Adrenocortical Regulation, Eating in the Absence of Hunger and BMI in Young Children

L. A. Francis[a], **D. A. Granger**[b], and **E. J. Susman**[a]

[a]Department of Biobehavioral Health, The Pennsylvania State University, 315 East Health and Human Development Building, University Park, PA 16802

[b]Center for Interdisciplinary Salivary Bioscience Research, School of Nursing, Bloomberg School of Public Health and School of Medicine, The Johns Hopkins University, 525 N. Wolfe Street, Baltimore, MD 21205

Abstract

The purpose of this study was to examine relations among adrenocortical regulation, eating in the absence of hunger, and body mass index (BMI) in children ages 5 to 9 years (N = 43). Saliva was collected before and after the Trier Social Stress Test for children (TSST-C), and was later assayed for cortisol. Area under the curve with respect to increase (AUC_i) was used as a measure of changes in cortisol release from baseline to 60 minutes post-TSST-C. Age- and sex-specific BMI scores were calculated from measured height and weight, and eating in the absence of hunger was assessed using weighed food intake during a behavioral procedure. We also included a measure of parents' report of child impulsivity, as well as family demographic information. Participants were stratified by age into younger (5 to 7 years) and older (8 to 9 years) groups. In younger children, parents' reports of child impulsivity were significantly and positively associated with BMI; cortisol AUC_i was not associated with BMI or eating in the absence of hunger. In older children, however, greater stress-related cortisol AUC_i was related to higher BMI scores and greater energy intake in the absence of hunger. The results suggest that cortisol AUC_i in response to psychosocial stress may be linked to problems with energy balance in children, with some variation by age.

Keywords

Stress; psychobiological; disinhibited eating; obesity; binge; self-regulation

INTRODUCTION

In the past two decades, a major focus in developmental science has been to advance our understanding of why, when exposed to stressful experiences or circumstances, some

children are placed at risk for emotional and behavioral problems but others are resilient (Boyce & Ellis, 2005; Cicchetti, 2002; Rutter, Dunn, Plomin, Simonoff, & et al., 1997). The effects of stress on children's health and developmental outcomes are well-documented (Evans & English, 2002; Gunnar & Quevedo, 2007), and these effects extend across the lifespan (Anda, et al., 2006; Dube, Felitti, Dong, Giles, & Anda, 2003; Evans & Kim, 2007). Obesity is one such health outcome whose biobehavioral underpinnings have been linked with stress (Bjorntorp, Rosmond, 2000; Chrousos, 2000; Gundersen, Mahatmya, Garasky, & Lohman, 2011).

Research attention has been drawn to the adrenocortical component of the physiological stress response as a potential mechanism in the development of obesity through its effects on the accumulation of central adiposity and dysregulated eating behavior, or eating in the absence of hunger (Bjorntorp & Rosmond, 2000; Drapeau, Therrien, Richard, & Tremblay, 2003; Kyrou, Chrousos, & Tsigos, 2006; Rogers, 1999). Disruptions in the hypothalamic pituitary adrenocortical (HPA) axis and sympathetic nervous system (SNS) functioning, as indicated by cortisol and cardiovascular responses to stress, are linked to the accumulation of central adiposity, increased energy intake, and appetite regulation (Adam & Epel, 2007; Bjorntorp, 1997; Gluck, 2006; Gluck, Geliebter, Hung, & Yahav, 2004; Nieuwenhuizen & Rutters, 2008; Tataranni, et al., 1996; Wallerius, Rosmond, Ljung, Holm, & Bjorntorp, 2003). Adrenocortical regulation (i.e., cortisol) may be an underlying mechanism for the development of energy balance problems in children (Spencer & Tilbrook, 2011).

Findings from a study in preadolescent youth (average age ~10 years) show that cardiovascular reactivity (i.e., heart rate) to a laboratory-based stressor was linked to a higher percent body fat; increases in perceived stress after exposure to the stressor was also related to higher levels of body fat (Roemmich, Smith, Epstein, & Lambiase, 2007). Dockray, Susman and Dorn (2009) confirmed a link between cortisol reactivity and obesity in 8- to 13-year-old boys and girls. A large majority of the studies that have provided evidence for a link between cortisol and obesity or eating behavior have been conducted with adult populations of men and women (Epel, et al., 2000; Evans, Boxhill, & Pinkaya, 2008; Gluck, Geliebter, & Lorence, 2004; Rowland & Antelman, 1976; Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). These studies suggest that cortisol reactivity may be linked to dysregulated eating, and eating in response to stress (Epel, Lapidus, McEwen, & Brownell, 2001; Gluck, Geliebter, Hung, et al., 2004).

Eating in the absence of hunger, a measure of dysregulated eating behavior, has consistently been associated with weight gain, overweight, and obesity, in both adults and children (Bellisle, et al., 2004; Fisher, Birch, 2002). As such, eating in the absence of hunger has been proposed as a behavioral phenotype of obesity (Faith, 2006). Findings from a study by Rutters and colleagues (Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2009) revealed that men and women ate more in the absence of hunger after exposure to an acute stressor; these effects were particularly strong for those participants with a disinhibited eating style. Similar findings have been reported more recently, in a sample of non-overweight women (Born, et al., 2010). Stress, both perceived and manipulated, also has been found to affect eating/dietary choices and behaviors in youth (Cartwright, et al., 2003; Jenkins, Rew, & Sternglanz, 2005; Roemmich, Wright, & Epstein, 2002) and adults (Born, et al., 2010; Gibson, 2006; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Zellner, et al., 2006). Roemmich and colleagues (2002) found that preadolescent boys and girls (average age ~9yrs) who reported high levels of dietary restraint, reported higher levels of perceived stress, and increased energy intake on days in which they were exposed to stress, compared to non-stress days. Findings from a study with boys and girls as young as age 8 show that perceived stress was positively related to children's reports of unhealthy eating behaviors, and the use of food as a coping mechanism for stress, nervousness and worry

(Jenkins, et al., 2005). There is a dearth of information on the biobehavioral processes underlying these dysregulated eating behaviors in young children. Understanding individual differences in the sensitivity of the physiological stress response may provide more information on the factors that contribute to the development of dysregulated eating and obesity in children.

Findings from recent studies show that eating appears to be a coping response to stress in youth (Balantekin & Roemmich, 2012; Roemmich, Lambiase, Lobarinas, & Balantekin, 2011). In these studies, adolescents ages 8–12 years consumed more calories on a stress day compared to a non-stress day, and this finding was particularly pronounced in adolescents with greater adiposity, and those who reported lower levels of dietary restraint. Similar findings from animal and human models studies suggest that eating may be a coping response to stress, particularly when the food is a comfort food (Dallman, et al., 2003; Greeno & Wing, 1994; Tomiyama, Dallman, & Epel, 2011). Ulrich-Lai and colleagues (2010) showed that intake of palatable foods can buffer the biobehavioral effects of stress via reward pathways in the brain. The maladaptive, long-term outcome of this biobehavioral dysregulation is the weight gain and fat accumulation that may result from consumption, or overconsumption, of energy-dense foods.

Using a longitudinal sample of youth in the U.S., Francis and Susman (2009) examined links between behavioral regulation (measured using laboratory-based self-control and delay of gratification tasks) and BMI changes from age 3 to 12 years. Results revealed that youth who showed self-regulation failure at ages 3 and 4 had the most rapid increases in BMI from age 3 to age 12, compared to those who showed evidence of self-regulatory success at ages 3 and 4. We propose that cortisol output in response to a stressor is not only a marker for HPA-axis functioning, but may also be linked to regulation across domains, particularly as it relates to biobehavioral regulation of eating and body mass. Given that childhood obesity is a major public health concern, with more than 30% of children ages 6 to 11 years in the U.S. classified as overweight or obese (Ogden, Carroll, Kit, & Flegal, 2012), a better understanding of the biobehavioral factors involved in the etiology of childhood obesity is needed. The purpose of this study was to examine adrenocortical regulation (as indexed by cortisol output in response to psychosocial stress) as an underlying mechanism for the development of problems with energy balance in 5- to 9-year-old children, through its effects on eating in the absence of hunger and body mass index (BMI). We tested the hypothesis that greater cortisol output in response to psychosocial stress would be linked to more eating in the absence of hunger and higher BMI scores.

METHODS AND PROCEDURES

Participants

Participants included 43 children (61% boys; 68% White, 16% Black, 16% other) ages 5- to 9-years (mean age = 7.1 ± 1.5 yrs) and their biological parents, recruited for a study on growth and development in young children in a mid-Atlantic state; the sample was not recruited or selected based on weight status, stress levels, or eating behavior. Eligibility criteria for participation included living with at least one biological parent, and the absence of any biological, physical or developmental problem that would preclude participation in the food procedures, activities, or interviews (e.g., food allergies, heart abnormality or developmental delays). Families were recruited using flyers and newspaper advertisements. Parents were well-educated and predominantly middle income; most parents reported having attained a bachelor's degree or higher (69.2%) and more than 75% of families reported combined family incomes over $50,000/yr.

Procedure

The study protocol appears in Table 1. Families visited a university General Clinical Research Center (GCRC) at approximately 1600 hrs. Parents provided written consent for their family's participation, and completed scales on child health (e.g., respiratory infection, stomach ache, medications, etc.), and whether or not the child ate or drank anything within the past 2 hours. After providing assent, children were taken to a private location to rotate through six 20- to 25-min interview sessions. To reduce boredom and break up the monotony of the interview sessions, children participated in 15- to 20-min craft and activity breaks after each interview session, unless the interview session included a fun activity (e.g., television-viewing procedure, not discussed here). The study was approved by the university's Office for Research Protections

Measures

Adrenocortical Output—was assessed by cortisol output in response to the *Trier Social Stress Test for Children (TSST-C)* (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST-C modification for children consists of an anticipation period (5 min.) and a test period (10 min.) in which children were asked to deliver a 4-min speech and perform a mental arithmetic (serial subtraction) task for 4 minutes. We substituted the block design task from the Wechsler's Intelligence Scale for Children-Revised (WISC-R) for the serial subtraction task so as to be more appropriate for use with children under 8 years old (Tsukayama, Duckworth, & Geier, 2010). Analyses revealed no significant age differences in cortisol levels at each time point during the procedure. The TSST-C tasks were completed in the presence of two confederate judges.

Five saliva samples were collected via passive drool (Granger, Kivlighan, el-Sheikh, Gordis, Stroud, 2007) at baseline (Times 1 and 2), and after the TSST-C (Times 3, 4 and 5). Using the passive drool saliva collection protocol, children are instructed to gently force saliva pooling in their mouths into a collection vial for 2 minutes, or until the saliva has reached the fill line. Cortisol samples were collected over the same time frame for all children; the entire protocol is shown in Figure 1. To avoid sample contamination (Granger, et al., 2012), participants were asked to refrain from eating, drinking (other than water), and tooth brushing for at least 2 hours before arriving at the laboratory. The first baseline measure was collected at approximately 1615 hrs, and the second baseline measure was collected approximately 25 minutes later. The TSST-C began at approximately 1700 hrs; 3 saliva samples were collected approximately 20 minutes apart following exposure to the final stressor. Saliva samples were assayed in duplicate using highly-sensitive enzyme immunoassays for salivary cortisol without modification to the manufacturer's recommended protocol (Salimetrics LLC, State College, PA). The test used 25 ul of saliva and had a lower limit of sensitivity of .003 ug/dl, and a range of sensitivity from .003 to 3.0 ug/dl, and average intra- and inter-assay coefficients of variation of less than 5% and 10%.

Eating in the Absence of Hunger—was measured using the *Free Access Procedure* (Fisher & Birch, 1999a). Approximately 20 minutes following a standard, ad-libitum lunch, children were individually interviewed and asked to indicate their hunger level on a 3-point scale (hungry, in-between or full. Following the hunger assessment, children were given free access to generous portions of 10 snack foods, along with a variety of toys/activities, and were told that that they can use the toys/activities or eat any of the foods while the interviewer took care of some homework in the adjacent room for 10 minutes. Using manufacturer's nutrient information, caloric intake during the 10-minute period was calculated by summing the caloric intake of all the snack foods consumed.

Weight Status—Height and weight measurements were obtained by trained research assistants in order to determine body mass indices (BMI percentiles and z-scores) using age- and gender-specific reference data for children (Kuczmarski, 2000). Children with BMI percentiles 95 were classified as overweight. BMI z-score (BMIz) was used as the main BMI criterion variable in regression models.

Child Temperament and Behavior—Caregivers' reports on various dimensions of their child's self-regulation and behavior were assessed using the *Children's Behavior Questionnaire (CBQ,* (Rothbart, Ahadi, Hershey, & Fisher, 2001) For the purposes of this study, the inhibitory control and impulsivity subscales were used. *Inhibitory control* is defined as "the capacity to plan and to suppress inappropriate approach responses under instructions or in novel or uncertain situations." An example of an item from the inhibitory control scale is: "My child is usually able to resist temptation when told he/she is not supposed to do something." *Impulsivity* is defined as "the speed of response initiation," or the inability to think before you act. An example of an item from the impulsivity subscale is, "My child usually rushes into an activity without thinking about it." Internal consistency estimates for caregivers in this sample were 0.72 and 0.77 for the inhibitory control and impulsivity subscales, respectively. Both inhibitory control and impulsivity have been linked to BMI and dysregulated eating in young children (Anzman & Birch, 2009; Nederkoorn, 2006).

Statistical Analyses

Data were available on 43 children, and 38 caregivers. Using the formula described by Pruessner and colleagues (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003), area under the curve was calculated to provide a repeated-measures indicator of total cortisol secretion over the course of the procedure. Area under the curve with respect to the ground (AUC_g) is a measure of the total area under the curve for all 5 cortisol measurements. As described by Fekedulegn et al. (2007) and Pruessner et al. (2003), AUC_g provides a measure of total hormonal output by calculating the differences between each cortisol measurement (sensitivity), as well as intensity, which is calculated as the distance of all cortisol measures from zero, or the point at which change occurs. Area under the curve with respect to the increase (AUC_i) does not take the distance from zero of all measurements into account. This measure highlights changes over time from baseline, thus providing a measure of person-specific changes in cortisol output over the course of the procedure, which is related to sensitivity of the system (Granger, et al., 2012). For the purposes of this paper, we focused analyses on AUC_i.

Five children did not have a fifth saliva sample collected, thus, the AUC_i score includes only four measures for these children. Given the abnormal distribution of the data, natural logarithmic transformations were performed on cortisol values, before AUC calculations (Gordis, Granger, Susman, & Trickett, 2006), and high cortisol values for one extreme outlier were changed to 3 SD above the mean. Analyses were conducted with and without this outlier, and the main pattern of results did not differ. Given that the stressor we used was validated on children as young as 8 years, we chose to stratify the sample by age in order to examine within-group associations between AUC_i, eating in the absence of hunger, and BMIz. Children were group into younger (5 to 7 years; N=32) and older (8 to 9 years; N=11) age groups. Because we were interested in grouping children based on age, the small sample size limited our ability to conduct further analyses by gender or race.

Descriptive and correlation analyses were conducted using SAS version 9.1 (SAS Institute Inc., Cary, NC). Multiple-group regression analysis was conducted using AMOS version 18 (SPSS Inc., Chicago, IL), and was used to examine relations between AUC_i, eating in the

absence of hunger, and BMI. Using this approach, identical regression models are run separately and simultaneously by group. Regression models were adjusted for the potential influence of parental education.

RESULTS

Sample Characteristics

Table 2 presents descriptive data on children in the sample. Overall, children in the sample were within the normal weight range based on BMI percentiles; approximately 19% of children in the sample were categorized as overweight (BMI percentile   85th). On average, children consumed just over 250 kcals during the Free Access procedure, although some children consumed very little (20 kcals) while other consumed upwards of 700 kcals during the 10-minute period.

Figure 2 displays the overall pattern of cortisol reactivity and recovery by groupings based on child age (younger/older). Preliminary analyses revealed that there were no significant differences in baseline cortisol by age (F=0.39; p=.54). In general, regardless of age, most children appeared to initially react to the novel, clinical setting upon arrival. Differential patterns of adrenocortical reactivity showed that some children did not exhibit high levels of cortisol upon arrival. Approximately 26% (11/43) of children showed at least a 10% increase in cortisol in response to the stressor (baseline to ~40 min post-stressor).

Cortisol, BMIz, and Eating in the Absence of Hunger by Age

Standardized regression estimates appear in Table 3. Results revealed that AUC_i was significantly and positively associated with BMIz and energy intake in the absence of hunger, but only in older children. That is, older children who exhibited greater cortisol release over the course of the procedure had higher BMI z-scores scores, and consumed a greater number of calories in the absence of hunger. Parental education was also a significant predictor of BMIz in older children, with higher BMIz scores being associated with lower reported levels of parental education ($p<.01$). Impulsivity was the only variable associated BMIz in younger children; children with higher BMIz had parents who rated them higher in impulsivity; no other associations were seen for younger children. For younger children, the model R^2 estimates were .41 and .01 for BMIz and eating in the absence of hunger, respectively, and .52 and .50 for older children.

We also dissected the cortisol curve to better understand pre- and post-stress responses (data not shown). In older children, greater cortisol output from baseline to immediate post-stress was related to higher levels of eating in the absence of hunger (r=.66, p   .05). Furthermore, greater cortisol output after the stressor had passed (low recovery) was associated with higher levels of eating in the absence of hunger (r=.71, p   .05).

DISCUSSION

This study was designed to examine associations between individual differences in cortisol output in response to stress, eating in the absence of hunger and BMI z-scores in 5- to 9-year-old children. The results from the study provide evidence for adrenocortical output as a potential marker for problems beyond general behavioral and psychological domains of child development. Area under the curve with respect to increase (AUC_i) was related to children's eating in the absence of hunger and BMI, but only in children ages 8 to 11 years. Specifically, 8- to 11-year old children who exhibited greater AUC_i over the course of the stress procedure had higher BMI z-scores and exhibited greater eating in the absence of hunger. In 5- to 7-year-old children greater levels of mother-reported impulsivity were related higher BMI z-scores. Although we did not experimentally examine the effects of

exposure to an acute stressor on energy intake in this study, all children were exposed to the stressor, and later participated in the eating in the absence of hunger protocol.

We found no similar published studies on stress reactivity and eating in the absence of hunger in young children, however, findings from studies with adult samples provide partial corroboration for our results. In an experimental study on stress eating in adults, Epel and colleagues (2001) found that women who exhibited greater cortisol output on a day in which they encountered a psychosocial stressor, consumed more calories during a snacking procedure compared to women who exhibited lower cortisol output on the stress day. Similarly, Gluck and colleagues (2004) reported that women with binge-eating disorder (BED) exhibited greater cortisol output in response to a physiological stressor, compared to non-BED women. Similar findings have also been reported in women with other disordered eating profiles, and show that cardiovascular reactivity also distinguishes between disordered and non-disordered eaters (Koo-Loeb, Costello, Light, & Girdler, 2000; Koo-Loeb, Pedersen, & Girdler, 1998). Results from another study revealed that peak cortisol levels after an injection of corticotropin-releasing hormone (CRH) were related to increased energy intake in healthy, non-obese adults (George, Khan, Briggs, & Abelson, 2010); this study provides evidence for the causal effects of cortisol on food intake. Taken together, these findings highlight cortisol output as an important factor in the energy balance equation.

Early childhood self-regulatory problems (i.e., self-control and delay of gratification) have been shown to be important determinants of risk for overweight (Graziano, Calkins, & Keane, 2010). Of note is that self-regulatory problems were shown to be longitudinal predictors of rapid weight gain from early childhood through early adolescence (Seeyave, et al., 2009; Tsukayama, Toomey, Faith, & Duckworth, 2010). Given that adrenocortical reactivity is considered a biomarker for self-regulation, the findings from the current study suggest that self-regulation failure in early childhood may predispose children to excessive weight gain, indicating a shared mechanism between the behavioral and energy balance domains of development. Our results show that children's cortisol response to stress was related to children's eating in the absence of hunger, a context in which energy-dense food is plentiful and easily accessible, but this was only true for older children (ages 8 to 9 years). If we were to extrapolate from the eating in the absence of hunger paradigm to the larger, obesigenic environment in which palatable, energy-dense foods are highly available, the results from the study suggest that children with maladaptive responses to stress are at a heightened risk for developing problems with obesity, partly due to dysregulated eating behavior.

Findings from several rodent studies provide evidence for the link between stress, HPA-axis activity, increased food intake, and adiposity (Dallman, Pecoraro, & la Fleur, 2005; Ely, et al., 1997; Hagan, et al., 2002; Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004; Rowland & Antelman, 1976; Wallach, Dawber, Mcmahon, & Rogers, 1977). These studies show that both acute (e.g., tail pinch) and chronic stressors (e.g., noise) can result in increased or hyperphagic eating. Furthermore, factors such as previous restriction or hunger can exacerbate these effects (Hagan, Chandler, Wauford, Rybak, & Oswald, 2003; Hagan, et al., 2002). Translated to human development and behavior, it is possible that such factors as living in high-stress environments (e.g., poverty, crime), and exposure to restriction (e.g., food insecurity, or controlling child-feeding practices) may increase eating in the absence of hunger, and increase children's risk for obesity. These environmental factors have previously been found to be related to children's dysregulated eating behavior (Birch, Fisher, & Davison, 2003; Fisher & Birch, 1999a, 1999b) and risk for obesity (Faith, 2006; Fisher, Birch, 2002; Francis, Ventura, Marini, & Birch, 2007; Larson & Story, 2011).

We are limited by our small, racially and economically homogenous sample, which precludes generalizability of the findings to more diverse populations. In addition, whereas approximately 26% of children in this study showed more than a 10% increase in cortisol in response to the stressor, the large majority of children showed decreases from baseline to immediate post-stressor. The decrease in reactivity suggests that children may have been reacting to the novel, research environment upon arrival. We also did not have a self-report measure of children's perceived stress following the stressor, which may have provided an opportunity to better understand characteristics of children who appeared to be most sensitive to the stressor. Although some developmental studies have yielded consistent mean-level differences in patterns of cortisol response following exposure to a stressful or novel event, a wide range of inter-individual differences in stress-related reactivity is expected. Granger and colleagues note that in response to the mild-to-moderate type stressors typically employed in developmental science, approximately 20–30% of individuals will exhibit a salivary cortisol increase over pre-task levels that are at least greater than 10% of the pre-task level (Evans, Fuller-Rowell, & Doan, 2012; Granger, et al., 2012). Unfortunately, there are many individual differences in prior life experience that we were unable to assess in the present study (e.g. insensitive parenting; (Roisman, et al., 2009). Those background differences would certainly contribute to how children individually approached, responded to and coped with the stressors.

In sum, the findings from this study indicate that individual differences in HPA-axis reactivity in response to psychosocial stress may be related, in different ways, to eating in the absence of hunger and BMI in young children. The approach to understanding relations between cortisol output and regulation of food intake and body weight is a promising area of inquiry with regard to obesity. Our findings confirm a link between cortisol output and energy balance in young children, and corroborate similar findings in adults and animal models. The results also highlight the promise of a minimally invasive technique to measure individual differences in salivary hormones, which afforded the opportunity to sample and study this same relationship in children. This collection technique enables us to measure this phenomenon in children's everyday social worlds. Increased attention has been focused on developing a better understanding of individual differences in biological sensitivity to context, which suggests that an individual's response to stress is highly dependent on the context in which they exist (Boyce & Ellis, 2005; Ellis & Boyce, 2008Ellis & Boyce, 2008; Ellis, Essex, & Boyce, 2005; Obradovic, Bush, Stamperdahl, Adler, & Boyce, 2010). While the current study was not designed to test this theory, the findings suggest the need to account for an individual's biological and behavioral reactivity in a specific context in order to better understand health and developmental outcomes, as well as their antecedents and correlates.

Acknowledgments

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Highlights

- We examined links between stress reactivity, eating behavior and body mass index in 5- to 9-year-old children

- Greater increases in cortisol in response to stress were related to dysregulated eating and higher body mass

- Stress reactivity may be a marker for energy balance dysregulation
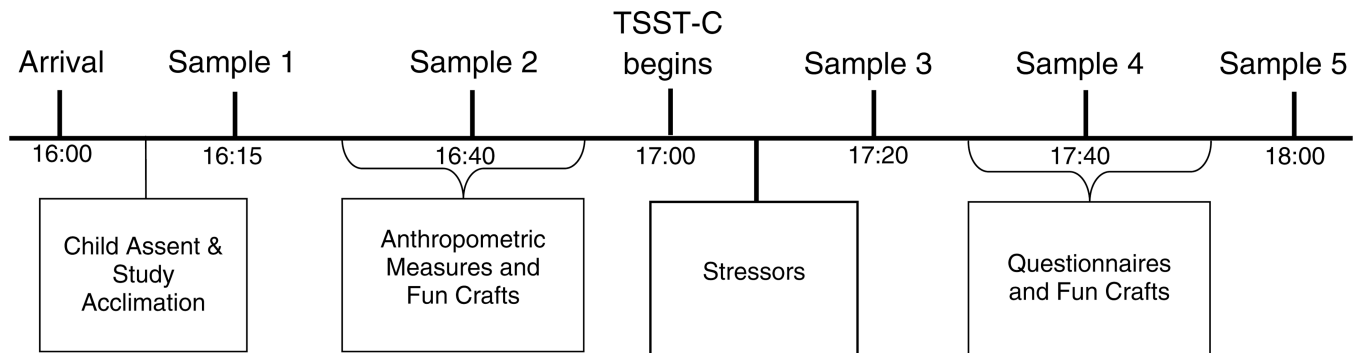
Figure 1.
Saliva sample collection periods. Two baseline samples were collected prior to administration of the Trier Social Stress Test for Children (TSST-C). Three additional samples were collected 20 minutes apart after the TSST-C ended.
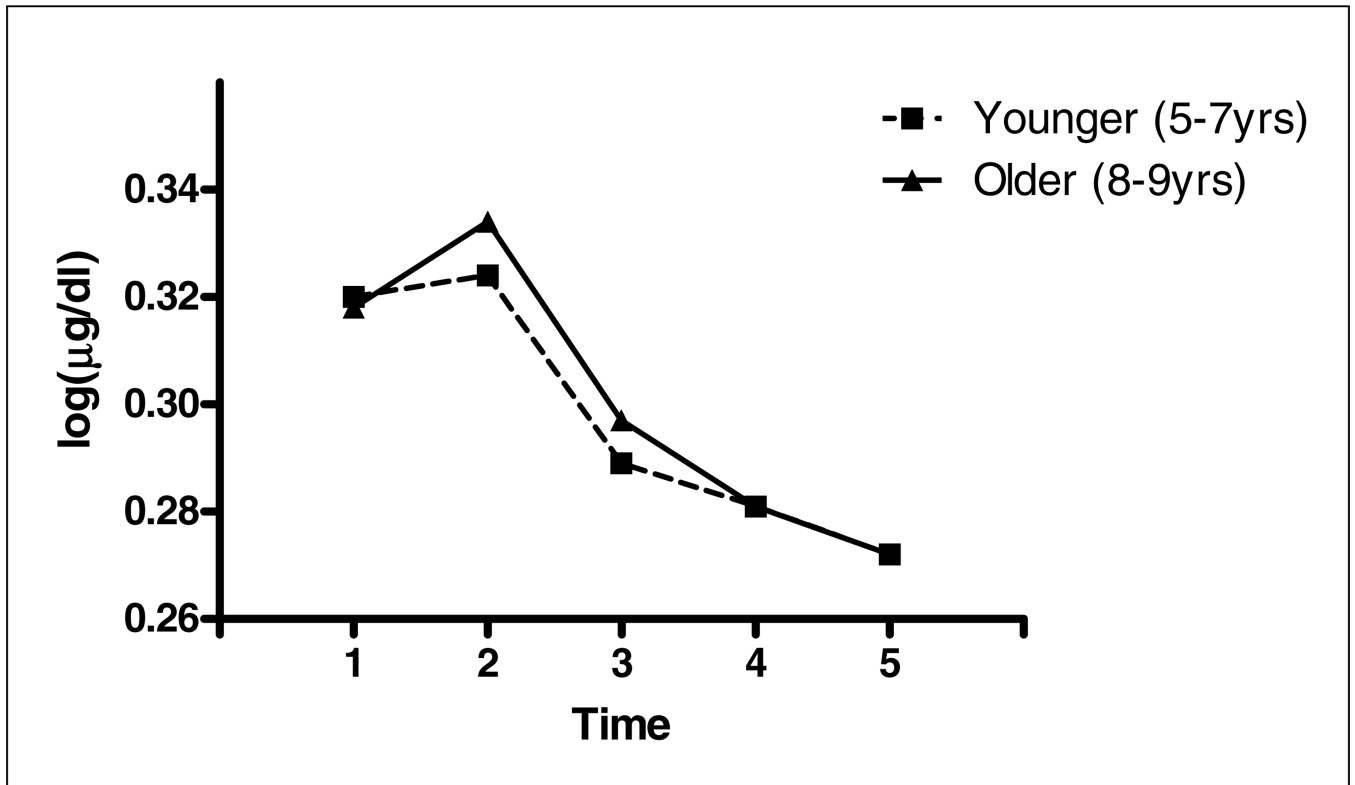
Figure 2.
Cortisol output by age.

Table 1

Schedule of study procedures

TIME	CHILD	PARENT
3:30pm – 4:00pm	Arrival times will vary	
	Meet interviewing buddy	Child health and wellness assessment; consent forms and health history form
	Anthropometric Measurements – Questionnaires (~40min)	Parent Questionnaires Anthropometric Measurements
	Stress Procedure Questionnaires/Crafts (75 min)	
	Dinner (25 min)	
	Activity Break (20 min)	
	Food Preference Procedure (15 min)	
	Break (15 min)	
	TV Viewing Procedure (30 min)	
7:30pm – 8:00pm	Questionnaires/Crafts (20 min)	
	Games, activities, incentives and pickup	

Table 2

Descriptive Sample Information

Variable	Mean (SD)	Range
Child Age (yrs)	6.6 (1.4)	5.0 – 9.0
Child BMIz	0.3 (1.3)	−3.1 – 4.4
Child BMI percentile	58.8	0.1 – 99.9
Child eating in the absence of hunger (kcals)	257.1 (164.8)	20.0 – 704.1

Table 3

Regression coefficients for relations among area under the curve with respect to increase (AUC_i), child body mass index z-score (BMIz), eating in the absence of hunger, and impulsivity; by age group.

	YOUNGER (5–7yrs) (N=32)		OLDER (8–9yrs) (N=11)	
	B (SE)	**β**	**B (SE)**	**β (SE)**
Relations with AUC_i				
BMIz (kg/m^2)	0.07 (.20)	.05	**1.38 (.47)** [**]	**.89** [**]
Eating in the Absence of Hunger (kcals)	8.54 (27.98)	.06	**253.68 (109.43)** [*]	**.69** [*]
Relations with Parents' Report of Child IMPULSIVITY				
BMIz (kg/m^2)	**0.96 (.22)** [***]	**.63** [***]	0.47 (.53)	.31
Eating in the Absence of Hunger (kcals)	12.78 (32.21)	.08	40.20 (124.26)	.11
R^2 for BMIz	.41		.52	
R^2 for Eating in the Absence of Hunger	.01		.50	

Note: All models are adjusted for parental combined education.

SE = Standard error of measurement

[*] p .05,

[**] p .01,

[***] p .001