative emotions, while coping is considered to be a problem-specific response to a stressful or otherwise negative situation, which is why the questionnaires are distributed among categories as follows (John & Gross, 2007).

Participants completed the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003), a well-established measure of trait emotion regulation strategies. The ERQ assesses both cognitive reappraisal and emotion suppression strategies. The ERQ consists of six reappraisal questions and four suppression questions. Participants answered according to 7-point Likert scale ranging from Strongly Disagree to Strongly Agree and answers were averaged into composite measures of Reappraisal and Suppression. Reliability was acceptable for both the reappraisal (Cronbach's alpha = .71) and suppression (Cronbach's alpha = .68) subscales. Three PI youth had incomplete ERQ data and were excluded from analyses examining emotion regulation strategies.

Coping. To measure coping strategies, participants completed the peer stressors version of the Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000), a measure of how often an individual responds in various ways to a specific type of stressor. The 57 items in the questionnaire ask how often the participant does a given response when faced with "Problems getting along with other kids" (4-point Likert scale ranging from Never to Almost Always). The RSQ contains three primary subscales:

volitional engagement coping, volitional disengagement coping, and involuntary stress responses. To control for differences in the base-rate endorsement of responses to stress, proportion scores were calculated as the total score for each factor divided by the total score on the RSQ (Compas, et al., 2001). Scores for each subscale ranged from 0 to 1; higher scores reflect higher levels of each type of response to stress. Reliability was acceptable for all three subscales: Engagement Coping [Cronbach's alpha = .84], Disengagement Coping [Cronbach's alpha = .77], and Involuntary Responses [Cronbach's alpha = .92]. Five NA youth and five PI youth had incomplete RSQ data and were excluded from analyses testing coping strategies.

Statistical Analysis

Preliminary analyses were conducted to identify outliers in cortisol concentrations, check normality of distributions, and calculate internal consistency of questionnaire measures. A 2 (Group) by 2 (Age) by 2 (Gender) repeated-measures ANOVA with five ratings of stress appraisal was examined as a manipulation check for the TSST-C paradigm. Two 2 (Group) by 2 (Age) by 2 (Gender) MANOVAs with Emotion Regulation subscales and Coping subscales tested group, age, and gender differences and interactions in trait emotion regulation and coping strategies.

The cortisol response curve was analyzed via hierarchical linear modeling using SAS 9.3 PROC MIXED procedure (see Singer, 1998), a model that is ideal for examining change over time and maximizing statistical power. The Level 1 model represents individual change in the cortisol response as a function of linear and quadratic terms,

Time and Time². Time represents the cortisol increase in response to the stressor task (positive slope) and Time² models the decrease in cortisol following the stressor (negative slope). The Level 2 model represents between-subjects differences in the cortisol response based on several independent variables: Age, Gender, Group and continuous measures of emotion regulation (Reappraisal, Suppression) and coping (Engagement, Disengagement, Involuntary Responses) that were entered as level two predictors in separate models.

All two- and three-way interactions were included in the original models and non-significant interactions were removed from final models. Age, Gender, and Group were dummy coded with female, adolescents, and non-adopted as the reference groups, respectively. Time since wake-up to the laboratory session, speech preparation support condition, and medication code were entered as covariates in all cortisol analyses. Although all sessions were scheduled in the late afternoon to reduce effects of the diurnal cortisol rhythm on cortisol reactivity, time since wake-up varied widely across participants. Time since wake-up was included as a covariate on the intercept term, because cortisol set point varies with diurnal rhythm. Speech preparation condition (parent versus stranger support) was expected to impact cortisol response (see Hostinar, 2013) and was included as a covariate on intercept, linear, and quadratic terms. Medication code was included as a covariate on intercept, linear, and quadratic terms. The mixed model was fit using restricted maximum likelihood estimation (REML) and degrees of freedom were computed using the Kenward and Roger (1997) method. Type 3

F tests of fixed effects are reported in the text, and estimated parameters are reported in the tables. Figures depict observed data, not estimated values.

Results

Preliminary Analyses

As expected, a RM-ANOVA with stress appraisal ratings demonstrated that ratings of stress appraisal varied significantly across the five time points in session 1, $F(3.68, 552.42) = 192.77, p < .001$ (M Arrival = 1.87, M Speech Preparation = 2.71, M Speech = 3.48, M Math = 3.76, M Recovery = 1.49), indicating that the participants perceived the TSST-C to be stressful. With the between-subjects factors of Age, Gender, and Group in the, there was a main between-subjects effect of group, $F(1, 150) = 5.64, p < .05$. Across the session, PI youth ($M = 2.54, SD = .074$) reported lower levels of perceived stress than NA youth ($M = 2.78, SD = .072$). There was also a significant between-subjects age by sex interaction, $F(1, 150) = 7.24, p < .01$, for which male adolescents ($M = 2.37, SD = .67$) rated the session as less stressful than female adolescents ($M = 2.76, SD = .63$) and children (girls: $M = 2.683, SD = .60$; boys: $M = 2.82, SD = .68$).

Across all participants, the growth curve model demonstrated a significant linear [Time: $F(1, 414) = 4.67, p < .05$] and curvilinear [Time² $F(1,317) = 35.30, p < .0001$] cortisol response, indicating that the TSST-C elicited expected increases in cortisol level. Paired-samples t-tests demonstrated that cortisol levels were significantly different at each time point as expected, namely time 1 levels ($M = .14, SD = .11$) were lower than

time 2 levels ($M = .17$, $SD = .13$) [$t(158) = -1.99$, $p < .05$], time 2 levels were higher than time 3 levels ($M = .13$, $SD = .11$) [$t(158) = 8.43$, $p < .001$], and time 3 levels were higher than time 4 levels ($M = .10$, $SD = .06$) [$t(157) = 7.81$, $p < .001$].

Cortisol Response

Age and group effects. Descriptive statistics for cortisol samples in PI and NA groups are listed by age and gender in Table 8.

Table 8

Means (SD) of Cortisol ($\mu\text{g/dl}$) Variables for PI and NA Youth by Age and Gender

	Children		Adolescents	
	Males	Females	Males	Females
<u>Post-Institutionalized</u>				
Session 1				
Adaptation (S1, T1)	.10 (.04)	.15 (.15)	.20 (.12)	.13 (.12)
Reactivity (S1, T2)	.11 (.09)	.18 (.16)	.20 (.14)	.14 (.12)
Recovery 1 (S1, T3)	.08 (.05)	.13 (.13)	.16 (.11)	.12 (.11)
Recovery 2 (S1, T4)	.08 (.07)	.10 (.07)	.13 (.06)	.11 (.08)
Session 2				
Basal 1 (S2, T1)	.10 (.04)	.09 (.07)	.14 (.06)	.10 (.05)
Basal 2 (S2, T2)	.10 (.05)	.08 (.07)	.14 (.06)	.10 (.08)
<u>Non-Adopted</u>				
Session 1				
Adaptation (S1, T1)	.11 (.08)	.13 (.10)	.19 (.08)	.15 (.09)
Reactivity (S1, T2)	.10 (.08)	.18 (.15)	.23 (.12)	.20 (.15)
Recovery 1 (S1, T3)	.09 (.05)	.14 (.13)	.16 (.07)	.14 (.13)
Recovery 2 (S1, T4)	.07 (.05)	.10 (.07)	.12 (.04)	.10 (.06)

Session 2

Basal 1 (S2, T1)	.09 (.05)	.10 (.08)	.18 (.08)	.12 (.07)
Basal 2 (S2, T2)	.09 (.06)	.09 (.06)	.17 (.07)	.12 (.08)

Note: For descriptive purposes the non-transformed values are shown.

First, a 2 (Group) by 2 (Age) by 2 (Gender) ANOVA with mean session 2 cortisol was conducted to test age and group differences in basal cortisol level. As expected, adolescents had higher basal cortisol levels than children, $F(1, 147) = 17.51, p < .001$ (M adolescents = .27, $SD = .14$; M children = .18, $SD = .12$). There was also a main effect of gender, with higher basal cortisol in males compared to females, $F(1, 147) = 9.45, p < .01$ (M males = .13, $SD = .07$; M children = .10, $SD = .07$). Early experience groups did not differ in basal cortisol level, $F(1, 147) = 1.62, ns$ (M PI = .21, $SD = .12$; M NA = .24, $SD = .15$). In the growth curve model, there was an interaction of age and gender on the intercept, $F(1, 230) = 7.91, p < .01$, with higher intercept cortisol levels in male adolescents compared to adolescent females and children (see Table 9).

To examine age and group differences in cortisol reactivity, cortisol was modeled via HLM as a function of Time and Time², with Age, Gender, Group, and their interactions as between-subjects factors. As in Chapter 2, there was a significant age by gender interaction on the quadratic term, Time² $F(1, 462) = 4.08, p < .05$, and a trend level effect on the linear term, Time $F(1, 462) = 3.00, p = .08$ (see Table 9). Male children did not demonstrate a significant cortisol response to the stressor. For early experience groups, there were trend level effects on the linear, Time $F(1, 462) = 3.09, p =$

.079, and quadratic terms, $\text{Time}^2 F(1, 462) = 3.12, p = .078$ (see Table 9). PI youth tended to show a blunted cortisol response, characterized by a less peaked response to the TSST-C (see Figure 4). This group trend did not interact with age or gender.

Table 9

Parameter Estimates for Growth Curve Model of Cortisol Reactivity with Age, Gender, and Group

Effect	Estimate	SE	Df	<i>t</i> value	<i>p</i>
Intercept	.71	.11	168	6.64	<.0001*
Time	.07	.05	458	1.32	.12
Time ²	-.04	.02	458	-2.80	.003*
Age	-.02	.07	227	-1.00	.69
Age x Time	.06	.06	458	1.02	.22
Age x Time ²	-.02	.06	458	-0.81	.20
Gender	.16	.07	227	1.84	.01*
Gender x Time	.01	.06	458	0.05	.91
Gender x Time ²	-.01	.02	458	-0.14	.66
Age x Gender	-.24	.09	227	-2.74	.005*
Age x Gender x Time	-.12	.07	458	-1.73	.08
Age x Gender x Time ²	.05	.02	458	1.99	.04*
Group	.002	.07	227	-0.87	.97
Group x Time	-.07	.06	458	-1.07	.08
Group x Time ²	.02	.02	458	1.49	.08

* $p < 0.05$

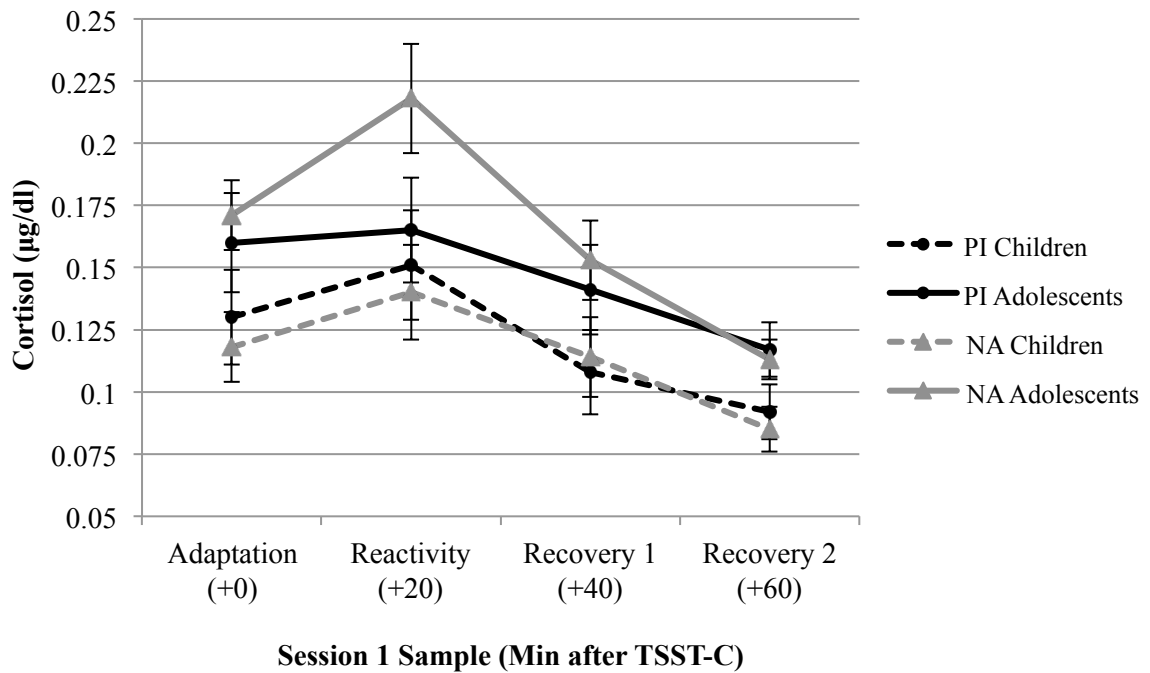


Figure 4. Mean cortisol response to the TSST-C for children and adolescents, split by early experience group. Error bars represent \pm SEM.

Severity of deprivation. To further examine the impact of early stress on cortisol reactivity, variables that index severity of early life deprivation within the PI group were tested as predictors of the cortisol growth curve. Age at adoption, duration of institutionalization, and physical growth stunting did not predict cortisol reactivity to the TSST-C.

Cognitive-Affective Strategies

Descriptive statistics for emotion regulation and coping variables in PI and NA youth are listed by age and gender in Table 10.

Table 10

Means (SD) of Cognitive-Affective Strategies for PI and NA Youth by Age and Gender

	Children		Adolescents	
	Males	Females	Males	Females
<u>Post-institutionalized</u>				
Emotion Regulation				
Reappraisal	4.85 (1.13)	5.04 (.79)	4.26 (1.17)	4.97 (.71)
Suppression	4.32 (.87)	3.38 (1.00)	2.97 (1.31)	3.39 (1.24)
Coping				
Engagement	.37 (.05)	.39 (.06)	.40 (.07)	.38 (.05)
Disengagement	.22 (.03)	.23 (.03)	.20 (.03)	.21 (.03)
Involuntary Responses	.41 (.05)	.38 (.05)	.39 (.07)	.41 (.05)
<u>Non-Adopted</u>				
Emotion Regulation				
Reappraisal	4.80 (1.01)	4.80 (1.03)	4.98 (.85)	4.89 (.91)
Suppression	4.14 (1.37)	3.75 (.93)	3.62 (.98)	3.35 (1.01)
Coping				
Engagement	.36 (.05)	.37 (.05)	.40 (.06)	.40 (.07)
Disengagement	.23 (.03)	.22 (.03)	.20 (.03)	.21 (.02)
Involuntary Responses	.42 (.06)	.41 (.06)	.40 (.05)	.38 (.06)

Emotion regulation. A 2 (Group) by 2 (Age) by 2 (Gender) MANOVA with Reappraisal and Suppression as dependent variables revealed a significant multivariate effect for age, Wilks' $\lambda = .93$, $F(2, 147) = 5.17$, $p < .05$. Univariate tests identified a main effect of age on Suppression, $F(1, 148) = 10.08$, $p < .01$, in which children ($M = 3.89$, SD

= 1.11) reported higher levels of trait suppression than adolescents ($M = 3.35$, $SD = 1.14$). Reported reappraisal did not differ by age or gender, and there were no main effects or interactions with group for either emotion regulation strategy.

Coping. A 2 (Group) by 2 (Age) by 2 (Gender) MANOVA with Engagement, Disengagement, and Involuntary Responses as dependent variables found a significant multivariate effect for age, Wilks' $\lambda = .88$, $F(2, 140) = 9.50$, $p < .001$. Univariate tests revealed that for engagement coping there was a main effect of age, $F(1, 141) = 8.46$, $p < .01$, with higher levels of engagement in adolescents ($M = .40$, $SD = .06$) compared to children ($M = .37$, $SD = .05$). For disengagement coping, there was a main effect of age, $F(1, 141) = 14.07$, $p < .001$, in which children ($M = .22$, $SD = .03$) reported higher levels of disengagement than adolescents ($M = .21$, $SD = .03$). No main effects or interactions with group or gender were observed for either engagement or disengagement coping. Reports of involuntary responses did not differ by age, gender, or group (see Table 10).

Associations Between Cognitive-Affective Strategies and Cortisol Reactivity

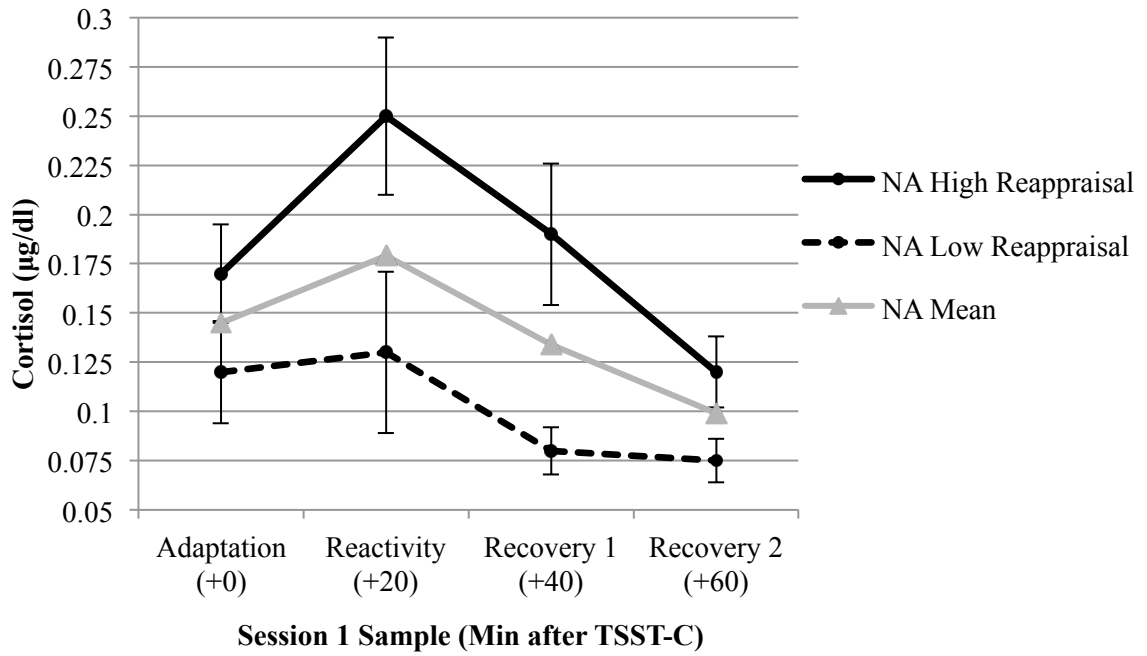
Emotion regulation. The growth curve model with Reappraisal, Age, Gender, and Group revealed a significant reappraisal by group interaction on the linear, Time $F(1, 452) = 4.93$, $p < .05$, and quadratic terms, Time² $F(1, 452) = 4.79$, $p < .05$ (see Table 11). Greater trait reappraisal predicted a more peaked cortisol response to the TSST-C in NA youth but was not related to cortisol reactivity in PI youth (see Figure 5). There was also a trend level reappraisal by group interaction on the intercept, $F(1, 228) = 3.78$, $p = .053$; higher reappraisal predicted higher intercept cortisol level in the NA group only.

Table 11

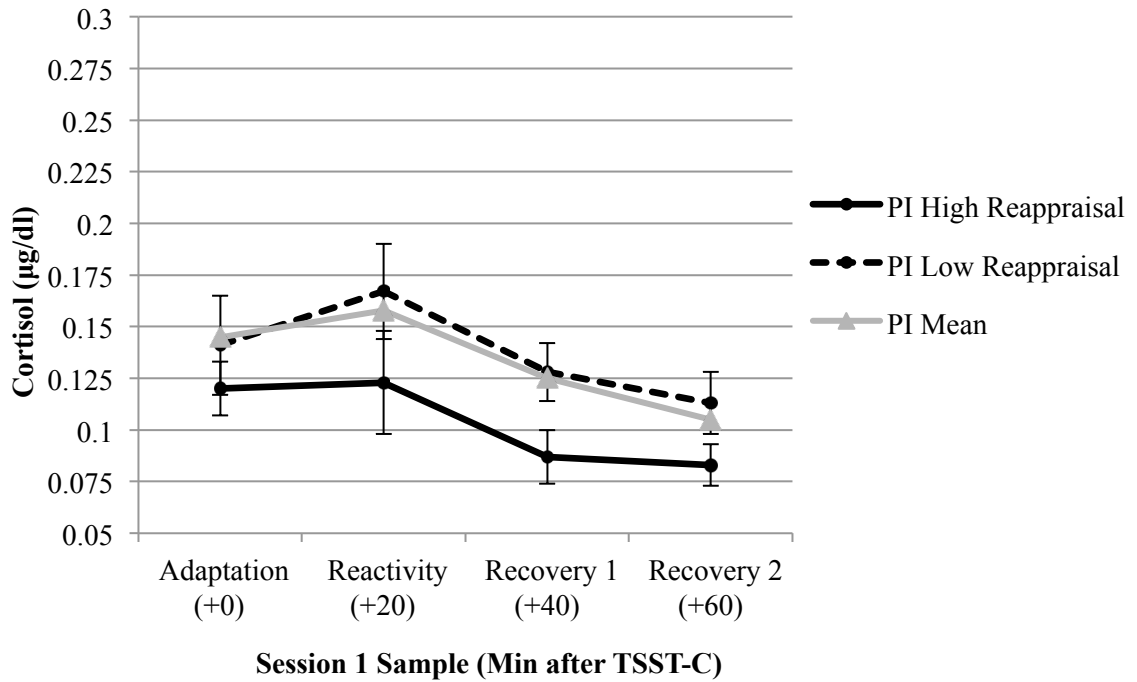
Parameter Estimates for Growth Curve Model of Cortisol Reactivity with Age, Gender, Group, and Reappraisal

Effect	Estimate	SE	df	<i>t</i> value	<i>p</i>
Intercept	.70	.10	163	6.76	<.0001*
Time	.07	.04	452	1.69	.09
Time ²	-.04	.01	452	-3.11	.002*
Age	-.02	.06	228	-0.27	.79
Age x Time	.05	.05	452	1.07	.28
Age x Time ²	-.02	.02	452	-1.15	.25
Gender	.15	.06	228	2.49	.01*
Gender x Time	-.005	.05	452	-0.10	.92
Gender x Time ²	-.004	.02	452	-0.28	.78
Age x Gender	-.25	.08	228	-2.95	.004*
Age x Gender x Time	-.11	.07	452	-1.49	.14
Age x Gender x Time ²	.04	.02	452	1.81	.07
Group	-.001	.04	228	-0.03	.98
Group x Time	-.07	.04	452	-1.99	.05*
Group x Time ²	.02	.01	452	2.01	.04*
Reappraisal	.07	.03	228	2.35	.02*
Reappraisal x Time	.06	.03	452	2.43	.02*
Reappraisal x Time ²	-.02	.01	452	-2.74	.01*
Reappraisal x Group	-.10	.04	228	-1.94	.05*
Reappraisal x Group x Time	-.09	.04	452	-2.22	.03*
Reappraisal x Group x Time ²	.03	.01	452	2.19	.03*

* $p < 0.05$



A)



B)

Figure 5. Mean cortisol response to the TSST-C for A) PI and B) NA youth high in reappraisal versus youth low in reappraisal. High and low reappraisal are depicted here as 1 SD above and below the mean, respectively; however, reappraisal was analyzed as a continuous variable. Error bars represent \pm SEM.

Suppression did not predict cortisol reactivity to the TSST-C as measured by linear or quadratic terms (see Table 12). There was a suppression by age interaction on the intercept, $F(1, 217) = 4.56, p < .05$. Suppression predicted higher intercept cortisol level in adolescents and was not related to intercept cortisol in children.

Coping. None of the RSQ coping subscales (i.e., Engagement, Disengagement, Involuntary Responses) predicted cortisol reactivity and there were no interactions with age, gender, or group (see Table 12).

Table 12

Type 3 Tests of Fixed Effects for Suppression and Coping Predictors of Cortisol Reactivity

Effect	Type 3 Test of Fixed Effects
Suppression	$F(1, 217) = 0.79, p = .38$
Suppression x Time	$F(1, 449) = 0.99, p = .32$
Suppression x Time ²	$F(1, 449) = 1.19, p = .28$
Engagement	$F(1, 205) = 0.10, p = .75$
Engagement x Time	$F(1, 429) = 0.21, p = .65$
Engagement x Time ²	$F(1, 429) = 0.15, p = .70$
Disengagement	$F(1, 204) = 0.30, p = .59$

Disengagement x Time	$F(1, 429) = 0.20, p = .65$
Disengagement x Time ²	$F(1, 429) = 0.01, p = .94$
Involuntary Responses	$F(1, 206) = 0.01, p = .94$
Involuntary Responses x Time	$F(1, 429) = 0.06, p = .81$
Involuntary Responses x Time ²	$F(1, 429) = 0.14, p = .70$

Note: Suppression, Engagement, Disengagement, and Involuntary Responses were tested in separate HLM growth curve models with Age, Gender, and Group in each model (as depicted for Reappraisal in Table 11). The statistics reported here are the Type 3 Test of Fixed Effects from each separate growth curve model.

Discussion

The goal of the current study was to extend the normative developmental findings described in Chapter 2 by examining the impact of early life stress on cortisol reactivity and related cognitive-affective regulatory strategies in late childhood and adolescence. Across age and early experience groups, the TSST-C elicited an increase in both perceived stress and cortisol levels. In both groups, adolescents had higher basal cortisol compared to children and groups did not differ in basal cortisol level. Groups did tend to differ in cortisol reactivity to the TSST-C. At a trend level of significance, PI participants tended to demonstrate low cortisol reactivity to the TSST-C compared to NA youth, characterized by a less peaked response to the stressor. Early experience groups did not differ in trait emotion regulation or use of coping strategies. Children reported higher levels of trait suppression and disengagement coping, while adolescents reported higher levels of engagement coping. These developmental effects did not differ by group. Reappraisal and involuntary responses did not differ by age or group. Regarding the third

and primary aim, trait cognitive reappraisal predicted cortisol reactivity in the NA group but was not related to cortisol reactivity in PI youth. As in Chapter 2, suppression and coping types were not related to cortisol reactivity.

The current results regarding group differences in cortisol reactivity support previous research demonstrating that early life stress could have long-term effects on HPA axis activity. However, these findings do not provide support for the hypothesis that the pubertal transition will impact the effect of early life stress on stress reactivity, either negatively or positively. PI participants tended to demonstrate lower cortisol reactivity both before and after the pubertal transition. This pattern of hypo-reactivity is in line with stress resilience models and results from studies of psychiatrically healthy individuals who experienced early adversity (e.g., Carpenter et al., 2007; Cicchetti & Rogosch, 2001; Elzinga et al., 2008; Heim et al., 2001, 2002). In Gunnar and colleagues' (2009) study, hypo-reactivity was observed among the children who experienced mild to moderate early adversity prior to adoption. Notably, when excluding participants were taking psychotropic medications (all of whom had a psychiatric diagnosis), the groups did not differ in cortisol reactivity. Previous research would predict the opposite effect, as individuals who have experiences early adversity and who have psychological disorders are more likely to demonstrate hyper-reactivity of stress systems (e.g., Heim et al., 2002). Importantly, because psychotropic medications may interfere with cortisol physiology, it is difficult to separate the influence of psychiatric health versus the biological effect of the medication.

Regarding the stress-inoculation hypothesis, individual child factors associated with severity of deprivation did not predict cortisol responses. Because the PI group was recruited to represent moderate to severe preadoptive stress (adopted after 11 months of age having spent the majority of preadoptive life in institutional care), the range of severity of deprivation was likely too narrow to detect this effect. To best examine the stress-inoculation hypothesis, groups should be specifically recruited to represent a range of severity in early life stress (see Gunnar et al., 2009a).

This is the first study to assess cortisol reactivity in PI adolescents and therefore, provides the first evidence that early deprivation in the form of institutional rearing might impact HPA axis activity beyond the pubertal transition. Some studies that have found HPA axis hypo-reactivity following chronic stress have interpreted as this pattern as a reflection of vulnerability and dysregulation of the HPA axis (e.g., Carpenter et al., 2007), while others suggest that lower cortisol reactivity might be a marker of resilience and adaptation (Gunnar et al., 2009a). Future research should include assessments of behavior and emotional problems and mental and physical health to better understand how low cortisol reactivity is related to vulnerability or resilience in other domains of self-regulation and well-being.

Contrary to predictions, PI and NA youth did not differ in self-reported trait emotion regulation or use of coping strategies. These results suggest that although PI youth may show deficits or delays in emotion understanding and regulation (e.g., Fries & Pollak, 2004; Tottenham et al., 2010), the strategies that they report using to manage their

emotions and stress responses are not different from their non-adopted peers. One explanation for this pattern of results could be that PI youth are less successful at using their emotion regulation and coping strategies to regulate their behavior. Alternatively, the same cognitive-affective strategies might not be most effective in regulating emotion and stress in youth who have experienced high stress versus low stress environments. Along this line, children and adolescents develop emotion regulation and coping strategies through socialization from parents and family. For PI youth, the socialization of these strategies within the context of the adoptive home may not match the emotion and behavioral systems developed early within the context of institutional care. On the other hand, PI children and adolescents in the current study may have demonstrated emotion regulation and emotion understanding skills similar to their non-adopted peers. Future studies will need to explore these associations further by including behavioral assessments of emotion regulation or understanding along with measures of trait emotion regulation and coping strategies.

Moving to the primary goal of the study, reappraisal of emotion predicted cortisol reactivity to the TSST-C in NA youth, but was unrelated to cortisol reactivity in PI youth. This does not support the hypothesis that PI adolescents would show typical patterns (i.e., similar to NA adolescents) between cognitive-affective strategies and cortisol reactivity. Instead, in both PI children and PI adolescents, no cognitive-affective strategies were related to cortisol reactivity. One explanation for this finding could be that due to blunted cortisol reactivity in PI youth, there was not enough variability in the cortisol curve to

detect significant associations with cognitive-affective strategies. However, despite lower reactivity on average, there was a good amount of variability in cortisol reactivity within the PI group. Alternatively, it was hypothesized that PI youth would have lower levels of trait reappraisal, which may affect the relationship between this emotion regulation strategy and cortisol reactivity in the PI group. PI and NA youth did not differ in level of trait reappraisal. However, trait reappraisal was measured via self-report; it is possible that the PI and NA youth interpreted the questions differently (note: Cronbach's alphas for the Reappraisal subscale of the ERQ were acceptable in each group).

Finally, it is critical to remember that the HPA axis works in concert with limbic and cortisol regions, creating a “neuro-symphony of stress” (Joëls & Baram, 2009) and chronic stress impacts the development of cortico-limbic-HPA networks. Several studies demonstrate detrimental effects of chronic stress on cortical and limbic regions that influence both higher-order cognitive functioning as well as regulatory connections with stress systems (Arnsten, 2009; Ulrich-Lai & Herman, 2009). Due to documented effects of stress on the development of cortico-limbic networks, it is not surprising that the relationship between cognitive-affective strategies and cortisol reactivity would be different among PI compared to NA youth years after adoption.

Future research is needed to understand both the relationship between reappraisal and cortisol reactivity in typically developing youth and the lack of association between these variables in PI youth. Years after adoption in supportive homes, PI children show a range of developmental outcomes, from vulnerability and risk to adaptation and

resilience. To date, research with this population has not clearly identified the role that severity of deprivation and individual difference factors play in predicting long-term developmental outcomes. The current results favor individual differences over severity of deprivation, as severity of deprivation did not predict cortisol responses. This study examined cognitive-affective strategies as potential individual difference factors that might predict stress responses. However, cognitive-affective strategies were not related to cortisol reactivity in the PI group. Future research focused on identifying other individual difference factors that could predict cortisol reactivity in PI youth is needed to better understand the observed pattern of possible hypo-reactivity of the HPA axis and vulnerability versus resilience in biological regulatory systems more generally.

In addition to the limitations discussed in Chapter 2, the current study has several limitations. First, although PI youth provide a natural experiment for early life stress that is circumscribed to a specific length of time, it is difficult to gain information about the type and severity of deprivation within the institutional setting. Additionally, it is very likely that PI youth experience increased prenatal risks compared to NA youth. With the current design, we are unable to separate the impact of prenatal and postnatal risks that contribute to experiences of early life stress. These limitations could be addressed via a logistically challenging prospective design that examined children in the institutional setting and followed their development post-adoption.

Second, by sampling age groups that fall before and after the pubertal transition, it was not possible to directly test the impact of pubertal development on outcome

measures. Specifically, pubertal recalibration models posit that pubertal hormones drive stress systems to recalibrate to the conditions of the current environment. Because puberty and age are confounded in the current design, the impact of pubertal hormones cannot be tested. Despite this limitation, the current study was able to adequately test developmental changes associated with the transition to adolescence. Future research should directly test the pubertal recalibration hypothesis by implementing a longitudinal study that examines reactivity and regulation within stress and emotion systems among PI youth before, during, and following the pubertal transition.

Third, interpretation of the current results is impaired by the presence of medications in the PI sample. Because both medications and psychological functioning impact activity of the HPA axis, it is difficult to tease apart these effects in PI youth who have psychiatric diagnoses and who are taking psychotropic medications. However, excluding all individuals who are taking psychotropic medications would greatly bias the sample, as this would in turn exclude individuals who have emotional and behavioral problems. The current findings are also limited by the inclusion of the social support manipulation. Although the social support condition was included as a covariate in all analyses, the current study should be replicated with a larger sample of children and adolescents who will all complete the traditional TSST-C (stranger support).

Fourth, the findings are limited to PI children and adolescents and cannot be generalized to other populations who have experienced early life stress. PI youth are a unique population, because they experience chronic, multifaceted stress as infants and

young children, then are adopted into highly resourced, highly stable homes. This experience of childhood stress is very different from experiences of poverty, maltreatment, abuse, or trauma, and it is therefore expected that developmental patterns will be unique to this group. Lastly, this study would be strengthened by the inclusion of other measures of self-regulation and well-being. The findings highlighted potential hypo-reactivity of the HPA axis in PI children and adolescents, but without additional measures it is difficult to interpret whether this trend toward hypo-reactivity is indicative of stress resilience or vulnerability.

The present study makes several contributions to further the understanding of how early life stress impacts the long-term development of regulatory systems. This is the first study to examine the associations between trait cognitive-affective strategies and stress reactivity in a sample of children and adolescents who experienced early life stress. Further, this research provides new evidence regarding what strategies youth use to regulate emotion and stress reactivity before and after the pubertal transition, and was designed to directly compare these regulatory and reactive process in typically developing youth and youth who experienced early life stress. This study builds on previous research in post-institutionalized youth that focused either on stress reactivity or on emotion regulation and in one age group.

In summary, the results demonstrate a tendency for PI youth to have a hypo-reactive response to the TSST-C. Although PI and NA youth did not differ in self-reported trait emotion regulation or coping strategies, cognitive reappraisal of emotion

did not predict cortisol reactivity within the PI group. These findings provide a glimpse into the complexity of the self-regulatory system before and after the pubertal transition. Future research is needed to examine individual factors that predict physiological reactivity in NA and PI youth, with an appreciation for the role of early life stress on the development of these regulatory systems. The current results have implications for the development of prevention and intervention efforts aimed at promoting self-regulation during the transition to adolescence. Specifically, these results highlight the importance of considering the impact of early life experiences on both reactivity and regulatory processes through late childhood and mid-adolescence.

Chapter 4: Conclusions and Future Directions

The organization of an adaptive internal regulatory system for responding to stress is a critical developmental task. However, research regarding the developmental processes involved in establishing this system, particularly during middle childhood and adolescence, is lacking. Emotion regulation and coping involve cognitive-affective neural networks that are rapidly developing during adolescence (Goldin et al., 2008; Ochsner & Gross, 2008). This developmental window offers the potential for continued neurobiological plasticity and sensitivity to intervention or training of these processes (Giedd, 2004; Spear, 2000).

This dissertation examined cognitive-affective strategies and early life experiences as predictors of physiological reactivity to a social stressor before and after the pubertal transition. Chapter 2 demonstrated that individual differences in how typically developing youth regulate their emotions predict psychosocial stress reactivity. Specifically, across age and gender, higher trait levels of cognitive reappraisal of emotions predicted higher cortisol reactivity. Chapter 3 examined the impact of early life stress and cognitive-affective strategies on cortisol reactivity. Overall, PI youth tended to demonstrate blunted cortisol reactivity to the stressor. PI and NA youth did not differ in self-reported emotion regulation and coping strategies. While cognitive reappraisal predicted increased cortisol reactivity in NA youth, no cognitive-affective strategies were associated with cortisol reactivity to the TSST-C in PI participants.

Overall, these results highlight the importance of examining relations among cognitive, emotional, and physiological processes, as well as early life experiences, to better understand the development of self-regulatory systems during the transition to adolescence. In addition to the need for future research that will strengthen the current study, as described in the discussion sections of Chapters 2 and 3, several important lines of research will stem from this research question. First, the current study and the vast majority of empirical studies in this area to date do not sufficiently address issues regarding directionality of the interactions among prefrontal, limbic, and HPA systems. Of course, these networks involve transactional effects creating a system of “top-down” and “bottom-up” regulatory and reactive effects. Questions regarding the directionality of signals across regulatory systems will be particularly critical for modeling how these systems develop over time. Future research should combine multiple levels of analysis for cognitive, affective, and physiological processes involved in stress reactivity, and include trait and state measures in combination with experimental manipulations of both regulatory and reactivity processes. Based on the current results, the impact of experimentally manipulated cognitive reappraisal on cortisol reactivity could be examined against the backdrop of individual differences in trait and state strategies in children and adolescents. The inclusion of additional behavioral (e.g., emotion regulation and executive function tasks) and neurobiological (e.g., autonomic reactivity or neuroimaging) measures would help clarify the processes underlying the observed association between cognitive reappraisal and cortisol reactivity. Ideally, this study would

implement a longitudinal design that followed participants before, during, and after the pubertal transition.

Along those lines, although the current study focused on the HPA axis, future research should integrate multiple physiological systems to best understand regulatory systems (for examples see Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011; Gunnar et al., 2009b; Stroud et al., 2009). The gold standard for the examination of individual differences in psychobiological stress responses has called for multiple biological processes to be measured in tandem (e.g., Bauer, Quas, & Boyce, 2002; Donzella, Gunnar, Krueger, & Alwin, 2000). The examination of interactions across biological measures will be particularly important for understanding how these develop as an integrated system across the life span.

Next, the socialization of emotion and the experience of positive social support are important components of youths' development of adaptive self-regulation (Gottman, Katz, & Hooven, 1996). Research indicates that parents' positive responsivity to their child's emotions and stress contributes to improved emotion and stress regulation abilities of their child (e.g., Abaied & Rudolph, 2011; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Ramsden & Hubbard, 2002). Further, supportive social behaviors attenuate stress-related activity in neural networks associated with fear and threat, the autonomic nervous system, and the HPA axis (e.g., Coan, Schaefer, & Davidson, 2006; DeVries, Glasper, & Detillion, 2003; Kirschbaum, Klauer, Filipp, Hellhammer, 1995). Although the socialization of emotion and social support are important components of

youth's development of the cognitive-affective skills involved in regulation of stress reactivity, research has only begun to elucidate these socialization processes during adolescence. Parents' socialization of coping provides cognitive, affective, and behavioral strategies that reinforce or redirect youths' coping behaviors, and this monitoring is particularly beneficial when youths' regulatory resources are strained, such as during the challenging transition to adolescence (Abaied and Rudolph, 2010; 2011). For example, mothers' disengagement and engagement coping suggestions predict youths' maladaptive and adaptive responses to peer stress, respectively, and these effects are strongest among adolescents who report experiencing high levels of peer stress (Abaied & Rudolph, 2011). Future research should examine the relationship between parental emotion socialization and children and adolescents' 1) cognitive-affective strategy use and 2) cortisol reactivity to a social stressor. Additionally, research is needed to examine what types of socialization behaviors are most effective for children versus adolescents and for different types of stressors.

The long-term goal of this line of research is to inform the development of interventions that promote adaptive self-regulation during childhood and adolescence. Although more research is needed to fully understand the implications of these results, these findings provide a first glimpse into how cognitive-affective regulatory strategies and physiological reactivity are related in late childhood and adolescence. In addition to current regulatory and reactivity patterns, individual differences in early life experiences appear to impact the development of reactivity and regulatory systems during this period.

Although the current results do not provide direct evidence for puberty as a sensitive period for either vulnerability or opportunity, this line of research might inform the development of interventions that promote adaptive functioning before the pubertal transition and potentially take advantage of the pubertal transition as a window of opportunity for positive developmental change. Changes in physiological and neurobehavioral function in adolescence may make individuals more susceptible to perturbations (Andersen, 2003; Dahl, 2004), but may also indicate an opportunity to intervene and promote positive adaptation (Romeo, 2010a).

Cognitive-affective regulatory strategies include a set of relatively straightforward targets for intervention. The opportunity to train regulatory systems during adolescence highlights the need for empirical identification of individual difference factors that promote adaptive and maladaptive responses across regulatory domains, types of stressors, and contexts of challenge. And lastly, prevention and intervention efforts should also target teaching parents, educators, and other social support figures effective means to provide emotional support and guidance during children's normative experiences of stress and challenge in middle childhood and adolescence (Abaied & Rudolph, 2011; Klimes-Dougan et al., 2007).

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