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CORTISOL, DAILY HASSLES AND OVERWEIGHT STATUS IN ADOLESCENCE

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ABSTRACT

Obesity may be one of the most pervasive current challenges to the health of adolescents. Obesity has diverse etiologies, but there are critical periods of life at which time weight status may be affected by experiences, and the individuals responses to their experiences. This study examined the associations between stressors, in the form of daily hassles, response to stressors (specifically cortisol secretion) and body mass index in adolescents. A sample of male (n=36) and female adolescents (n=42) and a parent or care-giver of each adolescent participated in the study. The adolescent participants collected saliva for cortisol assay and completed daily experiences diaries for four consecutive days. Participants also completed a laboratory stress test, and completed measures of psychosocial function. Three hierarchical linear regression models were evaluated in which cortisol reactivity to a stressor, childhood vulnerabilities, specifically depression and high cortisol reactivity, exercise and daily hassles were hypothesized to predict adolescent body mass index. The stability of daily cortisol profiles from childhood to adolescence, and across consecutive days in adolescence were evaluated using trajectory analyses, and the associations between daily hassles, total daily cortisol secretion, and cortisol reactivity were evaluated using HLM procedures. The results indicated that daily hassles, cortisol reactivity and symptoms of depression in childhood were not associated

with adolescent weight status after controlling for childhood weight status. Habitual exercise was associated with weight status in both male and female adolescents. Daily cortisol trajectories were stable across consecutive days in adolescence, but were not stable from childhood to adolescence. The results of the HLM analyses indicated that hours of sleep and daily hassles were associated with total daily cortisol secretion, but the strength of this association was not predicted by the magnitude of cortisol response to a laboratory stress challenge. These results demonstrate the impact of daily experiences in adolescence on levels of cortisol, and illustrate the change in daily cortisol secretion patterns with age and exposure to daily hassles. In summary, this study provides evidence that studying the associations between daily hassles and cortisol reactivity to a lab stressor in adolescence may have utility in expanding models of stress and weight status.

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Chapter 1

Introduction

Obesity may be one of the most pervasive challenges to the health of adolescents, and obesity has serious implications for an individual's current health status, and also has ramifications for the future well-being of the person. Obesity has diverse etiologies, but there are critical periods of life at which time weight status may be affected by experiences, and the individual's responses to their experiences. Trajectories to obesity may be established in childhood but these trajectories may be deflected upward at adolescence by behaviors, hormones and daily events experienced against a background of family and social contexts. The period of adolescence is aligned with developmental transitions that may pose a significant risk for a deflection upward in a trajectory of obesity given preexisting risks and the rapid and dramatic physical growth associated with this period and hormonal and social-contextual changes that adolescents experience.

This project examined the associations between stressors, in the form of daily hassles, responses to stressors (specifically cortisol secretion) and weight status in adolescents. Explicit in the theoretical model (Figure 2.2) is that stress is positively associated with cortisol levels, and then, in turn, cortisol levels, in

combination with behavioral change, are predictive of weight status. This model is adapted from the conceptual model of allostatic load, wherein repeated perturbations to cortisol secretion, as part of the stress response, are predictive of changes in physiological parameters, including weight status. The results of this study relate the existing bodies of research on the relationship between daily hassles and cortisol, and the relationship between cortisol and weight status in adolescence. The research design allows for the testing of hypotheses relating associations between hassles and cortisol secretion during adolescence, the associations between biobehavioral responses to stress and weight status, and the stability of, and influences on, daily cortisol secretion.

Relevance

The transition between childhood and adolescence is marked by developmental changes that may influence weight status via several mechanisms, including the adoption of certain health behaviors. Trajectories of weight status are influenced by voluntary behaviors, such as eating patterns and physical activity, and also by genetic predisposition and repeated perturbations to physiological functioning, such as those of the stress response. Thus, to understand specific health outcomes, it is vital that theory driven multilevel models of childhood weight status are developed that incorporate behavioral,

biological and contextual factors (Susman, Dorn, & Schiefelbein, 2003; Susman, Reiter, Ford, & Dorn, 2002).

The problem of childhood overweight is one in which multilevel theories are especially warranted. The prevalence of childhood overweight status has almost tripled over the past two decades, and the Youth Risk Behavior Survey (YRBS, 2003) indicated that 42% of high school students did not participate in physical activity, and that only 22% had eaten the recommended number of fruits and vegetables in the previous week. The trajectories of weight status that are established in childhood are vulnerabilities for later obesity, and are proposed to be influenced by behaviors, hormones and daily experiences, within the context of the family and social environment. The degree to which a child is responsive to daily challenges is influenced by individual psychological attributes, such as trait anxiety or depression, and the physiological response to challenge has been linked to health behaviors and outcomes, including higher BMI (Epel et al., 2004; Epel et al., 200a; McEwen 1998b). Very little research has focused on understanding how physiological responses to experiences, especially hassles of daily life, indexed by cortisol secretion, interact with psychosocial attributes and contextual factors to influence weight status in early life.

The person-centered and holistic interactionism perspectives elucidated in the work of Magnusson (1996) consider the individual as a part of a dynamic person-environment system, affirming the interactions between the multiple levels of the person, within the larger social and cultural context (Cairns, Costello, & Elder, 1996; Cairns, Elder, & Costello, 1997; Magnusson & Stattin, 1998; Susman, 2001). This interdisciplinary approach is guided by the discipline of developmental science that affirms the integration of perspectives from many fields to examine individual change (Cairns et al., 1996; Cairns et al., 1997; Magnusson & Stattin, 1998; Rutter, 1989; Susman, 2001).

Specific to the current study, weight status, especially as associated with responses to stress experiences, can be considered using a variant of the allostatic load paradigm. This model provides an approach with which to examine the multiple levels on which experiences and the associated biobehavioral responses influence weight status. Many theorists and studies have used the allostatic load construct as a model to describe and test the relationships between responses to environmental, psychological or social challenge and biological markers of health risk, including weight status (Bjorntorp, 2001; Bjorntorp & Rosmond, 2000b; Braet & Van Strien, 1997; Cartwright et al., 2003b; Dallman et al., 2004; Dimitriou, Maser-Gluth, & Remer, 2003; Drapeau, Therrien, Richard, & Tremblay, 2003; Epel et al., 2000a; Halbreich, 1976; Kendall-Tackett, 2002; Mastorakos & Zapanti, 2004; McEwen, 2001, 2004;

McEwen & Seeman, 1999; Mota et al., 2004; Pasquali & Vicennati, 2000; Roemmich, Gurgol, & Epstein, 2003; Rosmond & Bjorntorp, 2000b; Rosmond, Dallman, & Bjorntorp, 1998; Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Thus the interactions between daily hassles, biological and behavioral responses to these, and weight status can be examined from the perspective of cumulative risk, a concept inherent in the allostatic load model.

Studies of stress-associated eating behavior have suggested there are moderators and mediators of the association, including sex (Grunberg & Straub, 1992; Klein, Faraday, Quigley, & Grunberg, 2004), perceived control over the stressor, and efforts at control over eating (Cools, Schotte, & McNally, 1992; Haynes, Lee, & Yeomans, 2003; Heatherton, Polivy, Herman, & Baumeister, 1993; Klein et al., 2004; Polivy, Heatherton, & Herman, 1988; Schotte, Cools, & McNally, 1990). Positive associations between stress and dis-inhibited eating have been reported in both laboratory and naturalistic settings (Crowther, Sanftner, Bonifazi, & Shepherd, 2001; Roemmich, Wright, & Epstein, 2002; Wardle, Steptoe, Oliver, & Lipsey, 2000) and the effects of stress on eating behaviors in younger populations indicate these patterns may be established early in life. Adolescents with higher levels of perceived stress are more likely to have a higher caloric intake, especially from foods high in fat, independently of background factors such as sex, ethnicity and socioeconomic status (Cartwright et al., 2003b; Pollard, Steptoe, Canaan, Davies, & Wardle, 1995;

Weidner, Kohlmann, Dotzauer, & Burns, 1996). None of these studies included cortisol as an objective measure of experiences of stress.

Associations between patterns of physical activity and stress have been documented in some samples of adolescents, although most studies have included very small numbers, or are completely laboratory based. A series of analyses from the HABITS Project in the United Kingdom suggests that the higher the stress reported by adolescents, the less likely they are to participate in exercise (Simon, Wardle, Jarvis, Steggles, & Cartwright, 2003), although it has been difficult to isolate the effects of stress from the effects of opportunity and access to exercise/recreation facilities (Cradock et al., 2005).

In a review of the trends in overweight status, and the relatively small changes in patterns of diet and activity, it is proposed that there is an alternative permissive or moderating phenomenon contributing to overweight status in children. Within the context of the model of stress and stress response, this pathway has been postulated as activation of the hypothalamic-pituitary-adrenal (HPA) axis under conditions of stress, with the subsequent release of cortisol, and effects on metabolism and weight.

Determinants of the magnitude and persistence of stress response

Psychological attributes and resources. Many explorations of the stress response have focused on, if not predicated on, the contribution of individual

psychological attributes to the magnitude and duration of the stress response. The magnitude and persistence of stress responses, especially those reflective of activation of the HPA axis, have been associated with many individual attributes, including, but not limited to, anxiety and depression (Biondi et al., 1994; Bohnen, Nicolson, Sulon, & Jolles, 1991; Falaschi et al., 2003; Luby et al., 2003; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Pruessner et al., 1997; Tull, Sheu, Butler, & Cornelious, 2005; van Eck, Nicolson, Berkhof, & Sulon, 1996). The stress response has been demonstrated to occur across settings, and studies of the contextual and individual effects on response magnitude have also indicated the interacting effects of age, depression and social support resources. Adolescences who report satisfaction with their support networks, especially family members, may be less likely to respond to daily hassles, and so have a different degree of cortisol reactivity to daily hassles.

Stress-induced changes in diet and physical activity. The seemingly incontrovertible associations between diet, exercise and weight status have recently been challenged. Eisenmann's demonstration of the stability of caloric intake and exercise yet rising prevalence of obesity represents a paradox (Eisenmann, 2003), contrary to the expected energy intake, energy expenditure formula for estimating weight maintenance. There are other indications that the association between caloric intake and weight status in children is not as fixed as it has been proposed (Francis, 1981; Rolland-Cachera, Deheeger, Akrout, &

Bellisle, 1995; Rolland-Cachera, Deheeger, & Bellisle, 1997, 2001). The formula of energy expenditure and energy intake provides a framework for understanding changes in weight status, but dietary composition may be more predictive of weight status, at least in children. Diets in which a high percentage of calories come from fat may be more predictive of overweight status than diets where the bulk of calories are derived from proteins or carbohydrates, as dietary fat is more efficiently stored as body fat than these other macronutrients (Bray & Popkin, 1998). This, in combination with a stress related increase in preference for high fat foods (Cartwright et al., 2003b; Epel, Lapidus, McEwen, & Brownell, 2001; Klein et al., 2004; Oliver, Wardle, & Gibson, 2000; Roemmich et al., 2002; Simon et al., 2003; Wardle & Huon, 2000; Wardle et al., 2003) and the hormonal events that occur through childhood and adolescence (e.g. the changes in sex steroids that may promote fat deposition) (Chrousos, Torpy, & Gold, 1998; Perks et al., 2000; Roemmich, Clark, Berr et al., 1998; Roemmich, Clark, Mai et al., 1998; Roemmich & Rogol, 1999; Rogol, Roemmich, & Clark, 2002; Susman et al., 2003; Torpy & Chrousos, 1996; Tsigos & Chrousos, 2002; Veldhuis et al., 2005) may interact to accentuate the effects of behavioral responses to stress.

There is evidence that dietary factors are not the sole effectors of change in weight status, and that levels of physical activity must be considered in concert with dietary patterns. Patterns of physical activity will influence energy balance, and thus weight status. Children who engage in high levels of

vigorous activity require higher total calories, and may also require adjustment to the relative proportions of macronutrients. The associations between energy intake, energy expenditure and weight status are intuitive, and have been demonstrated in a series of studies of activity levels and weight status (including measures of body mass, fat mass and body mass index (Epstein et al., 1995; Epstein, Wing, & Valoski, 1985; Goran, Hunter, Nagy, & Johnson, 1997; Goran et al., 1998; Hernandez et al., 1999; Obarzanek et al., 1994; Wolf et al., 1993) however not all studies support a linear association between the two (Gazzaniga & Burns, 1993; Treuth et al., 1998) and the relationship may be less precise than proposed by some studies.

Stress-induced HPA Axis Activity and Weight Status

The putative primary pathway between physiological response to stress and weight change is cortisol (Bjorntorp, 1996, 2001; Bjorntorp & Rosmond, 2000a, 2000b; Drapeau et al., 2003; McEwen, 1998b; McEwen & Stellar, 1993; McEwen & Wingfield, 2003). Cortisol acts at multiple sites in the body, and one of the effects is to increase the amount of energy stored as fat; excessive cortisol secretion is a classic endocrine cause of obesity.

There is a dearth of studies reporting empirically tested associations between cortisol and weight status in children. Few studies have examined changes in cortisol in response to stress in a natural setting, and even fewer have

examined changes in cortisol and weight status in early life. There is a multiplicity of potential influences on weight status, including the behavioral and biological responses to stress. The allostatic load model variant, as proposed in the current study, incorporates the concept that stress, specifically, daily hassles predicts both changes in behavior, specifically exercise, and in cortisol secretion, and that these changes are associated to predict weight status.

The current study has two aims. The first aim is to examine the associations between stress, cortisol and weight status, including an examination of vulnerabilities that may enhance these associations The second aim to test the stability of daily cortisol levels, both on consecutive days, and across different developmental periods, and to test if laboratory responses to stressors are analogous to responses in naturalistic settings,

Aim 1: To examine the relationships between daily experiences, cortisol secretion, diet and weight status in adolescents.

The archetypal relationship between stress exposure and increased cortisol secretion is well documented, and much has been written of the stress-weight status relationship. More recently the effects of cortisol, either chronically elevated, or repeated transient elevations, have been associated with weight status. Elevated cortisol has been positively associated with increased weight status, both via increases in food intake and changes in the metabolic processing

of macronutrients, and it is notable that both post-stressor cortisol levels, and heightened morning cortisol levels have also been positively associated with body mass index (Wallerius, Rosmond, Ljung, Holm, & Bjorntorp, 2003). Metabolic actions of cortisol include the activation of lipase, effects on leptin, a protein hormone with effects of feeding behavior and satiety, and effects on insulin resistance. Add to this the tendency of people to select higher fat foods and eat more snacks when under stress (Cartwright et al., 2003b), the fat deposition promoted by cortisol and the positive energy balance created by behavioral change, and the risk of overweight and obesity is enhanced.

Drawing links between cortisol and weight status is intuitive, the more stress that a person experiences, the more cortisol they will release, the less they will exercise, the more calories they will ingest and ultimately the more they will weigh. Thus, positive associations between stress and weight status are expected, that is, the more stress, in the form of daily hassles that are reported, the higher the body mass index of the person. Experiences of stressful events are expected to impact health behaviors, namely food selection and exercise, and these behaviors are well-documented predictors of weight status. It is likely that psychosocial attributes of the person, for example, symptoms of depressive affect, predict whether these changes are promotive of weight gain. It is also expected that adolescents with a greater response to daily hassles will have a greater cortisol secretion, both as measured across the day, and in response to a

stressor. Hypothesis 1: Higher body mass index will be associated with higher reported responses to daily hassles; this relationship will be mediated by cortisol reactivity to a laboratory stressor.

Hypothesis 2: Higher cortisol reactivity to a laboratory stressor will be associated with a higher body mass index, and this relationship will be mediated by diet selection and exercise patterns.

Hypothesis 3: Childhood vulnerability, characterized by high cortisol reactivity to a laboratory stressor and symptoms of psychopathology, will predict higher body mass index, and this relationship will be mediated by daily hassles.

Aim 2: To examine diurnal patterns of cortisol secretion in an adolescent sample.

The circadian rhythm of cortisol secretion is usually characterized by an early morning peak, an afternoon plateau and a nadir in late afternoon and evening hours with a typical pattern of increase, and then decline in the first 30 minutes of awakening (Schmidt-Reinwald et al., 1999; Weitzman et al., 1971) . Recent evidence of individual differences in trajectories of cortisol secretion over the initial waking period, and also across the day (Schmidt-Reinwald et al., 1999) represented in whole by a measure of total day secretion, suggest that people have different patterns and levels of secretion across a day, and these patterns are predicted by, and predictive of, changes in weight status in adults.

The diurnal pattern of cortisol secretion may be established during the adolescent period, although there are few longitudinal studies of the population. The stability of within day cortisol trajectories in adolescence has not been established, and there is evidence of effects of emotional functioning on patterns of cortisol secretion. There are reports that the diurnal rhythm of cortisol changes with age (Buckley & Schatzberg, 2005; Kiess et al., 1995; Lupien et al., 1994; Lupien et al., 1996) and may be influenced by changes in sleep patterns (Dahl, 1996; Dahl et al., 1992; Vgontzas et al., 1999). Emerging evidence suggests that cortisol may have a developmental profile although levels of cortisol transact with emotional and physiological states and traits, and so it is expected that cortisol levels are variable.

People experience stressors in context, transacting with the environment (Cohen, Kessler, & Gordon, 1995) and responses to a stress challenge may be amplified dependent on the attributes of the person, and the temporal and social context in which the challenge is experienced. The subjective report of experiences may be more predictive of biological responses to daily experiences, further, stressors are embedded within psychosocial and environmental contexts, and there may be amplification of the person's response or the salience of stressors dependent on the context.

Hypothesis 4: Patterns of cortisol secretion across the day will demonstrate effects of development; daily trajectories will be more stable in late and post-pubertal adolescents.

Hypothesis 5. Total cortisol secretion across a day will be predicted by greater reported responses to daily hassles.

Summary

This study extends previous examinations of the associations between cortisol responses and weight status at adolescence and incorporates measures of cortisol secretion across consecutive days, temporally tied to experiences of daily hassles. The inclusion of assessments of the stability of daily secretion of cortisol, and stability of daily cortisol secretion across developmental periods enables this work to draw conclusions about the utility of cortisol reactivity, measured in laboratory settings, as a measure in other contexts, and as a direct predictor of weight status in a sample of adolescents. This study will generate findings that may contribute to the development of biopsychosocial models of adolescent development.

Chapter 2

Literature Review

The current epidemic of obesity is widely acknowledged with 30% of US youth now considered overweight (15%) or at risk of overweight (15 %) (Gordon-Larsen, Adair, Nelson, & Popkin, 2004). The etiology of obesity can be established by the biological and behavioral responses to experiences, embedded within the social, cultural and societal milieu during childhood and adolescence. Trajectories of health status, specifically, weight status, are predicted by repeated or profound perturbations to physiological functioning, such as those of the stress response. There are critical periods for the upward deflection of weight trajectories, including adolescence, as the biological and psychosocial changes associated with this period may contribute to patterns of biobehavioral responses that influence weight status.

Theoretical Framework

The problem of childhood overweight is one in which multilevel theories are especially warranted. The allostatic load model provides a paradigm with which to examine the multiple levels on which experiences and the associated

biobehavioral responses influence weight status. Numerous studies have used the allostatic load paradigm from which to develop and test the relationships between responses to environmental, psychological or social challenge and biological markers of health risk (Evans, 2003a; Johnston-Brooks, Lewis, Evans, & Whalen, 1998; Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002; Seeman et al., 2004; Seeman, McEwen, Singer, Albert, & Rowe, 1997; Steptoe, Cropley, & Joekes, 1999; Steptoe, Lundwall, & Cropley, 2000). This review examines the interactions between stress, biological and behavioral components of the stress response, and weight status from the perspective of the allostatic load model.

The elemental allostatic load model is illustrated in Figure **2-1** , the stress, biological response and weight status variant of the model is presented in Figure **2-2**. Although not made explicit in its original form (McEwen, 1998a, 1998b; McEwen, 2003; McEwen & Lasley, 2003), the importance of individual differences is embedded in the allostatic load model, and the model is set within a context that may serve to amplify or attenuate the pathways between the stress and the stress response. The integral pathways in the allostatic load model of stress, biological and behavioral response and weight status, as presented in Figure 2.2, provide a structure for examining the extant literature on the associations between stress and weight status.

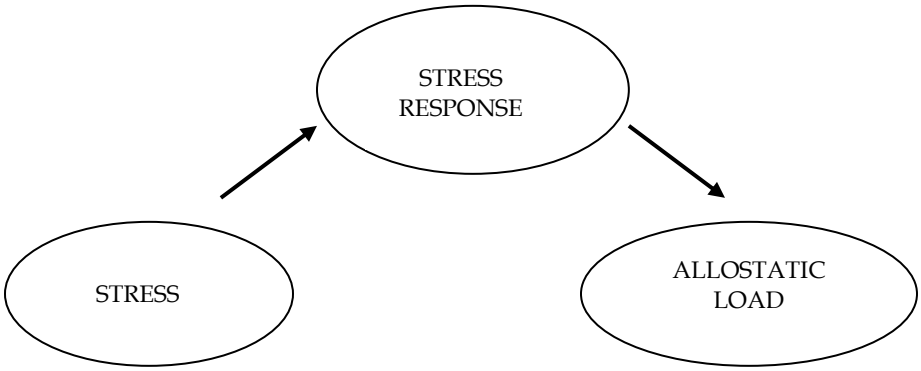

Figure **2-1**: The elemental allostatic load model (adapted from McEwen, 1988). In this model, the experience of stress activates the stress response system with a subsequent initiation of a cascade of neuroendocrine factors. Repeated activation of this system represents an accumulation of allostatic load, expressed in enduring changes to the health of the person.

The allostatic load model variant (Figure 2.2) provides the basis for the current study and draws on two bodies of literature; the literature on the biological responses to stress, and the literature on the determinants of intensity of stress experience, and of the magnitude and persistence of the stress response. This interdisciplinary approach is patterned on the discipline of developmental science that endorses the importance of integrating perspectives from many fields to examine change within the individual (Bergman, Cairns, Nilsson, & Nystedt, 2000; Cairns, Costello, & Elder, 1996; Magnusson & Cairns, 1996; Susman, 2001). Change within the individual necessarily represents multiple levels of the whole person, and occurs within a context.

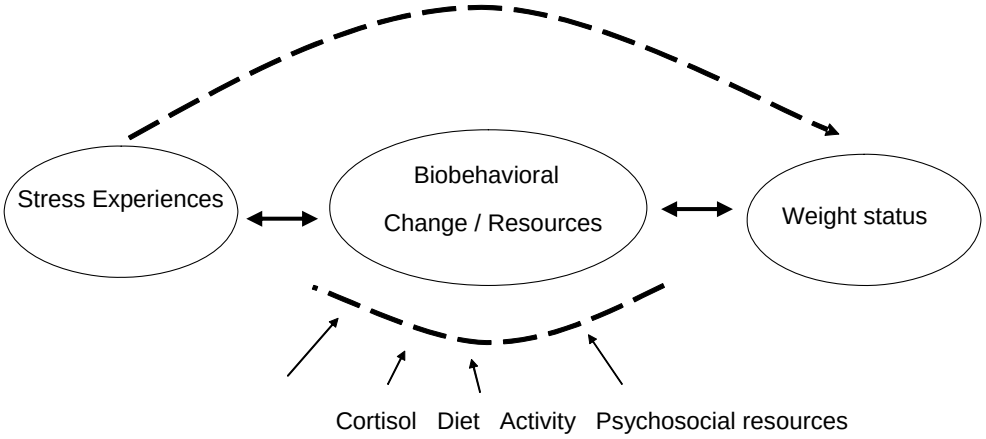

Figure **2-2**: The stress experiences, biobehavioral change/resources and weight status model (Dockray, 2006). The experience of stress causes a range of biological and behavioral responses which influence weight status. The biological and behavioral responses are dynamically integrated, so that each may serve to establish or dampen the other.

The person-centered and holistic interactionism perspectives exemplified in the work of Magnusson (1996) consider the individual as a part of a dynamic person-environment system and is predicated on the interactions between the multiple levels of the person, within the larger social and cultural context (Bergman et al., 2000; Cairns, Bergman, & Kagan, 1998; Cairns, Elder, & Costello, 1997; Magnusson, 2000; Rutter, 1989; Susman, 2001). Within the person, systems are dynamically integrated, and the person is dynamically and reciprocally interacting with the environment and context such that there is constant

reshaping of one by the other. These three theoretical perspectives have guided the research questions and design of this study, and are integral in the current examination of the relationships between stress and weight status at adolescence. This section reviews the intrinsic concepts of these perspectives and relates them to the current study.

The prime principle of developmental science is the integration of concepts and models from other disciplines, including the biobehavioral, psychological and social disciplines are integrated to guide the examination of intra-individual change. This perspective emphasizes: 1) time and timing, 2) multiple levels of analysis, and 3) contextual effects (Cairns et al., 1996). The emphasis on time and timing is integral to examinations of development and the holistic interactionism perspective posits that the development of the individual can occur in different ways. Inherent in this perspective is a caution against codifying development, for example, the assumption implicit in cross-sectional designs, that the individual considered at one time, necessarily has developed from the individual at an earlier time, or vice versa. This highlights the importance of longitudinal research designs and methods that examine the development of the individual (Collins & Sayer, 2001). The modeling of the associations between cortisol and weight status from childhood to adolescence, and the use of statistical models to explore consistency and change was directed by the developmental science, integrative perspective.

Another tenet of developmental science is the value of multiple levels of analysis (Cairns et al., 1996), which seek to capture the interactions among components that make up the whole, and considers it imperative to examine a component of the person by examining how that component operates in the larger social, cultural and societal milieu. This principle necessitates that biological processes are not examined for their intrinsic value, but are examined with reference to other processes of the individual. The current study incorporates this philosophy by considering the activity of the hypothalamic pituitary adrenal (HPA) axis and how it impacts other biological systems, and also how it can be impacted by processes internal (e.g. anxiousness) and external (e.g. daily hassles) to the individual. In addition the activity of the HPA axis was also examined to assess the stability of cortisol parameters across different time periods.

The third emphasis of developmental science is on context. The person-oriented approach stresses that the individual is a purposeful part of a dynamic person-environment system that reciprocally affects and is affected by the environment. Within the person, the different systems are dynamic: interacting with each other in meaningful ways to help the individual develop into an integrated person. Outside of the person, similar principles apply. The person shapes and changes his/her environment, and is shaped and changed in turn. Social, cultural, and physical characteristics of the environment are different

aspects of context which each may interact with multiple levels of the individual Biological processes are one level of the individual that are influenced by the context of the individual. In sum, developmental science, person-oriented approaches, holistic interactionist perspectives and the biosocial model contributed to the underlying theoretical theme. Multiple levels of analysis, the focus on a salient developmental period, a consideration of interactive processes and an individual difference perspective on biosocial processes formed the conceptual basis for a biobehavioral model of stress and weight status in adolescence.

The Stress System

The allostatic load model is predicated on the effects of perturbation to parameters of physiological systems of the body, and perturbation of the HPA axis is of particular importance in this theory. This section first reviews the physiology of stress, with a focus on the HPA axis, and then examines the influences and correlates of individual differences in the magnitude and duration of the stress response. This examination of the extant literature supports a central component of the conceptual model; that an individual's responses to daily hassles are predictive of increases in cortisol secretion, and individual differences explain the differential response to these hassles.

The Anatomy and Physiology of the Stress System

The human body reacts to stress by activating a repertoire of physiologic responses that originate in the central nervous system (CNS). The stress system is comprised of interconnected tissues, organs and neuroendocrine pathways. Components and pathways extend through the central nervous system and the peripheral limbs of the stress system - the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic (sympathetic) nervous system.

The stress system is comprised of components of both the central and peripheral nervous systems. The two principal components of the stress system are the corticotropin-releasing hormone (CRH) system and the locus ceruleus-norepinephrine (LC-NE) nervous system. The hypothalamic-pituitary adrenal (HPA) axis and the efferent fibres of the sympathetic adreno-medullary system form the peripheral components of the stress system. The CRH system extends to extra-hypothalamic sites of the brain but there is a significant accumulation in the paraventricular nucleus of the hypothalamus (Tsigos & Chrousos, 2002). The LE-NE system is located in the brainstem with a network of projections to the amygdala, hippocampus and prefrontal cortex (Charney, 2004) .

Effectors of the Stress Response

The neuroendocrine circuitry involved in the stress response allows a multiplicity of sites to which components of the stress system can communicate. There are bi-directional connections between the CRH and noradrenergic neurons of the CNS, and there are feedback loops in the PVN and the brainstem noradrenergic neurons. The CRH and noradrenergic neurons are innervated through serotoninergic and cholinergic systems, and may be inhibited by glucocorticoids, the product of the HPA axis. CRH regulates the pituitary-adrenal axis, and recent evidence suggests it may have a central coordinator of the stress system response (Chrousos & Gold, 1992; Torpy & Chrousos, 1996). CRH is released into the hypothalamic-pituitary portal system and activates the HPA axis with the release of adrenocorticotropic hormone (ACTH) (Figure **2-3** . Vasopressin (AVP) has a potent synergistic relationship with CRH, although very little independent ACTH secretagogue activity (Charmandari, Tsigos, & Chrousos, 2005) . ACTH is the regulator of glucocorticoid secretion by zona fasciculata and reticularis of the adrenal gland. Other hormones, cytokines, and neuronal information from the autonomic nerves of the adrenal cortex may also participate in the regulation of cortisol secretion (Charmandari et al., 2005).

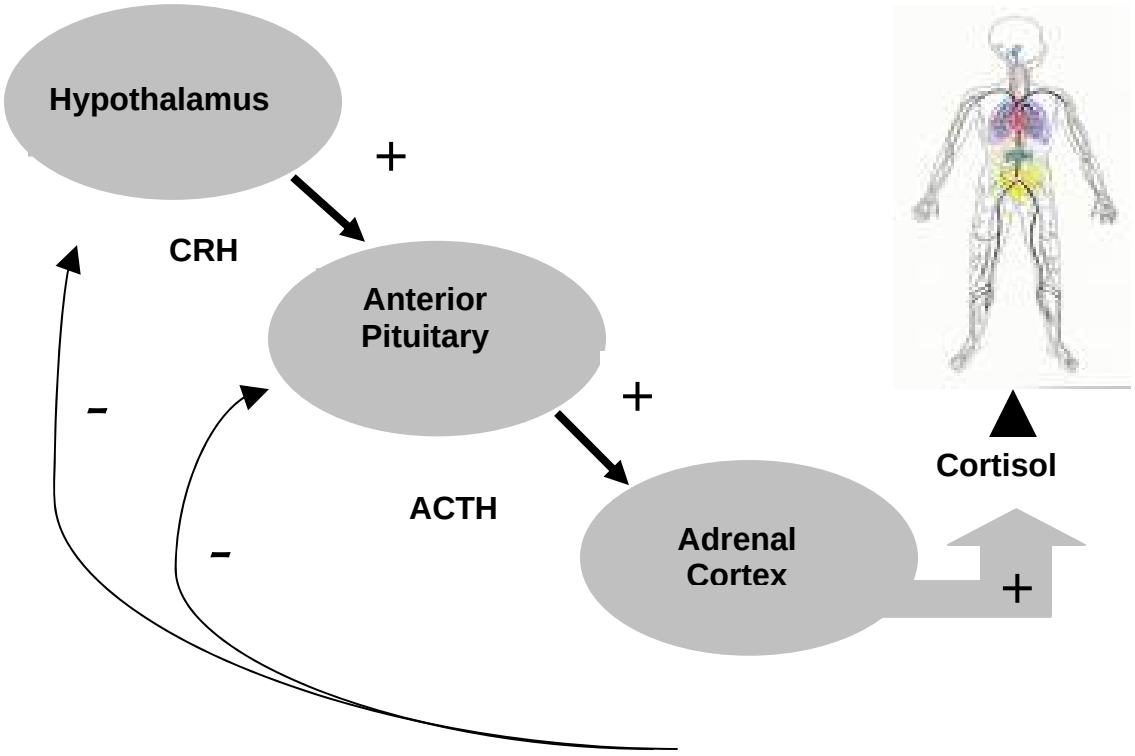

Figure **2-3**: The hypothalamic pituitary adrenal axis. Note. Stressors initiate a cascade of events. Corticotropin-releasing hormone is released from the hypothalamus into the portal system causing the release of adrenocorticotropic hormone from the anterior pituitary. In turn adrenocorticotropic hormone causes the release of cortisol from the adrenal cortex into the general blood supply and has the potential to affect all cells of the body. A negative feedback mechanism, cortisol causes the release of corticotropin releasing hormone to be reduced.

Glucocorticoids (GCs) are the final effectors of the HPA axis. Almost all cells of the body are potentially affected by GCs through intracellular receptors. Cortisol is stimulated by ACTH and is a steroid hormone that increases the rate and strength of heart contractions, sensitizes blood vessels, and affects many metabolic functions in order to prepare the body to adapt to a stressful situation (McEwen, 1998b; McEwen & Stellar, 1993). The stress system interacts with the

immune system, reproductive axis, somatotropic axis, thyroid axis, and metabolic and gastrointestinal systems. The interactions between these systems play an important role in organism allostasis, or the ability to maintain stability through change (McEwen, 1998b). Chronic over-activity or under-activity, or repeated activation of the stress system increases the potential for allostatic load (McEwen & Stellar, 1993). The negative feedback loop of glucocorticoids on the secretion of CRH and ACTH serves to minimize the catabolic, lipogenic, anti-reproductive, and immunosuppressive effects of GCs (Chrousos & Gold, 1992; Tsigos & Chrousos, 2002) and subsequently reduces the risk of allostatic load.

Cortisol influences an organism through permissive, suppressive, stimulatory and preparative actions on a wide variety of organ systems (Sapolsky, Romero, & Munck, 2000). Cortisol has an integral role in glucose metabolism, and maintains fat and carbohydrate metabolism. Cortisol also has regulatory effects on immune system processes and influences vascular responsivity (Al'Absi et al., 1997; Gregg, James, Matyas, & Thorsteinsson, 1999). The HPA cascade operates through both feed-forward and negative feedback mechanisms. Cortisol suppresses CRH and ACTH levels (Buckley & Schatzberg, 2005b; Charmandari et al., 2005; Chrousos, 2000b; McCann et al., 2000; Tsigos & Chrousos, 2002), thus returning cortisol levels to near baseline after the stress experience subsides. Cortisol levels return to near baseline levels 40 to 60 minutes after a stressor (Kirschbaum & Hellhammer, 1989, 1994; Kudielka,

Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004b; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Frequent sampling of cortisol during stress shows that cortisol peaks 10 to 20 minutes after a stressor (Young & Nolen-Hoeksema, 2001), although there are individual differences in adrenocortical reactivity (Altemus et al., 2001; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Kunz-Ebrecht, Kirschbaum, Marmot, & Steptoe, 2004; Lupien et al., 1996; Stroud, Papandonatos, Williamson, & Dahl, 2004; Tout, de Haan, Campbell, & Gunnar, 1998; Uhart, Chong, Oswald, Lin, & Wand, 2006; Wust, Federenko, van Rossum, Koper, & Hellhammer, 2005) . Not all people show perceptible changes in initial levels of cortisol, and others show an extended time to return to baseline levels. These differences are attributed to individual differences in appraisal, coping mechanisms, genetic influences and HPA axis profiles (Seeman, Singer, & Charpentier, 1995; Seeman, Singer, Wilkinson, & McEwen, 2001). If cortisol levels fail to return to baseline before the individual experiences another event that activates the HPA axis, the risk of allostatic load increases (Johnston-Brooks et al., 1998; Korte, Koolhaas, Wingfield, & McEwen, 2005; McEwen, 2002; McEwen & Seeman, 1999; Vanitallie, 2002). Chronic exposure to cortisol has a negative impact on a wide variety of systems and functions, including the nervous system (McEwen, 1997; Seeman et al., 1997), cardiovascular system (Cohen et al., 2000; Gregg et al., 1999; Kapuku, Treiber, & Davis, 2002; Matthews, Gump, & Owens, 2001; Rosmond & Bjorntorp, 2000), metabolism and insulin function (Bjorntorp,

1997a; Roy, Kirschbaum, & Steptoe, 2001), gastrointestinal function (see Sapolsky, 1998), reproduction (see Sapolsky, 1985), immune function (Biondi et al., 1994; Cohen et al., 2000; Cohen, Miller, & Rabin, 2001; Filaire, Bonis, & Lac, 2004; Kunz-Ebrecht, Mohamed-Ali, Feldman, Kirschbaum, & Steptoe, 2003), growth (Abplanalp, Livingston, Rose, & Sandwisch, 1977; Fernald & Grantham-McGregor, 2002; Rosmond, Dallman, & Bjorntorp, 1998), learning and memory (e.g., Buckley & Schatzberg, 2005a; Lupien et al., 1994; Seeman et al., 1997; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000), mood (Bay, Hagerty, Williams, & Kirsch, 2005; Hanson, Maas, Meijman, & Godaert, 2000; Pruessner, Hellhammer, Pruessner, & Lupien, 2003; Smyth et al., 1998; van Eck, Berkhof, Nicolson, & Sulon, 1996) and behavior profiles (e.g., (Battaglia et al., 1997; Moss, Vanyukov, Yao, & Kirillova, 1999; Rosmond, Baghei, Holm, & Bjorntorp, 2001; Susman, 2006; Susman et al., 1999; Watamura, Donzella, Alwin, & Gunnar, 2003).

Individual Differences in Products of the Stress System

Activation of the stress system may follow established pathways but there is evidence of individual differences in patterns of secretion. For example, individual differences have been reported in baseline and reactive levels of cortisol, and these differences may be explained by a multitude of factors, including programming by early life experiences, processes of stress appraisal, sex , age, developmental stage and life event stress (Anisman, Zaharia, Meaney,

& Merali, 1998; Cameron, 2004; Charmandari et al., 2005; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kudielka, Buske-Kirschbaum et al., 2004b; Peeters, Nicholson, & Berkhof, 2003; Stroud et al., 2004; Weise, Eisenhofer, & Merke, 2002). The stress system has many effectors, and changes in these reflect the activation of the stress system, and also reflect underlying psychological and physiological states.

Individual differences in cortisol levels have been reported in many studies, and are often used as the ultimate indicator of stress system activation. Cortisol levels are often compared between groups in order to explain individual differences in physiological and psychological function. That is, individual differences in cortisol are not unexpected, and there are particular patterns of response that are related to the functioning of the person. Increased and prolonged activation of the HPA axis has been associated with physiological functioning, especially in the disease states of diabetes and truncal obesity. An attenuated cortisol response to stress has also been explained by existence of disease, such as arthritis and fibromyalgia (Gur, Cevik, Sarac, Colpan, & Em, 2004), although it is likely that these are the sequelae of hypoarousal of the HPA axis rather than predictive of individual differences in arousal (Chrousos & Gold, 1992; Tsigos & Chrousos, 1994) . Individual differences in cortisol levels, including in the stress response, may also be predicted by psychological states and traits such as depression and obsessive-compulsive disorder (Tsigos &

Chrousos, 2002). Some forms of depression, for example atypical depression, and other psychological attributes such as patterns of antisocial behaviors have been associated with individual differences in HPA axis activity, specifically hypoarousal of the HPA axis (Burke, Fernald, Gertler, & Adler, 2005; Dorn et al., 1996; Dorn et al., 2003; Kaufman et al., 1997; Kurina, Schneider, & Waite, 2004; Pruessner et al., 2003; Sher, 2003; Tse & Bond, 2004; van Eck, Berkhof et al., 1996). Individual differences in stress responses may be anchored in differences of anatomy, and determined by physiological and psychological functioning. Individual differences in the effectors of the stress response offer insight into the complexity of the stress system, and are useful in developing models of health and well-being.

Individual Differences in Cortisol: Mechanisms

Mechanisms that lead to individual differences may operate at multiple levels, and at many stages of development. These differences have been demonstrated in both animal and human models and indicate the plasticity and intricacy of connections between the stress systems and of other tissue and organ systems. Many mechanisms that lead to differential expression of the stress response are possible, including mechanisms that program activity of the HPA axis, and mechanisms that operate via differences in sex steroids. These two mechanisms are described below.

Prenatal programming of the HPA axis system is postulated to occur under the stimulus of glucocorticoids, arising from placental, fetal or trophoblast/amnion sources (Dean, Yu, Lingas, & Matthews, 2001; Gutteling, de Weerth, & Buitelaar, 2004; O'Connor et al., 2005; Susman, 2006; Takahashi, 1998). Exposure to glucocorticoids in the fetal period may result in enduring organizational effects on the brain, and alter glucocorticoid receptor gene expression in the central nervous system (Bertram & Hanson, 2001, 2002). This alteration may result in attenuation or exaggeration of the HPA axis, via altering the structures of the negative feedback pathways. Bertram and Hanson (2001, 2002) provide evidence that this programming may have sex-specific effects, further contributing to individual differences (Klein & Corwin, 2002). There is evidence that the early years of life may also be susceptible to reprogramming effect (McEwen, 2003). As an example , children experiencing deprivation in the early childhood years have consistently higher cortisol levels than children raised in other environments (Gunnar, Morison, Chisholm, & Schuder, 2001; Rutter & O'Connor, 2004). This is suggestive of changes in the functioning of the HPA axis in response to persistent stress, although the actual mechanisms remain to be elucidated. The speculated mechanism is that there are changes in the sensitivity of the HPA axis as a result of sustained exposure to glucocorticoids in early life.

Differences in systems and hormones aligned to sex difference have also been postulated as a mechanism that predicts individual differences in the stress response (Klein & Corwin, 2002; Taylor et al., 2000). Gonadal steroids may exert specific modulatory effects on the HPA axis, sensitizing the axis to feedback. One lab group has conducted experimental studies in the effects of sex steroids on the stress response, comparing the stress response of male adults and female adults in both the luteal and follicular phase of the menstrual cycle and females on oral contraceptives (Kirschbaum et al., 1999). The results of this study indicate that sex and menstrual cycle phase exerted effects on the stress response. Sex differences in levels of ACTH released in the stress response were observed, with men having a greater response than women, and effects of menstrual cycle phase on cortisol levels, with women in the luteal phase significantly different from women in the follicular phase. Others have reported that estradiol binds to particular sites on the CRH gene, and upregulates CRH gene transcription , thus altering the stress response in a potentially enduring manner (Vamvakopoulos & Chrousos, 1993). Adrenal androgens have also been postulated as a mechanism of individual difference in the stress response. In a review of animal studies Kudielka and Kirschbaum (Kudielka & Kirschbaum, 2005) suggest that evidence is suggestive of the effects of androgens at multiple sites of the HPA axis. Kudielka has examined this model across several lab stress studies (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004a; Kudielka et al., 1998;

Kudielka, Schmidt-Reinwald, Hellhammer, & Kirschbaum, 1999). The most direct examination compared the stress response to a laboratory based challenge in two groups of older adults, one group received DHEA treatment and the other a placebo. The women in the DHEA treatment group had a significantly greater HPA response to the stressor compared to the women in the placebo group. These studies are suggestive of a mechanism of adrenal androgens influencing the HPA axis activity during times of stress. The observed differences may also represent different mechanisms of the stress system itself. Taylor and colleagues (Taylor et al., 2000) have provided evidence from animal models that suggest individual differences in the stress system response may have evolved differently so that men and women have different patterns of responding to stress . Specifically it is proposed that men will have greater activation of the HPA and NE-LC system, whereas women have a response that is characterized by increases in oxytocin and endogenous opioids.

Individual differences in the stress system response to challenge may be explained with reference to psychological attributes of the person (e.g. coping skills, depression), stressor salience (e.g. social-evaluative versus cognitive challenge) and the experience of particular life events (e.g. trauma). Further individual differences may also reflect appraisal processes and memory mechanisms. Differences between individuals may predict the magnitude and time course of HPA axis activation during stress, and may be rooted in

differences in the functional capacity of the HPA axis, in the levels of endogenous steroids and age associated changes in the HPA axis and central nervous system.

Influences on the Magnitude and Persistence of the Stress Response

The nature and intensity of the stressor is assumed to influence the stress response; however, there are a multiplicity of moderators and mediators of the stress experienced, and thus, the response. There is a substantial accumulation of evidence from both laboratory based and naturalistic setting studies that the magnitude and time course of the stress response is predicted by individual attributes and contexts. Research with diverse foci has revealed influences of context, chronic stress, psychological attributes, genes, stressor salience, sex, age and race/ethnicity on the magnitude of biological response to stress.

Contextual Influences

The stress response is shaped by interacting forces and experiences including the family, social and environmental contexts. For instance, familial context has influences on the regulation of the stress system (i.e. HPA axis). Taylor and colleagues (Taylor, Lerner, Sage, Lehman, & Seeman, 2004) identified

a pathway from family contexts characterized as chaotic and conflictual, to changes in the regulation of the stress response via changes in anxiety and hostility of the child. In other studies of the influences of environmental context on the stress responses, it has been demonstrated that there may be differential activation of the HPA axis in and across settings. Watamura and colleagues (Watamura et al., 2003) suggest that there is a marked difference in the patterns of cortisol secretion between a home and a child care context. In a sub-sample of the larger population for which measures of cortisol in the home environment were available (n=35) and a difference score between the morning and afternoon samples obtained in the home and child care setting, Watamura and colleagues conclude that the pattern of cortisol secretion may be context sensitive. In the child-care setting, 35% of the infants and 71% of the toddlers had a rise in cortisol across the day, whereas in the home environment most infants and toddlers showed a decrease. A caveat for these findings was the small sample that were willing to provide samples (of saliva) for determination of cortisol levels, and indications that those who provided samples may be demonstrably different from others in the sample. For example, in the home group of children there was a high average rating of (child) positivity and ability for, and engagement in, complex forms of social play.

Other work is suggestive of other environmental contextual effects on the activity and the reactivity of the stress system, especially those contexts

associated with chronic, albeit low- level experiences of stress, such as those of high neighborhood risk or physical contexts characterized by high levels of noise or crowding (2001). Neighborhoods that are characterized by noise and overcrowding are also more likely to be characterized as neighborhoods with residents who have less economic resources and lower levels of educational attainment. In a series of studies of the effect of socioeconomic status on stress system activity, Lupien and colleagues undertook a comparison of cortisol levels in children with higher and lower socioeconomic resources(Lupien, King, Meaney, & McEwen, 2001) . Children in families of lower socioeconomic status had higher levels of cortisol. However, this relationship is short-lived, dissipating by the time of high school transition. This finding is reminiscent of the age-context interactions reported by others, for example, Watamura and colleagues (Watamura et al., 2003) and serves as a reminder of the complexity of the stress response, and the caution with which results must be interpreted.

The evidence on contextual effects on the stress system activity and reactivity is equivocal with some studies showing no relationship and others showing relationships between context and stress system responses. This is partly attributable to the relatively small number of studies with context as a specific and empirically testable focus. The evidence is suggestive of complex interactions between context, including familial, social and environmental, age and the magnitude of HPA axis activation. Context and its relationship to the

stress response in the current study refers to the daily experiences of adolescents, specifically, daily hassles, experienced in their usual environments.

Experiences of Chronic Stress

Chronic stressors persist over time and have been associated with chronically elevated levels of cortisol (Ockenfels et al., 1995; Pruessner, Hellhammer, & Kirschbaum, 1999; Wright & Steptoe, 2005; Wust, Federenko, Hellhammer, & Kirschbaum, 2000a; Wust, Wolf et al., 2000a). Conceptualizing work with high demand yet low latitude in decision making as a chronic stressor, Kunz-Ebrecht and colleagues demonstrated the mediating effect of chronic stressors on the response to more acute stressors in a sample of adults drawn from the Whitehall II cohort, (Kunz-Ebrecht, Kirschbaum, Marmot et al., 2004; Kunz-Ebrecht et al., 2003). Participants collected saliva samples for cortisol determination at ten time points across the day, and reported their experience of stress in the 20 minutes prior to saliva sampling. Cortisol levels over the course of the day (representing the diurnal rhythm and reactivity to stressors) were greatest in women with low job control and high job demand, although job control alone influenced daily cortisol level in men. The results suggest complex interrelationships between chronic stressors, response to acute stress and differences between males and females.

In a study of job stress as a paradigm of chronic stress, cortisol variability was compared across the working day in a sample of teachers who had previously (12 months prior) been classified as high or low job strain (Steptoe, Cropley, Griffith, & Kirschbaum, 2000). Those in the high strain group had significantly higher cortisol levels early in the work day, but the variability across the day was not significantly different. Steptoe and colleagues explain these unanticipated findings with respect to individual psychological attributes, such as anger expression, of the person.

Other components of HPA axis activity may be influenced by chronic stress, including the usual diurnal cycle of cortisol (Smyth et al., 1998). Wüst and colleagues, (Wust, Federenko, Hellhammer, & Kirschbaum, 2000a) in a study of twins, estimated the contribution of genetic factors to diurnal pattern of cortisol secretion, and report that there is evidence of a genetic influence on the morning rise in cortisol, however, the pattern of cortisol secretion at wakening and across the day is also predicted by chronic stress. There is a series of other studies that demonstrate the influence of chronic stress on the diurnal and within-day variability of the activity of the HPA axis (Cicchetti & Rogosch, 2001; 2001; Heim et al., 2000; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Marshall & Garakani, 2002; Moss et al., 1999; Ockenfels et al., 1995; Steptoe, Brydon, & Kunz-Ebrecht, 2005; Taylor et al., 2004). The influence of chronic stressors on the reactivity of the HPA axis to stress is well-established, and there

are developmental effects that are evident at periods of transition, or across age (Dettling, Gunnar, & Donzella, 1999; Gunnar, Bruce, & Hickman, 2001; Gunnar & Donzella, 2002; Gunnar, Morison et al., 2001; Lupien et al., 2001; Tarullo & Gunnar, 2006; Watamura, Donzella, Kertes, & Gunnar, 2004).

Psychological Attributes

Many explorations of the stress response are interested in, if not predicated on, the contribution of individual psychological attributes to the magnitude and duration of the stress response. The magnitude and persistence of the stress responses, especially those reflective of activation of the HPA axis, has been associated with many individual attributes, including, but not limited to, coping style, life event experiences, degree of neuroticism, anxiety and depression [for examples see (Biondi et al., 1994; Bohnen et al., 1991; Falaschi et al., 2003; Luby et al., 2003; Nachmias et al., 1996; Pruessner et al., 1997; Tull et al., 2005; van Eck, Nicolson et al., 1996). Associations between psychological attributes and cortisol have been demonstrated across settings, and have also indicated the interacting influences of context, age, task, repetition and social support. The effects of individual psychological attributes are acknowledged within the framework of holistic interactionism, and in the current instance serve to both mediate and moderate the relationship between stressor and stress

response, and are thus may have formative influences on the stress-weight status model.

Stressor Salience

There are differences in the stress response that arise from the differences in meaning of the stressor to the person. Stressors with low emotional valence or those that are perceived as posing little threat to the function and goal related behavior of the person will not evoke a significant stress response (Kirschbaum et al., 1999; Kunz-Ebrecht, Kirschbaum, & Steptoe, 2004; Stroud et al., 2004; Stroud, Salovey, & Epel, 2002). The meaning of the stressor can only be measured against the characteristics and attributes of the person, and so the pathway described in the model from stressor to stress response exists only when the stressor is meaningful.

A neatly constructed examination of the effect of stressor salience is provided by the work of Stroud and colleagues (Stroud et al., 2002) . A relatively small sample of young adults (N=50, mean age = 19 years) were assigned to either a rejection or achievement stressor group. The rejection stressor comprised of two social interaction challenges and the achievement comprised of two tasks, a mathematical and a verbal challenge. There was a clear interactive relationship between stressor type and sex, with women more responsive (indexed by salivary cortisol) than men to the rejection stressor, and men more

responsive to the achievement challenge. It is argued that the different challenges are differentially salient, and so stress responses will be determined by experiences, goals and priorities of the person.

Sex Differences

Sex differences in stress responses have only very recently begun to be revealed. Culture and context have driven the research paradigm under which stress responses have been examined resulting in a wealth of work on males. This trend, combined with the rhetoric of gender politics, impeded the development of theory on sexual differentiation of the stress response that may have concealed a stress response sex dimorphism. This speculated differentiation has neuroendocrine and behavioral correlates that may have utility in explaining the differential responses that have been reported between males and females. Taylor and colleagues (Taylor et al., 2000) have proposed a biobehavioral model of the stress response that may explain sexually dimorphic stress responses. Describing a model with a base in evolutionary theory, Taylor and colleagues propose that forces of natural selection exert different pressures on males' and females' stress responses. Different responses may be more adaptive for females, especially those that enhance the safety of the offspring. These responses may include behavioral components of affiliation with others for support and access to resources, and reducing vulnerability to self or

offspring. Affiliative behavioral responses may be anchored in a physiological stress response that differs from the 'prototypic' response of 'fight/flight' (F/F) response that is anchored in the sympathetic nervous system and the release of the catecholamines. Taylor and colleagues propose that these F/F responses, both behavioral and neuroendocrine, may be less adaptive for females who might have been limited in their capacity to flee or fight in times of challenge. It is proposed that sustaining the sympathetic-adrenomedullary system pattern of stress response may have been maladaptive. Taylor and colleagues propose that a more appropriate model may be the 'tend-and-befriend' model, a model based in a neuroendocrine system that includes the secretion of oxytocin and endogenous opioids (Taylor et al., 2000). In this model, females under stress are likely to have a neuroendocrine profile that differs from males. These sex differences in the endocrine response to stress offer an explanatory model for understanding the sex differences in the development of obesity.

The model of stress response in females, as proposed by Taylor and colleagues (2000), describes the secretion of oxytocin and endogenous opioids in response to a stressor. The profile of hormones has also been associated with health protective effects, although there is continuing debate on this relationship (Gnatuk, 2002; Hodis, Mack, & Lobo, 2003; Rosano & Fini, 2002) and these may be mechanisms that adjust the cumulative risk of repeated stress as proposed by the allostatic load model. Oxytocin may ameliorate the increases in cortisol

associated with stress, thus conferring some additional degree of protection from the effects of stress on the development of obesity. Taylor and colleagues (2000) report an association between oxytocin and cortisol, such that there is a reduced cortisol response to stress and that, in the context of a laboratory challenge, higher levels of oxytocin were associated with a shorter HPA recovery period. Further, Taylor and colleagues (2000) also propose a role for endogenous opioids in a differential stress response for females, and these have also been reported to be associated with the moderation of the release of norepinephrine and cortisol (Williams, Ko, Rice, & Woods, 2003). If oxytocin moderates the release of norepinephrine and cortisol, oxytocin release during stress may mediate the relationships between stress and health. A stress response characterized by increased in levels of oxytocin, especially with the co-occurring presence of estrogen may explain the differences in stress and weight status associations between the sexes, via different pathways of stress responses.

Sex differences in cortisol responses to stress are not consistently reported and sex is not routinely explored as a potential individual differences explaining patterns of cortisol response to challenge, although there is evidence that cortisol responses to challenge may differ by age and sex (Kudielka, Buske-Kirschbaum et al., 2004a; Kudielka et al., 1998; Kudielka & Kirschbaum, 2005; Uhart et al., 2006). A series of studies by Kirschbaum and colleagues (Kirschbaum, Klauer et al., 1995; Kirschbaum et al., 1999; Kirschbaum, Pirke, & Hellhammer, 1995;

Kirschbaum, Wust, & Hellhammer, 1992; Kudielka, Buske-Kirschbaum et al., 2004b; Kudielka et al., 1998) have documented higher cortisol responses to psychological stress in younger males than females, although age may be a confounder, as in older populations females may have a greater stress response (Kirschbaum, Klauer et al., 1995; Kirschbaum et al., 1999; Kirschbaum, Pirke et al., 1995; Kirschbaum et al., 1992; Kudielka, Buske-Kirschbaum et al., 2004b; Kudielka et al., 1998). The effects of appraisal processes and psychosocial resources may contribute to sex differences. Males and females may be differentially responsive dependent on the type of stressor, and there is evidence that males show higher response to stress challenge paradigms that involve conflict, win/loss scenarios and performance evaluation tasks (Kirschbaum, Klauer et al., 1995; Kirschbaum et al., 1999; Kirschbaum, Pirke et al., 1995; Kirschbaum et al., 1992; Kudielka, Buske-Kirschbaum et al., 2004b; Kudielka et al., 1998; Seeman et al., 1995; Seeman et al., 2001). It has also been shown that females may be more responsive to laboratory stress paradigms involving threat to the social self, (Stroud et al., 2002) and more responsive to hostility, discord and social rejection in their interpersonal relationship (see review by Kiecolt-Glaser et al., 1996). Thus, individual differences in stress responses to daily life may be examined by sex differences in social and cognitive processes. In sum, the cortisol response to stress may be different between males and females, and some of this difference may be attributed to difference in stressor salience and

appraisal processes. The variance in cortisol responses to challenge may be explained in some part by sex differences on physiological and psychosocial processes (Dorn et al., 1996; Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997), and thus sex differences in cortisol responses to daily hassles are predicted and explored in the current study.

Effects of Age and Sex

Stroud and colleagues (Stroud et al., 2004) have attempted to unpack the variance in the stress response, specifically to determine the effects of sex and age by eliminating the contribution of stressor salience. Using a CRH challenge paradigm in children and adolescents, moderate effects of sex and age were revealed (Dorn et al., 1996). This study, however, was designed to examine the effects of pubertal stage, not age, and although these are usually grossly correlated, the results may reflect influences of estrogen, gonadal steroids or adrenal androgens on the activation of the HPA axis.

There is other evidence, in adult populations, of the effects of age and sex on the stress induced activation of the HPA axis. Seeman and colleagues have reported the interactive effects of age and stress (Seeman et al., 2001). In this study 40 participants completed a standardized stress challenge over a 30-minute period. The younger male participants (20-40 years) had a greater HPA axis response to stress than younger female participants, but in the older cohort (65+

years) females had a greater HPA axis response. This study has a small sample size, especially in the group of older women. The effects of sample size may explain the contrast with the results of other studies who report no age or sex associated change in HPA axis activity post stress, under similar laboratory conditions (Kirschbaum et al., 1999; Kudielka, Buske-Kirschbaum et al., 2004a, 2004b; Kudielka et al., 1998).

There is evidence that there are age-sex interactions on the HPA axis response to stress during adolescence, and more specifically, that puberty is a period associated with change in activity of the HPA axis in the stress response (Hardie, Moss, Vanyukov, Yao, & Kirillovac, 2002; Romeo, Lee, & McEwen, 2005; Viau, Bingham, Davis, Lee, & Wong, 2005). Animal models are particularly suggestive of changes in the stress system response at puberty (Consten et al., 2002; Dean et al., 2001; Dorn et al., 1996; Dorn & Chrousos, 1997; Wommack & Delville, 2003; Wommack, Salinas, Melloni, & Delville, 2004). In review, in humans there is some evidence for sex differences in the magnitude of HPA axis response to stress, and evidence that these sex differences interact with age, however, the paucity of studies in children make the influence of sex and age on the magnitude and persistence of the stress response in early life speculative.

Race/ethnicity

The associations between race/ethnicity and the stress response have received little direct attention. Few studies have specifically examined the influence of race/ethnicity on the magnitude or persistence of the HPA axis mediated stress response, although there is a large body of work examining race/ethnicity and cardiovascular reactivity. A series of studies by Yanovksi and colleagues are suggestive of intrinsic differences between racial/ethnic groups in the activation patterns of the HPA axis (Yanovski et al., 2000; Yanovski, Yanovski, Cutler, Chrousos, & Filmer, 1996; Yanovski, Yanovski, Friedman et al., 1996; Yanovski, Yanovski, Gold, & Chrousos, 1993; Yanovski, Yanovski, Harrington, Gold, & Chrousos, 1995). In a comparison of 18 African-American and 30 Caucasian weight- and age-matched non-obese and obese women, plasma cortisol responses to dexamethasone and plasma ACTH and cortisol responses to (ovine) CRH were determined. In the African-American women the CRH-stimulated ACTH concentrations were significantly greater, and this group also had persistent elevations of ACTH (area under the curve) in comparison to the Caucasian women. Using the same research paradigm of intravenous administration of ovine corticotropin-releasing hormone (CRH) and dexamethasone administration, this lab group also examined the differences between African American and Caucasian men, and African-American and Caucasian girls and report similar patterns of differences in response. The

physiological underpinnings of differences remain to be elucidated, and it should be noted that there were no racial differences in baseline free or total plasma cortisol levels, baseline ACTH concentrations, or the plasma cortisol response to CRH. However this series of studies does indicate differences that potentially contribute to the response to a stress experience.

There may also be effects of the type of challenge , and as a point of comparison, others have reported no significant differences between African-American and Caucasian post-menopausal women in the cortisol response to exercise (Giannopoulous, Carhart, Sauro, & Kanaley, 2003), and the magnitude of cortisol reactivity to an interpersonal stressor has been reported to be greater in African-American post-menopausal women than in Caucasian post-menopausal women (Wilcox, Bopp, Wilson, Fulk, & Hand, 2005). Implicit in the design of many studies yet seldom tested, the contribution of race to the physiology of stress may be an important moderating force on the model of stress and weight status. Studies that explicitly test the influence of race/ethnicity on stress responses will allow the development of a model of stress-weight status that allows for the dynamic interaction between the multiple levels of the person, including physiological dispositions associated with genotypes (Magnusson & Stattin, 1998; Susman, 2006; Susman, Nottelmann, Dorn, Inoff-Germain, & Chrousos, 1988).

The pathway between stressor and response is multi-layered, and multi-faceted, as there is significant potential and likelihood of the individual attributes to interact with contexts. The potential interactions and effects on the pathway between stressor and stress response form an essential part of the model of stress relationships with on weight status. The theoretical framework of holistic interactionism suggests that change within the individual is predicated on interactions between multiple levels of the function of the person, including those influenced by genes and gene-environment interactions. The relationships between stress, stress responses and weight status as postulated by the allostatic load (variant) model, and conceptualized through the holistic interactionism theoretical framework, necessitate that all levels of function are incorporated and acknowledged, although not necessarily measured in the same study.

In summary, the physiological stress response is characterized by a profile of changes in neuroendocrine factors and neurotransmitters that have myriad effects on psychological function. The influences also extend to, and associate with, psychological function and behavior. The expression of the physiological stress response has been posited as a device that guides behavior [see Charney (2004) or De Kloet (2004)] . The altered patterns of neuroendocrine function as a result of experience have also been associated with psychopathological and illness states [see Meyer, Chrousos and Gold (2001)] and

so there exists potential for the experiences of stress to occur in two connected dimensions, the biological and the psychological.

Stress-induced HPA Axis Activity and Weight Status

The putative primary pathway between a physiological response to stress and weight change is cortisol (Bjorntorp, 1996, 2001; Bjorntorp & Rosmond, 2000a, 2000b; Drapeau et al., 2003; Epel et al., 2000b; Epel et al., 1999; McEwen, 1998b; McEwen & Stellar, 1993; McEwen & Wingfield, 2003). Cortisol acts at multiple sites in cells and organs systems, and one of the effects of cortisol is to increase the amount of energy stored as fat, especially fat stored in the abdominal region. Excessive cortisol secretion, either repeated acute elevations or chronic elevation, is one of the classical endocrine causes of obesity, empirically demonstrated in animal models involving either treatment with glucocorticoids or adrenalectomy (York & Bray, 1972). Psychosocial stress, with increased cortisol secretion with increasing stress has also been demonstrated to predict obesity in a primate model (Jayo, Shively, Kaplan, & Manuck, 1993). In the current study associations between cortisol reactivity and weight status will be examined.

Exaggerated or persistent elevations in cortisol, as a result of stress, are also associated with a redistribution of fat to the viscera and the abdomen, as well as overall recruitment of dietary fat to the fat depots of the body,

(Bjorntorp, 1990, 1995a; Rosmond et al., 1998) and so causing change in weight status. Cortisol activates lipoprotein lipase, an enzyme important in lipid metabolism and regulating lipid accumulation in adipocytes, cortisol also inhibits lipid mobilization (release for energy) in the presence of insulin. These two actions of cortisol are mediated by glucocorticoids receptors, receptors which are denser in number in the adipose tissue of the intra-abdominal viscera than in other sites of adipose tissue of the body. Thus, the influence of cortisol on lipid metabolism and storage is greater in the abdominal area than in other area. This initiates a cycle, as the increase of visceral and abdominal fat has an associated increase in cortisol receptor density in the abdominal region. The greater density of receptors mediates an increase in the accumulation of triglycerides in fat depots, which then increases receptor density and so on.

There is a dearth of studies reporting empirically tested associations between cortisol and weight status in children. Knutsson and colleagues (Knutsson et al., 1997) report that although there is large intra-individual and inter-individual variability of cortisol in an adolescent sample, they found no association between basal cortisol levels and body composition. A lack of an association between cortisol and body fat mass, irrespective of age or pubertal stage has also reported (Sudi et al., 2000). One study, involving 300 children, does provide support for the association between cortisol and weight status. This study, the DONALD (Dortmund Nutritional and Anthropometric

Longitudinally Designed) tracks children's development as they mature, but the report on associations between cortisol and weight status utilizes only the cross-sectional data (Dimitriou, Maser-Gluth, & Remer, 2003). Children collected 24 hour samples of urine that were used to determine urinary free cortisol secretion, and then carried out a series of measurements that allowed the calculation of body mass index, percentage body fat and fat/fat free mass. Analyses of variance procedures, with each sex considered separately, indicated that in girls in all age groups (4-5 years, 8-9 years and 12-14 years) there was a significant association between glucocorticoid excretion and fat mass, and for percentage body fat, and a significant positive association with body mass index. In a simple linear regression model, variation in glucocorticoids in girls explained 10-32% of the variation in body fat. In boys, glucocorticoids and fat mass were only associated in the 8-9 years group, although glucocorticoids were positively associated with BMI in both 8-9 and 12-14 year old boys. These results indicate an association between glucocorticoids and fat mass and weight status in a sample of children, although causality cannot be determined.

The causal association between psychological attributes and weight status has been tested in a study of almost 1600 children and adolescents by Ravaja and Keltikangas-Jarvinen (1995). In this study measures of temperament and body mass index were obtained three years part. At baseline weight status was significantly correlated with negative emotionality and responsivity (a

measure of sociability) in 15-year-old boys, and negative emotionality and anger predicted metabolic syndrome risk 3 years later. The results are suggestive of complex and interactive pathways between psychological attributes and weight status. In a predictive model of psychological attributes and body mass index, the sole significant relationship was between responsivity and weight status in 6-year-old girls. Accepting the temperament measures (negative emotionality, aggression, anger, mental vitality) as proxies of psychological attributes (as stress reactivity is purported to be) this study provides little evidence that there is a developmental transformation of feelings into changes in physiognomy in later life.

A role for stress-induced cortisol in predicting weight status is suggested by examinations of differential experiences of stress, the concomitant physiological profile of stress and weight status in twins discordant for total body mass and body fat mass. In a study of adult identical twins drawn from the Finnish Twin Study, measures of psychosocial stress, 24 hour urinary free cortisol, percentage body fat and visceral body fat were obtained. The twins were separated into groups based on the relationship of their fat mass measures to the gender specific median. Differences in cortisol were observed between non-obese and obese pairs; the larger the amount of visceral fat a person had compared to their twin, the higher the levels of cortisol secretion. This difference did not reach statistical significance, perhaps as a result of the large inter-

individual variation. Psychosocial distress scores were also higher in the obese co-twin, and this was associated with visceral fat accumulation (Marniemi et al., 2002).

These reports represent preliminary attempts to characterize the cortisol–weight status relationship in humans free from disease, and currently the reported associations are inconsistent but suggestive of a role of cortisol reactivity in determining weight status. In the current study, inconsistencies in extant findings are considered attributable to the use of basal cortisol levels, collected over a single day period, rather than measures of the intra-individual variability, especially variability related to stressor exposure. The diurnal cortisol profile may be more useful in predicting obesity vulnerability as a time-lagged effect, but cortisol reactivity profiles may be more proximally associated with weight status, especially in adolescence.

Stress and Obesogenic Behavior

In response to experiences of stress individuals may also alter their usual behavior, and may adopt behaviors that are associated with changes in health, including weight status. In the following section, the literature examining stress and behavioral responses will be reviewed, with a specific focus on the behavioral responses implicated in weight change.

The seemingly incontrovertible association between diet and exercise and weight status has recently been challenged. Eisenmann's demonstration of the stability of caloric intake and exercise yet rising prevalence of obesity represents a paradox (Eisenmann, 2003). There are other indications that the association between caloric intake and weight status in children is not as fixed as it has been proposed (Francis, 1981; Rolland-Cachera et al., 1995; Rolland-Cachera et al., 1997, 2001). The formula of energy expenditure and energy intake provides a framework for understanding changes in weight status, but although a parsimonious explanation, it may not necessarily capture the intricacies of interactions among stress, metabolic processes and behavior that also influence weight status.

Stress and Eating Behavior

The scientific discourse on the stress-eating relationship surrounds two apparently antithetical relationships, stress induced over-eating, hyperphagia, and a stress induced hypophagia. Examinations of the influence of stress experiences on eating behavior have been conducted in both controlled and naturalistic settings, and the results indicate the complexity of the associations between stress and eating behaviors (Cools et al., 1992; Crowther et al., 2001; Grunberg & Straub, 1992; Haynes et al., 2003; Heatherton et al., 1993; Klein et al., 2004; Polivy et al., 1988; Schotte et al., 1990). Haynes and colleagues (Haynes et

al., 2003) examined the interaction of restraint and disinhibition on stress associated eating behavior in women. In a comparison of groups (combinations of high/low restraint and high/low disinhibition) under conditions of stress or no-stress, Haynes and colleagues report that women in the high restraint/high disinhibition group, and women in the low restraint/low disinhibition group ate more in the stress condition, whereas women in the low restraint/high disinhibition group ate less under the stress condition. Levels of disinhibition were also associated with stressor responsivity, as the high inhibition group experienced greater affective response to the stressor. In a study comparing the experience of stress (daily hassles) and binge eating, Crowther and colleagues (Crowther et al., 2001) report that women with a history of binge eating report greater responsivity to the experience of daily hassles, and were more likely to eat greater amounts on days characterized as high stress. In one of the very few laboratory based explorations of stress induced eating in children and adolescents, Roemmich and colleagues (2002) explored the interactions between restrained eating and stress. Children completed two lab sessions, one was a neutral experience and the other was designed as a stressor involving a videotaped speech task. Children with high restraint (an indicator of dietary restriction or dieting) and high stress reactivity ate significantly more snacks after the stress experience, although children with low restraint and low reactivity ate fewer snacks than in the control condition.

The overall theme in the results of these studies is that there is an interaction of sex and stressor such that women may eat more than men under conditions of stress, and that stress may result in disruption to active efforts of restrained eating. In related studies, it has been shown that people who are overweight or obese are more likely to eat greater amounts of food under conditions of anxiety, and this behavior is accentuated for those with greater levels of state anxiety. Thus, anxiety disorders may also moderate the hyperphagic response to stress, which has been reported in longitudinal studies (Hasler et al., 2005).

Physical activity and Weight Status

Patterns of physical activity will influence the amount of energy stored as fat, and so compensate for increased caloric intake. In a similar fashion, those children who engage in high levels of vigorous activity require higher total calories, and may also require adjustment to the relative proportions of macronutrients. The associations between energy intake, energy expenditure and weight status is intuitive, and has been demonstrated in a series of studies of activity levels and weight status (including measures of body mass, fat mass and body mass index, (Epstein et al., 1995; Epstein et al., 1985; Goran et al., 1997; Goran et al., 1998; Hernandez et al., 1999; Obarzanek et al., 1994; Wolf et al., 1993) however not all studies support a linear association between the two (Gazzaniga

& Burns, 1993; Treuth et al., 1998) and the relationship may be less precise than proposed by some studies. There exists a need for a holistic approach to examinations of the associations between physical activity and weight status in children and adolescents, as weight status is not necessarily only predicted by energy intake.

Obesogenic Behavior in Naturalistic Settings

The controlled provocation of a stress response and the limitations on the full range of possible behavioral responses in a laboratory setting offer insight, yet require caution in the translation to the dynamic conditions of usual life, and thus to the development of models of stress and weight status. There is some evidence to suggest daily stress may influence eating behaviors, and so affect weight status. In a study of 90 people tested over a period of 6 months, Wardle and colleagues (Wardle et al., 2000) compared periods of high and low stress (indexed by hours worked) and identified an effect of stress on eating patterns. Periods of high stress were associated with a greater overall caloric intake, of especial note is the higher intake of fats and sugars, and this effect was greater in those who practiced restrained eating (i.e. dieting to manage weight status). That is, in higher stress conditions in usual life, people ate more, with a greater predilection for fats and sugars, and this effect was particularly evident for people who practiced some form of dietary restraint. This effect has been

noted across different work settings. In a small study of women (N=14) in an office work setting McCann (2004) reports that periods of higher stress were associated with greater caloric intake, with a similar pattern of greater intake of foods high in fats.

The effects of stress on eating behaviors in younger populations indicate these patterns may be established early in life. In one of the largest studies that allows explication of the association between stress and eating behaviors, Cartwright and colleagues (Cartwright et al., 2003a) report that in adolescents (N=4320) a similar pattern of stress and hyperphagia is evident. The participants from the Health and Behavior in Teenagers Study (HABITS), a longitudinal study implemented in schools in South London, England, completed measures of their level of perceived stress over the preceding month, and a battery of food frequency measures. The results demonstrate that independently of background factors such as sex, ethnicity and socioeconomic status, adolescents with higher levels of perceived stress were more likely to have a pattern of poor health practice such as eating less fruits and vegetables, with a strong linear association between stress and eating foods high in fats. Acute periods of heightened stress characterize the lives of many school students, and as patterns of dietary restraint and balance are established in early life, they provide an insight into the development of the stress-eating relationship. Results from other studies of younger populations suggest the

relationship is borne out, evidenced in the increase in food intake during periods of heightened stress (Pollard et al., 1995; Weidner et al., 1996)

Stress Experiences and Changes in Activity Patterns

Internationally there are changing profiles of activity in young people, which are often presented as the primary reason for the increasing prevalence of obesity in the US and in many other countries. There is evidence that there is a secular trend to lower levels of physical activity (Eisenmann, 2003) but few empirical studies have examined the association between stress and physical activity levels in children and adolescents. There is equivocal support for the association between patterns of physical activity and stress. A laboratory based study of a small sample of children illustrated the potential association between exercise and stress, although the study indicates this association may be mediated by individual attributes. Children were classified as either high or low stress reactive on their reports of perceived stress, or in a separate analysis, based on their heart rate reactivity, to a videotaped speech task (Roemmich, Gurgol, & Epstein, 2003). Children then were asked to exercise with monetary reward given for longer periods of exercise. Exercise tasks were completed both on the control and the stressor day. Analyses of group differences based on differences in perceived stress indicated no effect of perceived distress, however, when grouped by physiological reactivity, the children who were more reactive

completed significantly less exercise. A series of analyses from the HABITS Project in the United Kingdom (Simon et al., 2003) also demonstrate negative associations between stress and exercise in both boys and girls, and a positive association with intake of foods high in fat in both boys and girls. These results indicate the pathway between behavioral reactivity and weight status, and provide support for stress induced changes in activity levels that associate with weight status (Roemmich et al., 2003). Brodersen and colleagues (Brodersen, Steptoe, Williamson, & Wardle, 2005), however, have shown that laboratory observations may not parlay into the naturalistic setting.

Using data from the HABITS project Brodersen and colleagues (2005) report that after inclusion of environmental predictors, such as access to facilities, age and weight status in stepwise regression analyses the amount of stress reported by the child did not predict physical activity or sedentary behavior. The access to recreational/exercise facilities is not equitable, not all children have access to venues or resources where they may be active, and so access to facilities may be serving as some other proxy measure of neighborhood or family resources (Cradock et al., 2005) . The changes in the child's desire to be active as a response to changes in perceived stress may be mediated by the resources available. This relationship of access and choice to be active is demonstrated by Epstein and colleagues (2004) who demonstrate empirically that a reduction in choice, or barriers to particular activities, may override other underlying forces.

Effects of Stress on Sleep Patterns

It is not only exercise that is predictive of obesity in adolescence; the quality and amount of sleep may also be predictive of overall health, including weight status. Adequate sleep in adolescence is considered to be approximately 6 to 8 hours per night, and obtaining adequate sleep is promotive of good health, whereas sleep deprivation is positively associated with behavior problems, emotional lability and attention problems in adolescence [e.g. (Dahl, 2004; Dahl & Lewin, 2002; Dahl et al., 1992)]. Adolescents who have inadequate hours of sleep are also more likely to be overweight (Chen, Wang, & Jeng, 2006; Sekine et al., 2002). The increasing risk of overweight status with lesser hours of sleep, especially cumulative sleep deprivation, has been explained with reference changes in the diurnal pattern of cortisol (Spiegel, Leproult, & Van Cauter, 1999). With sleep deprivation, or poor quality of sleep, there is a rise in the cortisol levels in the evening hours, a time when cortisol is usually at its lowest levels. Poor quality sleep may also have increased the cortisol responses to stress. The potential increase in cortisol, both through higher levels of cortisol secreted across the day, and higher levels secreted in responses to stress may promote weigh gain through the pathways already described. Changes in weight status linked to inadequate sleep may also be explained by dysregulations in leptin and ghrelin which regulate satiety and appetite respectively (Spiegel, Leproult et al., 2004). Leptin levels are dependent on sleep duration, and leptin has relationships

with carbohydrate regulation and with cortisol (Chen et al., 2006; Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005; Spiegel, Tasali, Penev, & Van Cauter, 2004). Sleep curtailment in healthy young men is associated with decreased leptin levels, and also with elevated ghrelin levels and subsequently with increases in hunger and appetite (Copinschi, 2005; Neff, 2005; Spiegel, Tasali et al., 2004). In a review of the trends in overweight status and the traditional predictors of weight status, Eisenmann (2003) concludes that changes in diet and activity may be necessary but are not sufficient explanations of the increasing prevalence of overweight and obesity in children, and suggests there is an alternative permissive or moderating phenomenon contributing to overweight status in children. Within the context of the model of stress and stress response, the current study postulates that activation of the HPA axis under conditions of stress, with the subsequent release of cortisol and metabolic consequences, may be implicated in overweight status.

Stress-induced Behavior and Weight Status

In this section a review of the literature examining stress and behavioral responses to it is presented, with a specific focus on the behavioral responses implicated in weight change.

The seemingly incontrovertible association between diet and exercise and weight status has recently been challenged. Eisenmann's demonstration of

the stability of caloric intake and exercise yet rising prevalence of obesity represents a paradox (Eisenmann, 2003). There are other indications that the association between caloric intake and weight status in children is not as fixed as it has been proposed (Francis, 1981; Rolland-Cachera et al., 1995; Rolland-Cachera et al., 1997, 2001). The formula of energy expenditure and energy intake provides a framework for understanding changes in weight status, but although a parsimonious explanation, it may not necessarily capture the intricacies of interactions among stress, metabolic processes and behavior that also influence weight status.

Dietary composition may be more predictive of weight status, at least in children. Diets in which a high percentage of calories comes from fat may be more predictive of overweight status than diets where the bulk of calories are derived from proteins or carbohydrates, as dietary fat is more readily stored as body fat than these other macronutrients (Bray & Popkin, 1998). This, and the children's reported preference for foods high in fat , in combination with a stress related increase in preference for high fat foods (Cartwright et al., 2003b; Simon et al., 2003; Wardle & Huon, 2000; Wardle et al., 2003) and the hormonal events that occur through childhood and adolescence (e.g. the changes in sex steroids that may promote fat deposition) (Roemmich, Clark, Berr et al., 1998; Roemmich, Clark, Mai et al., 1998; Roemmich & Rogol, 1999; Rogol, Roemmich, & Clark, 2002) may interact to accentuate the effects of behavioral responses to stress.

Although dietary factors are necessary to explain weight status, they are not the sole effectors of change in weight status, and levels of physical activity must also be considered.

Few studies have systematically examined associations between stress and physical activity in adolescents. Psychological functioning, especially depression and anxiety, have been linked to levels of physical activity, however the relationship has often been tested using cross-sectional studies, and most have assessed the benefits of exercise therapy on stress and depression, and most (Steptoe, Kimbell, & Basford, 1998; Steptoe, Wardle, Pollard, Canaan, & Davies, 1996), although not all, (Allison et al., 2005), report that physical activity is inversely related to depression.

Patterns of physical activity will influence the amount of energy stored as fat, and so compensate for increased caloric intake. In a similar fashion, those children who engage in high levels of vigorous activity require higher total calories, and may also require adjustment to the relative proportions of macronutrients. The associations between energy intake, energy expenditure and weight status is intuitive, and has been demonstrated in a series of studies of activity levels and weight status (including measures of body mass, fat mass and body mass index, (Epstein et al., 1995; Epstein et al., 1985; Goran et al., 1997; Goran et al., 1998; Hernandez et al., 1999; Obarzanek et al., 1994; Wolf et al., 1993) however, not all studies support a linear association between the two (Gazzaniga

& Burns, 1993; Treuth et al., 1998) and the relationship may be less precise than proposed by some studies. There exists a need for a holistic approach to examinations of the associations between physical activity and weight status in children and adolescents, as weight status is not necessarily only predicted by energy intake.

Responses to Stress in the Laboratory and in Natural Settings

The purpose of the next section is to describe the value of laboratory measures and measures collected in the natural setting, and to support the use of these measures in concert to explore associations between cortisol reactivity and weight status.

Laboratory stressors have many advantages, such as control over context and stressor type, but do not capture the dynamics of life, and how these dynamics may mediate stress responses. Extrapolating the dynamics of stress responses from the controlled setting of the lab to the usual daily experiences of the person is a risky undertaking, and it is generally accepted that stress response profiles indicated by lab stressors may not hold any value outside the lab. Negrao and colleagues (Negrao, Deuster, Gold, Singh, & Chrousos, 2000) suggest that the link between individual reactivity to stress determined by lab response and health outcomes is not clear. Stress profiles generated in laboratory settings may not be useful except to characterize that one person'

response in that setting, to that one particular task (Cohen et al., 2000; Manuck & Garland, 1979; Negrao et al., 2000). There is a large body of evidence indicating substantial individual differences, both within and between individuals, and a person's stress response at a given time may be dependent on recent stress experiences (Dickerson & Kemeny, 2004; Roy, Kirschbaum, & Steptoe, 2003) degree of chronic stress (Matthews et al., 2001; Schlotz, Hellhammer, Schulz, & Stone, 2004) and mood states, including positive affect (Raikkonen, Matthews, Flory, Owens, & Gump, 1999; Steptoe, Wardle, & Marmot, 2005).

One of the challenges in examining the effects of cortisol reactivity on weight status is mapping reactivity in daily life, with people in their natural environment. Daily stressors may be more useful predictors of future functioning, due to their frequent occurrence and chronic nature (DeLongis, 1982; DeLongis, Folkman, & Lazarus, 1988). Studies in children and adolescents have demonstrated associations between daily stress and lower levels of psychological well-being. Perceived stress and blood pressure in early adolescent children (Banez & Compas, 1990; Caputo, Gill, Tseh, Jamurtas, & Morgan, 2000; Schanberg et al., 2000). The functioning of the HPA axis and its transactions with context and with behavior are complex, and an individual's cortisol reactivity to a laboratory stressor may not necessarily have ecological validity. Recent models have been proposed that suggest stress responses

evoked in a laboratory setting may have limited utility in understanding characteristic stress response profiles.

Exploring the relationship between cortisol and weight status using responses to laboratory challenges as the measure is not antithetical, laboratory measures are of value in examinations of individual change in other settings. However, laboratory measures have the most value when used in concert with measures of stress collected in the individual's daily life. With some exceptions most stress reactivity work in adolescence has been completed using lab paradigms with few studies designed to explore both in parallel.

Conclusions

This chapter has reviewed evidence of the effects of stress on weight status, including an examination of the individual differences, such as sex and exercise patterns, that may mediate the association between stress, cortisol reactivity and weight status in adolescence. Also reviewed was the ecological validity of responses to laboratory stress challenges in explaining the associations between daily experiences of stress, cortisol and weight status in adolescents. The putative primary pathway between physiological response to stress and weight change is cortisol, although studies that examine the associations between stress in natural settings, changes in cortisol levels and changes in weight status are few. There is a multiplicity of potential influences on weight status,

including the behavioral and biological responses to stress. The allostatic load model variant, as proposed, incorporates the concept that stress, specifically daily hassles, predicts both changes in behavior, and in cortisol secretion, and that these changes are associated with weight status.

Chapter 3

Methods

Participants.

Participants were 78 healthy adolescents and a parent or caregiver (73 mothers, 4 fathers and 1 grandmother) who had participated in a larger longitudinal study of puberty and behavior (N= 135). The sub-sample consisted of 42 female adolescents and 36 male adolescents. The female adolescents were, on average, younger than the males, the mean age for female participants was 13.4 years (*SD* = 1.6) and males were aged 14.5 years (*SD* = 1.7). The age difference was a planned aspect of the larger study as the aim was to include boys and girls at similar stages of pubertal development and girls mature earlier than boys. The racial/ethnic composition of the adolescents based on adolescent reports was non-Hispanic Caucasian (n = 71), Hispanic (n=2), African American/Black, 2 (n =2), Asian (n=2) and Other (n =1). The family socioeconomic status (SES) and other demographic characteristics of the sample appear in Tables 3.1 and 3.2.

Sample Recruitment.

Participants were recruited from a longitudinal study *'Physiology of Puberty & Antisocial Behavior'* (NIH 5 RO1 58393-03). The sample included in the current analysis had participated in the larger longitudinal study of the relationships between hormones and problem behavior in early puberty. The design of the original study consisted of three assessments at 6-month intervals on measures that included cortisol reactivity, psychological attributes, hormones, health and physical development. Some measures of behavior obtained during the original study (e.g. depressive affect) and stress response (e.g. cortisol reactivity to a laboratory stressor) are used in the current analyses. A description of the original recruitment strategy is provided, and then the recruitment procedures used for the current study are detailed below.

Sample Recruitment for the '*Physiology of Puberty & Antisocial Behavior*' Study

The recruitment strategy for the original sample was designed to obtain a sample that was heterogeneous in terms of occupational and educational status of parents from a non-urban area. To ensure heterogeneity in the sample, the recruitment strategy consisted of obtaining a list of children from designated ZIP codes from the American Student List (ASL), a commercial enterprise that provides lists of names of school age children and youth. The list of names was

generated by ASL from ZIP codes supplied to ASL by the investigator. The ZIP codes were chosen from the county in which the research lab was located and adjacent counties that had easy accessibility to the lab. The list from ASL included the name, address, and phone number (in some cases) of children enrolled in the education system in the designated ZIP codes. Names were chosen at random from the list until all names were exhausted. A letter was mailed to the parents of the 966 children on the list. The parents then either called the lab to ask about the study or families were contacted via phone calls by the research staff to inquire if the adolescent and parent were interested in participating in the study, or, if the adolescent was ineligible, if a sibling were interested in participating. Eligibility criteria were: boys age 9 -, 11 -, or 13 - years; girls age 8 -, 10 -, or 12 - years; not on medications that would interfere with hormone levels (e.g. oral steroids); and free from chronic health problems (e.g. diabetes, cancer) or serious mental health problems that would interfere with completing the questionnaires. Children on psychotropic medications were included as eliminating these children would have excluded an important group at risk for antisocial behavior. Eighty-five children were enrolled based on responses from the parents of the children on the list. Of the remaining children, research staff were unable to contact 584 (e.g., returned letters, no published phone number, not able to contact by phone), and 89 were ineligible based on study criteria. Other reasons for nonparticipation included: discomfort with

research procedures (n = 11) such as answering sensitive questions or having a physical exam; family problems (n = 3), such as death in the family; lack of time (n= 41); or not interested (n= 39). One family gave multiple reasons (e.g., lack of time and also not interested).

The remaining participants were obtained from flyers distributed throughout the community and from parental telephone response to e-mails distributed to staff at a large university. These efforts resulted in an additional 48 families contacting the research project and of these contacts, 26 participated. Of the non-participants from these off-list families, research staff was unable to re-establish contact with one family and seven were ineligible to participate. Other reasons for nonparticipation included: discomfort with research procedures (n = 6) such as answering questions or having a physical exam; lack of time (n= 1); or not interested (n =5). Two families did not give a reason.

The original sample is heterogeneous with regard to occupational status and the education level of the sample also is diverse. The sample has a disproportionate number (54%) of families employed at the university. However, this statistic is representative of the number of county citizens employed by the university. In brief, the sample is of interest as it is heterogeneous with regard to occupation and educational status and the sample was drawn from non-urban, large-city communities.

If the adolescent and a parent or guardian were interested in participating, a parent was administered a telephone screening interview by a pediatric nurse or a graduate student to establish the adolescent's eligibility for the study. The screening interview also asked questions about the child's overall health, behavior problems, use of prescribed and over-the-counter medications and school performance.

Sample Recruitment to the *'Hassles and Health'* Study

A letter extending an invitation to participate in further research sessions was mailed to all families who had participated in the original study of relationships between hormones and problem behavior in early puberty. This initial contact was followed by a telephone call from a researcher, who offered detailed information on the study, determined eligibility for the study and ascertained interest in participating.

Of the 135 adolescents enrolled in the original longitudinal study, 125 completed the second time of measurement and 123 completed three times of measurement. Of these 123, 78 agreed to participate in the current study. Analyses of differences by t-test procedures between the current group (N=78) and those who chose not to participate, indicated no significant differences in

demographic characteristics, sex, age, measures of psychosocial characteristics or cortisol reactivity to a laboratory stressor.

The protocols for both the '*Physiology of Puberty & Antisocial Behavior*' and the *'Hassles and Health'* study were approved by The Pennsylvania State University Institutional Review Board (IRB # 21374) and the advisory committee of the GCRC. All methods and procedures were executed in accordance with a written protocol.

Methods

Procedures

The adolescent and their parent/care-giver completed an initial research ('Home') session conducted either at the family home, or at the PSU General Clinical Research Center (GCRC), according to the family's wish. During this session, the researcher explained the procedures, then instructed the adolescent and parent in the completion of the measures to be collected at the adolescent's home, detailed below, including collection of saliva samples and activity monitors. The adolescent and parent completed pen and paper questionnaires. The date on which the home collection period was to begin was confirmed at this session, and the research session scheduled. A schematic of the data collection time-line

procedure is presented in Figure **3-1** . Female participants were scheduled for the research session between day 5 and 9 of the girl's menstrual cycle to minimize sex differences.

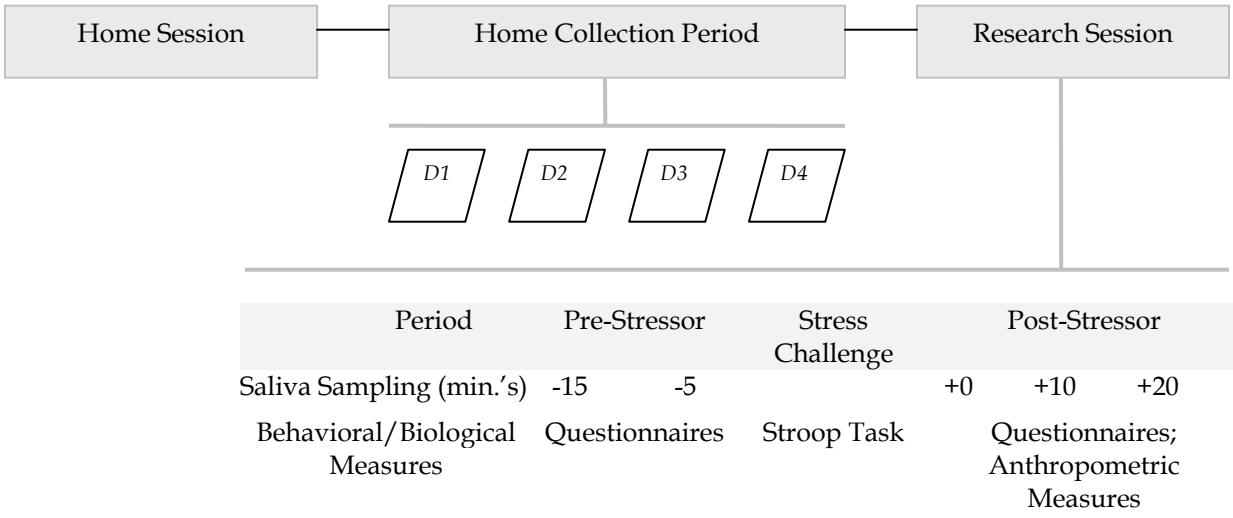

Figure **3-1**: The data collection procedures. The Home Session was scheduled no more than one week prior to the Home Collection Period. The Research Session was scheduled no more than one week after completion of the Home Collection.

On the evening preceding Day 1 of the home collection period for the adolescent a reminder call was made to the adolescents and/or their parent/guardian to remind them of the collection procedures for the following day. These reminder calls were made to participants on each afternoon or evening for the four-day collection period, unless the family requested not to

receive the reminder call. During the home data collection period, consisting of 4 consecutive days, 2 school and 2 non-school days, the adolescent completed measures of daily experiences, collected saliva samples and completed pen and paper questionnaires. The parent/care-giver also completed pen and paper questionnaires during this period.

The second research session ('Research Session') was conducted either at the family home, or at the PSU General Clinical Research Center, according the family's wish. The sessions were scheduled at 4:00 p.m. ($\pm$ 1. 5 hours) depending on the adolescent's activity schedule. During the research session, the adolescents and their parent completed questionnaires and structured interviews that collected information about the adolescent's traits, emotional state, behavior and health. The adolescent collected saliva samples during the research session, and completed a stress challenge task, the modified Stroop Task (Antonius, Nolan, & Weiss, 2001; Stroop, 1935). A series of measures of the anthropometry of the adolescent was also obtained, and the adolescent completed a self-report measure of pubertal status. The parent/guardians also completed measures that collected information about their emotional state. Only the measures germane to the theoretical model postulated in the current study are described herein.

Measures

Socioeconomic Status

Family socioeconomic status (SES) was assessed using the Hollingshead scale (Hollingshead, 1975.), a scale based on survey data of social status tied to U.S. census categories. The participating parent reported on the education and occupation of the parent/caregivers in the household. For each head of household, education (1 = "less than seventh grade" to 7 = "graduate school") is multiplied by a weight of 3 and occupation (1 = farm labor and "menial service" to 9 = executives and major professionals) is multiplied by a weight of 5. Thus, the range of possible SES scores is from 8 to 66 (Hollingshead, 1975). For one-parent households, the education score and occupation score were summed to yield an overall SES score. In two-parent families, with one parent in paid employment, the family SES score was derived by adding the mean of the parents' education scores to the occupation score for the employed parent. If both parents were employed outside the home, the family SES score was the mean of the parents' SES scores. The family SES was heterogeneous, ranging from unemployed to professionals, (range 21 – 66, mean 46.9, SD 12.4).

Daily Experiences Diary

The Daily Experiences Diary (DED) was developed to assess the range of experiences and feelings an adolescent might have over the course of a day (Bolger, Davis, & Rafaeli, 2003; Fuligni, Yip, & Tseng, 2002). The DED has 7 sections, including sections that collect information on sleep, activities, daily experiences and emotional states. The DED is presented in Appendix A.

Sleep.

Participants recorded the number of hours and minutes they had slept the previous night. This was used as the total time of sleep.

Daily Experiences.

The list of daily experiences was based on existing measures of daily hassles and interpersonal interactions experienced by adolescents, that includes stressors such as family demands on time and negative interpersonal interactions (Fuligni et al., 2002; Fuligni & Zhang, 2004) extended to include other daily stressors such as time demands and work overload (Almeida, 2004; Almeida, Wethington, & Kessler, 2002; Grzywacz, Almeida, Neupert, & Ettner, 2004; Mroczek & Almeida, 2004) and peer and academic hassles (Walker, Garber, Smith, Van Slyke, & Claar, 2001) . The daily experiences listed 29 items in total,

including items such as "Harassed, picked on, or teased by a student in school", "Did poorly on a test, quiz, or homework "Had a lot of demands made by your family". Of the 29 items, 17 items formed the Hassles scale. The other 12 items listed positive experiences (e.g. "Something good happened to you or you were treated well") and were not used in the current analyses. For each item in the Hassles Scale, participants indicated if and when (morning, afternoon, evening) the experience had occurred. It was possible for participants to indicate the same experience at more than one item in the day, for example they could report an argument with another person in the morning, afternoon and evening. The items were summed to create a Daily Hassles Scale, with possible scores ranging from 0 to 48 for each day.

Daily Emotions.

A list of 30 items measured the extent to which participants felt anxiety (e.g. couldn't concentrate), depressive feelings, (e.g. hopeless, sad), happiness (joyful) and achievement (e.g. successful). The list of items was derived from Fuligni's measure (Fuligni et al., 2002) and drew on the diary of stress and coping in adolescents by Bolger and Zuckerman (Bolger & Zuckerman, 1995). For each item participants reported the extent to which they had experienced each item using a 5-point scale (1 = not at all, 5 = extremely) in the morning, day and evening of each day. Principal components analysis of the daily emotions

yielded 76.1% of the total variance accounted for by five factors; "Internal Source Positive Emotions", "External Source Positive Emotions", "Internal Source Negative Emotions", "External Source Negative Emotion" and "Anxious/Depressed". The "Anxious/Depressed" factor is used in the current analyses as a measure of anxiety and symptoms of depressed affect for the day given the interest in associations of depression with cortisol and daily hassles.

Cortisol

Saliva offers a noninvasive route for the measurement of the unbound fraction of cortisol, and is highly correlated with the circulating unbound fraction, r = .54 to r = .97) with changes in plasma levels reflected in salivary samples within minutes (Kirschbaum & Hellhammer, 1989; Kudielka, Buske-Kirschbaum et al., 2004b). At the Training Session each adolescent and parent was trained in the protocol for collection of saliva at home. Participants were instructed to collect samples by passive drool into a 5 ml tube. The tubes were labeled by day and time of collection (e.g. Day 2; 4p.m.) and were presented in a holder that arrayed the tubes in sequence for the designated collection times. Tubes were also color-coded to the day of collection (e.g. red = Day 4) to minimize mistakes in collection. Participants were instructed to rinse their mouths with water before passively drooling into a 5 ml tube and to collect saliva to the 4 ml mark on the tube within five minutes and then put the tube in

the refrigerator. Participants were requested not to eat or drink (except water) during the collection procedure, and not to have caffeine, meals or exercise in the 2 hours prior to collection.

Five samples of saliva were collected on each sampling day. The timing of each sample was designated to capture the morning rise in cortisol levels, and to indicate the expected afternoon decline and nighttime nadir (Kirschbaum & Hellhammer, 1989, 1994; Kudielka, Schommer et al., 2004; Pruessner et al., 1997; Wust, Wolf et al., 2000b). Three samples of saliva were collected at 20-minute intervals upon awakening and prior to breakfast or teeth-brushing: Sample 1 was obtained immediately on wakening, Sample 2 at 20 minutes post-wake time, Sample 3 at 40 minutes post-wake time. Additional samples were collected at 4:00 p.m. and at 9 p.m. Participants and/or their parents recorded the time of collection for each sample on the collection form and also in the Daily Experiences Diary.

Saliva samples were refrigerated (≈4^{o} C) after collection at home and later transferred to a -70^{o} C ultra low freezer until assay. All samples were assayed using a highly sensitive enzyme immunoassay specifically designed for use with saliva (Cat. No.1-0102/1-0112, Salimetrics, PA). The test requires only 25 µl. of saliva (for singlet determinations) and has a range of sensitivity from 0.007 to 1.2 μg/dl, and average intra- and interassay coefficients of variation (CV) of 5.34% and 9.86%. The standard curve was highly reproducible (mean R^2=. 993). In each

assay, controls representing low and high salivary levels were included, and all samples and controls were assayed in duplicate and the average used in analyses. Where the duplicate test value varied by more than 7% error, the sample was re-assayed.

Cortisol levels for each day were calculated using an area under the curve formula (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) which provides a representation of total cortisol secretion for the day of interest. Cortisol reactivity to a laboratory stressor, the Stroop Test, was calculated as AUC-*I*, indicating the area under the curve increase of cortisol after the administration of the laboratory stress challenge. Using the method of Pruessner and colleagues, (Pruessner et al., 2003), change in salivary cortisol was calculated by averaging Samples 1 and 2, to provide a baseline measure, and then Samples 3, 4 and 5 were entered as subsequent time points, that is, post-stressor. In contrast to total area under the curve, this formula deletes the distance from zero for all measurements, thus changes subsequent to an event are emphasized (see Eq. **3.1** and Eq. **3.2**) .

$$\text{AUCGround} = \left(\sum_{i=1}^{n-1} \frac{(m_{(i+1)} + m_i) \cdot t_i}{2} \right) \qquad \textbf{3.1}$$

Eq. 3.1 The formula for calculation of Area Under the Curve with respect to ground. Individual time between measurements is denoted as t_i, individual measurements are denoted as m_i and n denotes the total number of measurements.

$$\mathrm{AUC}I = \left(\sum_{i=1}^{n-1} \frac{(m_{(i+1)} + m_i) \cdot t_i}{2} \right) - \left(m_1 \cdot \sum_{i=1}^{n-1} t_i \right) \qquad \textbf{3.2}$$

Eq. 3.2 The formula for calculation of Area Under the Curve with respect to increase. This formula is derived from the formula for AUCGround. The area between ground and the first measure is removed for all time points is removed to capture change form the ground measurement.

Stroop Challenge

The Stroop Task was used as a stressor to evoke a cortisol response (Dickerson & Kemeny, 2004). The Stroop Task presents participants visual stimuli, to which participants are required to provide a correct verbal response (Antonius et al., 2001; Stroop, 1935). There were two parts to the task. Challenge A presented congruent and incongruent color-word stimuli. Participants were required to correctly identify the color of the word that was presented on the screen, responses that indicated the textual content are considered incorrect. Challenge B presented congruent and incongruent directional stimuli (e.g. left, top). Participants were required to correctly identify the direction indicated by location (the congruent direction), not the word that was presented. Each Challenge was presented twice, in a series of timed trials; each trial ran for 2 minutes and twenty seconds. The total time of the Stroop Task, inclusive of instructions and an understanding check, was 15 minutes. Challenge A requires that the participant have full color vision. All students enrolled in schools in Pennsylvania are tested for color vision. Prior to

instructing the participant on Challenge A, participants were asked if they had ever been told they have color vision impairment ("color blindness"). No participant reported having color vision impairment.

Body Mass Index

The average of three measures of height and weight were used. Measures were obtained by the researcher with participants wearing light clothing without coats, jackets or shoes. Height was measured to the nearest 0.1 cm using a stadiometer (SECA Equipment) using standard techniques (Frishanco, 1990). Participants were weighed to the nearest 0.1 gram on a digital scale (Model BC533, Tanita Corporation, USA). BMI was calculated using the standard formula of weight (kg)/height^2 (mm), and used to calculate the age and sex specific BMI for age percentile. In this sample, 6% of the sample was underweight (<5th percentile) and 21% of the sample was overweight, with 15% of the adolescents between the 85th and 95th percentile, or at risk for overweight, with 6% above the 95th percentile for weight according to the CDC (2000) growth charts, from the National Health Interview Survey (Kuczmarski et al., 2002).

Percentage of Body Fat

Percentage body fat was determined by using a function on the digital scale (Model BC533, Tanita Corporation, USA). This function uses electrical impedance and an algorithm preset in the scale. The average of three measurements was calculated. The mean body fat percentage for this sample was 27.1 % with a SD of 14.3%.

Waist Circumference

Waist circumference measurements were obtained by measuring the total circumference of the torso at a level equidistant between the iliac crest of the hip and the lower rib cage. Measurements were taken over light clothing**.** The average of three measurements was used in analyses. The mean waist circumference for this sample was 23.9 c.m., with a SD of 10.54.

Pubertal Stage.

Pubertal Stage was assessed by self-report. The adolescent was provided with pictures of the five stages of puberty (Marshall & Tanner, 1968, 1969, 1970) and an explanation of the five stages depicted. The adolescent then independently assessed which picture most closely represented their level of development for both pubic hair stage and breast or genital stage.

Diagnostic Interview for Children

The Diagnostic Interview for Children (DISC-IV) is a structured interview used to assess symptoms of psychiatric disorders in children and adolescents in accordance with DSM-IV criteria (Lucas et al., 2001; Piacentini et al., 1999a; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The DISC is a commonly used and completely pre-constructed, computerized interview, administered by a researcher. It incorporates approximately 2500 questions, including 358 stem questions, with contingent questions used to ascertain the duration and severity of symptoms. The DISC has been reported to be reliable and accurate in the diagnosis of psychiatric symptoms, and has been shown to be even more reliable in diagnoses than those given by clinicians (Angold & Fisher, 1999).

There are parallel versions for parents (the DISC-P for 6 - to 17-year-olds) and youth (the DISC-Y for 9 - to 17-year-olds). The DISC-Y was administered to all adolescent participants. Parent reports are used in the current analysis given the higher reported correlation of parent scores across time (Piacentini et al., 1999b). In the current analysis only the section measuring symptoms of depression was used.

Child Behavior Checklist

The behavior of the adolescent over the six months prior to completion of measures was assessed with the Child Behavior Checklist/4-18 (CBCL), a norm-referenced behavior rating scale completed by parents (Achenbach & Edelbrock, 1983). Parents rate the adolescent's behavior on a 3-point scale for 113 behavioral and emotional problems that have occurred during the past six months (0 = not true , 1 = somewhat or sometimes true, 2 = very true or often true). In the current analyses, the internalizing behavior subscale 'Withdrawn/Depressed' at the first occasion of measurement, that is, the first time of the original study. The Cronbach's alpha for this scale for this sample was .97. This scale was selected for use given the reported associations between depressed affect and cortisol as already discussed. The normative mean T-score for the scale is 50, with a standard deviation of 10; these scores are normed separately for male and female adolescents based on a nationally-representative sample. The T score on the Withdrawn/Depressed CBCL scale for this sample is 53.10, and is comparable to the published norms (Achenbach, Verhulst, Baron, & Althaus, 1987).

Child Depression Inventory

The Child Depression Inventory (CDI) (Kovacs, 1985) was used to assess cognitive, affective, and behavioral symptoms of depression. The pen and paper

measure indicates levels of depression on a continuum and so captures sub-clinical levels of symptoms of depression. The CDI has 27 items, and each item consists of three statements graded in order of increasing severity from 0 to 2. The total score ranges from 0 to 54. Children select one sentence from each group that best describes themselves for the past 2 weeks (e.g., "I am sad once in a while," "I am sad many times," or "I am sad all the time"). The CDI has high levels of internal consistency in non-clinical populations (Smucker, Craighead, Craighead, & Green, 1986) . In the current sample, Cronbach's alpha was .91.

Accelerometry Measurements

The Actigraph accelerometer (Model AM7164-2.2C, Manufacturing Technologies, Inc., Fort Walton Beach, FL) was used to measure activity across the four days of data collection. The Actigraph is a small (5.1 × 3.8 × 1.5 cm) accelerometer unit that measures movement and stores data over specified time intervals. Acceleration detection ranges from 0.05 to 2.00 g in magnitude and the frequency responses ranges from 0.25 to 2.5 Hz, so that motion outside normal human movement is rejected by a filtered bandpass. The acceleration-deceleration signal is numerically integrated, and signals were collected 10 times per second over the defined 30 second epoch interval. The rate of change of acceleration is sampled and the data were stored in the internal memory; then the integrator is reset to zero for the next 30 second epoch. A real-time internal

clock allows the data collection time and date to be pre-set, and the Actigraph was programmed to start data collection at 4 a.m. on Day 1 of the data collection period to ensure any activity of early risers was captured. The output from the Actigraph is recorded in counts per epoch. Counts represent the summed amount and magnitude of acceleration during each 30-second epoch. That is, higher numbers represent a combination of higher frequency and intensity of movement. The resultant data were reduced by calculating total activity counts (counts per epoch expressed as minutes) for each 24 hour period. The total minutes spent in different levels of activity, vigorous, moderate, sedentary was then calculated for each 24 hour period. The total activity counts for the four days of data collection were calculated. The Actigraph provides a reliable and valid measure of physical activity (Acebo et al., 1999; Jean-Louis et al., 1997; Le Masurier, Keup, & Tudor-Locke, 2004; Patterson et al., 1993)

Analysis Strategy

Three separate analytical strategies were employed to address the study's hypotheses. Hypotheses 1, 2 and 3 were tested with a series of hierarchical linear regression models using procedures in SPSS. Hypothesis 4 was tested by determining the stability of cortisol patterns using a procedure for determining the likelihood of the cortisol secretory patterns remaining similar across different times of measurement using procedures in both the SAS and SPSS packages.

Hypothesis 5 was evaluated using a multi-level modeling approach in HLM to evaluate intra-individual and inter-individual patterns of cortisol secretion.

Testing Mediation Models: Hypotheses 1, 2 and 3.

A series of two hierarchical regression models, applying the methods of Baron and Kenney (Baron & Kenny, 1986) were used to determine any significant associations between the predictor and outcome variables, and the associations between these and the mediating variables for hypotheses 1, 2 and3. These hypotheses were tested separately in males and females. To test for mediation, the first model tested for effects of the predictor on the outcome, after inclusion of any control variables. The second model tested for effects of the mediator on the outcome, and the third model tested for effects of the predictor on the mediating variable. These first three models were used to establish direct correlational associations between the variables. Lastly, the final model tested for the change in the magnitude of effect of the predictor on the outcome after the inclusion of the mediator. If the significant direct association between the predictor and outcome variables declines (after including both the mediator and predictor variable) the obtained pattern was considered consistent with the mediation hypothesis. If the direct effect of the predictor approaches zero, the mediator was considered to completely, although not necessarily exclusively,

account for the relation between the predictor and the outcome (Baron & Kenny, 1986)

Specifically, Hypothesis 1 was tested using bivariate correlations between responses to daily hassles, cortisol and body mass index. The variables of sex, age and pubertal stage were also included in the correlational analysis. Then, the first model, (Model 1) tested for the direct effects of daily hassles on body mass index, in a hierarchical linear regression model, with sex, age and pubertal stage entered in steps 1, 2 and 3 respectively before the addition of daily hassles to the model. Model 2 tested for the direct association between cortisol reactivity and body mass index, and Model 3 tested for an association between daily hassles and cortisol. The final model included sex, age and pubertal stage in steps 1, 2, and 3 respectively, followed by cortisol reactivity in step 4, and finally by daily hassles. The utility of retaining each component of the model was assessed by examination of the significance of change in variance explained for each subsequent step of the model.

Initial examination of the bivariate correlations between cortisol reactivity, activity patterns, daily cortisol secretion and body mass index for Hypothesis 2 indicated significant correlations between the variables. Then, the first model (Model 1) tested for the direct effects of cortisol reactivity on body mass index, in a hierarchical linear regression model, with age and pubertal stage entered in steps 1 and 2 before the addition of cortisol reactivity to the model. Model 2

tested for the direct association between activity patterns and daily cortisol secretion on body mass index, and Model 3 tested for effects of cortisol reactivity on activity patterns and daily cortisol secretion. The final model included age and pubertal stage in steps 1, 2, and 3, followed by activity patterns and daily cortisol secretion in steps 4 and 5, and finally by cortisol reactivity. The utility of retaining each component of the model was assessed be examination of delta R^2 for each new step of the models. This approach was also used to test the mediating effects of activity levels on the associations between cortisol reactivity and body fat percentage and the mediating effects of activity levels on the associations between cortisol reactivity and waist circumference.

Initial examination of the bivariate correlations between childhood vulnerability, operationalized as childhood cortisol reactivity and symptoms of depression, stress experiences, in the form of daily hassles, cortisol reactivity and body mass index for Hypothesis 3 indicated associations between the variables. The variables of age and pubertal stage were also included in the correlational analysis. Then, the first model (Model 1) tested for the direct effects of childhood cortisol reactivity and symptoms of depression, in a hierarchical linear regression model, with sex, age and pubertal stage entered in steps 1, 2 and 3 respectively before the addition of childhood cortisol reactivity and symptoms of depression in steps 4 and 5 respectively to the model. Model 2 tested for the direct association between daily hassles and body mass index, and Model 3 tested for

effects of childhood cortisol reactivity and symptoms of depression on daily hassles. The final model included sex, age and pubertal stage in steps 1 , 2, and 3, followed by cortisol reactivity in step 4, and then childhood cortisol reactivity and symptoms of depression in steps 5 and 6. The utility of retaining each component of the model was assessed be examination of delta R^2 for each new step of each regression model.

Testing the Stability of Cortisol Trajectories

Hypothesis 4 was tested using a developmental trajectory model developed by Nagin (Nagin, 1999, 2005). A series of two analyses were performed; the first analysis determined the stability of the trajectory across the four consecutive days of cortisol collection (Days 1, 2, 3, 4 of the current data collection period), the second determined the stability of the cortisol trajectory over development using the previously collected (Times 1, 2, and 3) and current Time 4 data. The first step of these analyses determined patterns of cortisol trajectories using all obtained cortisol values, and indicates groups of patterns and the proportion of the population in each group. The optimal number of groups was selected by running a series of models starting with a one-group model. Subsequent models were compared using the change in the Bayesian Information Criterion (BIC) to evaluate change in model fit (Jones, Nagin, & Roeder, 2001.). Negative changes in BIC suggest decrements in fit. The output of

the model provided a set of conditional probabilities for group assignment for each participant.

As this approach does not allow absolute identification of group membership, participants were assigned to a group for which they has the largest probability estimates for each period of interest (i.e. Days 1, 2, 3 and 4; Time, 1, 2, 3 and 4). Following group assignment the stability of trajectories within individuals, across time periods, was evaluated in two ways. The first approach used cross-tabulation procedures to evaluate the stability of group assignment. In this approach, the group assignment on Day 1, for analysis of consecutive day trajectory, and Time 1, for the analysis of development of diurnal trajectory (Times, 1, 2, 3, and 4) was determined. Group assignment at Day 1, for analysis of consecutive day trajectories, and Time 1, for analysis of data from Times, 1, 2, 3 and 4, was used in inter-correlation procedures to evaluate group membership probability stability from the first time of measurement for each period).

Intra-individual and Inter-individual Patterns of Cortisol Secretion.

This analysis tests Hypothesis 5, and addresses research objectives aimed to describe and analyze the change in daily cortisol secretion over a four-day period with reference to the type of daily experiences the adolescent reported, and the hours of sleep obtained, and to explore the effects of activity levels and

intensity of cortisol response to a stressor on the associations between daily cortisol secretion, hassles and sleep. The analyses applied individual growth models using HLM (Bryk & Raudenbush, 1992). In these analyses separate (first level) linear models are fitted for each predictor. These models are then linked together by a second-level model in which the regression coefficients of the first level model are used as outcomes and the explanatory variables are used at the second level (Kreft & de Leeuw, 1998). The multilevel model was obtained using the Hierarchical Linear Model (HLM) framework of Raudenbush and Bryk (2002). Bryk and Raudenbush (Bryk & Raudenbush, 1987) proposed that individual change can be conceptualized using a two-level hierarchical model where, in level-1, each individual's development is represented by his or her own trajectory of change that depends on a unique set of parameters (i.e. hassles and sleep). These individual growth parameters were then used as outcomes in the level-2 model, where they were regressed on person level characteristics (e.g. sex, cortisol reactivity and activity levels). Participants were grouped into high or low cortisol reactivity, by percentile ranking, with those above the 75th percentile classified as 'higher' reactors, and those below the 75th percentile as low reactors (Tout et al., 1998). The unique relationship between daily hassles and AUCg was represented by a within person slope generated using HLM. Higher slopes represented a stronger association between amount of cortisol secreted across a day and the number of daily hassles. The effect of sex, cortisol reactivity (higher

or lower) and activity levels on the intercept and slope for these associations were then examined.

Chapter 4

Results

The results of the descriptive and hypothesis testing analyses are presented in this section. The data are first described with descriptive statistics, intercorrelations among the variables, and statistical concerns are addressed. The order of the analyses is structured by the hypotheses. Specifically, a summary of the mediation models is described and then the results for linear regression analyses for these models are presented. Next, the results for the trajectory analysis of diurnal cortisol secretion are presented, followed by the results of analyses for the stability of patterns of cortisol secretion across time. Finally, the results of the multi-level model analyses of patterns of daily cortisol secretion and daily hassles are presented.

Preliminary Analysis

The distribution of all variables was examined to determine the need for transformations and outliers. Body mass index, cortisol reactivity and reports of hassles experienced are often reported to vary with sex, age, socioeconomic status (SES), race and pubertal stage and so bivariate correlations were calculated to assess the need to include the latter variables as covariates in further analyses.

BMI

The BMI scores ranged from 15.2 to 43.2, with a mean BMI of 22.8. The mean BMI for males was 23 (sd = 65) and for females the mean BMI was 22 (sd = 5) The distribution of BMI for this sample are presented in Figure **4-1** and Figure **4-2** for males and females respectively.

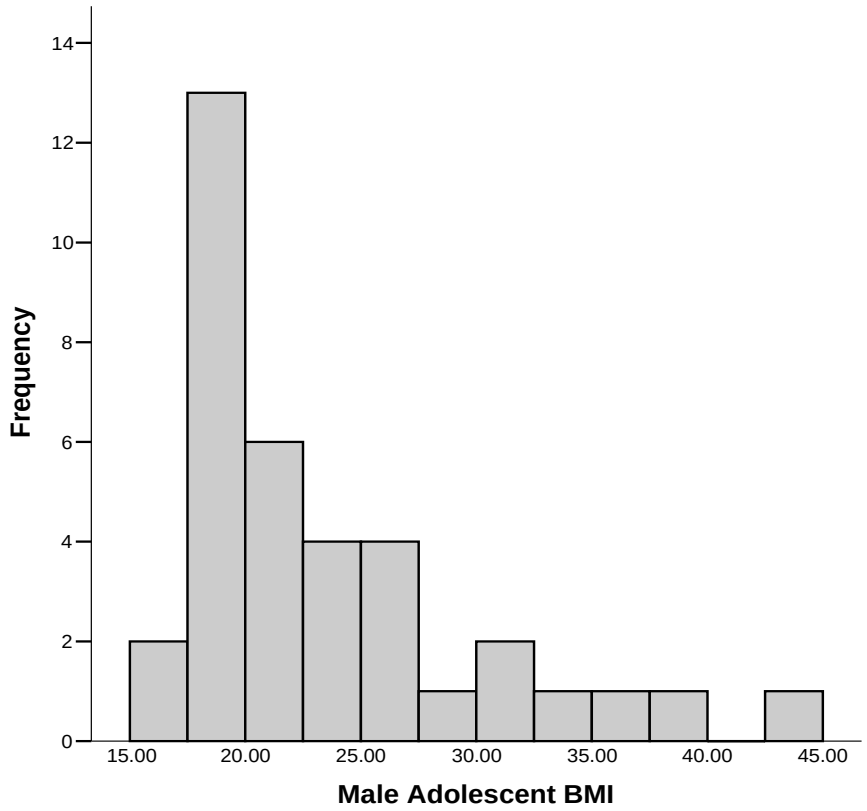

Figure **4-1**: The distribution of BMI in male adolescents. The range of scores for males was 16 – 43.

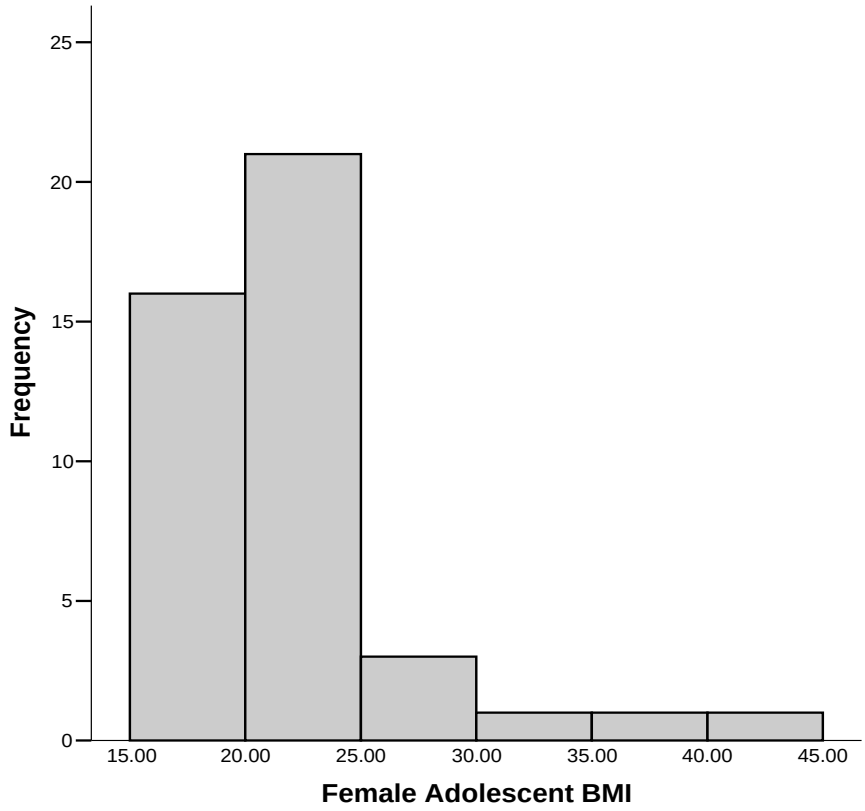

Figure **4-2**: The distribution of BMI in female adolescents. The range of scores for males was 15 - 42.

Sex

Examination of the bivariate correlations for the sample (see Table 1 in Appendix B) indicates there are few associations between variables, however, when the correlations are examined for males and females separately, correlations were revealed (see Table 4.1). Analysis of mean differences between males and females after applying a Bonferroni correction for the number of tests

indicate there are differences only in Activity Level on Day 2 (see Table **4-3**). However, the different pattern of correlations for males and females prescribed that the contribution of sex to patterns of relationships between variables be examined. Within-sex differences thus were evaluated by running separate regression models for males and females for tests of hypotheses when mediation was examined, or by the inclusion of sex as a predictor in analysis of trajectories and the multi-level models of daily hassles and patterns of cortisol secretion.

Table **4-1**: Correlations between Variables[1] by Gender

		1	2	3	4	5	6	7	8	9	10	11
1	Childhood BMI	-	.65**	.63**	.56**	.10	-.09	.00	.57**	-.25	-.11	-.08
2	Adolescent BMI	.91**	-	.96**	.94**	.05	-.40*	-.13	.05	.09	.23	-.16
3	Body Fat %	.82**	.91**	-	.91**	.05	-.35	-.18	.14	.06	.27	-.14
4	Waist Circumference	.85**	.94**	.84**	-	-.04	-.47**	-.15	.07	.11	.24	-.14
5	Total Daily Hassles	-.22	-.22	-.22	-.24	-	.17	-.03	.41*	.19	.20	.23
6	Total Activity Count	-.50**	-.52**	-.45*	-.53**	.43*	-	-.02	-.01	.02	-.12	.09
7	AUC*I* Childhood	.35	.28	.33	.22	.29	.17	-	-.02	-.06	-.14	-.06
8	AUC*I* Adolescence	.56**	.42*	.42*	.37*	-.23	-.38*	.24	-	.07	-.02	-.03
9	Childhood Symptoms of Depression - DISC	-.15	-.12	-.01	-.22	.13	.18	-.13	-.17	-	.54**	.12
10	Childhood Child Report Depression - CDI	-.07	-.01	.10	.03	.04	.06	-.13	-.34	.33	-	.56**
11	Childhood CBCL Withdrawn/Depressed	.54**	.51**	.50**	.45*	-.25	-.39*	.48**	.15	.08	.23	-

Note. * $p < 0.05$, ** $p < 0.01$; Correlations presented above the diagonal are for female adolescents, correlations presented below the diagonal are for male adolescents.

1. Only variables for which associations were tested separately for males and females are presented.

Age

As discussed, age is a possible confound with several variables in the models, and so correlations between age and the variables of cortisol reactivity, daily hassles and BMI were examined. Age was not associated with any of these three variables.

Socioeconomic Status

Socioeconomic status may also be a confound with several variables in the models, and so correlations between SES and the variables of cortisol reactivity, daily hassles and BMI were examined. Socioeconomic status was not associated with any of these three variables.

Race

The majority of participants identified themselves as non-Hispanic White and so the numbers of participants in each group disallows testing for differences between each group. The non-White participants were subsequently grouped, and mean differences between this group and the non-Hispanic White group were examined. The results indicate that no variable differed significantly by race. This does not preclude the possibility that relationships between

variables could vary by race, however, the small number of participants who identified as non-White disallows most analytic approaches, including by reason of severely limited statistical power, and so race was not examined further in the analyses.

Pubertal Stage

Examination of the correlations between pubertal stage and timing of puberty indicated that pubertal stage was not consistently associated with reported hassles. However, pubertal stage and reports of interpersonal hassles in the morning by boys were significantly correlated (r = - 0.39, p <.05), and for girls, pubertal stage and reports of positive interpersonal interactions were significantly correlated (r = 0.21, p <.05). The number of males and females in each pubertal stage, based on self-report are presented in Table **4-2.** Given these significant correlations, the potential of pubertal stage as a confound in further analyses was accepted, and so pubertal stage was included as a covariate in all hypothesis tests.

Table **4-2**: Chi-square tests for composition of sample for males and females by pubertal stage and race.

	N		χ^2	*p*-value
	Males	Females		
Race			53.09	.00
Non-Hispanic Caucasian	35	38		
Hispanic	1	1		
African American	0	2		
Asian	1	1		
Other	0	1		
Pubertal Stage			35.05	.00
Stage 1	1	0		
Stage 2	7	12		
Stage 3	8	18		
Stage 4	16	9		
Stage 5	4	2		
Refused	1	1		

Cortisol

The sets of cortisol values had non-normal distributions (skew > |1|) and so raw values of cortisol were log-transformed. Reported values in tables and figures are raw values, unless noted as otherwise.

Descriptive Statistics and Correlations

Means and standard deviations are shown in Tables **4-3** and Table **4-4**. All variables were tested for sex differences using *t*-test procedures. As described in the methods section, as a group, male participants were older when first enrolled in the study and continue to be older.

Table **4-3**: Means and Standard Deviations of Psychosocial Variables by Sex and *t*-tests for Gender Differences

	Mean (*SD*)		Gender *t*-test	
	Boys	Girls	*t*	*p*
Age	14.5 (1.7)	13.45 (1.6)	2.9	.006
Hollingshead SES	48.4 (10.6)	45.8 (13.9)	.765	.447
T1 DISC Symptoms of Depression	.17 (1.01)	.15 (.93)	-.46	.64
T1 CBCL Withdrawn / Depressed	1.3 (1.6)	1.0 (1.2)	.76	.446
T4 STAI Trait Anxiety	37.3 (8.5)	37.7 (9.5)	-.23	.810
Day 1 Total Activity Counts	401631 (201598)	352655 (196949)	.859	.395
Day 2 Total Activity Counts**	370763 (142528)	246835 (110836)	3.378	.05
Day 3 Total Activity Counts	336221 (180222)	281251 (123982)	1.204	.235
Day 4 Total Activity Counts	252948 (121354)	306521 (127266)	-1.317	.196
Day 1 Sleep (hours, minutes)	8.15 (1.33)	8.48 (1.17)	-.89	.37
Day 2 Sleep (hours, minutes)	7.20 (1.50)	8.8 (1.53)	-2.5	.08
Day 3 Sleep (hours, minutes)	8.8 (1.48)	8.03 (1.51)	2.08	.09
Day 4 Sleep (hours, minutes)	9.50 (2.00)	8.57 (2.58)	1.6	.111
DED Daily Hassles Day 1	1.6 (2.1)	2.1 (2.9)	-.59	.55
DED Daily Hassles Day 2	2.1 (2.2)	1.9 (2.4)	.25	.79
DED Daily Hassles Day 3	2.4 (4.2)	2.7 (2.9)	-.43	.69
DED Daily Hassles Day 4	1.8 (3.5)	2.5 (3.1)	-.77	.44
Day 1 DED Anxious / Depressed	8.1 (3.1)	8.7 (2.6)	.54	.58
Day 2 DED Anxious / Depressed	8.2 (2.3)	8.2 (2.1)	.12	.89
Day 3 DED Anxious / Depressed	6.9 (1.6)	7.3 (2.1)	1.4	.15
Day 4 DED Anxious / Depressed	7.5 (1.02)	7.8 (1.9)	1.7	.10

Note. Asterisks indicate mean gender difference by *t*-test, *= p <.05, ** = p < .01

Table **4-4**: Means and Standard Deviations of Biological and Anthropometric Variables by Sex and *t*-tests for Gender Differences.

	Mean (*SD*)		Gender *t*-test	
	Boys	Girls	*t*	*p*
Adolescent Body Mass Index	23.3 (6.2)	22.2 (5.2)	.9+04	6.64
Waist Circumference (cm)	80.1 (15.1)	74.1 (12.9)	1.863	1.08
Body fat (%)	19.8 (11.1)	26.7 (8.6)	-3.03	.05*
Childhood Body Mass Index	20.2 (5.4)	19.1 (2.7)	1.09	5.04
Childhood Time One AUC*I* Laboratory	11.90 (10.08)	13.34 (11.82)	-.458	11.52
Adolescent Time Four AUC*I* Laboratory	19.95 (6.30)	18.47 (8.24)	.873	6.93
Time 4 Day 1 AUCg Cortisol	778.15 (269.11)	841.19 (364.53)	-.851	6.87
Time 4 Day 2 AUCg Cortisol	810.14 (292.75)	821.36 (373.54)	-.145	15.83
Time 4 Day 3 AUCg Cortisol	947.97 (393.39)	981.36 (384.08)	-.378	13.86
Time 4 Day 4 AUCg Cortisol	851.10 (360.31)	1022.82 (534.64)	-1.623	1.96
Time 1 cortisol, sample 1 (A.M. 1), ug/mL	0.40 (0.21)	0.36 (0.22)	1.00	5.77
Time 1 cortisol, sample 2 (A.M. 2), ug/mL	0.44 (0.22)	0.42 (0.25)	0.32	0.749
Time 1 cortisol, sample 3 (A.M. 3)ug/mL	0.31 (0.15)	0.40 (0.23)	-2.08	0.07
Time 1 cortisol, sample 4 (4 P.M.), ug/mL	0.11 (0.07)	0.17 (0.21)	-1.85	1.24
Time 1 cortisol, sample 5 (9 P.M.), ug/mL	0.05 (0.05)	0.10 (0.19)	-1.80	1.41
Time 2 cortisol, sample 1 (A.M. 1), ug/mL	.3853 (.2577)	.4026 (.2125)	-.261	14.31
Time 2 cortisol, sample 2 (A.M. 2), ug/mL	.45410 (.3462)	.4820 (.2919)	-.314	13.52
Time 2 cortisol, sample 3 (A.M. 3)ug/mL	.3837 (.3379)	.3060 (.1600)	1.113	4.87
Time 2 cortisol, sample 4 (4 P.M.), ug/mL	.1083 (.0711)	.1046 (.0639)	.188	15.33
Time 2 cortisol, sample 5 (9 P.M.), ug/mL	.0750 (.0781)	.0817 (.1181)	-.222	14.39
Time 3 cortisol, sample 1 (A.M. 1), ug/mL	.3826 (.2953)	.3818 (.1234)	.014	17.45
Time 3 cortisol, sample 2 (A.M. 2), ug/mL	4256 (.2484)	.5464 (.2938)	-1.45	2.28
Time 3 cortisol, sample 3 (A.M. 3)ug/mL	.5263 (.4640)	4299 (.3134)	.87	7.11
Time 3 cortisol, sample 4 (4 P.M.), ug/mL	1425 (.0732)	.1151 (.0732)	1.27	3.90
Time 3 cortisol, sample 5 (9 P.M.), ug/mL	.1130 (.1529)	.0685 (.1039)	.121	4.23
Time 4 Day 1 cortisol, sample 1 (A.M. 1), ug/mL	.3111 (.1628)	.2584 (.1472)	1.38	3.43
Time 4 Day 1 cortisol, sample 2 (A.M. 2), ug/mL	.3417 (.1534)	.4007 (.2876)	-.481	6.37
Time 4 Day 1 cortisol, sample 3 (A.M. 3)ug/mL	.3099 (.1662)	.3463 (.1913)	1.06	8.01
Time 4 Day 1 cortisol, sample 4 (4 P.M.), ug/mL	.1229 (.0997)	.1195 (.0719)	.080	15.93
Time 4 Day 1 cortisol, sample 5 (9 P.M.), ug/mL	.0878 (.0473)	.0978 (.0939)	-.168	11.37
Time 4 Day 2 cortisol, sample 1 (A.M. 1), ug/mL	.3186 (.1955)	.2660 (.1862)	.742	10.35
Time 4 Day 2 cortisol, sample 2 (A.M. 2), ug/mL	.3891 (.2238)	.3840 (.2327)	.59	16.77
Time 4 Day 2 cortisol, sample 3 (A.M. 3)ug/mL	.2843 (.1956)	.3890 (.2638)	-.711	17.46
Time 4 Day 2 cortisol, sample 4 (4 P.M.), ug/mL	.1377 (.1123)	.1157 (.1151)	-.146	8.36
Time 4 Day 2 cortisol, sample 5 (9 P.M.), ug/mL	.1026 (.1307)	.0855 (.0964)	-.662	10.04
Time 4 Day 3 cortisol, sample 1 (A.M. 1), ug/mL	.2714 (.1472)	.3125 (.1534)	.110	2.21
Time 4 Day 3 cortisol, sample 2 (A.M. 2), ug/mL	.3348 (.2876)	.3426 (.1662)	.274	8.34
Time 4 Day 3 cortisol, sample 3 (A.M. 3)ug/mL	.3592 (.1913)	.3991 (.0997)	.111	10.04
Time 4 Day 3 cortisol, sample 4 (4 P.M.), ug/mL	.1670 (.0719)	.1637 (.0473)	.278	8.36
Time 4 Day 3 cortisol, sample 5 (9 P.M.), ug/mL	.1108 (.0939)	.1029 (.1955)	-.164	16.02

Time 4 Day 4 cortisol, sample 1 (A.M. 1), ug/mL	.2576 (.1862)	.2643 (.2238)	-.165	9.16
Time 4 Day 4 cortisol, sample 2 (A.M. 2), ug/mL	.3179 (.2327)	.4127 (.1956)	.-1.52	16.45
Time 4 Day 4 cortisol, sample 3 (A.M. 3)ug/mL	2872 (.2638)	.4230 (.1123)	-1.57	14.05
Time 4 Day 4 cortisol, sample 4 (4 P.M.), ug/mL	.1388 (.1151)	.1767 (.1307)	-1.30	15.786
Time 4 Day 4 cortisol, sample 5 (9 P.M.), ug/mL	.0893 (.0484)	.0801 (.0964)	.43	2.44

Note. Asterisks indicate mean gender difference by *t*-test, * =$p < .05$, **= $p < .01$

Tests of Hypotheses

The integral pathways in the model of stress, biological and behavioral response and weight status, as presented in Figure 2, provided the basic model for examining the associations between daily hassles, cortisol reactivity and weight status (see Figure **4-3**). The model provides the structure for the first three hypotheses, detailed below. These three hypotheses were tested using a series of regression analyses applying the methods of Baron and Kenney (1986).

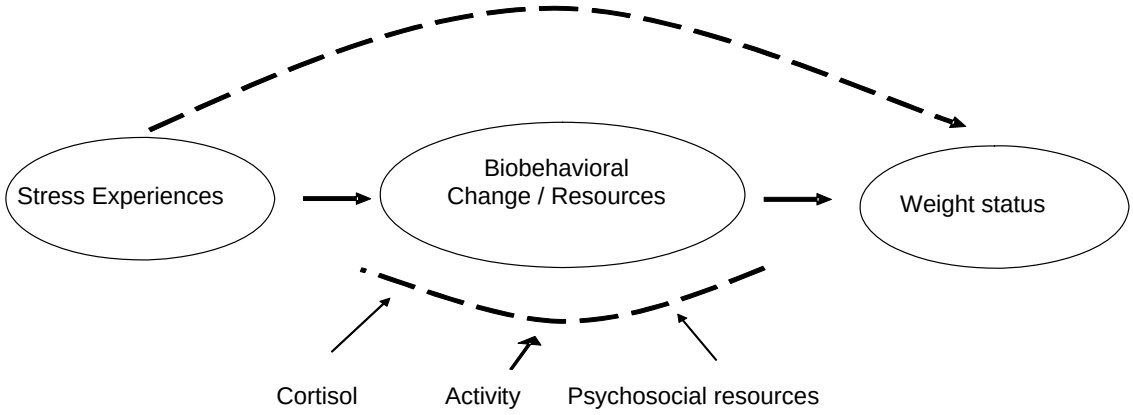

Figure **4-3**: The Stress Experiences, Biobehavioral Change/Resources and weight status model. Stress experiences are positioned as a diathesis, in the case of early vulnerability, or as a direct, more proximal effect, on weight status. Changes in biological systems, for example, an increase in cortisol secretion, or in behavior, or diet mediate the effects of stress on weight status.

Hypothesis 1: Higher body mass index will be associated with greater reported responses to daily hassles; this relationship will be mediated by cortisol reactivity to a laboratory stressor.

The general pattern of interrelationships between BMI, daily hassles and cortisol reactivity were examined for male and female adolescents separately. Associations between cortisol reactivity and BMI are evident for male

adolescents; associations were not evident between daily hassles and BMI, or in other indices of weight status (body fat percentage, waist circumference) for male adolescents. Therefore the prime criteria for a mediation model, that the proposed predictor variable, that is, daily hassles, and outcome variable, that is, weight status, be significantly associated was not established. A test of the association between cortisol reactivity and male adolescent BMI, after controlling for age and pubertal stage, indicates that higher cortisol reactivity is significantly associated with higher BMI in male adolescents. The final regression model is presented in Table **4-5**.

Table **4-5**: Results of the hierarchical regression model of adolescent BMI on age, pubertal stage and cortisol reactivity in male adolescents.

	Adolescent BMI						
Variable	*B*	*SE B*	β	df	F	R2	ΔR2
Step 1							
Age	.66	.59	.18				
				1, 35	1.25	.04	.04
Step 2							
Age	.53	.61	.15				
Pubertal Stage	.05	.06	.15				
				2, 34	.97	.06	.02
Step 3							
Age	-.101	.49	-.03				
Pubertal Stage	.05	.06					
Cortisol Reactivity	.39	.15	.41*				
				3, 33	2.97*	.14	.12*

Note. # = p <.08, *= p < .05, ** = p< .01

Associations between cortisol reactivity, BMI and daily hassles were not indicated for female adolescents. Examination of the bivariate correlations indicated no associations between cortisol reactivity, daily hassles and overweight (i.e. body fat percentage, waist circumference) for girls.

Hypothesis 2: Higher cortisol reactivity to a laboratory stressor will be associated with a higher body mass index and this relationship will be mediated by activity patterns.

An examination of the bivariate correlations between body mass index, cortisol reactivity and activity patterns indicated significant associations between these variables for male adolescents. The initial model (Model 1) regressed BMI on age and then cortisol reactivity. The direct relationship between cortisol reactivity and BMI was established. The second and third models (Models 2 and 3) tested the associations among activity patterns and cortisol reactivity, and activity levels and BMI, respectively (see Table **4-6**). The final model indicates that activity levels completely mediates the effects of cortisol reactivity on BMI for male adolescents; activity levels and BMI are negatively correlated, the higher the level of activity, the lower the BMI.

Examination of the table of correlations indicates no association between cortisol reactivity and BMI in female adolescents. Levels of activity explains 30% of the variance in BMI, after controlling for age in female adolescents (β = -.56, F = 6.92, p = .00).

Table **4-6**: Hierarchical regression models of activity level or BMI on age, pubertal stage and cortisol reactivity in male adolescents.

Variable	Activity Level							BMI						
Variable	*B*	*SE B*	β	df	F	R2	ΔR2	*B*	*SE B*	β	df	F	R2	ΔR2
Step 1														
Age	-15003	9421	-.27					.447	.66	.12				
				1, 33	2.53	.07	.07				1, 33	.45	.01	.01
Step 2														
Age	-13559	8880	-.24					.34	.62	.09				
Cortisol Reactivity	-5270	2283	-.36*					.37	.16	.38*				
				2, 32	4.10*	.20	.14*				2, 32	6.92**	.30	.28**
Step 3														
Age								-.09	.58	-.02				
Activity Level								-3.3	.00	-.50**				
Cortisol Reactivity								.19	.15	.20	3, 31			
												5.23**	.33	.03

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

Hypothesis 3: Childhood vulnerability, characterized by high cortisol reactivity to a laboratory stress challenge and symptoms of psychopathology, will predict higher body mass index, and this relationship will be mediated by daily hassles.

To determine the general pattern of interrelationships, bivariate correlations were examined between cortisol reactivity, symptoms of depression in childhood, reports of daily hassles and overweight status, namely BMI, waist circumference and body fat percentage. An association between symptoms of depression (CBCL Withdrawn/Depressed) in childhood and BMI, body fat percentage and waist circumference in adolescence was evident. As the three indicators of overweight status were so highly correlated in male adolescents (BMI and body fat percentage, r = .90, p = .00, BMI and waist circumference r = .94, p = .00), BMI was used as the prime indicator of overweight status and used as the sole outcome variable. No correlations are indicated for any indicators of overweight status (BMI, waist circumference or body fat percentage) and childhood cortisol reactivity, or for the proposed mediator, daily hassles. Thus, the prime criteria for a mediation model, that the proposed predictor variables and outcome variable be significantly associated, was not established. A hierarchical linear model estimated the effects of symptoms of childhood depression on male adolescent BMI, after controlling for age and pubertal stage. Significant effects are evident for childhood depression, but after controlling for

the effect of childhood BMI, these effects are no longer evident. The final regression model is shown in Table **4-7** .

Table **4-7**: Results of the hierarchical regression model of adolescent BMI regressed on age, pubertal stage, childhood depression and childhood BMI in male adolescents.

	Adolescent BMI						
Variable	*B*	*SE B*	β	df	F	R2	ΔR2
Step 1							
Age	080	.71	.21			.04	.04
				1, 29	1.42		
Step 2							
Age	-.64	.75	.16				
Pubertal Stage	.05	.07	.14				
				2, 28	.91	.06	.02
Step 3							
Age	-.56	.32	-.14#				
Pubertal Stage	.04	.02	.117				
Childhood BMI	1.15	.09	.95**				
				3, 27	54.70**	.86	.80**
Step 4							
Age	-.50	.36	-.13				
Pubertal Stage	.04	.03	.124				
Childhood BMI	1.117	.110	.93**				
CBCL Withdrawn/ Depressed	.02	.38	.01				
				4, 26	39.81*	.86	.00

Note. # = p <.08, * = p< .05, ** = p< .01

For female adolescents there were no significant correlations between childhood depression, childhood cortisol reactivity, daily hassles or adolescent BMI.

Hypothesis 4: Patterns of cortisol secretion across the day will demonstrate effects of development; daily trajectories will be more stable in late and post-pubertal adolescents.

Two separate models for fitting trajectories were run. In the first model (Model A) and subsequent tests of stability, trajectory group probabilities were calculated and trajectory group was indicated by the likelihood ratio for the four consecutive sampling days at Time 4, and then the stability of trajectories was evaluated, and a test of the effect of pubertal development was assessed. This model is presented first. Then a second model (Model B) evaluated the stability of cortisol trajectory probability across Times, 1, 2, 3 and 4. The first non-school day of sampling in the Time 4 collection period was selected for inclusion in these analyses. Sampling days in Times 1, 2 and 3 were all non-school days and most commonly Saturdays, and the selection of the first non-school days reduces the potential variance related to daytime activities.

Model A: Consecutive Days of Measurement

The BIC, AIC, and population proportions for Model A are shown in Table **4-8** . Based on the BIC, the best-fitting model for both Models A was a 4-

group model and visual inspection confirmed that each group represented sufficiently unique parameters. The anticipated quintic order of the parameters was confirmed as the parameters in each group were significant at $p < 0.01$.

Table **4-8**: Trajectory Model Fit by Number of Groups

Number of Groups	BIC (casewise)	BIC (point-wise)	AIC	Population Proportions (%)*
1	416.02	401.23	479.61	100
2	363.33	341.81	436.77	87.3, 13.0
3	489.96	462.41	524.42	52.6, 38.1, 10.3,
4	563.43	512.05	626.32	50.4, 38.7, 7.8, 3.0

*Population proportions are listed in descending order; values may not add to 100% due to rounding.

The obtained trajectories for Time 4, four consecutive days of sampling, are illustrated in Figure **4-4** . The most common cortisol pattern is represented by Group 2 (50.4% of the population). The shape of the trajectory for this group approximates the expected diurnal pattern with a rise, albeit low, soon after awakening, and plateaus to the third sample, followed by decline across the day.

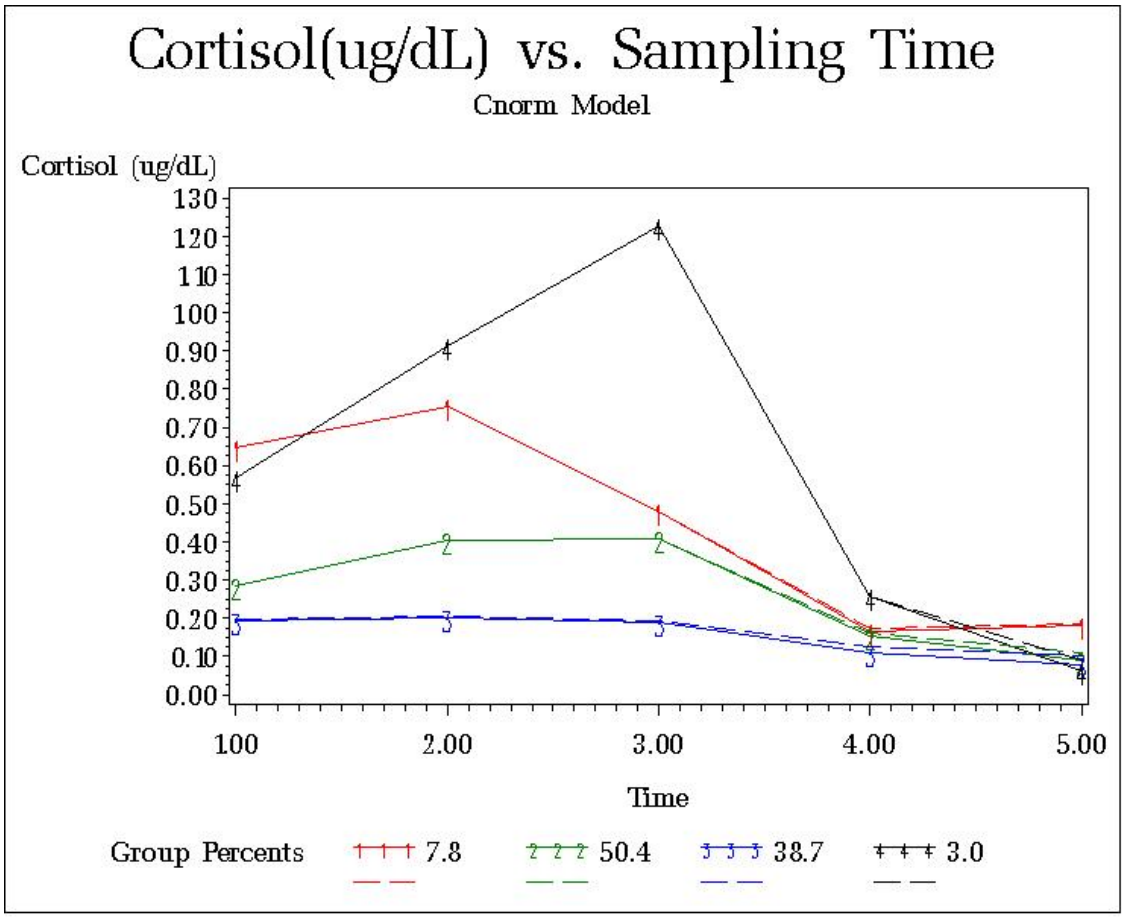

Figure **4-4**: Cortisol trajectories showing four groups of trajectory shape. Samples 1, 2 and 3 represent 3 morning samples obtained 15 minutes apart, Sample 4 is obtained at 4 p.m. and Sample 5 is obtained at 9 p.m.

Group 3 (38.7%) has no evident morning peak, with the three morning values changing little across the first 45 minutes of waking, but followed by a slight decline across the day to the evening. Group 1 (7.3%) has the classical pattern with cortisol peaking 20 minutes after waking, declining sharply at 40 minutes after waking, and declining gradually across the day. Group 4 (3%) has

a cortisol marked rise in cortisol across the first three samples, and then declines sharply to the afternoon, and again to the evening sample.

The stability of trajectory probability across four consecutive days of measurement, based on group assignment was good. The trajectory of Group 1 had the highest probability of stability. Test-retest correlations for the probabilities within each trajectory across the four consecutive days are presented in Table **4-9** . The correlations are stronger between Days 3 and 4, the two non-school days.

Table **4-9**: Test-Retest Correlations for Trajectory Probabilities

	Day 1 – 2	Day 1 – 3	Day 1 – 4	Day 2 – 3	Day 2 – 4	Day 3 - 4
Group 1	.93**	.79**	.88**	.74**	.63*	91**
Group 2	.14	.31**	.13	.41**	.26*	.18
Group 3	.53**	.47**	.44**	.45**	.29*	.63*
Group 4	.38**	.13	.16	.05	.21*	.48**

* = p <.05, ** = p <.01

The separate trajectory analysis with pubertal stage as a predictor of the cortisol trajectory indicated no association. The same procedure using age as a predictor indicated no association between trajectory and age.

Within group cortisol trajectories were stable across all days of measurement. Associations across the four days were moderate, although significant (p<.05) for all days of testing when testing for group assignment based on Day 1 assignment. The obtained Lambda (Λ), representing the proportional reduction in error when the likelihood ratio of group status for a

previous day was used to predict likelihood ratio of groups status on subsequent days, and the uncertainty coefficient (UC), indicating the proportional reduction in error when the likelihood ratio of group status for a previous day was used to predict likelihood ratio of group status on subsequent days, are presented in Table **4-9.**

Table **4-9**: Within-group cortisol trajectory stability

	Day 2	Day 3	Day 4
Λ	.34**	.24*	.38**
UC	.33**	.28**	.37**

* = p <.05, ** p<.01

Model B: Measurement across time

The next series of analyses tested the stability of cortisol trajectories across Times 1, 2, 3 and 4 of measurement. The BIC, AIC, and population proportions for Model B are shown in Table **4-10**. Based on the BIC, the best-fitting model for Model B was a 4-group model and visual inspection confirmed that each group represented sufficiently unique parameters. The anticipated quintic order of the

parameters was confirmed as the parameters in each group were significant at $p < 0.01$.

Table **4-10**: Trajectory Model B Fit by Number of Groups

Number of Groups	BIC (casewise)	BIC (point-wise)	AIC	Population Proportions (%)*
1	312.02	301.23	379.61	100
2	376.23	341.81	472.77	75.6, 24.3
3	499.90	462.41	564.42	60.3, 29.9, 12.1
4	571.43	512.05	594.62	60.3, 28.2, 9.8, 1.8

*Population proportions are listed in descending order; values may not add to 100% due to rounding.

The obtained trajectories are illustrated in Figure **4-2** . The most common cortisol pattern is represented by Group 1 (60.3% of the population). The shape of the trajectory for this group approximates the expected diurnal pattern with a rise, albeit low, soon after wakening, and plateaus to the third sample, followed by decline across the day. Group 3 (28.2) has the classical pattern with cortisol peaking 20 minutes after waking, declining sharply at 40 minutes after waking, and then declining gradually across the day. Group 2 (9.8%) has a marked rise in cortisol across the first two samples, and rises again at the third sample, with a sharp decline to the afternoon, and a gradual declines across the remainder of the day. Group 4 (1.8%) has a lower cortisol value 20 minutes after wakening, but

then a marked rise at 40 minutes after wakening, with a sharp decline to the late afternoon sample, followed by a decline to the evening sample.

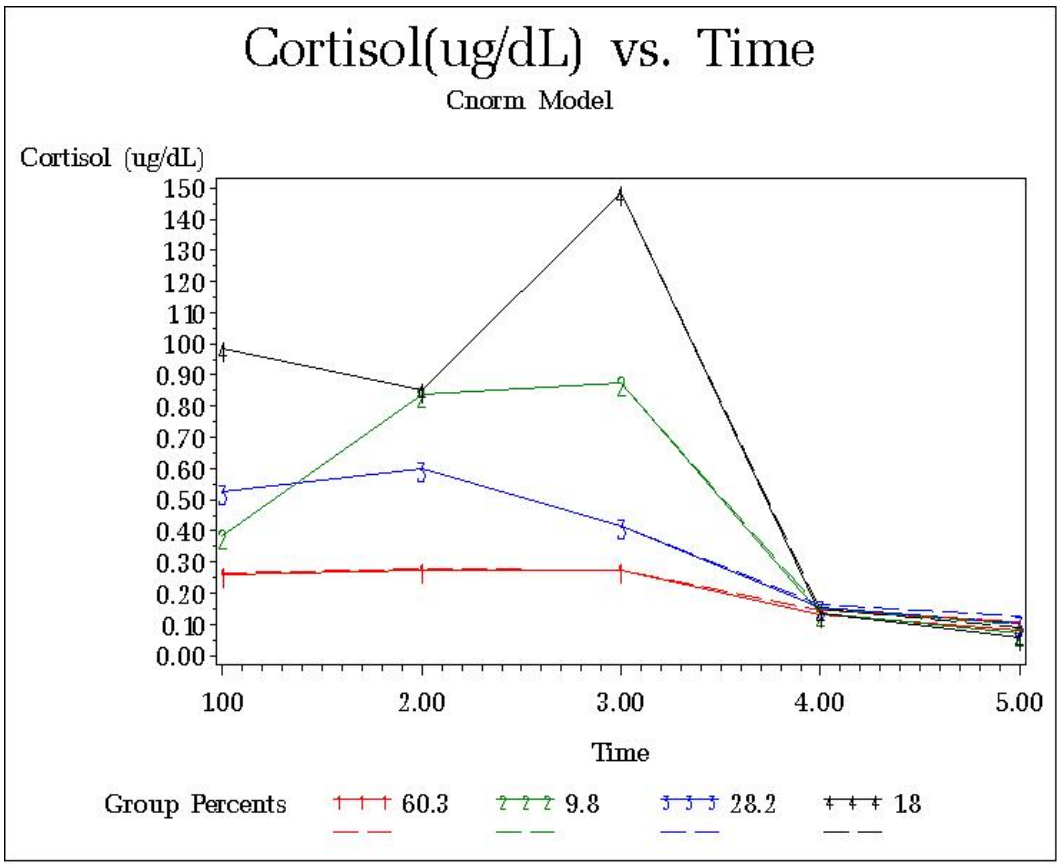

Figure **4-2**: Cortisol trajectories showing four groups of trajectory shape. Samples 1, 2 and 3 represent 3 morning samples obtained 15 minutes apart, Sample 4 is obtained at 4 p.m. and Sample 5 is obtained at 9 p.m

The stability of trajectory probability across Times, 1, 2, 3, and 4 (day 3) (see Table **4-1**) indicate that cortisol trajectory probabilities were moderately, although significantly correlated (p<.05) across equidistant 6 month intervals. The trajectory probability at Time 4 was not associated with probabilities at Times 1, 2 or 3. The stability of the cortisol trajectory stability across the 4 times

of measurement was not indicated except for an association between Time 1 and Time 3 (UC = .14, p<.05)

Table 4-11**:** Test-Retest Correlations for Trajectory Probabilities

	Time 1 - 2	Time 1 – 3	Time 1 – 4	Time 2 – 3	Time 2 – 4	Time 3 - 4
Group 1	.32**	.37**	.19	-.06	-.16	.21
Group 2	.06	.54**	-.02	-.13	.02	.02
Group 3	.10	.31*	.27*	.05	-.18	-.03
Group 4	.74**	.16	-.11	.43**	.05	.70

* = p <.05, ** = p <.01

Hypothesis 5. Total cortisol secretion across a day will be predicted by greater reported responses to daily hassles.

Examination of the differences in reports of daily hassles and AUCg by participants with higher versus lower cortisol reactivity to a laboratory stressor indicates no significant differences (Means and SD are presented in Table **4-12**).

Table **4-12**: Means and standard deviations for high and low cortisol reactivity groups, and t-test results for group differences.

	Mean (*SD*)			
	High Reactivity	Low Reactivity	*t*	*P*
Age	13.8 (1.7)	14.3 (2.1)	-1.13	.27
Day 1 Total Activity Counts	367190 (209777)	377562 (193980)	.169	.86
Day 2 Total Daily Activity Counts	385260 (451729)	32044 (151318)	-.75	.45
Day 3 Total Activity Counts	2777873 (136121)	313678 (164757)	.72	.47
Day 4 Total Activity Counts	263030 (131392)	285848 (127959)	.53	.60
Day 1 Sleep (hours, minutes)	8.22 (1.88)	8.37 (1.22)	.39	.69
Day 2 Sleep (hours, minutes)	8.25 (1.67)	8.50(1.51)	.65	.52
Day 3 Sleep (hours, minutes)	8.57 (1.85)	8.27 (1.48)	-.73	.46
Day 4 Sleep (hours, minutes)	8.46 (1.64)	8.39 (1.32)	-.19	.84
Day 1 AUCg Cortisol	898.25 (325.74)	7775.78 (320.57)	-.15	.12
Day 2 AUCg Cortisol	868.26 (354.41)	789.34 (330.34)	.96	.34
Day 3 AUCg Cortisol	1060.58 (358.27)	917.95 (394.62)	-1.5	.13
Day 4 AUCg Cortisol	1032.48 (635.09)	902.87 (367.79)	-1.14	.26
Day 1 DED Hassles	2.65.22 (3.53)	1.52 (1.97)	-1.7	.08
Day 2 DED Hassles	2.79 (2.71)	1.69 (2.09)	-1.9	.05
Day 3 DED Hassles	2.16 (2.16)	2.73 (3.98)	.65	.51
Day 4 DED Hassles	1.5 (2.48)	2.49 (3.65)	1.20	.23
Day 1 DED Anxious / Depressed	8.34 (3.02)	8.7 (2.6)	.54	.58
Day 2 DED Anxious / Depressed	8.20 (2.34)	8.2 (2.5)	.12	.89
Day 3 DED Anxious / Depressed	7.9 (1.95)	8.9 (2.9)	1.4	.15
Day 4 DED Anxious / Depressed	7.5 (1.02)	8.2 (1.8)	1.7	.09
STAIC Trait Anxiety	39.2 (9.8)	36.9 (8.5)	2.30	.07

A test of the full model (see Eq. **4.1) i**n HLM indicated that the within-person factors feelings of anxiety/depressed mood and activity levels were not significantly related to change in AUCg and were subsequently removed from the model. Of the Level 2 variables, sex, cortisol reactivity group and activity levels, only cortisol reactivity group was potentially important in predicting parameters of the AUCg – Hassles relationship. The final models are presented in Equation 4.2.

$$AUCg_{ij} = 0_{ij} + 1_{ij}\,(\text{Sleep}) + 2_{ij}\,(\text{Daily Anxiety/Depression}) + 3_{ij}\,(\text{Activity}) + 4_{ij}\,(\text{Hassles})$$

4.1

$$0_j = 00 + 01(\text{Sex}) + 02(\text{Cortisol Reactivity})$$

Eq. 4.1 The initial two-level model. In the level 1 model the value of AUGg_{ij} is for adolescent *i* and time *j* is a linear function of sleep, daily anxiety/depression, activity levels and hassles for that occasion. The level 2 model estimates the effects of sex and cortisol reactivity group status on the level 1 model.

$$AUCg_{ij} = 0_j + 1_j\,(\text{Sleep}) + 2_j\,(\text{Hassles})$$

4.2

$$0_j = 00 + 01(\text{Cortisol Reactivity})$$

Eq. **4.2** The two-level model. In the level 1 model the value of AUGg_{ij} is for adolescent *I* and time *j* is a linear function of sleep and hassles for that occasion. The level 2 model estimates the effects of cortisol reactivity group status on the level 1 model.

Intra-individual effects (Model 1) Higher numbers of daily hassles were associated with daily cortisol secretion, as daily hassles increased, AUCg cortisol also increased. The amount of sleep obtained the preceding night was also a significant contributor to AUCg cortisol, the more sleep the participant had, the lower AUCg cortisol value.

Inter-individual effects (Model 2) There was not a significant main effect for group; higher or lower cortisol reactivity to a lab stressor was not a significant predictor of the associations between AUCg and daily hassles or sleep. Cortisol reactivity group status was, however, important in predicting the association between sleep and AUCg. The association between sleep and AUCg was stronger in those who had a higher cortisol response to the laboratory stressor. The final model is presented in Table **4-13.**

Table **4-13**: HLM model predicting AUCg values and slopes from daily hassles, sleep and cortisol reactivity.

Fixed Effects	Coefficent	SE	t-ratio	p-value
Predicting AUCg intercept	972.96	108.72	8.9	.000
Intercept	56.63	35.04	1.6	.11
Reactivity Group				
Predicting AUCg slope				
Intercept	23.53	6.74	3.48	.00
Reactivity Group				
Sleep	-21.10	11.98	-1.7	.08
Random effects	Variance		X^2	
Component				
AUCg intercept	56134.11		271.41	.00
AUCg slope	86891.28			

* = p <.05, ** = p <.01

Chapter 5

Discussion

This study examined the associations between daily hassles, cortisol reactivity, activity and weight status in adolescents. The use of diary measures and the combination of cortisol reactivity to laboratory stress and daily cortisol levels in the natural setting are particular strengths of this study. The findings have value in advancing knowledge of daily cortisol patterns in adolescence, and how these daily patterns may be affected by daily experiences. This section will first summarize the support for the hypotheses before discussing the results and the interpretation of the current results for understanding weight status in adolescence. The limitations of the study will be discussed, and recommendations for future work presented.

The first aim of this study was to examine, using a variant of the allostatic load model, the associations between daily hassles, cortisol reactivity and weight status in adolescence. The first hypothesis was that higher numbers of reported daily hassles would predict a higher body mass index (BMI), and that the degree of cortisol response to a stress challenge would mediate the association. This hypothesis was not supported for male or female adolescents. In male adolescents, daily hassles and cortisol reactivity were associated with BMI.

The second hypothesis was that higher cortisol reactivity would be associated with a higher body mass index, and this relationship would be mediated by activity levels. This hypothesis was supported only for male adolescents. In male adolescents a higher level of cortisol reactivity to a laboratory stress challenge was associated with higher weight status, although this association was no longer significant once activity levels were accounted for. In female adolescents there were no associations between cortisol reactivity, activity and weight status.

The third hypothesis was that childhood vulnerability, characterized by high cortisol reactivity and symptoms of psychopathology, would predict higher body mass index, and that this relationship would be mediated by daily hassles. In male adolescents, childhood cortisol reactivity was not associated with body weight status or with daily hassles in adolescence. In male adolescents symptoms of childhood depression were associated with adolescent BMI, although this association was no longer evident after childhood BMI was included in the regression model. For female adolescents there were no significant correlations between symptoms of childhood depression, childhood cortisol reactivity, daily hassles in adolescence or adolescent body weight status. The hypothesized associations between childhood depression and adolescent weight status were not supported.

The second aim of the study was to examine diurnal patterns of cortisol secretion in an adolescent sample. It was hypothesized that daily trajectories of cortisol secretion would be more stable in late and post-pubertal adolescents. This hypothesis was not supported. Although there were different groups evident in the cortisol trajectories, group assignment was not explained by age, or by pubertal stage. A second hypothesis was that an individual's cortisol trajectory profile would not be stable from childhood to adolescence was supported. The daily cortisol profile of a person in childhood was stable across three equidistant 6 month intervals, but the cortisol profile in childhood was not associated with cortisol profiles in adolescence.

The final hypothesis, that daily hassles would predict daily cortisol secretion was supported. The number of hours of sleep the preceding night also predicted daily cortisol secretion. However, there was no support for the hypothesis that the degree of cortisol reactivity to a stressor would predict the level of association between daily hassles and daily cortisol secretion.

Daily Hassles, Cortisol Reactivity and Weight Status

The first hypothesis was that higher numbers of reported daily hassles would predict a higher body mass index (BMI), and that the degree of cortisol response to a stress challenge would mediate the association. This hypothesis was not supported, daily hassles were not associated with BMI in either male or

female adolescents, and cortisol reactivity was associated with BMI only in male adolescents.

The allostatic load models pioneered by Björntorp and colleagues (Bjorntorp, 1992, 1995b, 1997b; Rosmond, 2004b) and Pine and colleagues (Pine, Cohen, Brook, & Coplan, 1997; Pine, Goldstein, Wolk, & Weissman, 2001) posit stress as a prime agent in the development of obesity, and propose that repeated activation of the HPA with the accompanying increase in cortisol secretion is the pathway through which stress exerts effects on weight status. This was not found in the present study. The lack of an association between daily hassles and BMI in the current study may be explained in two several ways. Daily hassles may cause repeated activation of the stress system, but it is possible that these activations are insufficiently large to affect body weight. Daily hassles may not elicit exaggerated cortisol responses, and so are not profound enough in magnitude to shift cortisol levels outside of the normal range. Daily hassles may associate with changes in body weight over time, and so effects of daily hassles on body weight may not be evident until later in life. Testing concurrent associations between daily hassles and body weight in adolescence may constitute a constrained model, which fails to capture distal effects of hassles on weight status.

Cortisol has a normal fluctuating range and it may be that anticipated and usual hassles, or stressors, do not push cortisol above normal operating levels,

and so the profundity of change as required of the hypothesized association between cortisol and weight status model is not met. Ham and Larson (Ham & Larson, 1990) report that in adolescence it is unexpected daily events that cause more distress than the anticipated hassles of life. Further, Schneiders and colleagues (Schneiders et al., 2006) report that there may be age effects on sources of stress such that in adolescence school and peer stressors are more salient than at other periods of life. It can be argued that although these sources of stress are more salient, these stressors in adolescence may be regularly occurring and so are anticipated, and the individual becomes habituated to them, and so they do not constitute stressors.

A central tenet of the allostatic load theory from which this hypothesis is drawn is that there is repeated or profound stress causing cortisol levels to rise above normal levels, or to be consistently elevated. The effects of repeated perturbation may have a time lagged effect so daily hassles in adolescents may be a more salient predictor of weight status in later life. This is consistent with findings that the effects of early life stress effects on health are not apparent until midlife (Schneiders et al., 2006; Seeman et al., 2004; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Steptoe & Cropley, 2000; Steptoe et al., 2003).

The lack of an association between daily hassles and cortisol reactivity to a laboratory stressor indicates the importance of careful matching of cortisol measure to research aims, and it is suggested that a coupling of cortisol reactivity

to laboratory stressors and cortisol measures obtained in naturalistic settings may offer the most utility in explanations of responses to daily experiences. It is intuitive that those who are high reactors might be sensitive to, and report more daily hassles, but it is also plausible that those who are high reactors self select into situations and environments where their controllable hassles (e.g. time demands, work load) are reduced or avoided. People who are highly responsive to daily hassles may construct their time or select their social group to minimize the number of daily hassles they experience (Aboud & Mendelson, 1996; Haselager, Hartup, van Lieshout, & Riksen-Walraven, 1998; Mariano & Harton, 2005).

The association between cortisol reactivity and BMI in male adolescents, but not in female adolescents, may be explained with reference to the theory of different hormonal responses to stress in males and females. The 'tend-and-befriend' model, a model based in a neuroendocrine system that includes the secretion of oxytocin and endogenous opioids (Taylor et al., 2000)) might explain the differences found in the current study. The model of stress response in females, as proposed by Taylor and colleagues (2000), describes the secretion of oxytocin, endogenous opioids and estrogen in response to a stressor. These factors have also been associated with health protective effects, although there is continuing debate on this issue (Gnatuk, 2002; Hodis et al., 2003; Rosano & Fini, 2002) and these may be mechanisms that adjust the cumulative risk of repeated

stress as proposed by the allostatic load model. Oxytocin may ameliorate the increases in cortisol associated with stress, thus conferring some additional degree of protection from the effects of cortisol. This effect, especially in conjunction with estrogen may explain much of the variance in the development of disease, including obesity, between males and females. Taylor and colleagues (2000) report an association between oxytocin and cortisol, such that there is a reduced cortisol response to stress and that, in the context of a laboratory challenge, higher levels of oxytocin were associated with a shorter HPA recovery period. If oxytocin moderates the release of norepinephrine and cortisol, oxytocin released during stress may mediate the relationships between stress and health. These sex differences offer an explanatory model for understanding the sex difference found in the association between cortisol reactivity and weight status in the current study.

Cortisol Reactivity, Activity Levels and BMI

The positive association between cortisol reactivity and BMI in male adolescents and not in female adolescents is consistent with the 'tend-and-befriend' theory already outlined. There were no mean differences in males or females in BMI or cortisol reactivity in the current study; however, for male adolescents cortisol reactivity was associated with BMI. The association between activity levels and BMI needs little explanation, the more energy that is

expended, the lesser the likelihood of weight gain, providing energy intake does not exceed expenditure. Cortisol reactivity and activity might be related in such a way to produce effects on BMI. Male adolescents who are more stress reactive may avoid activities that evoke feelings of stress, such as activities that involve competition and comparison to others. Consistent with findings that motivations to engage in physical activity, including sports and exercise, that offer opportunities for competition and comparison to others, are important for male adolescences (De Bourdeaudhuij et al., 2005) and it is plausible that male adolescents who are more responsive to psychosocial stress might avoid physical activities that entail competition and comparison (Epstein, Roemmich, Saad, & Handley, 2004; Margetts, Rogers, Widhal, Remaut de Winter, & Zunft, 1999; Norton, Burns, Debra, Hope, & Bauer, 2000; Pratt, Macera, & Blanton, 1999; Salguero, Gonzalez-Boto, Tuero, & Marquez, 2004; Sirard, Pfeiffer, & Pate, 2006), and that there are lesser opportunities to engage in non-competitive exercise for males.

Males and females may be differently responsive dependent on the type of stressor, and there is evidence that males show higher responses to stress challenge paradigms that involve conflict, win/loss scenarios and performance evaluation tasks (Kudielka, Buske-Kirschbaum et al., 2004b; Kudielka et al., 1998; Kudielka & Kirschbaum, 2005; Seeman, Singer, & Charpentier, 1995; Uhart et al., 2006). It has also been shown that females may be more responsive to laboratory

stress paradigms involving threat to the social self (Stroud et al., 2004; Stroud et al., 2002) and more responsive to hostility, discord and social rejection in their interpersonal relationships (Kiecolt-Glaser et al., 1996; Stroud et al., 2002). This, individual differences in stress responses may predict levels of involvement in exercise in a different fashion for males, and thus contribute to differences in the relationship between cortisol reactivity and activity levels.

The association between cortisol reactivity and BMI for males was not evident after activity level was included in the regression model. This suggests that the effects of cortisol alone are not sufficient to produce weight change, especially change over a short period of time. That is, repeated and or chronic elevation in cortisol may enhance the risk of obesity synergistically with other risk factors such as sedentary lifestyle and excess caloric intake. Thus, the weight change as a consequence of cortisol oversecretion as proposed in the allostatic load model may be insufficient and require potentiation from lifestyle and behavioral factors for changes in BMI to occur.

The association between activity level and BMI in female adolescents was much stronger than in males, and this was unexpected. The effects of exercise on weight status, when caloric intake is stable, should be to maintain or to reduce weight; however there are reports that there are sex differences in these effects and that the association between exercise and weight is less straightforward in females than in males. In a study of healthy adults Novotny and colleagues

(Novotny et al., 2003; Paul, Novotny, & Rumpler, 2004) evaluated the effect of sex and food intake on the association between physical activity and body fat. They report that the association between exercise and body fat is not strong, and apparent only in males. In this study there was no association between activity levels and body fat in women, and Paul and colleagues (2004) attribute this to increases in energy intake in response to increased exercise, and different processes of metabolism in females. The findings are consistent with others that report that exercise has lesser effects of body fat in women than in men (Gleim, 1993; Gornall & Villani, 1996) . The stronger effects in female adolescents may also be explained with reference to the different motivational forces for exercise by males and females. Female adolescents may exercise specifically for weight management, and so engage in exercise that increases heart rate with effects on weight. In a study of differences in motivations for exercise (Tiggemann & Williamson, 2000) report that women are more likely to exercise to control weight and tone muscle, and these effects are more apparent in younger women. These results have been paralleled in other study of motivations to exercise (Furnham, Badmin, & Sneade, 2002; Silberstein, Striegal-Moore, Timko, & Rodin, 1998). The activity levels in males may represent general patterns of activity, which may be different in intensity level, and so not associated with weight status.

Childhood Depression, Daily Hassles in Adolescence and Adolescent Weight Status

The current study found no evidence for an association between symptoms of childhood depression, childhood cortisol reactivity, daily hassles and weight status in adolescence. There is emerging evidence that depression is predictive of obesity, although traditionally, obesity has been viewed as an antecedent to depression, with social stigmatization producing shame, guilt and peer isolation leading to affective disorder, especially in early life (Field, Camargo, Taylor, Berkey, & Colditz, 1999; Field, Camargo, Taylor, Berkey, Frazier et al., 1999; Field et al., 2001; Strauss, Doyle, & Kreipe, 1994; Vila et al., 2004). In contrast, recent research provides support for a model wherein obesity is but a physical manifestation of the neuroendocrine processes associated with depression (Bjorntorp, 1997a, 1997b; Goodman & Whitaker, 2002; Mustillo et al., 2003; Pine et al., 1997; Rosmond, 2004a; Wurtman, 1993). As evidence that depression may be a causal factor in obesity, recent prospective studies demonstrated an association between childhood depression and obesity in later life (Pine et al., 1997; Pine et al., 2001; Richardson et al., 2003). In a longitudinal study depressed children were much more likely to be obese at follow-up, however, obese children were not at any increased likelihood of being depressed (Goodman & Whitaker, 2002). The increased likelihood of obesity for depressed children remained even after controlling for physical activity. In sum, both

depression and obesity may have multiple biological and social determinants, and there is mounting evidence that childhood depression is associated with overweight and obesity, although this was not found in the current study.

The lack of findings for depression in the current study may be attributed to the time frame of analysis; there may be time lagged effects that were not captured by the design of the current study. Much of the previous work examining depression and obesity has drawn on populations of adults, or measured outcomes in adulthood, rather than in the pubertal years when preventive measures may be most effective. There is accumulating evidence indicating that depression and obesity share a common neurobiological pathway, specifically, the HPA axis, and are both associated with the over secretion of cortisol (Bjorntorp, 2001; Chrousos, 2000a, 2000b; McEwen, 1998; Wurtman, 1993). It is plausible that neurobiological pathways and the socio-environmental and familial milieu may contribute equally to predict and maintain both depression and obesity, but that these effects do not emerge until later in life, such as early or mid-adulthood.

Cortisol profiles, especially cortisol reactivity profiles, have been postulated as a predictor of weight gain. Results in adolescents are, however, equivocal, and inclusion of baseline weight status into models has often subsumed most of the variance in BMI in later years. However, there is emerging evidence that exaggerated reactivity, interacting with other factors,

such as behavioral patterns, predicts later risk of obesity. These other factors are perhaps not surprisingly, parental weight status, exercise patterns and diet selection – all notable independent risk factors. The risk conferred by exaggerated cortisol reactivity to later weight status may be less direct than originally hypothesized.

Daily Trajectories of Cortisol

Cortisol trajectories were quite stable across four consecutive days of measurement, especially for those who had the classic morning rise followed by gradual decline across the day. Cortisol trajectories were also stable in those who had a flattened low pattern across the four days. These results indicate that although there is some flux between consecutive days, cortisol trajectories maintain the same profile, at least across discrete, short periods of time. The results parallel other reports of low variability in cortisol profile over a short period of time (e.g. 6 months). Selmaoui and colleagues (Selmaoui & Touitou, 2003) measured cortisol profiles 3 times over a 6 week period, at time zero, time two weeks and time six weeks. The results indicate that stability was good across all three measurements. This finding, and those of the current study attest to the trait-like nature of the diurnal rhythm of cortisol, and there is a genetic influence on cortisol levels (Clow, Thorn, Evans, & Hucklebridge, 2004; Hucklebridge, Hussain, Evans, & Clow, 2005). Bartels (Bartels, de Geus,

Kirschbaum, Sluyter, & Boomsma, 2003; Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003). Notably the genetic influence may be most evident in morning cortisol levels and the cortisol level in the afternoon, although not necessarily at other times of the day, adding weight to the conceptualization of the diurnal rhythm of cortisol being trait-like.

The circadian rhythm of cortisol secretion is usually characterized by an early morning peak, an afternoon plateau and a nadir in late afternoon and evening hours with a typical pattern of increase then decline in the first 30 minutes of wakening (Schmidt-Reinwald et al., 1999; Weitzman et al., 1971) . Recent evidence of individual differences in trajectories of cortisol secretion over the initial waking period, and also across the day (Schmidt-Reinwald et al., 1999) suggest that people have different levels, if not different diurnal trajectories, of secretion, and these patterns are predicted by and predictive of changes in health and well-being.

The morning rise in cortisol has been well documented in adults, although the magnitude of the rise may vary, with increases of between 50-100 percent reported between people (Schulz & Knabe, 1994) although there is stability within individuals (Wust, Federenko, Hellhammer, & Kirschbaum, 2000b; Wust, Wolf et al., 2000b). This morning rise may be at least partly genetically determined with the remaining variance associated with aspects of the individual, including psychological attributes and pathologies (Federenko et al.,

2004; Federenko, Nagamine, Hellhammer, Wadhwa, & Wust, 2004; Mroczek & Almeida, 2004; Pruessner et al., 1997; Pruessner, Hellhammer, & Kirschbaum, 1999; Roy, Kirschbaum, & Steptoe, 2003; Wust, Federenko et al., 2000a; Wust, Wolf et al., 2000a).

There is however inconsistency in reports of the association between awakening cortisol levels and the trajectory (expected decline) over the course of the day. These inconsistencies may be due to the influence of genetic predisposition or the complex interactions between chronic stress, sex, psychopathology or pathophysiology (Pruessner et al., 2003; Wust, Federenko et al., 2000a; Wust, Wolf et al., 2000a) and researchers have often separated out the morning increase from the daily decline. Cortisol secretion is expected to decline across the day, although this expectation may reflect the effect of regression to the mean in large samples. Recently Smyth and colleagues (Smyth et al., 1998) reported that in a sample of adults only half of the sample had the expected pattern of decline, approximately 30% had inconsistent patterns and about 17% had no discernable pattern, others have also reported samples where a proportion of people did not have the expected daily decline or cycle. It may be that the people who show inconsistent and unexpected patterns have some underlying disorder, or that there are more variable patterns of cortisol than originally believed. Activities across the day such as eating, exercise, sleep (including napping) influence cortisol secretion, in addition to, or interacting

with, stressors (Elimam & Marcus, 2002; Follenius, Brandenberger, & Hietter, 1982; Watamura et al., 2004). In addition, incorporating measures of daily decline in models of health require that one examines the patterns carefully and account for variance in other aspects of life, otherwise the associations between daily stressors and daily cortisol trajectories may go undetected.

The relative stability of diurnal cortisol trajectory does not preclude effects of development. There is an increase in cortisol levels with age so that cortisol levels rise in mid-childhood and rise again, in mid-adolescence (Kiess et al., 1995; Lupien et al., 1998; Lupien, King, Meaney, & McEwen, 2000; Smyth et al., 1998) although these age associated changes have not been consistently found (Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997). Kiess (1995) found that sex maturation predicts changes in cortisol levels, particularly levels of cortisol reactivity, a finding also reported by Walker and colleagues (Walker, Bonsall, & Walder, 2002; Walker, Walder, & Reynolds, 2001). These findings are suggestive of change in the diurnal pattern of cortisol secretion across development, such that the pattern and total secretion of cortisol may change throughout early life. The current finding is that that cortisol profiles measured at six months apart show similarity, but are much less similar than cortisol trajectories measured at closer time points, such as consecutive days, and it is surmised that this indicates effects of age on the diurnal cortisol profiles.

Age may thus be considered an intra-individual difference, and cortisol levels may change across the lifespan, not only in childhood and adolescence but in later life (Gunnar, Bruce et al., 2001; Kudielka, Buske-Kirschbaum et al., 2004a, 2004b; Kudielka et al., 1998; Lupien et al., 1994; Lupien et al., 1996; Seeman, Singer, Wilkinson, & McEwen, 2001). This would explain why the pattern of cortisol trajectories within the individual was more highly correlated the closer the time points of measurement and not correlated with measurements taken at a more distal time (Shirtcliff, Granger, Booth, & Johnson, 2005). Future work that rigorously evaluates the contribution of age and development and of behavioral and contextual effects on the diurnal cortisol trajectory profile would elucidate the stability of the HPA axis, and contribute to a model of HPA axis activity that acknowledges the effects of development. If aging has effects on the activity of the HPA axis, the schema of the association between diurnal cortisol patterns and biobehavioral vulnerability outcomes may need to be re-conceptualized, with development considerations included in models of health.

Daily Hassles and Cortisol Secretion

The finding that daily hassles were associated with total cortisol secretion across the day supports the assertion that the experience of daily hassles can cause measurable increases in hormonal parameters. The association of daily hassles, or challenge, with changes in cortisol has been reported in adults. Van

Eck and colleagues (van Eck, Nicolson, Berkhof, & Sulon, 1996) examined the association between perceived stress of male adults and cortisol, where cortisol was sampled 10 times a day for 5 days. Findings indicated no association between perceived stress and cortisol, but stressful daily events such as network stress, transport problems and interpersonal interactions were associated with increases in cortisol. Van Eck (van Eck, Berkhof, Nicolson, & Sulon, 1996) reported that the magnitude of cortisol change to daily stress was also influenced by familiarity and previous experience with the stressor, which supports the notion that it is unexpected daily events that cause more distress than the anticipated hassles of life (Ham & Larson, 1990). Similar results were found in a sample of adolescents that tracked momentary state emotions and paired with salivary cortisol values (Adam, 2006). The results indicate that there is a relationship between momentary mood state and concurrent salivary cortisol levels, and of especial interest was that 'worry/stress' was positively associated with higher levels of cortisol. Adam (2006) also reported finding individual differences in the size of the association between mood and cortisol, including an effect of age.

The amount of sleep obtained the previous night was also associated with cortisol, the longer the duration of sleep, the lower the total cortisol output. It has been shown that deep sleep has an inhibitory effect in HPA axis activity, and sleep deprivation increases cortisol secretion the following day (Leproult,

Copinschi, Buxton, & Van Cauter, 1997; Vgontzas et al., 1999). It is hypothesized by McEwen (McEwen, 2006) that sleep may be considered an essential task in the maintenance of health and that sleep deprivation, with concomitant increases in cortisol, can be considered a contributor to allostatic load.

The association of daily stressors and change in the levels of cortisol at a momentary level were reflected in area under the curve (AUC) which represents both magnitude and persistence of cortisol across the period of interest. AUC may therefore offer an omnibus measure reflecting daily total secretion and activation of the HPA axis, and serve especially well in examinations of daily stressors and health. Daily hassles have only recently received attention for their potential contribution to health. It is plausible that repeated, even low magnitude, stress responses confer health risk though the same pathways as more chronic stress, that is, through a mechanism of allostatic load (Evans, 2003; McEwen, 2003).

Sex Differences

There were few mean differences between male and female adolescents in the variables of interest, although differences in correlations within males and females were apparent when correlations were split by sex. In the regression model testing associations between daily hassles, cortisol reactivity and weight status, an association between cortisol reactivity and BMI was evident only for males. This suggests there may be differential effects of stress dependent

increases in cortisol between males and females, and that males may be more vulnerable to the effects of cortisol on weight status. This may be explained by the 'tend-and-befriend' theory as already discussed, but it may also be attributable to differences in the hormonal milieu between the sexes. There are influences of menstrual cycle phase on the cortisol response to a stressor, where there is no difference between females in the luteal phase of the menstrual cycle and men, although females in the follicular phase have a lesser response than both these groups (Kirschbaum et al., 1999). The effects of cortisol act in concert with other hormones, such as testosterone, which may amplify or moderate the effects of cortisol on metabolism and weight gain (Bjorntorp, 1997a).

Sex differences were also evident in associations between activity level and BMI. The negative association between activity level and BMI was stronger in female adolescents than in males. Reasons for this finding are discussed above. Although the predictive value of cortisol reactivity on BMI was nullified after the inclusion of activity level in the regression model, cortisol reactivity was correlated with BMI in males but not in females. This relationship might also be explained with reference to the different levels of other hormones, for example, sex steroids, that enhance the effects of cortisol on weight status in males. The results also suggest that childhood obesity may constitute a greater risk for adolescent obesity in males than in females. This difference may be explained with reference to the differences in body image and motivation theory between

males and females (Davison & Birch, 2001; Davison, Markey, & Birch, 2000; Faith, Leone, Ayers, Heo, & Pietrobelli, 2002; McCabe, Ricciardelli, & Finemore, 2002; Ricciardelli, 2003; Sands, 2003). At adolescence females may be more sensitive to contextual cues and peer influences to modify their weight, through diet or exercise as already discussed, and so there may be a shift in the trajectory of BMI. The sex differences that have emerged in the current study, and other studies of predictors of weight status are yet to be adequately explained, although may be a function of the endocrine milieu, gender role socialization or psychosocial factors.

Limitations

The relatively small sample size of this study may constrain the power to detect significant differences and associations in the results. In addition, the size of the sample is smaller than usually recommended for hierarchical linear modeling approaches, although the numbers are adequate to test the two level model such as the one tested (Willett, Singer, & Martin, 1998).

The majority of the adolescent participants were White, which is reflective of the racial composition of the area from which the sample was drawn. Intrinsic differences between racial/ethnic groups in the activation patterns of the HPA axis have been found (Yanovski et al., 2000; Yanovski, Yanovski, Cutler et al., 1996; Yanovski, Yanovski, Friedman et al., 1996; Yanovski et al., 1993; Yanovski

et al., 1995), however, no differences in baseline levels of cortisol have been reported. The small number of non-White adolescents precluded analyses for race/ethnicity differences, and so the results of the current study may contribute only to knowledge of cortisol patterns in White adolescents. Similarly, the number and experience of daily hassles may demonstrate effects of race/ ethnicity, although this is likely to be a reflection of the confound of socioeconomic variables (Nazroo & Williams, 2006), however, the current study was not able to test for differences between groups.

A common concern in studies that use in-home saliva sample collection is that there may be a drift from the expected time of collection. Two strategies were designed to preempt this in the current sample. First, all participants were required to record the actual time of collection of the sample in two ways, once in the daily diary and once on a saliva collection time-sheet. At each phone contact during the home collection period, the adolescents were asked what time they had collected their samples that day and the time was recorded. Although there were some inconsistencies, there were high correlations between these three records of sampling time, and suggest that most participants collected their sample within one hour of the designated sampling time. Second, during the training session it was explained to the participants that the data derived from the Actigraphs could be used to check on minute-to-minute activity levels across the day, and this data could be linked to the salivary cortisol measurements, and

it was very important that these correlated. Third, each participant was provided with a "Spit Kit", which included a clock, reminder notices that were hung in prominent places in the home, and a "Spit Holster" which arrayed the collection tubes in order of designated collection time and was placed in a prominent part of the home to serve as a visual reminder. These methods were designed to increase compliance, however, there is some possibility that there are individuals, or sub-groups who were not compliant, and that this non-compliance was not detected. The use of time-stamped collection tubes may provide the most reliable indicator of collection time, although not possible for the current study, may provide the most accurate indicator of compliance to designated sampling times.

Staging of puberty by physical examination using Tanner Criterion (Marshall & Tanner, 1968, 1969, 1970) is the gold standard for assessment of pubertal development; however it was not feasible in the current study. The use of self-report of pubertal status has been reported to be an valid measure when estimations of pubertal stage are acceptable and appropriate for the aim of the research (Dorn, Dahl, Woodward, Rojahn, & Biro, 2006). Self-reported pubertal stage may be less valid in adolescents who are overweight, especially in females who may overestimate breast development (Dorn et al., 2006), and this is a noted caveat for the analyses, which examined effects of pubertal development on associations between stress and weight status in this study.

Finally, the use of paper diaries offers participants an opportunity to retrospectively complete the diary, that is, to recall their experiences and emotions of the previous day, rather than complete the diary on the designated day. Known as retrospection error, participants may reconstruct or fabricate entries so as to provide a 'complete' set of measures (Bolger et al., 2003; Bolger & Zuckerman, 1995). This issue is common to paper diary measures and it is difficult to assess the degree to which retrospection error exists in the data obtained. This potential problem may be minimized by supplementing the paper diary with signals (pagers, telephone calls) that remind the participant to complete the diary in the specified time frame. In the current study this approach was adopted by telephoning participants each evening during the home collection period, but it is still possible that retrospection error is present in the current results. Another caution in diary methods is the participants modifying their responses for fear that others may read the diary contents. Each participant was provided with individual sealable envelopes to address these concerns, although it is still possible that some may have been less truthful and forthcoming in their responses due to this concern.

Conclusions

In summary, this study provides evidence that studying the associations between daily hassles, cortisol reactivity and weight status in adolescence may

have utility in exploring models of stress and health. Adolescents' everyday situations and hassles were related to changes in their daily cortisol levels, and changes in cortisol levels in response to stress may contribute to weight status. The cortisol trajectory profile is less stable over long periods of time, and changes are associated with age, if not pubertal stage. The cortisol trajectory profile may be consistent across a short period, but absolute levels of cortisol may change according to the daily experiences and sleep pattern of the individual.

Future research may further explicate the complex interplay between experiences, cortisol levels and weigh status in adolescence, and would benefit from testing associations in a larger, more diverse sample. It is also recommended that future work examine the stability in both trajectory pattern and absolute level of daily cortisol secretion across the adolescent period, and test for effects of pubertal development. Developing an understanding of the dynamic interplay between daily experiences in adolescence and cortisol levels in the natural setting may help to elucidate the pathways through which daily stressors influence health status, in particular, weight status in adolescence.

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Appendix A

The Daily Experiences Diary

Hassles & Health Study

Daily Experiences Diary

Day 2

ID _____

Date_____

Day_____

Daily Diary Instructions.

You should add to the Diary several times over the day; once in the morning, once in the afternoon and once in the evening.

If you need more space to write in your answers, you can use a separate sheet of paper and insert this into the back of the Diary.

You may use pencil or pen to fill out this diary.

At the end of the day, you should seal the Diary in the provided envelope and place it with your Research Kit.

Collecting your saliva samples.

Instructions for collecting your morning samples.

- **Wake up at your usual waking time for a school day. As soon as you wake up, rinse your mouth and collect the** AM1 **Sample. Do this before you brush you teeth or have anything to eat or drink.**
- **Collect your sample for 5 minutes.**
- **Wait 15 minutes then collect the** AM2 **sample. Collect this for 5 minutes or until the tube is full.**
- **Wait 15 minutes then collect the** AM3 **sample. Collect this for 5 minutes or until the tube is full. Place all the tubes in the bag in your fridge.**
- **Write the times you collected the sample in the box below.**

Instructions for collecting your 4 p.m. sample.

- **At 4 p.m. rinse your mouth and collect your sample for 5 minutes. Place the tube in the bag in your fridge.**

Instructions for collecting your 9 p.m. sample.

- **At 9 p.m. rinse your mouth and collect your sample for 5 minutes. Place the tube in the bag in your fridge.**

We know that although you will try your best, sometimes you won't be able to collect the sample exactly at the scheduled time – that's okay but we really need to know the actual time you collected it.

Scheduled Time	Actual Collection Time
AM1	
AM2	
AM3	
4 p.m.	
9 p.m.	

Today is Monday/Tuesday/Wednesday/Thursday/Friday/Saturday/Sunday

Today's date _____

1. What time did you go to bed last night?

2. What time did you wake up this morning?

3. How did you wake up?

   Alarm clock Just woke up Someone woke me Noises Another way?

   Please tell us how_____

4. How long did you sleep last night? ____ Hrs ____ minutes

5. Did you go to school today? ____ **YES** ____ **NO**

6. Did you have a sports game or training today? ____ **No** ____ **YES** - if yes, what time? _____

7. The next box lists the kinds of activities you might have done today. Please use the code listed next to the activity to fill in the times when you did these activities.

   Remember, you can come back and add to this table at any time in your day. Codes for some activities you might do are listed below. <u>If you did something that is not listed, please add it to the list.</u>

SL = Sleeping	**Sch**– in classes	**T** = sat a test	**Arg** = had an argument	**Sp** = did sports
CTV = Using a computer, watched TV/DVD etc	**Ml** – ate meal/snack	**RST** = resting (includes listening to music)		

Morning	Afternoon	Evening	Night
A.M.	P.M.	P.M.	A.M.
5:00	12:00	7.00	Midnight
6:00	1.00	8.00	1.00
7:00	2.00	9.00	2.00
8:00	3.00	10.00	3.00
9:00	4.00	11.00	4.00
10:00	5.00		
11:00	6.00		

8. Lots of different things can happen in one day. We would like to know if the things listed below happened to you, and if they did, whether they happened in the morning, during the day or in the evening.

Please ***CHECK*** each of the following events or situations that you **experienced today.**

For each item you check, please tell us **WHEN** it happened using this code:

M = Morning, that is, from the time you woke up until 10 am

D = Day, that is, from 10 am – 4 pm

E = Evening, that is, from 4 pm until you went to bed.

☑	M	D	E	
				studied or did homework with friends
				something bad happened to someone else in your family
				did well on test, quiz, or homework
				argued with your parent/s / guardian/s about something
				Punished or disciplined by parents
				argued with another family member about something
				got along with your parents
				had difficulty getting to school or class on time

			went to a party
			got along with your friends
			did not understand something taught in class
			argued with a close friend, boyfriend, or girlfriend
			had a lot of demands made by friends
			absent from school with permission (for example, you were sick)
			skipped or cut a class or school
			had a test or a quiz at school
			did not get along with adults at school
			had homework due (e.g., a paper, problem set, project or presentation)
			Parents/guardians had an argument with each other
			something good happened to you or you were treated well
			did poorly on a test, quiz, or homework
			had an argument or were punished by an adult at school
			harassed, picked on, or teased by a student in school
			did not turn in homework that was due
			something bad happened to you or you were treated poorly
			got along with adults at school
			had a lot of demands made by your family
			had a lot of work at school or at home (please circle where)
			harassed, picked on, or teased by someone outside of school

9. The following is a list of feelings or experiences that people may have. We would like to know if you had any of these feelings or experiences today. We would also like to know when this was, in the morning, the day or the evening. If you h the feelings or experiences across several times of the day, you should circle both times. *Remember…*

M = Morning, that is, from the time you woke up until 10 am

D = Day, that is, from 10 am – 4 pm

E = Evening, that is, from 4 pm until you went to bed.

1 **2** **3** **4** **5**

Not At All		A Little		Moderately		Quite a Bit		Extremely
Successful	M	1	2	3	4	5	**Frustrated**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Exhausted	M	1	2	3	4	5	**Joyful**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Optimistic	M	1	2	3	4	5	**Under Pressure**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Bored	M	1	2	3	4	5	**Uneasy**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Couldn'tConcentrate	M	1	2	3	4	5	**Energetic**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
In Control	M	1	2	3	4	5	**Happy**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Discouraged	M	1	2	3	4	5	**Guilty**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Appreciated	M	1	2	3	4	5	**Scared**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Nervous	M	1	2	3	4	5	**Rejected**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Hopeless	M	1	2	3	4	5	**Loved**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Encouraged	M	1	2	3	4	5	**Sad**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Regretful	M	1	2	3	4	5	**Calm**	M	1	2	3	4

	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Upset	M	1	2	3	4	5	**Strong**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Ashamed	M	1	2	3	4	5	**Excited**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4
Jittery	M	1	2	3	4	5	**Proud**	M	1	2	3	4
	D	1	2	3	4	5		D	1	2	3	4
	E	1	2	3	4	5		E	1	2	3	4

10. In the last 24 hours did you have an argument or disagreement with anyone? If so, please tell us:

Who was it with?	**What was the main topic of the argument?**	**What time was it?**			**How stressful was this for you?**			
e.g. Mom, Dad, sister, brother, friend,	*e.g. school, chores, friends, other*	*M*	*D*	*E*	Not at all. 1	A little. 2	Somewhat. 3	Very. 4
1.								
2.								
3.								

11. In the past 24 hours, did you experience any of the following physical symptoms? (Check all that apply.)

	Headache	Heart Pounding
	Constipation/Diarrhea	**Nausea/Upset Stomach**
	Muscle Soreness	Hot or Cold Flashes
	Shortness of Breath	**Congestion**
	Tightness in Chest	Poor Appetite
	Trembling/Shaking	**Sore Throat**
	Backache	Dizziness
	Cold Symptoms	**Flu Symptoms**

12. Have you had any of the following problems with your work or other regular daily activities as a result of your physical health today? Please circle the number in the appropriate box

	No	Yes, Slightly	Yes, Very Much
Did you cut down the amount of time you spent on school or other activities?	1	2	3
Did you accomplish less than you would like?	**1**	**2**	**3**
Were you limited in the kind of school- work or other activities you did?	1	2	3
Did you have difficulty performing the school-work or other activities?	**1**	**2**	**3**

13. How many cigarettes did you smoke today? _____ Cigarettes _____ I don't smoke

14. How many DRINKS of alcohol did you have today? 0 / 1 / 2 / 3 / 4 / 5 / 6 / 7plus

(One drink = bottle of beer, a glass of wine, or shot of liquor. Please do not include wine used in religious ceremonies.)

15. We are interested to know about the kinds of things you eat, and where you eat meals. Please answer the following questions by circling the truest response, or by writing your answer.

a. Did you eat breakfast? NO YES If yes, Where? (e.g. home, school, restaurant)_____

b. Did you eat lunch? NO YES If yes, Where? (e.g. home, school, restaurant)_____

c. Did you eat dinner? NO YES If yes, Where? (e.g. home, school, restaurant)_____

16. Did your family eat at least one meal together today?	**Yes**	**No**
17. I was just too busy to eat dinner with my family.	**Yes**	**No**
18. Today in my family, different schedules made it hard to eat meals together.	**Yes**	**No**

Appendix B

Table of Correlations for the Whole Sample

Table B.1. Correlations between All Variables

		1	2	3	4	5	6	7	8	9	10	11	12	13
1	Child's Age	-												
2	Family Socioeconomic Status	-.19	-											
3	Self-Reported Pubertal Stage	.72**	.01	-										
4	Childhood Symptoms of Depression - DISC	-.06	.12	-.21	-									
5	Childhood Child Report Depression - CDI	-.14	-.02	-.16	.43**	-								
6	Childhood CBCL Withdrawn/Depressed	.31*	-.04	.23	.08	.30*	-							
7	Adolescent BMI	.12	-.14	-.07	.04	.13	.32*	-						
8	Body fat percentage	-.13	-.19	-.26*	.03	.20	.23	.83**	-					
9	Waist circumference (cm)	.28	-.17	-.02	.01	.14	.30*	.94**	.74**	-				
10	Day 1 Total Activity Count	-.28*	.08	-.20	.21	.06	-.22	-.04	-.08	-.08	-			
11	Day 2 Total Activity Count	.22	-.08	.09	.20	-.01	.59**	.21	.13	.19	.08	-		
12	Day 3 Total Activity Count	-.07	.10	.06	-.23	-.03	-.17	.12	-.01	.19	.30*	-.10	-	
13	Day 4 Total Activity Count	-.32*	-.09	-.25	.07	.12	-.33*	.31	.34*	.21	.22	-.16	.34*	-
14	Day 1 Daily Hassles	-.01	.22	.08	-.07	-.04	.08	-.05	-.04	-.05	-.02	-.02	-.09	-.24
15	Day 2 Daily Hassles	-.03	.01	-.01	.04	.16	.16	-.02	-.02	-.05	.00	-.09	-.06	-.13
16	Day 3 Daily Hassles	-.10	-.14	-.08	.16	.22	-.08	.01	.02	-.04	.17	.17	-.05	.13
17	Day 4 Daily Hassles	-.12	.14	-.02	.11	-.06	-.27*	-.08	-.03	-.17	.35*	-.02	.21	.13

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		1	2	3	4	5	6	7	8	9	10	11	12	13
18	Day 1 Time 4 Daily Anxious/Depressed	.11	.06	.12	.06	.03	.01	.03	-.06	.01	.13	.03	-.01	.15
19	Day 2 Time 4 Daily Anxious/Depressed	.06	.20	.08	.07	.10	.02	-.10	-.10	-.10	-.00	-.01	.07	.15
20	Day 3 Time 4 Daily Anxious/Depressed	-.17	.10	.05	.05	.12	-.11	-.16	-.03	-.21	.08	-.08	.12	.34*
21	Day 4 Time 4 Daily Anxious/Depressed	-.10	.23	.02	.06	-.00	-.07	-.17	-.10	-.24*	.06	-.00	-.01	.13
22	Day 1 Sleep	-.10	.07	-.03	-.18	-.18	-.00	-.26*	-.13	-.23*	-.09	-.08	-.09	.05
23	Day 2 Sleep	-.17	-.09	-.22	.07	.16	-.06	-.11	.04	-.18	.24	-.08	-.10	.04
24	Day 3 Sleep	.09	.10	.06	-.08	.06	.06	-.12	-.26*	-.16	.09	-.16	-.23	-.39*
25	Day 4 Sleep	-.14	.13	-.12	.12	.17	-.01	-.05	-.05	-.05	.07	.01	-.30*	-.11
26	AUC Increase Lab Stress Childhood	.34**	-.18	.23	-.09	-.13	.31*	.11	.07	.08	-.16	.63**	-.19	-.36*
27	AUC Increase Lab Stress Adolescent	.05	.00	-.01	.00	-.10	.09	.28*	.25*	.27*	.07	.14	-.04	.09
28	AUCg Day 1 Time 4	.08	.05	.08	-.10	-.16	.07	-.25*	-.19	-.24*	-.21	-.00	-.13	-.27
29	AUCg Day 2 Time 4	.07	.08	.05	-.09	-.13	-.00	-.14	-.16	-.11	-.21	-.02	.12	-.04
30	AUCG Day 3 Time 4	.10	.11	.01	-.03	.09	.04	-.14	-.13	-.10	-.00	-.08	.20	-.06
31	AUCg Day 4 Time 4	.06	.06	.05	-.06	-.11	-.02	-.05	-.01	-.11	.01	-.18	.27	-.03
32	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 4	-.10	.20	-.02	-.02	-.17	-.02	-.25*	-.17	-.29**	-.09	-.03	-.10	-.10
33	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 4	.02	-.13	-.02	-.12	-.14	-.02	-.19	-.12	-.17	-.18	-.06	-.17	-.32*
34	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 4	-.10	.20	-.02	-.02	-.17	-.02	-.24*	-.17	-.29**	-.09	-.03	-.10	-.10
35	Saliva Cortisol (ug/dL) Day 1 4pm Home Sample Time 4	.35**	.13	.27*	-.17	-.03	.29*	-.05	-.11	.01	-.18	.11	-.07	-.21

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		1	2	3	4	5	6	7	8	9	10	11	12	13
36	Saliva Cortisol (ug/dL) Day 1 9pm Home Sample Time 4	.19	-.11	.21	.04	-.01	.13	-.15	-.13	-.17	-.13	.06	.10	-.06
37	Saliva Cortisol (ug/dL) Day 2 AM1 Home Sample Time 4	.03	-.08	-.04	-.04	-.06	-.02	-.10	-.19	-.02	-.17	-.02	.01	-.06
38	Saliva Cortisol (ug/dL) Day 2 AM2 Home Sample Time 4	.03	.00	.00	-.06	-.13	-.03	-.20	-.19	-.15	-.18	.07	.13	-.12
39	Saliva Cortisol (ug/dL) Day 2 AM3 Home Sample Time 4	-.05	.03	-.00	-.06	-.10	-.02	-.01	.07	-.09	-.20	.05	-.11	.01
40	Saliva Cortisol (ug/dL) Day 2 4pm Home Sample Time 4	.11	.16	.06	-.04	-.07	.04	-.04	-.14	-.01	-.06	-.16	.01	.10
41	Saliva Cortisol (ug/dL) Day 2 9pm Home Sample Time 4	.16	.07	.17	-.06	.01	.00	-.08	-.17	-.03	.01	-.07	.43**	-.12
42	Saliva Cortisol (ug/dL) Day 3 AM1 Home Sample Time 4	.07	.02	-.05	-.01	.23	.14	-.09	-.02	-.04	-.09	.14	.05	-.08
43	Saliva Cortisol (ug/dL) Day 3 AM2 Home Sample Time 4	.025	.04	.06	-.06	.21	.08	-.16	-.13	-.12	.03	-.07	.30*	-.03
44	Saliva Cortisol (ug/dL) Day 3 AM3 Home Sample Time 4	.02	.16	-.00	.04	.04	-.02	-.23*	-.17	-.18	-.07	-.13	.17	-.13
45	Saliva Cortisol (ug/dL) Day 3 4pm Home Sample Time 4	.17	.20	.07	.06	-.05	.00	.04	-.05	.04	.14	-.01	.07	.02
46	Saliva Cortisol (ug/dL) Day 3 9pm Home Sample Time 4	.05	-.10	-.08	-.27*	-.12	-.03	.09	.05	.07	-.11	-.09	-.02	-.01
47	Saliva Cortisol (ug/dL) Day 4 AM1 Home Sample Time 4	.08	.06	.11	.00	-.02	.19	-.27*	-.22	-.25*	-.24	.05	.03	-.11
48	Saliva Cortisol (ug/dL) Day 4 AM2 Home Sample Time 4	.04	.10	.02	-.02	-.10	-.05	-.12	-.08	-.16	.08	-.23	.30*	-.00
49	Saliva Cortisol (ug/dL) Day 4 AM3 Home Sample Time 4	-.01	-.00	.01	-.04	-.12	-.04	-.00	.04	-.06	.04	-.16	.27	-.00
50	Saliva Cortisol (ug/dL) Day 4 4pm Home Sample Time 4	.13	.03	.04	-.11	-.01	.00	.09	.09	.04	-.09	-.11	.03	-.01
51	Saliva Cortisol (ug/dL) Day 4 9pm Home Sample Time 4	.08	.14	.14	-.16	-.03	-.05	-.02	-.12	-.00	.14	-.02	.06	-.10

Table B.1. Correlations between All Variables (cont.)

		1	2	3	4	5	6	7	8	9	10	11	12	13
52	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 1	-.07	-.07	-.03	.13	.38**	.05	.00	-.01	.00	.05	-.14	.04	.12
53	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 1	04	.02	-.06	.01	.08	.11	-.19	-.14	-.23	.00	.08	-.21	-.33
	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 1	.05	-.10	.05	-.09	-.10	.20	-.21	-.10	-.25	-.16	.16	-.27	-.30
55	Saliva Cortisol (ug/dL) Day 1 4 PM Home Sample Time 1	-.15	-.02	-.14	-.00	.28*	.03	-.03	.09	.02	.12	-.15	-.09	.01
56	Saliva Cortisol (ug/dL) Day 1 9 PM Home Sample Time 1	.06	-.22	.13	-.22	-.12	-.01	.04	-.01	.10	.23	.11	.08	-.03
57	Saliva Cortisol (ug/dL) AM 1 Home Sample Time 2	.21	-.17	.01	.18	.16	.17	-.11	-.11	-.15	-.06	.18	-.18	-.25
58	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 2	.10	-.09	-.06	.17	.18	-.02	-.26	-.22	-.30*	-.21	-.22	-.13	-.03
59	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 2	.10	.06	.02	.40**	.07	-.12	-.23	-.31*	-.29*	.14	.00	-.25	-.13
60	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 2	-.05	.13	-.07	.06	-.07	-.13	-.23	-.25	-.20	.11	.01	-.38*	-.19
61	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 2	-.01	-.30*	-.05	.07	-.06	-.14	-.04	-.04	-.01	.23	.05	-.10	-.03
62	Saliva Cortisol (ug/dL) AM 1Home Sample Time 3	-.04	-.15	-.00	.10	-.03	.08	-.22	-.21	-.28*	-.06	.12	-.09	.01
63	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 3	-.09	.00	.06	-.27	-.16	.01	-.22	-.08	-.31*	.05	-.07	-.01	-.04
64	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 3	.09	-.19	.04	-.20	-.24	-.03	-.21	-.24	-.19	-.09	-.01	.10	.02
65	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 3	.22	-.10	.12	-.10	-.08	-.13	-.03	-.10	.05	-.02	-.03	.05	.00
66	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 3	.12	.03	-.13	.09	.38**	.11	.36*	.23	.38**	-.08	-.09	-.07	-.09

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		14	15	16	17	18	19	20	21	22	23	24	25	26
1	Child's Age													
2	Family Socioeconomic Status													
3	Self-Reported Pubertal Stage													
4	Childhood Symptoms of Depression - DISC													
5	Childhood Child Report Depression - CDI													
6	Childhood CBCL Withdrawn/Depressed													
7	Adolescent BMI													
8	Body fat percentage													
9	Waist circumference (cm)													
10	Day 1 Total Activity Count													
11	Day 2 Total Activity Count													
12	Day 3 Total Activity Count													
13	Day 4 Total Activity Count													
14	Day 1 Daily Hassles	-												
15	Day 2 Daily Hassles	.54**	-											
16	Day 3 Daily Hassles	.12	.12	-										
17	Day 4 Daily Hassles	.07	.00	.52**	-									

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		14	15	16	17	18	19	20	21	22	23	24	25	26
18	Day 1 Time 4 Daily Anxious/Depressed	.39**	.25*	-.09	.05	-								
19	Day 2 Time 4 Daily Anxious/Depressed	.19	.25*	-.05	.15	.68**	-							
20	Day 3 Time 4 Daily Anxious/Depressed	.13	.11	.23*	.36**	.52**	.57**	-						
21	Day 4 Time 4 Daily Anxious/Depressed	.14	.19	.22	.39**	.45**	.46**	.63**	-					
22	Day 1 Sleep	-.36**	-.30**	-.20	.03	-.15	.00	.11	.05	-				
23	Day 2 Sleep	-.31**	-.21	-.02	.05	-.17	-.13	.09	.10	.26*	-			
24	Day 3 Sleep	.06	.10	-.18	-.12	.17	.03	-.11	-.02	-.02	.08	-		
25	Day 4 Sleep	-.06	.19	-.15	-.10	.02	.08	-.05	.01	.01	.27*	.26*	-	
26	AUC Increase Lab Stress Childhood	.09	-.00	.09	.13	-.09	.00	.08	-.06	-.03	-.01	-.01	-.08	-
27	AUC Increase Lab Stress Adolescent	.19	.22	-.05	.00	.05	-.11	-.07	-.08	.01	-.07	-.11	-.03	.11
28	AUCg Day 1 Time 4	.16	.11	-.12	-.06	-.08	-.05	-.08	.02	-.00	-.10	-.03	-.13	.02
29	AUCg Day 2 Time 4	.23*	.20	-.10	-.02	-.13	-.15	-.11	-.16	-.12	-.32**	-.10	-.24*	-.03
30	AUCG Day 3 Time 4	.05	.12	.08	.00	-.07	.08	.21	-.06	-.01	-.03	-.14	-.17	-.06
31	AUCg Day 4 Time 4	.08	-.02	.04	.19	-.07	-.08	.04	-.07	-.08	.02	-.14	-.38**	.16
32	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 4	.13	.13	-.16	-.06	-.00	.026	.02	.19	.05	-.07	.04	-.11	-.05
33	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 4	.01	.09	-.06	-.09	-.21	-.14	-.12	-.16	-.02	-.04	-.02	-.10	.00
34	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 4	.13	.13	-.16	-.06	-.00	.02	.02	.19	.05	-.07	.01	-.11	-.05
35	Saliva Cortisol (ug/dL) Day 1 4pm Home Sample Time 4	.19	-.10	-.15	-.10	-.03	-.09	-.10	-.06	-.02	-.11	.00	-.02	.19

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		14	15	16	17	18	19	20	21	22	23	24	25	26
36	Saliva Cortisol (ug/dL) Day 1 9pm Home Sample Time 4	.21	.18	.14	.19	.15	.10	.08	.13	-.09	-.08	-.18	-.12	.04
37	Saliva Cortisol (ug/dL) Day 2 AM1 Home Sample Time 4	-.09	-.03	.07	-.04	-.21	-.17	-.19	-.10	.00	-.13	-.08	-.09	-.22
38	Saliva Cortisol (ug/dL) Day 2 AM2 Home Sample Time 4	.00	.11	-.13	-.05	-.15	-.19	-.19	-.18	-.04	-.32**	-.08	-.20	-.01
39	Saliva Cortisol (ug/dL) Day 2 AM3 Home Sample Time 4	.21	.08	-.12	-.03	-.09	-.15	-.08	-.09	-.07	-.22	-.11	-.29*	.06
40	Saliva Cortisol (ug/dL) Day 2 4pm Home Sample Time 4	.28*	.27*	-.04	-.01	.01	.04	.08	.01	-.10	-.10	.01	.06	-.03
41	Saliva Cortisol (ug/dL) Day 2 9pm Home Sample Time 4	.23*	.14	.06	.11	-.01	.02	-.01	-.16	-.16	-.17	-.06	-.11	-.03
42	Saliva Cortisol (ug/dL) Day 3 AM1 Home Sample Time 4	-.05	-.03	.09	-.07	-.12	-.01	.11	-.07	-.00	.05	-.20	-.25*	-.0
43	Saliva Cortisol (ug/dL) Day 3 AM2 Home Sample Time 4	-.09	.02	.17	.03	-.19	-.02	.15	-.01	.05	.00	-.06	-.14	-.03
44	Saliva Cortisol (ug/dL) Day 3 AM3 Home Sample Time 4	-.03	.08	.02	-.04	-.01	.12	.33**	.01	.01	.09	-.17	-.13	-.04
45	Saliva Cortisol (ug/dL) Day 3 4pm Home Sample Time 4	.33**	.17	-.08	.10	.13	.19	.06	-.06	-.04	-.17	.01	-.01	-.03
46	Saliva Cortisol (ug/dL) Day 3 9pm Home Sample Time 4	.04	.14	.04	-.06	-.0	-.10	-.13	-.13	-.12	-.12	-.05	-.05	.00
47	Saliva Cortisol (ug/dL) Day 4 AM1 Home Sample Time 4	-.08	-.04	-.14	-.10	-.19	-.04	.07	.07	.03	.12	-.02	-.00	.13
48	Saliva Cortisol (ug/dL) Day 4 AM2 Home Sample Time 4	-.10	-.02	.00	.18	-.17	-.07	-.00	-.07	-.03	.11	-.14	-.23*	.07
49	Saliva Cortisol (ug/dL) Day 4 AM3 Home Sample Time 4	.12	-.03	.08	.23*	-.04	-.07	.12	-.06	-.06	.00	-.16	-.43*	.19
50	Saliva Cortisol (ug/dL) Day 4 4pm Home Sample Time 4	.15	.01	.06	.13	.04	.01	-.01	-.07	-.06	-.12	-.07	-.34**	.15
51	Saliva Cortisol (ug/dL) Day 4 9pm Home Sample Time 4	.26*	.03	-.05	-.04	.16	-.14	-.17	-.07	-.22*	.05	.13	.11	-.11

Table B.1. Correlations between All Variables (cont.)

		14	15	16	17	18	19	20	21	22	23	24	25	26
52	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 1	-.12	-.08	.14	.05	.07	-.06	.23	-.05	-.04	.11	-.00	.08	.06
53	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 1	.10	-.05	-.03	-.05	.01	-.08	.13	-.11	-.04	.17	.21	-.31*	.37*
	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 1	.16	.08	.19	.11	-.03	-.00	.10	.12	.19	.13	.13	-.23	.42**
55	Saliva Cortisol (ug/dL) Day 1 4 PM Home Sample Time 1	.14	.16	.06	-.09	-.00	-.02	-.02	.11	.18	-.01	-.05	-.17	-.08
56	Saliva Cortisol (ug/dL) Day 1 9 PM Home Sample Time 1	-.09	-.11	-.03	-.22	.05	-.26	-.09	-.08	-.22	-.01	.00	.09	.07
57	Saliva Cortisol (ug/dL) AM 1 Home Sample Time 2	.00	.09	.06	-.17	.00	.11	.07	.02	-.18	.08	-.02	-.05	.01
58	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 2	-.04	.27*	.10	-.07	.18	.26	.21	.12	-.16	.03	.05	.01	-.18
59	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 2	-.20	.02	.25	-.07	.00	.07	.08	-.07	-.01	.06	.18	-.03	-.12
60	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 2	-.04	-.14	-.08	-.04	-.11	.04	.03	-.10	.29*	-.05	-.07	.02	-.21
61	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 2	-.10	-.10	.16	-.12	-.05	-.00	.00	.02	.17	.05	-.21	-.16	-.07
62	Saliva Cortisol (ug/dL) AM 1Home Sample Time 3	-.02	.04	.27	.15	.08	.09	.20	.08	.14	-.22	-.03	-.28	-.04
63	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 3	.07	.12	.28	.24	-.02	.08	.10	.13	.10	-.11	-.05	-.34*	-.17
64	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 3	.00	.03	.23	.27	.20	.12	.16	.11	.03	-.28*	-.11	-.31*	-.07
65	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 3	.01	.11	.04	.01	-.12	-.04	-.08	-.02	.02	-.24	-.23	.08	.01
66	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 3	-.07	.11	.13	-.19	-.05	-.04	-.02	-.16	-.06	-.08	.09	.28	.06

Table B.1. Correlations between All Variables (cont.)

		27	28	29	30	31	32	33	34	35	36	37	38	39
1	Child's Age	.05	.08	.07	.10	.06	-.10	.02	-.10	.35**	.19	.03	.03	-.05
2	Family Socioeconomic Status	.00	.05	.08	.11	.06	.20	-.13	.20	.13	-.11	-.08	.00	.03
3	Self-Reported Pubertal Stage	-.01	.08	.05	.01	.05	-.02	-.02	-.02	.27*	.21	-.04	.00	-.00
4	Childhood Symptoms of Depression - DISC	.00	-.15	-.09	-.03	-.06	-.02	-.12	-.02	-.17	.04	-.04	-.06	-.06
5	Childhood Child Report Depression - CDI	-.10	-.16	-.13	.09	-.11	-.17	-.14	-.17	-.01	-.01	-.06	-.13	-.10
6	Childhood CBCL Withdrawn/Depressed	.09	.07	-.00	.04	-.02	-.02	-.02	-.02	.29*	.13	-.02	-.03	-.02
7	Adolescent BMI	.28*	-.25*	-.14	-.14	-.05	-.25*	-.19	-.24*	-.05	-.15	-.10	-.20	-.01
8	Body fat percentage	.25*	-.19	-.16	-.13	-.01	-.17	-.12	-.17	-.11	-.13	-.19	-.19	.07
9	Waist circumference (cm)	.27*	-.24*	-.11	-.10	-.11	-.29**	-.17	-.29**	.01	-.17	-.02	-.15	-.09
10	Day 1 Total Activity Count	.07	-.21	-.28	-.02	.01	-.09	-.18	-.09	-.18	-.13	-.17	-.18	-.20
11	Day 2 Total Activity Count	.14	-.00	-.04	-.08	-.18	-.03	-.06	-.03	.11	.06	-.02	.07	.05
12	Day 3 Total Activity Count	-.04	-.13	.12	.20	.27	-.10	-.17	-.10	-.07	.10	.01	.13	-.11
13	Day 4 Total Activity Count	.09	-.27	-.04	-.06	-.03	-.10	-.32*	-.10	-.21	-.06	-.06	-.12	.01
14	Day 1 Daily Hassles	.19	.16	.23*	.05	.08	.13	.01	.13	.19	.21	-.09	.00	.21
15	Day 2 Daily Hassles	.22	.11	.20	.12	-.02	.13	.09	.13	-.10	.18	-.03	.11	.08
16	Day 3 Daily Hassles	-.05	-.12	-.10	.08	.04	-.16	-.06	-.16	-.15	.14	.07	-.13	-.12
17	Day 4 Daily Hassles	.00	-.06	-.02	.00	.19	-.01	-.09	-.06	-.10	.19	-.04	-.05	-.03

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		27	28	29	30	31	32	33	34	35	36	37	38	39
18	Day 1 Time 4 Daily Anxious/Depressed													
19	Day 2 Time 4 Daily Anxious/Depressed													
20	Day 3 Time 4 Daily Anxious/Depressed													
21	Day 4 Time 4 Daily Anxious/Depressed													
22	Day 1 Sleep													
23	Day 2 Sleep													
24	Day 3 Sleep													
25	Day 4 Sleep													
26	AUC Increase Lab Stress Childhood													
27	AUC Increase Lab Stress Adolescent	-												
28	AUCg Day 1 Time 4	.25*	-											
29	AUCg Day 2 Time 4	.19	.47**	-										
30	AUCG Day 3 Time 4	.18	.41**	.51**	-									
31	AUCg Day 4 Time 4	.18	.34**	.41**	.42**	-								
32	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 4	.21	.82**	.41**	.24*	.28*	-							
33	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 4	.13	.77**	.40**	.39**	.25*	.42**	-						
34	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 4	.21	.82**	.41**	.24*	.28*	.89**	.42**	-					
35	Saliva Cortisol (ug/dL) Day 1 4pm Home Sample Time 4	.18	.56**	.23*	.29**	.21	.33**	.21	.33**	-				

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		27	28	29	30	31	32	33	34	35	36	37	38	39
36	Saliva Cortisol (ug/dL) Day 1 9pm Home Sample Time 4	.18	.51**	.17	.17	.18	.28*	.34**	.28*	.18	-			
37	Saliva Cortisol (ug/dL) Day 2 AM1 Home Sample Time 4	.16	.40**	.50**	.35**	.22*	.17	.57**	.17	.20	.06	-		
38	Saliva Cortisol (ug/dL) Day 2 AM2 Home Sample Time 4	-.10	.28*	.77**	.23*	.18	.28*	.27*	.28*	.08	-.02	.35**	-	
39	Saliva Cortisol (ug/dL) Day 2 AM3 Home Sample Time 4	.11	.29**	.72**	.15	.35**	.40**	.15	.40**	.03	.05	.05	.59*	-
40	Saliva Cortisol (ug/dL) Day 2 4pm Home Sample Time 4	.37**	.29**	.52**	.47**	.18	.27*	.09	.27*	.33**	.10	.25*	.04	.11
41	Saliva Cortisol (ug/dL) Day 2 9pm Home Sample Time 4	.11	.29**	.44**	.53**	.36**	-.02	.42**	-.02	.14	.45**	.36**	.15	-.03
42	Saliva Cortisol (ug/dL) Day 3 AM1 Home Sample Time 4	.09	.28*	.29**	.69**	.22*	.09	.34**	.09	.22	.12	.30**	.14	.13
43	Saliva Cortisol (ug/dL) Day 3 AM2 Home Sample Time 4	.03	.24*	.34**	.77**	.44**	.14	.20	.14	.28*	.00	.37**	.20	.06
44	Saliva Cortisol (ug/dL) Day 3 AM3 Home Sample Time 4	.00	.21	.32*)	.82**	.35**	.19	.17	.19	.12	.01	.10	.23*	.16
45	Saliva Cortisol (ug/dL) Day 3 4pm Home Sample Time 4	.33*)	.32**	.41**	.49**	.13	.27*	.13	.27*	.30**	.27*	.05	.04	.14
46	Saliva Cortisol (ug/dL) Day 3 9pm Home Sample Time 4	.19	.36**	.26*	.34**	.09	.04	.68**	.04	-.02	.26*	.50**	.09	-.01
47	Saliva Cortisol (ug/dL) Day 4 AM1 Home Sample Time 4	-.01	.26*	.38**	.41**	.42**	.23*	.15	.23*	.30**	-.06	.35**	.31**	.179
48	Saliva Cortisol (ug/dL) Day 4 AM2 Home Sample Time 4	.06	.26*	.35**	.44**	.85**	.23*	.22*	.23*	.12	.06	.23*	.25*	.21
49	Saliva Cortisol (ug/dL) Day 4 AM3 Home Sample Time 4	.19	.22*	.33**	.36**	.92**	.19	.14	.19	.10	.17	.07	.06	.35**
50	Saliva Cortisol (ug/dL) Day 4 4pm Home Sample Time 4	.22*	.43**	.29**	.19	.68**	.32**	.29**	.32**	.31**	.30**	.18	.10	.34**
51	Saliva Cortisol (ug/dL) Day 4 9pm Home Sample Time 4	-.00	-.00	.01	-.14	.07	-.08	.02	-.08	.07	.02	.15	-.04	-.08

Table B.1. Correlations between All Variables (cont.)

		27	28	29	30	31	32	33	34	35	36	37	38	39
52	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 1	-.07	-.15	-.00	.15	.18	-.14	-.13	-.14	.05	-.18	.04	-.02	-.05
53	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 1	-.04	.12	.14	.14	.41**	.14	.06	.14	.19	-.11	-.08	.07	.28*
	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 1	.00	.04	-.02	-.12	.13	.06	-.06	.06	.07	.13	-.19	-.02	.13
55	Saliva Cortisol (ug/dL) Day 1 4 PM Home Sample Time 1	.10	.05	-.19	-.11	-.12	.03	.06	.03	.04	-.00	-.06	-.08	-.14
56	Saliva Cortisol (ug/dL) Day 1 9 PM Home Sample Time 1	.01	.12	.07	-.05	-.02	-.02	.15	-.04	.20	.09	-.02	.01	.03
57	Saliva Cortisol (ug/dL) AM 1 Home Sample Time 2	-.17	-.01	-.05	.16	.01	-.02	-.10	-.02	.08	.10	-.08	.04	.02
58	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 2	-.06	.20	.08	.34*	.07	.10	.27*	.11	-.06	.28*	.31*	.10	-.11
59	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 2	-.02	.10	-.04	.23	.06	.15	.08	.15	-.09	.06	.18	.00	-.105
60	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 2	.077	.07	-.013	.17	.16	.02	.09	.02	.25	-.22	.26	-.07	-.00
61	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 2	-.01	-.08	-.20	-.05	-.23	-.10	.01	-.10	-.02	-.17	.05	-.14	-.08
62	Saliva Cortisol (ug/dL) AM 1Home Sample Time 3	-.08	.1	.23	.07	-.01	.18	.01	.18	-.02	.12	.26	.23	.25
63	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 3	.03	.30*	.18	.19	.27	.32*	.34*	.32*	-.13	.17	.39**	.02	.13
64	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 3	.11	.13	.16	.16	.20	.06	.19	.06	-.06	.20	.39**	.10	.01
65	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 3	.34*	.15	.13	.13	-.08	.14	.20	.14	-.11	.17	.34*	-.03	-.20
66	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 3	.30*	-.07	-.11	.14	-.06	-.11	-.02	-.11	.03	-.09	-.06	-.20	-.19

Correlations between All Variables (cont.)

		40	41	42	43	44	45	46	47	48	49	50	51	52
1	Child's Age	.11	.16	.07	.02	.02	.17	.05	.08	.04	-.01	.13	.06	-.07
2	Family Socioeconomic Status	.16	.07	.02	.04	.14	.20	-.15	.06	.10	-.00	.03	.14	-.07
3	Self-Reported Pubertal Stage	.06	.17	-.05	.06	-.00	.07	-.08	.11	.02	.01	.04	.14	-.03
4	Childhood Symptoms of Depression - DISC	-.04	-.06	-.01	-.06	.04	.06	-.27*	.00	-.02	-.04	-.11	-.16	.13
5	Childhood Child Report Depression - CDI	-.07	.01	.23	.21	.04	-.05	-.12	-.02	-.10	-.12	-.02	-.03	.38**
6	Childhood CBCL Withdrawn/Depressed	.04	.00	.14	.08	-.02	.00	-.03	.198	-.05	-.04	.00	-.05	.05
7	Adolescent BMI	-.04	-.08	-.09	-.16	-.23*	.04	.09	-.27*	-.12	-.00	.09	-.02	.00
8	Body fat percentage	-.14	-.17	-.02	-.13	-.17	-.05	.05	-.22	-.08	.06	.09	-.12	-.01
9	Waist circumference (cm)	-.0	-.03	-.04	-.12	-.18	.04	.07	-.25*	-.16	-.06	.04	-.00	.00
10	Day 1 Total Activity Count	-.06	.01	-.09	.03	-.07	.14	-.11	-.24	.08	.04	-.09	.14	.05
11	Day 2 Total Activity Count	-.16	-.07	.14	-.07	-.13	-.01	-.09	.05	-.23	-.16	-.11	-.02	-.14
12	Day 3 Total Activity Count	.01	.43**	.05	.30*	.17	.07	-.02	.03	.30*	.27	.03	.06	.04
13	Day 4 Total Activity Count	.10	-.12	-.08	-.03	-.13	.02	-.01	-.11	-.00	-.00	-.01	-.10	.12
14	Day 1 Daily Hassles	.28*	.23*	-.05	-.09	-.03	.33**	.04	-.08	-.10	.12	.15	.26*	-.12
15	Day 2 Daily Hassles	.27*	.14	-.03	.02	.08	.17	.14	-.04	-.02	-.03	.01	.03	-.08
16	Day 3 Daily Hassles	-.04	.06	.09	.17	.02	-.03	.04	-.14	.00	.08	.06	-.05	.14
17	Day 4 Daily Hassles	-.01	.11	-.07	.03	-.04	.10	-.06	-.10	.18	.23*	.13	-.04	.05

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		40	41	42	43	44	45	46	47	48	49	50	51	52
18	Day 1 Time 4 Daily Anxious/Depressed	.01	-.01	-.12	-.19	-.01	.13	-.08	-.19	-.17	-.04	.04	.16	.07
19	Day 2 Time 4 Daily Anxious/Depressed	.04	.02	-.01	-.02	.12	.19	-.10	-.04	-.07	-.07	.01	-.14	-.06
20	Day 3 Time 4 Daily Anxious/Depressed	.08	-.01	.11	.15	.33**	.06	-.13	.07	-.00	.12	-.01	-.17	.23
21	Day 4 Time 4 Daily Anxious/Depressed	.01	-.16	-.07	-.01	.01	-.06	-.13	.07	-.07	-.06	-.07	-.07	-.05
22	Day 1 Sleep	-.10	-.15	-.00	.05	.01	-.04	-.12	.03	-.03	-.06	-.06	-.22*	-.04
23	Day 2 Sleep	-.10	-.17	.05	.00	.09	-.17	-.12	.12	.11	.00	-.12	.05	.11
24	Day 3 Sleep	.019	-.06	-.20	-.06	-.17	.01	-.05	-.02	-.14	-.16	-.07	.13	-.00
25	Day 4 Sleep	.06	-.15	-.25*	-.14	-.13	-.01	-.05	-.00	-.23*	-.43**	-.34**	.11	.08
26	AUC Increase Lab Stress Childhood	-.03	-.03	-.09	-.03	-.04	-.07	.00	.13	.07	.19	.15	-.11	.06
27	AUC Increase Lab Stress Adolescent	.37**	.11	.09	.03	.00	.33**	.19	-.01	.06	.19	.22*	-.00	-.07
28	AUCg Day 1 Time 4	.29**	.29**	.28*	.24*	.21	.32**	.36**	.26*	.26*	.22*	.43**	-.00	-.15
29	AUCg Day 2 Time 4	.52**	.44**	.29**	.34**	.32**	.41**	.26*	.38**	.35**	.33**	.29**	.01	-.00
30	AUCG Day 3 Time 4	.478**	.53**	.69**	.77**	.82**	.49**	.34**	.41**	.44**	.36**	.19	-.14	.15
31	AUCg Day 4 Time 4	.18	.36**	.22*	.448**	.35**	.13	.09	.42**	.85**	.92**)	.68**	.07	.18
32	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 4	.27*	-.02	.09	.14	.19	.27*	.04	.23*	.23*	.19	.32**	-.08	-.14
33	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 4	.09	.42**	.34**	.20	.17	.13	.68**	.15	.22*	.14	.29**	.02	-.13
34	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 4	.27*	-.02	.09	.14	.19	.27*	.04	.23*	.23*	.19	.32**	-.08	-.14
35	Saliva Cortisol (ug/dL) Day 1 4pm Home Sample Time 4	.33**	.14	.22	.28*	.12	.30**	-.02	.30**	.12	.10	.31**	.07	.05

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		40	41	42	43	44	45	46	47	48	49	50	51	52
36	Saliva Cortisol (ug/dL) Day 1 9pm Home Sample Time 4													
37	Saliva Cortisol (ug/dL) Day 2 AM1 Home Sample Time 4													
38	Saliva Cortisol (ug/dL) Day 2 AM2 Home Sample Time 4													
39	Saliva Cortisol (ug/dL) Day 2 AM3 Home Sample Time 4													
40	Saliva Cortisol (ug/dL) Day 2 4pm Home Sample Time 4	-												
41	Saliva Cortisol (ug/dL) Day 2 9pm Home Sample Time 4	.29**	-											
42	Saliva Cortisol (ug/dL) Day 3 AM1 Home Sample Time 4	.16	.33**	-										
43	Saliva Cortisol (ug/dL) Day 3 AM2 Home Sample Time 4	.27*	.36**	.46**	-									
44	Saliva Cortisol (ug/dL) Day 3 AM3 Home Sample Time 4	.26*	.23*	.60**	.59**	-								
45	Saliva Cortisol (ug/dL) Day 3 4pm Home Sample Time 4	.64**	.43**	.09	.13	.18	-							
46	Saliva Cortisol (ug/dL) Day 3 9pm Home Sample Time 4	.13	.47**	.23*	.13	.02	.11	-						
47	Saliva Cortisol (ug/dL) Day 4 AM1 Home Sample Time 4	.30**	.08	.30**	.58**	.45**	-.09	-.08	-					
48	Saliva Cortisol (ug/dL) Day 4 AM2 Home Sample Time 4	.12	.33**	.20	.54**	.39**	.06	.06	.50**	-				
49	Saliva Cortisol (ug/dL) Day 4 AM3 Home Sample Time 4	.15	.34**	.20	.38**	.31**	.13	.05	.29**	.70**	-			
50	Saliva Cortisol (ug/dL) Day 4 4pm Home Sample Time 4	.05	.21	.10	.07	.09	.22*	.17	-.01	.39**	.53**	-		
51	Saliva Cortisol (ug/dL) Day 4 9pm Home Sample Time 4	.07	.08	-.06	-.21	-.16	-.01	.09	-.12	-.13	-.02	.09	-	

Table B.1. Correlations between All Variables (cont.)

		40	41	42	43	44	45	46	47	48	49	50	51	52
52	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 1	.00	.06	.12	.37**	.13	-.06	-.12	.19	.27*	.16	-.04	.02	-
53	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 1	.04	-.04	.16	.20	.20	-.05	-.11	.29*	.29*	.38**	.25	.12	.49**
	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 1	-.04	-.13	-.09	-.00	-.00	-.16	-.25	.16	.14	.10	.09	-.03	.06
55	Saliva Cortisol (ug/dL) Day 1 4 PM Home Sample Time 1	-.15	-.16	.03	-.00	-.16	-.14	-.03	-.05	-.11	-.12	-.04	-.06	-.09
56	Saliva Cortisol (ug/dL) Day 1 9 PM Home Sample Time 1	.11	.07	.08	-.05	-.02	-.16	.04	.01	-.08	.00	.00	.00	-.01
57	Saliva Cortisol (ug/dL) AM 1 Home Sample Time 2	-.10	-.13	.11	.23	.20	.02	-.17	.17	.11	-.01	-.03	-.21	.09
58	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 2	.00	.18	.27*	.31*	.29*	-.07	.35**	.17	.16	-.01	.03	-.06	.13
59	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 2	-.08	-.01	.05	.27*	.33*	-.06	.03	.09	.14	-.00	.02	-.00	.11
60	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 2	.03	-.17	.10	.28*	.18	-.02	-.06	.22	.16	.05	.22	-.01	.18
61	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 2	-.22	-.13	-.06	.12	-.05	-.12	-.10	-.14	-.11	-.18	-.21	-.18	.24
62	Saliva Cortisol (ug/dL) AM 1Home Sample Time 3	.03	-.07	.15	.04	.12	-.04	-.03	.02	-.06	-.05	.17	-.10	-.07
63	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 3	.01	.19	.23	.23	.05	-.04	.27	.09	.30*	.17	.27	-.00	.00
64	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 3	.00	.21	.25	.14	.08	-.07	.24	.07	.23	.18	.17	-.14	.01
65	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 3	.31*	.28*	.10	.04	-.06	.18	.31*	.00	.00	-.12	-.06	.00	-.00
66	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 3	.17	-.02	.03	.05	.03	.17	.21	-.16	-.10	-.08	.12	.00	.22

Correlations between All Variables (cont.)

		53	54	55	56	57	58	59	60	61	62	63	64	65	66
1	Child's Age	.04	.05	-.15	.06	.21	.10	.10	-.05	-.01	-.04	-.09	.09	.22	.12
2	Family Socioeconomic Status	.02	-.10	-.02	-.22	-.17	-.09	.06	.13	-.30*	-.15	.00	-.19	-.10	.03
3	Self-Reported Pubertal Stage	-.06	.05	-.14	.13	.01	-.06	.02	-.07	-.05	-.00	.06	.04	.12	-.13
4	Childhood Symptoms of Depression - DISC	.01	-.09	-.00	-.22	.18	.17	.40**	.06	.07	.10	-.27	-.20	-.10	.099
5	Childhood Child Report Depression - CDI	.08	-.10	.28*	-.12	.16	.18	.07	-.07	-.06	-.03	-.16	-.24	-.08	.38**
6	Childhood CBCL Withdrawn/Depressed	.11	.20	.03	-.01	.17	-.02	-.12	-.13	-.14	.08	.01	-.03	-.13	.11
7	Adolescent BMI	-.19	-.21	-.03	.04	-.11	-.26	-.23	-.23	-.04	-.22	-.22	-.21	-.03	.36*
8	Body fat percentage	-.14	-.10	.09	-.01	-.11	-.22	-.31*	-.23	-.04	-.21	-.08	-.24	-.10	.23
9	Waist circumference (cm)	-.23	-.25	.02	.10	-.15	-.30*	-.29*	-.23	-.01	-.27*	-.31	-.19	.05	.39**
10	Day 1 Total Activity Count	.00	-.16	.12	.23	-.06	-.21	.14	.11	.23	-.06	.05	-.09	-.03	-.08
11	Day 2 Total Activity Count	.08	.16	-.15	.11	.18	-.22	.00	.01	.05	.12	-.07	-.02	-.08	-.10
12	Day 3 Total Activity Count	-.21	-.27	-.09	.08	-.18	-.13	-.25	-.38*	-.10	-.09	-.01	.11	.05	-.07
13	Day 4 Total Activity Count	-.33	-.30	.01	-.03	-.25	-.03	-.13	-.19	-.03	.01	-.05	.02	.00	.11
14	Day 1 Daily Hassles	.10	.16	.14	-.09	.00	-.04	-.20	-.04	-.10	-.03	.07	.00	.02	-.07
15	Day 2 Daily Hassles	-.05	.08	.16	-.11	.09	.27*	.02	-.14	-.10	.05	.12	.04	.12	.13
16	Day 3 Daily Hassles	-.03	.19	.06	-.03	.06	.10	.25	-.05	.16	.27	.28	.23	.05	.14
17	Day 4 Daily Hassles	-.05	.11	-.09	-.22	-.17	-.07	-.07	-.04	-.12	.15	.24	.27	.01	-.19

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		53	54	55	56	57	58	59	60	61	62	63	64	65	66
18	Day 1 Time 4 Daily Anxious/Depressed	.01	-.03	-.00	.05	.00	.18	.00	-.11	-.05	.08	-.02	.21	-.12	-.05
19	Day 2 Time 4 Daily Anxious/Depressed	-.08	-.00	-.02	-.26	.11	.26	.07	.04	-.01	.09	.08	.12	-.05	-.04
20	Day 3 Time 4 Daily Anxious/Depressed	.13	.10	-.02	-.09	.07	.21	.08	.03	.00	.21	.11	.17	-.08	-.02
21	Day 4 Time 4 Daily Anxious/Depressed	-.11	.12	.11	-.08	.02	.12	-.07	-.10	.02	.08	.13	-.11	-.03	-.17
22	Day 1 Sleep	-.04	.19	.18	-.22	-.18	-.16	-.01	.29*	.17	.15	.10	.03	.03	-.06
23	Day 2 Sleep	.17	.13	-.01	-.01	.08	.03	.06	-.05	.05	-.23	-.11	-.28*	-.25	-.08
24	Day 3 Sleep	.21	.13	-.05	.00	-.02	.05	.18	-.07	-.21	-.03	-.05	-.11	-.23	.09
25	Day 4 Sleep	-.31*	-.23	-.17	.09	-.05	.01	-.03	.02	-.16	-.28	-.35*	-.31*	.08	.29
26	AUC Increase Lab Stress Childhood	.37**	.42**	-.08	.07	.01	-.1	-.12	-.21	-.08	-.05	-.17	.02	.07	.02
27	AUC Increase Lab Stress Adolescent	-.04	.00	.10	.01	-.17	-.06	-.02	.07	-.01	-.08	.09	.11	.35*	.30*
28	AUCg Day 1 Time 4	.12	.04	.05	.12	-.01	.20	.10	.07	-.08	.11	.31*	.13	.15	-.07
29	AUCg Day 2 Time 4	.14	-.02	-.19	.07	-.05	.08	-.04	-.01	-.20	.23	.19	.17	.13	-.17
30	AUCG Day 3 Time 4	.14	-.12	-.11	-.05	.16	.34*	.23	.17	-.05	.07	.19	.16	.13	.14
31	AUCg Day 4 Time 4	.41**	.13	-.12	-.02	.01	.07	.06	.16	-.23	-.01	.27	.21	-.07	-.06
32	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 4	.14	.06	.03	-.04	-.02	.10	.15	.02	-.10	.18	.32*	.06	.14	-.11
33	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 4	.06	-.06	.06	.15	-.10	.27*	.08	.09	.01	.02	.35*	.19	.21	-.02
34	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 4	.14	.06	.03	-.04	-.02	.10	.15	.02	-.10	.18	.32*	.06	-.14	.11
35	Saliva Cortisol (ug/dL) Day 1 4pm Home Sample Time 4	.19	.07	.04	.20	.08	-.06	-.09	.25	-.02	-.02	-.13	-.12	.03	.12

Note. * $p < 0.05$, ** $p < 0.01$

Table B.1. Correlations between All Variables (cont.)

		53	54	55	56	57	58	59	60	61	62	63	64	65	66
36	Saliva Cortisol (ug/dL) Day 1 9pm Home Sample Time 4	-.11	.13	-.00	.09	.10	.28*	.06	-.22	-.17	.13	.19	.20	.17	-.09
37	Saliva Cortisol (ug/dL) Day 2 AM1 Home Sample Time 4	-.08	-.19	-.06	-.02	-.08	.31*	.18	.26	.05	.26	.39**	.39**	.34*	-.06
38	Saliva Cortisol (ug/dL) Day 2 AM2 Home Sample Time 4	.07	-.02	-.08	.01	.04	.10	.00	-.07	-.14	.23	.03	.10	-.06	-.20
39	Saliva Cortisol (ug/dL) Day 2 AM3 Home Sample Time 4	.28*	.13	-.14	.03	.02	-.11	-.10	-.00	-.14	.23	.02	.10	-.06	-.20
40	Saliva Cortisol (ug/dL) Day 2 4pm Home Sample Time 4	.04	-.04	-.15	.11	-.10	.00	-.08	.03	-.08	.25	.13	-.01	-.20	-.19
41	Saliva Cortisol (ug/dL) Day 2 9pm Home Sample Time 4	-.04	-.13	-.16	.07	-.13	.18	-.01	-.17	-.225	.03	.05	.01	.31*	.17
42	Saliva Cortisol (ug/dL) Day 3 AM1 Home Sample Time 4	.16	-.09	.03	.08	.11	.27*	.05	.10	-.14	-.07	.19	.22	.28*	-.02
43	Saliva Cortisol (ug/dL) Day 3 AM2 Home Sample Time 4	.20	-.00	-.00	-.05	.23	.31*	.27*	.28*	-.06	.15	.24	.25	.10	.03
44	Saliva Cortisol (ug/dL) Day 3 AM3 Home Sample Time 4	.20	-.00	-.16	-.02	.20	.29*	.33*	.18	.12	.05	.23	.15	.04	.05
45	Saliva Cortisol (ug/dL) Day 3 4pm Home Sample Time 4	-.05	-.16	-.14	-.16	.02	-.07	-.06	-.02	-.05	.12	.05	.08	-.06	.04
46	Saliva Cortisol (ug/dL) Day 3 9pm Home Sample Time 4	-.11	-.25	-.03	.04	-.17	.35**	.03	-.06	-.13	-.04	-.07	.18	.17	.16
47	Saliva Cortisol (ug/dL) Day 4 AM1 Home Sample Time 4	.29*	.16	-.05	.01	.17	.17	.09	.22	-.12	-.04	.27	.24	.32*	.22
48	Saliva Cortisol (ug/dL) Day 4 AM2 Home Sample Time 4	.29*	.14	-.11	-.03	.11	.16	.1	.16	-.14	.03	.09	.07	.00	-.16
49	Saliva Cortisol (ug/dL) Day 4 AM3 Home Sample Time 4	.38**	.10	-.12	.00	-.01	-.01	-.00	.05	-.12	-.06	.31*	.23	.00	-.10
50	Saliva Cortisol (ug/dL) Day 4 4pm Home Sample Time 4	.25	.09	-.04	.00	-.03	.03	.02	.22	-.18	-.05	.17	.18	-.13	-.08
51	Saliva Cortisol (ug/dL) Day 4 9pm Home Sample Time 4	.12	-.03	-.06	.00	-.21	-.06	-.00	-.01	-.22	.17	.27	.17	-.06	.13

Table B.1. Correlations between All Variables (cont.)

		53	54	55	56	57	58	59	60	61	62	63	64	65	66
52	Saliva Cortisol (ug/dL) Day 1 AM1 Home Sample Time 1														
53	Saliva Cortisol (ug/dL) Day 1 AM2 Home Sample Time 1	-													
	Saliva Cortisol (ug/dL) Day 1 AM3 Home Sample Time 1	.49**	-												
55	Saliva Cortisol (ug/dL) Day 1 4 PM Home Sample Time 1	-.07	.19	-											
56	Saliva Cortisol (ug/dL) Day 1 9 PM Home Sample Time 1	.0	.06	.49**	-										
57	Saliva Cortisol (ug/dL) AM 1 Home Sample Time 2	.10	-.00	.03	-.29*	-	.								
58	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 2	-.00	-.12	-.01	-.36*	.45**	-								
59	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 2	.11	.05	-.02	-.37*	.25	.55**	-							
60	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 2	.21	-.01	.13	-.08	.30*	-.00	.25	-						
61	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 2	.05	.20	.37*	.05	.26	.04	.26	.36**	-					
62	Saliva Cortisol (ug/dL) AM 1Home Sample Time 3	.03	.06	-.12	-.09	.22	.33**	.43**	.24	.15	-				
63	Saliva Cortisol (ug/dL) AM 2 Home Sample Time 3	.06	.26	-.04	-.19	.01	.34*	.19	.03	.04	.54**	-			
64	Saliva Cortisol (ug/dL) AM 3 Home Sample Time 3	-.09	-.07	-.17	.03	.10	.39**	.11	.00	-.09	.51**	.71**	-		
65	Saliva Cortisol (ug/dL) 4 PM Home Sample Time 3	-.26	-.20	-.04	-.14	-.11	.10	-.10	-.04	-.01	-.08	.27	.23	-	
66	Saliva Cortisol (ug/dL) 9 PM Home Sample Time 3	.03	-.02	.19	.02	-.07	.01	-.03	-.05	-.07	-.30*	-.22	-.15	.15	-

Curriculum Vita

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Education History:

• B.S. 1997 in Psychology and Physiology at Victoria University, Australia

• Honors 1998 (Class 1) in Biomedical Science at Victoria University, Australia
Thesis: A preliminary investigation of the influence of psychological factors on immunologic reactivity to psychological stress. (Advisors: Catherine Kamphuis, Ph.D and Ona Lipkevcius, Ph.D.)

• Ph.D. May 2007 in Biobehavioral Health, at The Pennsylvania State University.
Thesis: Cortisol, daily hassles and overweight status in adolescence. (Advisor: Elizabeth J. Susman, Ph.D.)

Teaching Experience:

• Faculty Lecturer at Victoria University Australia in Physiology, Psychophysiology, Research Methods and Wellness: Mind-Body Medicine

• Guest Lecturer at The Pennsylvania State University in Adolescent Development

• Graduate Teaching Assistantships at The Pennsylvania State University for Biobehavioral Health, Biobehavioral Aspects of Stress, Foundations in Health Promotion

Selected Awards:

• Hintz Fellowship for Graduate Students in the Department of Biobehavioral Health, Pennsylvania State University 2004-2005

• Hintz Fellowship for Graduate Students in the Department of Biobehavioral Health, Pennsylvania State University 2005-2006

• Kligman Graduate Fellowship, Pennsylvania State University 2005-2006