STRESS-RELATED RISK FACTORS LINKED TO ADOLESCENT ADIPOSITY: A CUMULATIVE RISK APPROACH

A thesis submitted
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Fulfillment of the requirements for the
Degree of Master of Arts

by

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Introduction

Pediatric obesity is a national epidemic with approximately 34% of children and adolescents considered overweight or obese in the United States (Ogden, Carroll, Kit, & Flegal, 2014). Pediatric obesity research has primarily focused on children, but risk factors contributing to adolescent obesity are not as well understood (Adair, 2008; Gordon-Larsen, Adair, Nelson, & Popkin, 2004; Singh, Mulder, Twisk, van Mechelen, & Chinapaw, 2008). Overweight and obesity status during adolescence are highly predictive of later obesity in adulthood (Adair, 2008). The probable trajectory of pediatric obesity may consequently lead to severe physical (e.g. cardiovascular disease, metabolic syndrome) and psychosocial (e.g., poorer quality of life, lower self-esteem) negative outcomes as they move into adulthood (Krebs, et al., 2007; Israel & Ivanova, 2002).

A range of lifestyle factors (e.g., eating and exercise habits), genetic influences, and broader environmental influences (e.g., availability of fast food, marketing, parent modeling of health behaviors) influence risk for pediatric obesity (Davis & Carpenter, 2009; Harris, Gordon-Larsen, Chantala, & Udry, 2006; Wardle, Carnell, Hawort, Polomin, 2008). Stress – an aversive response to external stimuli affecting an individual - represents one important psychosocial predictor of obesity (DeVriendt, Moreno, DeHenauw, 2009; Koch et al., 2008; van Jaarsveld, Fidler, Steptoe, Boniface, & Wardle, 2009). A recent review of multiple meta-analytic studies found a small, but significant association in which psychological stress was longitudinally related to greater obesity risk into adulthood (Wardle et al., 2011). Although the stress-obesity linkage is known in adults, recent cross-sectional and longitudinal designs have also found this
relationship in youth (Nguyen-Michel, Unger, & Spruijt-Metz, 2007; van Jaarsveld et al., 2009). Cross-sectional designs assessing self-report and laboratory-induced psychological stressors found increased stress related to greater food intake (e.g., stress eating) and overweight and obese status in children (Nguyen-Michel et al., 2007; Balantekin & Roemmich, 2012). Fewer longitudinal studies have examined the role of stress relating to obesity trajectories across time. One longitudinal study found “moderate- and higher-stress” adolescents (highest tertile determined by mean scores of self-reported perceived overall stress) having higher BMI and waist-circumference compared to lower-stress adolescents across five years (van Jaarsveld et al., 2009).

The link between stress and obesity remains an understudied area in pediatric populations (Gundersen, Mahatmya, Garasky, & Lohman, 2010; van Jaarsveld et al., 2009 Wilson & Sato, 2013). Stress may appear in multiple domains of development, and further research is needed to investigate whether, and how multiple exposures to childhood adversities and psychological stressors may combine and contribute to greater adiposity (amount of human body fat) in adolescents. Adolescence may be a particularly vulnerable period for this stress-obesity relationship, due to physical (e.g. puberty) and psychosocial changes (e.g., increased autonomy, increased reliance on peers). Adolescents’ perceptions and reactions to various life stressors (e.g., poverty, internalizing symptoms, parent psychopathology) may consequently affect their health outcomes. This study examines how accumulated stress-related risk factors, or “stress pile-up,” are related to greater adolescent adiposity.

Stress and Pediatric Obesity

Bronfenbrenner’s socio-ecological model. The stress-obesity link can be conceptualized through Bronfenbrenner’s socio-ecological model of human development
Bronfenbrenner’s model proposes that multiple domains, including child-specific, parental, and environmental factors, influence child development. In the context of stress, stress-related risk factors from various domains of development have been previously related to pediatric obesity risk. Examples of individual-level risk factors include cortisol patterns and child psychopathology (i.e., depressive symptoms), which have been previously related to greater adolescent weight status. More distal domains of family- and environment-level stress also influence child health. At the family-level, maternal depression and familial conflict represent examples of stress-related risk factors that have been related to greater adolescent BMI. Broader environment-level factors, such as neighborhood safety and poverty, have also been linked to greater pediatric obesity. Exposure to multiple levels of risk factors may theoretically combine to produce an even greater risk, but this has not been adequately studied in past research. Using Bronfenbrenner’s socioecological framework, this study examined combined risk factors from three domains of child development including individual-, maternal-, and environmental-level risk related to adolescent adiposity.

**Individual child-level risk factors.** A range of child-specific risk factors have been associated with increased exposure to stress and increased obesity risk. These include both biological factors, including dysregulated hypothalamic-pituitary-adrenocortical (HPA) axis, and psychosocial risk factors such as stressful life events and child depression (Goodman & Whitaker, 2002; Koch, Sepa, & Ludvigsson, 2006; Shih et al., 2006).

Depressive symptomatology longitudinally predicts adolescents’ risk for obesity (Shih et al., 2006; Goodman & Whitaker, 2002). Depression and obesity are suggested to share biological stress-response pathways, as both are related to excessive cortisol release through the HPA axis (Marniemi et al., 2002). Stress reactivity, as measured through salivary cortisol, mediated the
relationship between depressive symptoms and obesity among adolescent females (Dockray et al., 2009). Thus, child depression may be seen as a child-specific stressor influencing obesity development.

Cortisol, a glucocorticoid stress hormone secreted through the HPA axis (Shimon & Melmed, 2005) represents a child-specific physiological risk factor that has also been related to child obesity risk. The majority of pediatric obesity literature examines cortisol in the context of stress reactivity (cortisol release in response to external stress) linked to greater energy intake and weight status (Dockray, 2009; Roemmich et al., 2007). Blunted awakening cortisol levels – seen in diurnal cortisol patterns – may provide an additional physiological marker identifying adolescents with prolonged exposures to stress and consequently greater adiposity. Normative diurnal cortisol patterns show increased levels of cortisol released immediately after awakening that gradually decrease throughout the day (Wessa et al., 2005). Atypical circadian patterns, such as blunted levels of awakening cortisol, have been found in adults and children with past experiences of prolonged interpersonal stressors (Carlson & Earls, 1997; Gunner & Vazquez, 2001; Rapetti, Taylor, & Seeman, 2002; Susman, 2006; Tarullo & Gunnar, 2006). However, less is understood regarding blunted cortisol patterns in the context of pediatric obesity. Lower cortisol levels may reflect the presence of prolonged stress and increased vulnerability for obesity, especially in accumulation with other maternal and environmental risk factors.

**Maternal risk factors.** A strong body of evidence suggests that parents play a substantial role in the maintenance of child weight (Clark, Goyder, Bissell, & Peters, 2007; Faith, Scanlon, Birch, Francis, Sherry, 2004). For example, parental feeding practices and modeling healthy behaviors are associated with pediatric obesity risk (Clark et al., 2007; Faith et al., 2004; Trost, Kerr, Ward, & Pate, 2001). Parental stress has also been connected to broad negative impacts on
parenting practices and increased risk for pediatric obesity (Parks et al., 2012; Garasky et al., 2009). Maternal depressive symptoms and single-parent home structure represent two maternal-level risk factors that may yield increased stress and have been related to pediatric weight status (Garasky et al., 2009; Gibson et al., 2007; Parks et al., 2012; Strauss & Knight, 1999).

Maternal depression has been related to higher levels of child BMI (Lane et al., 2013), as well as poorer parental modeling of healthy behaviors (Gross, Velazco, Briggs, & Racine, 2013). Depressed mothers may be less involved in monitoring their child’s health behaviors, as indicated in results showing maternal depression related to greater sedentary behaviors in children (Burdette, Whitaker, Kahn, & Harvey-Berino, 2003). Elevated salivary cortisol levels in children have also been related to greater maternal depressive symptoms, suggesting that maternal depression may act as a stressor in children’s development (Lupien, King, Meaney, McEwen, 2000). Past research found pre- and post-natal maternal depression related to externalizing and emotional problems in their offspring (Korhonen, Luoma, Salmelin, & Tamminen, 2011; Luoma et al., 2001). Although maternal depression has been found to impact negative mental health outcomes in children, more research is needed relating maternal depression and weight outcomes in adolescents.

A second maternal characteristic that may serve as a stressor is the household family structure of single parent homes. Single parents expectedly have greater psychological distress, as they more likely experience greater time demands with paid work and childcare compared to two-parent family structures (Avison, Ali, & Walter, 2007). Consequently, children reared in single-parent homes tend to experience greater stress relating to financial issues, increased parental time demands, and family disruption from divorce/separation (Gibson et al., 2007).
Environmental risk factors. Broader home and neighborhood environmental stress may also play a particularly important role in predicting obesity risk in adolescents. Low socio-economic status (SES) and related home and neighborhood predictors (e.g., emotional support in household, perceived neighborhood safety) represent sources of potential environmental stress contributing to pediatric obesity risk (Babey et al., 2010; Oreskovic, Goodman, Slap, & Hung, 2003; Kuhlthau, Romm, & Perrin, 2009).

A family’s low SES alone may indicate a significant stressor in a child or an adolescent’s life (Babey, Hastert, Wolstein, & Diamant, 2010). Lower SES poses risk for increased financial strain which may result in increased barriers to purchasing healthy foods, regular family mealtime routines, and adequate food accessibility, thus hindering healthy weight trajectories in youths (Burke et al., 2011). Although financial strain has also been conceptualized as a parental-level stressor, SES is determined by both household income and social factors (expenses needed for food and shelter), which incorporates broader environmental influences (Bornstein & Bradley, 2003). Therefore, SES is defined as an environmental risk factor in the present study. Low-SES represents a stressor, as low-income children and adolescents are more likely exposed to greater amounts of psychosocial stressors such as parent psychopathology (Garasky et al., 2009; Gundersen, Mahatmya, Garasky, & Lohman, 2011), in turn increasing their propensity for obesity (Burke, Hellman, Scott, Weems, & Carrion, 2011).

A second environment-level risk factor that may be reflected in a child’s home includes the physical home environment and social interactions in a household. A poorer home environment posits an additional source of stress associated with poorer pediatric health outcomes. Strauss and Knight (1999) used the well-established Home Observation for Measurement of the Environment (HOME-Short Form; Caldwell & Bradley, 1984) to assess
cognitive stimulation and emotional support provided from children’s home environments. Results showed that younger children receiving lower cognitive stimulation and lower emotional support at home were more likely to be overweight or obese, even after controlling for maternal obesity, gender, race, SES, and marital status (Strauss & Knight, 1999).

Expanding outside a child’s home environment, neighborhood safety has also been considered a stressor associated with pediatric obesity. Parents’ and children’s perceptions of less safe neighborhoods and higher rates of neighborhood violence were associated with less physical activity and overweight status in children (Lumeng, Appugliese, Cabral, Bradley, & Zuckerman, 2006; Molnar, Gortmaker, Bull, & Buka, 2004; Gómez, Johnson, Selva, & Sallis, 2004). Although some high poverty neighborhoods have accessibility to outdoor recreational spaces, these spaces tend to be less safe, less maintained, and less pleasurable than their counterparts (Franzini et al., 2009). Physical neighborhood characteristics and perceived neighborhood safety present environmental barriers to physical activity in high poverty areas, in turn increasing obesity risk.

**Cumulative Risk Index (CRI) Approach**

The majority of previous literature examining the association between stress and pediatric obesity risk has examined only one or more risk factors from one domain development (e.g., parent stressors), with limited consideration of cumulative risk (Garasky et al., 2009; Parks et al., 2012). Therefore, a comprehensive examination of stress (i.e., individual-, maternal-, and environment-levels) is needed to understand the cumulative effect of stress on adolescent weight outcomes. A small number of studies have adopted a CRI approach to understand the impact of accumulated stress, or “stress pile-up” on child health outcomes (Garasky et al., 2009; Koinis-Mitchell et al., 2010).
Two known studies have employed a CRI approach to examine pediatric obesity risk. Garasky and colleagues (2009) used a cross-sectional design and created six domains of stress indices with scores based on dichotomizing responses from self-report. Five risk indices were used in multivariate analyses examining family stressors related to child obesity, and results showed that family’s mental and physical health problems and financial strain were associated with higher weight status in adolescents (12-17 years old; Garasky et al., 2009). However, this study did not address child-specific risk factors that may also contribute to adolescent weight outcomes. A related study focused specifically on parental stressors in predicting pediatric obesity. A CRI was created consisting of parental stressors (e.g., mental illness, financial strain, unemployment), and results indicated a greater number of parent stressors related to higher BMI levels in children (ages 3-17). Although Parks and colleagues (2012) used a CRI approach, they lacked objective height and weight measurements and did not expand their examination beyond parental stressors related to adolescent health outcomes.

Measures of BMI normed for youth (e.g., zBMI, BMI %ile) are widely used objective measures of pediatric weight status. Waist circumference-to-height ratio, measuring visceral (abdominal) fat, represents an additional outcome measure that has not yet been examined in relation to cumulative stress and obesity in pediatric populations. Waist-to-height ratio has been strongly associated with indicators of obesity-related morbidity, including metabolic syndrome and cardiovascular risk (Kahn, Imperatore, & Cheng, 2005; Maffeis, Banzato, & Talamini, 2008; Moreno et al., 2002). Visceral fat development may be particularly susceptible to stress (Brambilla, Bedogni, Heo, & Pietrobelli, 2013). Therefore waist-to-height ratio should be included in future research examining accumulated stress related to adolescent adiposity.
The Present Study

In summary, various aspects of stress (e.g., poverty, child depressive symptoms) experienced in multiple domains of development have been related to pediatric obesity risk. Only a few studies have employed a CRI approach to examine the cumulative effects of stress on adolescent weight outcomes, and these studies have lacked an examination of multiple domains of in which stress may occur for adolescents. Objective measures of weight outcomes and predictor variables have also been lacking in past CRI research (Parks et al., 2012). The present study fills these gaps in past literature by using a CRI approach with subjective and objective predictor variables and multiple objective outcome measures of weight status (zBMI, waist to height ration) to more accurately assess cumulative risk linked to adolescent adiposity. This study examined cumulative stress from multiple domains of adolescent development including individual-, maternal-, and environment-level risk factors. The CRI approach in the present study was primarily guided by Koinis-Mitchell and colleagues’ (2007) approach, which examined cumulative risk factors related to asthma morbidity in urban settings (Koinis-Mitchell et al., 2010; Koinis-Mitchell et al., 2007). No known research has adopted this approach to comprehensively examine accumulated stress linked to adolescent adiposity.

The primary goal of this study was to assess whether greater cumulative stress (number of stress-related risk factors) predicted poorer adolescent weight outcomes. Higher CRI total scores were expected to predict greater levels of adolescent zBMI and waist-to-height ratio (hypothesis 1). The second goal of this study was to employ hierarchical regressions to examine the unique predictive value of stress from multiple domains of development related to adolescent adiposity. Greater levels of risk factors in each domain (individual-, maternal-, and environmental levels) were expected to associate with greater adolescent zBMI and waist-to-
height ratio (hypothesis 2). In addition, hierarchical regressions also examined unique associations with independent risk factors related to adolescent adiposity.
Methods

Participants

The present study used data from the longitudinal National Institute of Child Development (NICHD) Study of Early Child Care and Youth Development (SECCYD; NICHD Early Child Care Research Network, 2005). A cross-sectional sample from Phase IV of the larger NICHD-SECCYD study was used in this study to specifically examine the impact of stress on obesity risk in adolescents at age 15. Broadly, the 4-phase longitudinal NICHD-SECCYD study examined influences of childcare on child development from birth to adolescence. Data were collected from 10 U.S. locations and conditional sampling was used to ensure inclusion of at least 10% single mothers, 10% without high school degree, and 10% ethnic minorities (Bradley et al., 2008). Mothers were excluded if they were under 18 years old, did not speak English, reported having drug problems or a serious illness, or refused to be interviewed. Child participants were excluded if they had a serious illness or were part of a multiple birth. Families were also excluded if they lived more than one hour away from the site location, planned to move away from the area in the next three years, or their neighborhood was too dangerous to visit for data collection, which was determined by police. The initially enrolled sample included 1,364 children (54% male) 13% of adolescent participants were African American, 6% Hispanic, 2% Asian or Native American, and 3% Other. Detailed methodological information regarding the larger NICHD-SECCYD is publicly available at http://www.nichd.nih.gov/research/supported/seccyd/overview.cfm.
The sample for the present study (N = 621) included adolescents (52.5% female) and their mothers who completed all relevant study measures in Phase IV of the larger NICHD-SECCYD study. Parent and adolescent descriptive characteristics regarding demographic (e.g., household income, ethnicity, parental education), predictor (e.g., income-to-needs), and outcome variables (e.g., zBMI, waist-to-height ratio), are presented in Table 1. The Kent State University Institutional Review Board approved this study.

Table 1. Adolescent and Maternal Descriptive Characteristics of Demographic Variables, Risk Factors, and Weight Outcomes (N = 621)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N (or Mean)</th>
<th>% (or SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adolescent (age 15) Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biological sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>326</td>
<td>52.5</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>526</td>
<td>84.7</td>
</tr>
<tr>
<td>Black or African American</td>
<td>59</td>
<td>9.5</td>
</tr>
<tr>
<td>Asian or Pacific Islander</td>
<td>8</td>
<td>1.3</td>
</tr>
<tr>
<td>American Indian or Eskimo</td>
<td>1</td>
<td>.2</td>
</tr>
<tr>
<td>Other</td>
<td>27</td>
<td>4.3</td>
</tr>
<tr>
<td>BMI z-score for age-and-gender</td>
<td>M = .51</td>
<td>SD = .96</td>
</tr>
<tr>
<td>Under/healthy weight (≤ +1 SD)</td>
<td>438</td>
<td>70.5</td>
</tr>
<tr>
<td>Overweight (≥ +1 SD and ≤ +2 SD)</td>
<td>140</td>
<td>22.5</td>
</tr>
<tr>
<td>Obese (≥ +2 SD)</td>
<td>43</td>
<td>6.9</td>
</tr>
<tr>
<td>Waist-to-height ratio</td>
<td>M = .45</td>
<td>SD = .06</td>
</tr>
<tr>
<td><strong>Maternal Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother’s Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>536</td>
<td>86.3</td>
</tr>
<tr>
<td>Black or African American</td>
<td>59</td>
<td>9.5</td>
</tr>
<tr>
<td>Asian or Pacific Islander</td>
<td>14</td>
<td>2.3</td>
</tr>
<tr>
<td>American Indian or Eskimo</td>
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<td>5</td>
</tr>
<tr>
<td>Other</td>
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<td>1.4</td>
</tr>
<tr>
<td>Total combined household annual income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than $19,999</td>
<td>39</td>
<td>6.3</td>
</tr>
<tr>
<td>$20,000 - $39,999</td>
<td>74</td>
<td>11.9</td>
</tr>
<tr>
<td>$40,000 - $59,999</td>
<td>94</td>
<td>15.1</td>
</tr>
<tr>
<td>$60,000 - $79,999</td>
<td>105</td>
<td>16.9</td>
</tr>
<tr>
<td>More than $80,000</td>
<td>309</td>
<td>49.8</td>
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<tr>
<td>Maternal Education level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>17</td>
<td>2.8</td>
</tr>
<tr>
<td>Graduate high school or GED</td>
<td>87</td>
<td>14.0</td>
</tr>
<tr>
<td>Some college but no degree</td>
<td>142</td>
<td>22.9</td>
</tr>
<tr>
<td>AA degree or vocational school</td>
<td>98</td>
<td>15.8</td>
</tr>
<tr>
<td>Bachelor’s degree from college/university</td>
<td>139</td>
<td>22.4</td>
</tr>
<tr>
<td>Some graduate work</td>
<td>135</td>
<td>21.7</td>
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<tr>
<td>Missing</td>
<td>3</td>
<td>.5</td>
</tr>
</tbody>
</table>

*Note.*
All percentages may not equate to 100 due to rounding.
SD = standard deviation; M= sample mean
**Procedures**

In Phase IV, data were obtained through laboratory assessments, at-home observations, and self-report measures from multiple informants (e.g., mothers, adolescents). Data collection for Phase IV occurred in 2006, and laboratory visits and home visits took place as closely as possible to adolescents’ 15th birthdays. During laboratory assessments, researchers obtained objective anthropometric measurements (e.g., height, weight, waist circumference) for adolescents. At-home visits included home observations, self-report questionnaires, and salivary cortisol assay. Trained research assistants conducted semi-structured interviews and direct home observations to assess cognitive stimulation and emotional support in adolescents’ home environments. Mothers and adolescents also completed self-report measures during home visits. Researchers conducted phone interviews with primary caregivers to obtain updated demographic information (e.g., current marital status).

**Measures**

*Adolescent adiposity outcome measures.*

**Body mass index z-score for age-and-gender.** Trained researchers measured adolescent height and weight during the laboratory visit. Height was measured to the nearest 1/8 inch, using a 7-foot calibrated yardstick and T-square. Weight was measured in pounds and ounces, to the nearest .25 pounds, using a Detecto Physician’s Scale Model #437 that was calibrated monthly with certified weights. Adolescents wore light clothing and no shoes during anthropometric assessments. The Centers for Disease Control and Prevention (CDC) growth charts for the United States in 2000 were used to calculate BMI percentile scores and zBMI scores normed for age and gender (Kuczmarski et al., 2000). Normative growth must be considered when assessing pediatric adiposity to provide a reference to determine weight status in youth (Must & Anderson,
BMI z-scores were used in the present analyses, because z-scores allow for greater variability than BMI percentile scores. Although BMI percentile scores are easily interpretable, a ceiling effect occurs at the 99th percentile, truncating the variability in adolescents that fall above the 99th percentile (Daniels, 2009).

**Waist-to-height ratio.** Trained researchers obtained triplicate waist circumference (to nearest 0.1 centimeter) measurements. Adolescents were asked to lift up their shirts to the bottom of their ribs, in order to expose their abdomens for more accurate measurements. Waist circumference was measured at the slimmest part of participants’ waists, typically in the middle of the lower ribcage and iliac crest (top of pelvis on either side). The average value of waist circumference was then divided by height (cm) to determine an adolescent’s waist-to-height ratio. Higher waist-to-height ratios indicate greater levels of adolescent abdominal adiposity.

**Individual child-level risk factors.**

**Average awakening cortisol (μg/dL).** Cortisol is a glucocorticoid stress hormone secreted through HPA axis activity in a normative diurnal pattern and in response to external stress (Shimon & Melmed, 2005). Normative patterns of cortisol secretion occur in a diurnal circadian rhythm, in which cortisol levels are generally higher after awakening (within one hour of waking) and then decrease throughout the day (Roisman et al., 2009; Shimon & Melmed, 2005). Cortisol is measured in units of micrograms to deciliters (μg/dL), with higher values indicating greater cortisol secretion. For three consecutive school days, following the at-home visit, adolescents were asked to provide one saliva sample in the morning immediately after awakening, after rinsing their mouths with water, and before eating. Adolescents used cotton rolls in salivettes provided by the researchers. Participants were instructed to keep the cotton roll in their mouths for three minutes before returning it to the salivette. Completed saliva collections
were stored in -80°C freezers until shipped to Salimetrics (State College, PA, USA) for analyses in a calibration range of .012-3.000 μg/dL, with < 0.003 ug/dL sensitivity. Duplicate salivary assays for Day 1, 2, and 3 were conducted and the average determined the value for that given day. The overall average awakening cortisol value was computed by calculating the mean across the three days of saliva collection (Roisman et al., 2009).

**Child depressive symptoms.** Child depressive symptoms were assessed through the Child Depression Inventory – Short Form (CDI-S), one of the most widely used questionnaires for assessing children’s (ages 7-17) depressive symptomology (Kovacs, 1992). The self-report questionnaire is a shorter version of Kovac and Beck’s CDI (27-items). The CDI-S contains 10 items on a 3-point Likert scale, with higher total scores indicating greater levels of depressive symptoms experienced in the past two weeks. Example items include, “I am sad…” (1 = “once in a while”; 2 = “many times”; 3 = “all the time”) and “I do…” (1 = “most things OK”; 2 = “many things wrong”; 3 = “everything wrong”). Both the CDI-S and CDI demonstrate acceptable internal consistency and test-retest reliability (Kovacs, 1992; Smucker, Craighead, Wilcoxon, & Green, 1986), and yielded good internal consistency and in the present sample, α = .80.

**Maternal-level risk factors.**

**Maternal depressive symptoms.** Mother reports were obtained during at-home visits to assess maternal depressive symptomology. The Center for Epidemiologic Studies – Depression (CES-D) is a well-validated and commonly used 20-item short self-report assessing depressive symptoms in adults (Radloff, 1977). Items are rated on a 4-point Likert scale (ranging from 1 = “Rarely or none of the time (less than 1 day)”, 2 = “Some or a little of the time (1-2 days)”, 3 = Occasionally or a moderate amount of the time (3-4 days)”, and 4 = “Most or all of the time (5-7 days)”)). Items refer to how often they felt various depressive symptoms in the past week (e.g., “I
was bothered by things that usually don’t bother me,” I did not feel like eating; my appetite was poor”). Total scores were calculated using the sum of the items, with higher scores indicating greater maternal depressive symptomology. The CES-D demonstrates high internal consistency and reliability (Radloff, 1977), and yielded excellent internal consistency (α = .92) in the present sample.

**Marital Status.** Marital status was obtained from a demographic questionnaire item in which mothers reported whether they were married and lived with their spouse, had a partner and lived together, were separated/divorced/not living with their partner, or widowed. Marital status was dichotomized to distinguish two-parent and single-parent homes. Specifically, mothers reporting separation/divorce or widowed were considered to be single-parent households.

**Environmental-level risk factors.**

**Socioeconomic status (SES).** Socio-economic status was defined using households’ income-to-needs ratios, according to the 2005-2006 federal poverty threshold guidelines (Current Population Survey, 2006). Mothers reported their annual total household income at the time of their child’s age 15 assessment. Income-to-needs ratios account for federal estimates of minimally required expenses for food and shelter based on the number of adults and children in a given household (Duncan & Brooks-Gunn, 1997). A household’s income-to-needs ratio was computed by dividing the total family pre-tax income by the poverty threshold for the household determined from the self-report data collected from mothers.

**Home environment.** Trained observers completed an adapted version of the Early Adolescent Home Observation Measurement of the Environment (HOME Inventory, Bradley et al., 2000) to assess the amount and quality of emotional support, cognitive stimulation, and structure provided in adolescents’ home environment. A modified 44-item version (5 subscales)
was used in this study. Trained researchers conducted the HOME inventory during a 45-minute home visit consisting of a semi-structured interview and direct observations with the child and primary caregiver. Parent-child interactions and parenting behaviors were observed and scored throughout the interview as parents and adolescents ate a snack and completed an activity together. Each item was coded with a 1 = yes (behavior observed) or 0 = no (behavior not observed), with example items including, “Parent talks to child during visit” and “Child has access to materials for arts and crafts” (Caldwell & Bradley, 2004). The HOME inventory yields an overall total score and five subscales including Physical Elements, Learning Materials, Variety of Experiences, Acceptance and Responsivity, and Regulatory Activities. This study used the HOME total score, with higher scores indicating higher levels of cognitive stimulation and support in adolescents’ home environment (Bradley et al., 2000). The Early Adolescent HOME Inventory demonstrates high inter-observer reliability, and consistently yielded good internal consistency (α = .80) in the present sample.

**Neighborhood safety.** The 4-item subscale of the Self-Care Checklist (SCC-Child) assessed self-perceived neighborhood safety in the adolescent’s neighborhood (Shumow, Vandell, & Posner, 1998; Vandell & Pierce, 2000). Administered during the home visit, adolescents reported their perceived neighborhood safety using a 5-point Likert scale ranging from 1 = “not at all true” to 5 = “really true.” The subscale included the following items: 1) “It is safe to walk around my neighborhood;” 2) “It is safe in my neighborhood;” 3) “I am scared of some of the people in my neighborhood;” 4) “There are people in my neighborhood who might hurt me.” Perceived neighborhood safety was computed by calculating the mean of the 4 items (items 3 and 4 reverse coded), with higher mean scores indicating greater levels of perceived neighborhood safety (Shumow et al., 1998). Similar to past research, the neighborhood safety
SCC subscale yielded moderate internal consistency (α = .76) in this sample (Shumow et al., 1998).

**Data Analytic Plan**

The present study employed hierarchical regressions to assess influences of individual-, maternal-, and environment-level stress on adolescent weight outcomes. First, a CRI was constructed to examine the total number of risk factors associated with adolescent weight outcomes. Second, two hierarchical linear regression analyses further extended previous literature by examining unique associations between stress-related risk factors within various domains of development and adolescent weight outcomes. Weight outcomes included zBMI for age-and-gender and waist-to-height ratio. Race/ethnicity and gender were controlled for in regression analyses, as they have may covary with weight outcome variables and cumulative stress (Burke et al., 2011; McCarthy & Ashwell, 2006; Misra, Wasir, & Vikram, 2005; Ogden et al., 2014).

**Cumulative risk approach.**

*Construction of CRI.* Consistent with prior research utilizing CRI approaches to understand child health outcomes (e.g., Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2010), a CRI was constructed in this study as a composite score reflecting the total number of high-risk variables present for each adolescent (please see Figure 1). In this CRI, risk factors were assumed to have similarly weighted predictive values. A stress-related risk factor was coded as “1” to reflect greater risk for obesity. Inherent categorical variables, such as marital status, were coded as 1 = “high-risk” (i.e., single-parent home) or 0 = “no/low risk” (i.e., married/living with partner). Continuous variables were dichotomized using the top- or lowest-most quartile of the sample, which determined the cut-off points indicating the presence of risk.
Individual level risk factors were coded as follows: CDI total scores assessing child depressive symptoms (1 = top 25%, 0 = lower 75%) and awakening cortisol levels (1 = lowest 25%, 0 = upper 75%). Maternal risk factors included: CESD total scores assessing maternal depressive symptoms (1 = top 25%, 0 = lower 75%) and marital status (1 = single-parent home, 0 = married/living with partner). Environmental risk factors were coded as follows: income-to-needs ratios (1 = lowest 25%, 0 = upper 75%); HOME total scores assessing cognitive stimulation and emotional support (1 = lowest 25%, 0 = upper 75%); and SCC subscale scores assessing perceived neighborhood safety (1 = lowest 25%, 0 = upper 75%). An adolescent’s CRI total score was calculated by summing the risk factor scores, with possible CRI total scores ranging from 0 – 7. Higher total scores indicated a greater number of experienced stress-related risk factors.

Figure 1. Cumulative Risk Model of Stress-Related Risk Factors Associated with Adolescent Adiposity
CRI regression models. Two multiple regression analyses examined whether greater cumulative risk (as defined by the total number of stress-related risk factors) predicted adolescent zBMI and waist-to-height ratio. Step 1 in the regression model controlled for gender and race. Step 2 included CRI total scores.

Multi-domain hierarchical linear regressions. Hierarchical linear regressions were conducted to extend the cumulative risk literature by examining the unique variance explained by each domain (individual-, maternal-, and environment-level) and independent risk factors associated with adolescent adiposity. These regression analyses allowed for a deeper examination of the risk factors’ unique predictive power related to adolescent weight outcomes. Conceptualized through Bronfenbrenner’s socio-ecological model of development (Bronfenbrenner, 1992), individual-, maternal-, and environmental-level risk factors were placed in the regression model, after controlling for gender and race in Step 1. Individual risk factors were entered in Step 2 (child depressive symptoms, awakening cortisol levels). Maternal-level risk factors were entered in Step 3 (individual- and parent-level). Environmental risk factors were entered in Step 4 (income-to-needs ratio, household cognitive stimulation and emotional support, perceived neighborhood safety.
Results

Descriptive and Correlational Analyses

As shown in Table 1, approximately half (52.5%) of adolescents in this study were female and participants were primarily Caucasian. According to the World Health Organization cut-offs for zBMI for age-and-gender, zBMI scores above 1.00 (+1 SD) indicate overweight status, and above 2.00 (+2 SD) indicate obesity for children ages 5-19 (World Health Organization, 2014). The majority of adolescents in this study had a zBMI that fell in the underweight or healthy weight range (70.5%). However, 22.5% were overweight and 6.0% were obese. 16.9% of adolescents fell in the “at-risk” range for waist-to-height ratio (≥ .5; Ashwell & Hsieh, 2005). Please see Table 1 for a full summary of descriptive characteristics for adolescents and mothers.

As shown in Table 2, approximately one-third (27%) of the sample met criteria for three or more stress-related risk factors. Means, standard deviations, and frequency distributions for the CRI risk factors, and outcome variables are displayed in Table 2. Distribution plots of normality were evaluated for each of the risk factors. Income-to-needs ratio was positively skewed, so a square root transformation was applied to this variable by transforming larger values with greater variance to be constant to the mean (Field, 2013). The transformed income-to-needs ratio variable was then used in further analyses. The correlation matrix including CRI risk factors and weight outcomes are shown in Table 3.
Table 2. Descriptive Characteristics of Risk Factors in CRI (N = 621)

<table>
<thead>
<tr>
<th>CRI characteristic</th>
<th>N (or Mean)</th>
<th>% (or SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Individual-level</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol Average (ug/dL)</td>
<td><em>M</em> = .37</td>
<td><em>SD</em> = .18</td>
</tr>
<tr>
<td>Adolescent depressive symptoms (CDI)</td>
<td><em>M</em> = 1.95</td>
<td><em>SD</em> = 2.59</td>
</tr>
<tr>
<td><strong>Maternal-level</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal depressive symptoms (CESD)</td>
<td><em>M</em> = 10.21</td>
<td><em>SD</em> = 9.36</td>
</tr>
<tr>
<td>Marital status (mother)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single-parent home</td>
<td>114</td>
<td>18.4</td>
</tr>
<tr>
<td>Married/living with partner</td>
<td>507</td>
<td>81.6</td>
</tr>
<tr>
<td><strong>Environment-level</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income-to-needs ratio</td>
<td><em>M</em> = 5.16</td>
<td><em>SD</em> = 5.12</td>
</tr>
<tr>
<td>H.O.M.E. total score</td>
<td><em>M</em> = 37.03</td>
<td><em>SD</em> = 4.84</td>
</tr>
<tr>
<td>SCC total score</td>
<td><em>M</em> = 4.55</td>
<td><em>SD</em> = .59</td>
</tr>
<tr>
<td>CRI Total score (0-7 indices)</td>
<td><em>M</em> = 1.78</td>
<td><em>SD</em> = 1.46</td>
</tr>
<tr>
<td>0</td>
<td>126</td>
<td>20.3</td>
</tr>
<tr>
<td>1</td>
<td>178</td>
<td>28.7</td>
</tr>
<tr>
<td>2</td>
<td>146</td>
<td>23.5</td>
</tr>
<tr>
<td>3</td>
<td>89</td>
<td>14.3</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>7.9</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>3.7</td>
</tr>
<tr>
<td>6</td>
<td>9</td>
<td>1.4</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>.2</td>
</tr>
</tbody>
</table>

*Note.*
All percentages may not equate to 100 due to rounding.

*SD* = standard deviation; *M* = sample mean
### CRI Scores Associated with Adolescent Weight Outcomes

Please see Table 4 for a summary of regression analyses examining CRI total scores as a predictor of adolescent weight outcomes (i.e. zBMI, waist-to-height ratio).

**zBMI for age-and-gender.** After controlling for gender and race in Step 1 (\(R^2 = .02\), Adjusted \(R^2 = .02\)), an additional 4.7% of the variance in adolescent zBMI was explained by CRI total scores in Step 2. A greater number of risk factors (\(\beta = .23, p < .001\)) was associated with greater adolescent zBMI (\(\Delta F (1, 617) = 30.69, p < .001\)) in the final model. Additional independent samples t-test showed that overweight/obese adolescents (zBMI > +1 SD; \(M = 2.26\),...
SD = 1.71) had higher CRI total scores compared to under/healthy weight adolescents (zBMI ≤ 1 SD; M = 1.59, SD = 1.29), t(272.78) = -4.81, p < .001.

**Waist-to-height (W-to-H) ratio.** After controlling for gender and race in Step 1 (R² = .01), CRI total scores entered in Step 2 explained an additional 5.5% of the variance. In the final model, a greater numbers of risk factors (β = .25, p < .001) was associated with greater adolescent W-to-H ratio (ΔF (1, 617) = 36.37, p < .001). Results are shown in Table 4. An independent samples t-test showed that adolescents with at-risk W-to-H ratios (≥ .5 W-to-H ratio; M = 2.58, SD = 1.77) had higher CRI total scores compared to their counterparts (< .5 W-to-H ratio; M = 1.62, SD = 1.33), t(128.89) = -5.25, p < .001).

Table 4. *Multiple Regressions Relating CRI Total Scores and Adolescent zBMI and Waist-to-Height Ratio (N = 621)*

<table>
<thead>
<tr>
<th><strong>Adolescent zBMI</strong></th>
<th>R²</th>
<th>ΔR²</th>
<th>B</th>
<th>S.E.</th>
<th>Beta (β)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1 – Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.02</td>
<td>.02**</td>
<td>-.11</td>
<td>.08</td>
<td>-.06</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td>.17</td>
<td>.11</td>
<td>.06</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRI total score</td>
<td>.07</td>
<td>.05***</td>
<td>.15</td>
<td>.03</td>
<td>.23***</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Adolescent waist-to-height ratio</strong></th>
<th>R²</th>
<th>ΔR²</th>
<th>B</th>
<th>S.E.</th>
<th>Beta (β)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1 – Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.01</td>
<td>.01*</td>
<td>-.01</td>
<td>.01</td>
<td>-.10*</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td>.00</td>
<td>.01</td>
<td>.00</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRI total score</td>
<td>.07</td>
<td>.06***</td>
<td>.01</td>
<td>.00</td>
<td>.25***</td>
</tr>
</tbody>
</table>

*Note.*

*** p < .001; ** p < .01; * p < .05.

def (degrees of freedom) = (1, 613) for hierarchical regressions.

**Hierarchical Regressions**

**zBMI.** As shown in Table 5, a hierarchical regression examined multiple domains of stress-related risk factors related to zBMI. Step 1 controlled for gender and race, and explained
1.8% (Adjusted $R^2 = 1.5\%$) of the variance in adolescent zBMI; $\Delta F (2, 618) = 5.79, p < .01$. Step 2 included individual-level risk factors and explained an additional 2.0% of variance in adolescent zBMI ($\Delta F (2, 616) = 6.49, p < .01$). Step 3 included maternal risk factors and explained an additional 1.0% of the variance ($\Delta F (2, 614) = 3.26, p < .05$). Lastly, Step 4 included environment-level risk factors and further improved the model fit ($\Delta F (3, 611) = 6.38, p < .001$), explaining an additional 2.9% of the variance. In the final model (Step 4), lower awakening cortisol ($\beta = -.08, p < .05$), greater child depressive symptoms ($\beta = .09, p < .05$), and lower income-to-needs ratio ($\beta = -.14, p < .01$) were associated with greater adolescent zBMI.

Table 5. *Hierarchical Regression Relating Stress-Related Risk Factors and Adolescent zBMI (N = 621)*

<table>
<thead>
<tr>
<th></th>
<th>$R^2$</th>
<th>$\Delta R^2$</th>
<th>B</th>
<th>S.E.</th>
<th>Beta ($\beta$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1 – Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.02</td>
<td>.02**</td>
<td>-.10</td>
<td>.08</td>
<td>-.05</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td>.16</td>
<td>.11</td>
<td>.06</td>
</tr>
<tr>
<td><strong>Step 2 – Individual</strong></td>
<td>.04</td>
<td>.02**</td>
<td>-.43</td>
<td>.21</td>
<td>-.08*</td>
</tr>
<tr>
<td>Awakening cortisol (μg/dL)</td>
<td></td>
<td></td>
<td>.03</td>
<td>.02</td>
<td>.09*</td>
</tr>
<tr>
<td>Child depressive symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 3 – Maternal</strong></td>
<td>.05</td>
<td>.01*</td>
<td>.00</td>
<td>.00</td>
<td>.02</td>
</tr>
<tr>
<td>Maternal depressive symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single-parent home</td>
<td></td>
<td></td>
<td>.09</td>
<td>.10</td>
<td>.04</td>
</tr>
<tr>
<td><strong>Step 4 – Environmental</strong></td>
<td>.08</td>
<td>.03***</td>
<td>-.16</td>
<td>.05</td>
<td>-.14**</td>
</tr>
<tr>
<td>Income-to-needs</td>
<td></td>
<td></td>
<td>-.01</td>
<td>.01</td>
<td>-.06</td>
</tr>
<tr>
<td>Home environment</td>
<td></td>
<td></td>
<td>-.08</td>
<td>.07</td>
<td>-.05</td>
</tr>
<tr>
<td>Neighborhood safety</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.*

$*** p < .001; ** p < .01; * p < .05.$

**Waist-to-height ratio.** As shown in Table 6, a hierarchical regression examined multiple domains of risk factors related to waist-to-height ratios. Step 1 (controlling for gender and race) significantly explained 1.1% (Adjusted $R^2 = 0.8\%$) of the variance in waist-to-height ratios; $\Delta F (2, 618) = 3.49, p < .05$. Step 2, including individual-level risk factors, explained an additional
2.5% of the variance in waist-to-height ratio ($\Delta F (2, 616) = 7.94, p < .001$). Step 3, including maternal risk factors, explained an additional 1.2% of the variance ($\Delta F (2, 614) = 3.75, p < .05$). Lastly, Step 4 including environment-level risk factors, accounted for an additional 3.3% of variance explaining waist-to-height ratio ($\Delta F (3, 611) = 7.36, p < .001$). In the final model (Step 4), greater child depressive symptoms ($\beta = .11, p < .01$), lower income-to-needs ratios ($\beta = -.13, p < .01$), and lower household cognitive stimulation and emotional support ($\beta = -.11, p < .05$) predicted greater waist-to-height ratios in adolescents.

Table 6. Hierarchical Regression Relating Stress-Related Risk Factors and Adolescent Waist-to-Height Ratio (N = 621)

<table>
<thead>
<tr>
<th>Waist-to-height ratio</th>
<th>$R^2$</th>
<th>$\Delta R^2$</th>
<th>B</th>
<th>S.E.</th>
<th>Beta ($\beta$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1 – Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.01</td>
<td>.01*</td>
<td>-.01</td>
<td>.01</td>
<td>-.09*</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td>.00</td>
<td>.01</td>
<td>-.01</td>
</tr>
<tr>
<td><strong>Step 2 – Individual</strong></td>
<td>.04</td>
<td>.03***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awakening cortisol (µg/dL)</td>
<td></td>
<td></td>
<td>-.02</td>
<td>.01</td>
<td>-.06</td>
</tr>
<tr>
<td>Child depressive symptoms</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.11**</td>
</tr>
<tr>
<td><strong>Step 3 – Maternal</strong></td>
<td>.05</td>
<td>.01*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal depressive symptoms</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.01</td>
<td>.04</td>
</tr>
<tr>
<td>Single-parent home</td>
<td></td>
<td></td>
<td>.00</td>
<td>.01</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Step 4 – Environment</strong></td>
<td>.08</td>
<td>.03***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income-to-needs</td>
<td></td>
<td></td>
<td>-.10</td>
<td>.00</td>
<td>-.13**</td>
</tr>
<tr>
<td>Home environment</td>
<td></td>
<td></td>
<td>.00</td>
<td>.00</td>
<td>-.11*</td>
</tr>
<tr>
<td>Neighborhood safety</td>
<td></td>
<td></td>
<td>.00</td>
<td>.00</td>
<td>-.03</td>
</tr>
</tbody>
</table>

*Note.*

*** $p < .001$; ** $p < .01$; * $p < .05$. 

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Discussion

This study builds on existing literature relating stress and pediatric obesity, while also adopting a more comprehensive approach by including subjective and objective predictors from multiple domains of development – individual, maternal, and environmental risk factors – associated with increased stress. Beyond employing a CRI approach based on a cumulative score, this study extended past literature to further explore which stress-related risk factors most strongly associated with greater adolescent weight outcomes.

Cumulative Risk and Adolescent Adiposity

Using a CRI approach, the first goal of the study was to examine whether a greater number of risk factors was associated with greater adolescent zBMI and waist-to-height ratio. Hypothesis 1 was supported, in that a greater number of risk factors (higher CRI total score) were associated with greater levels of adolescent zBMI and waist-to-height ratios. Overweight and obese adolescents had higher cumulative risk scores compared to underweight/healthy weight individuals. Although these risk factors explained a small amount of variance, these findings were consistent with previous studies using CRI approaches to examine the influence of accumulated household and family stress on pediatric obesity risk (Garasky et al., 2009; Parks et al., 2012).

Past research using probit (probability unit) regression analyses showed adolescents in families experiencing financial strain (e.g., bankruptcy, unaffordable bill payments) .7% more likely to be overweight and 1.9% more likely to be obese (Garasky et al., 2009). Parks and colleagues (2012) used logistic regression models and also found that children with an additional
parent stressor were 1.13 times more likely to be obese than non-obese children, even after controlling for child’s race, age, race/ethnicity, health quality, and parent’s gender and education level. Odds ratios less than 1.5 suggest small effect sizes (Chen, Cohen, & Chen, 2010), indicating relatively small variance explained by stress pile-up in past CRI approaches. Probit and logit regression models examine independent variables predicting a categorical variable (i.e., stressors predicting healthy weight or overweight/obese). Categorical variables however, may restrict within-group variability, such as the variability within groups of “healthy weight” and “overweight or obese” youth (Osborne, 2014).

The present study extends past literature by using hierarchical regression models, allowing for greater variability in the continuous dependent variables – zBMI and waist-to-height – instead of restricting them to categorical outcome variables. We found similarly small, but significant effects, relating stress and adolescent adiposity. Although stress-related risk factors may play a role in predicting weight outcomes, a small amount of explained variance suggests the need to consider other genetic and psychosocial factors (e.g., genetic predispositions, parent feeding practices) as stronger predictors of obesity risk. Future research should evaluate the accumulation of stress in the context of sedentary behaviors, poor parental feeding, and family history of diabetes and metabolic syndrome to better examine the role of stress.

Notably, this study is the first known research examining cumulative stress related to adolescent waist-to-height ratio. Greater cumulative risk predicted greater waist-to-height ratios in adolescents. Past literature examining stress and pediatric obesity has focused on BMI outcome variables (Garasky et al., 2009; Parks et al., 2012). However, BMI does not account for varying muscle mass and body types across individuals (Brambilla et al., 2013; Kahn, et al., 2005). Waist-to-height ratio offers a complementary depiction of weight status in adolescents. In
both adults (Savva, Lamnisos, & Kafatos, 2013) and adolescents (Kahn, Imperatore, & Cheng, 2005; Maffeis, Banzato, Talamini, 2008) waist-to-height ratio has been strongly associated with risk for development of the metabolic syndrome, which is a precursor to the development of type II diabetes. Particularly important in the context of stress, visceral (abdominal) fat growth results from cortisol reactivity in response to external stress (Drapeau, Therrien, Richard, & Tremblay, 2003). In response to stress, increased blood flows to abdominal areas, which contain more glucocorticoid receptor sites than other areas of the body, and stimulates increased weight gain in abdominal regions (Adam & Epel, 2007; Drapeau et al., 2003). This study found that stress – especially combined stress – may potentiate heightened risk for greater waist-to-height ratio; however, these findings should be considered in light of the small amount of variance accounted for in the regressions. Despite the small magnitude of effect, our study alludes to the importance of including waist-to-height ratio as an outcome variable in future pediatric obesity research.

CRI approaches can be used to address the compounded effects of stress related to adolescent adiposity. However, CRIs limit the interpretability regarding which stress-related risk factors most strongly predict adolescent weight outcomes. When predictor variables are dichotomized and given equal weight in an index score, their unique predictive associations cannot be examined. The present study addressed this limitation through further analyses examining these domains of risk factors contributing to adolescent adiposity.

**Individual-, Maternal-, and Environment-Level Risk Factors Related to Adolescent Weight Outcomes**

To address the specificity limitations in CRI approaches, the second goal of this study was to examine unique associations from individual-, maternal-, and environment-level domains of risk factors related to adolescent zBMI and waist-to-height ratios. Guided by
Bronfenbrenner’s socio-ecological model of development (Bronfenbrenner, 1992), this study expected that greater levels of stress in these domains of adolescent development (individual, maternal, and environmental) would each explain greater adolescent zBMI and waist-to-height ratios. This hypothesis was supported, as each domain significantly explained an additional amount of variance in adolescent weight outcomes. This suggests the importance of examining risk factors not only for the individual adolescent, but also other possible sources of stress due to individuals’ parents and broader environments. Still, for both zBMI and waist-to-height ratio, the overall amount of variance explained at each level was small (1-3%), with a cumulative ~8% variance accounted for by all three levels combined. This is consistent with past research finding small magnitude effects relating subjective psychosocial stressors to obesity prevalence and development (Garasky et al., 2009). Eating patterns, physical activity, genetic predispositions, and parenting modeling behaviors should be included in future research to examine whether stress plays a role above and beyond other known risk factors (Clark et al., 2007; Krebs et al., 2007; Mitchell, Pate, Beets, & Nader, 2012; Patro et al., 2013).

**Unique Associations between Risk Factors and Adolescent Adiposity**

Hierarchical regressions allowed for an examination to determine which risk factors uniquely associated with greater levels of adolescent weight outcomes. Environment-level risk factors explained the largest amount of variance in adolescent zBMI and waist-to-height ratios.

**Low SES and weight outcomes.** Lower SES (income-to-needs ratio) in particular, was the strongest predictor for both weight outcomes. As suggested by past literature, poverty represents an influential psychosocial stressor related to greater pediatric obesity (Babey et al., 2010; Kendzor et al., 2012). Although income-to-needs ratio explained a small amount of variance in this study, these findings still suggest clinical significance based on previous research
regarding influences of poverty on pediatric health. Children in lower SES households likely experience multiple stressors including financial strain, limited food availability, less access to safe physical activity opportunities, and less parental monitoring and involvement (Babey et al., 2011; Burke et al., 2011). Low-income stress may particularly lead to adversity for adolescents who may be more aware of their family’s financial strain compared to younger children, thus, leading to negative health effects (Garasky et al., 2009).

Home environment and zBMI. Related to but distinct from SES in this study, broader home environment characteristics, including lower cognitive stimulation and lower emotional support, were associated with greater waist-to-height ratios. Interestingly, these home environment characteristics failed to predict adolescent zBMI. Past research found that younger children were at greater risk for obesity when they had lower household cognitive stimulation and emotional support; however, this relationship was not found in adolescents (Garasky et al., 2009). Just as this study found mixed results regarding these home environment characteristics and adolescent weight outcomes, the previous literature examining broad home environment characteristics and pediatric obesity risk has been inconsistent. Future research is needed to better understand whether and how the physical and emotional characteristics of adolescents’ home environments may influence health outcomes such as obesity.

Child depressive symptoms and weight outcomes. At the child-specific (individual) level, greater child depressive symptoms predicted greater levels of both adolescent zBMI and waist-to-height ratios, as seen in past longitudinal research observing this relationship (Goodman & Whitaker, 2002). Depressive symptoms may contribute to obesity risk for several reasons. Increased depression in children has been related to greater emotional eating and decreased physical activity (Goossens, Braet, Van Vlierberghe, & Mels, 2009; Motl, Birnbaum, Kubik, &
Dishman, 2004), which in turn may potentiate obesity risk. In the context of stress, depressive symptoms and obesity are proposed to share a similar stress-response pathway, in which cortisol reactivity explains the link between greater depressive symptoms and higher BMI (Dockray et al., 2009). Depressive symptoms may serve as a stressor related to dysregulated diurnal cortisol patterns and higher cortisol reactivity from stressful experiences.

**Awakening cortisol and waist-to-height ratio.** Findings from this study suggest that blunted levels of awakening cortisol may be an additional risk factor for greater adiposity (zBMI) in a relatively healthy sample of adolescents. Researchers largely agree that dysregulated HPA axis activity is related to obesity risk (Pasquali, Vicennati, Cacciari, & Pagotto, 2006), yet the literature examining associations between cortisol and obesity risk has been mixed and relatively little work has been done with pediatric populations. The majority of the research, conducted primarily in adults, suggests that elevated cortisol release is associated with greater obesity risk (Pasquali et al., 2006; Bose, Olivan, & Laferrere, 2009). However, lower awakening cortisol, as part of a diurnal pattern, has also been associated with obesity in adults (Kumari, Chandola, Brunner, & Kivimaki, 2010; Praveen et al., 2011). Blunted cortisol levels are typically found in populations with stress-related disorders (e.g., PTSD) and among those who have experienced chronic stress (Roisman et al., 2009; Tarullo et al., 2006; Wessa et al., 2005). This study conceptualized lower awakening cortisol as a marker of increased adolescent stress. However, given that awakening cortisol was not correlated with the other stress-related risk factors in this study and that we did not observe an association between awakening cortisol and waist to height ratio, alternative explanations should also be considered (e.g., that awakening cortisol is related to other stressors outside the scope of this study or that this finding is a measurement artifact).
This is the first study to report an association between lower awakening cortisol and greater adolescent adiposity. Due to the small amount of variance explained by cortisol levels in this, future research is needed—employing rigorous methodology of basal and diurnal cortisol collection—to better understand the role of cortisol activity as a stress biomarker of obesity risk. To date, pediatric obesity research has primarily focused on cortisol reactivity related to overweight and obesity status. Given the lack of previous literature examining associations between diurnal cortisol patterns and adiposity in relatively healthy adolescents, additional research is needed to better characterize the association between awakening cortisol and pediatric obesity. Further research should examine these inconsistencies, which may, in part, be due to methodological differences in cortisol measurement (e.g., basal conditions, stress-induced conditions).

**Limitations and Future Directions**

While this study extends the literature on cumulative stress and pediatric obesity, there are several limitations that should be kept in mind. First, the physiological and psychosocial risk factors utilized in this study were not all-inclusive. Future research should include other physiological markers of stress (e.g., skin conductance, cortisol reactivity to stress induction) as well as additional psychosocial predictors relevant in adolescence (e.g., peer victimization) that may predict adolescent obesity development (Foss & Dyrstad, 2011). As this study found cumulative stress explaining a small, but significant amount of variance in adolescent obesity, an examination of additional relevant stressors in future CRI research may elucidate a larger effect of cumulative stress related to pediatric obesity risk. Early childhood adversities (e.g., trauma) or prolonged exposure to stress represent risk factors that should also be included in future stress-obesity research. An examination of chronic stress related to obesity development would benefit
from longitudinal analyses; however, these analyses were not included in this study’s cross-sectional design. Longitudinal analyses may also consider potential interactive effects between interrelated risk factors that may further increase adolescent obesity risk.

Future research using longitudinal analyses is needed to explain complexities and bidirectional relationships between stressors and obesity risk. For instance, blunted awakening cortisol in diurnal patterns (Gunnar et al., 2010; Roisman et al., 2009; Wessa et al., 2005) and higher cortisol reactivity have both been related to poorer weight outcomes and behaviors (Roemmich et al., 2007). In this study, average awakening cortisol was calculated from daily saliva samples across three days; however, longitudinal research including both diurnal cortisol patterns (with multiple saliva assays throughout the day) and cortisol reactivity might further elucidate the impact of cortisol in predicting obesity development. Depressive symptoms offer a second example of a complex relationship with risk for obesity. While depression has been found to longitudinally predict obesity (Blaine, 2008), a bidirectional relationship may exist as obesity is often explained as a condition leading to increased depressive symptoms from experiences including perceived stigmatization and peer victimization (Friedman et al., 2005). This study conceptualized depressive symptoms as a stressor (Dockray et al., 2009). Future research should examine this relationship longitudinally to better understand the prominent directionality relating depression and pediatric obesity.

Lastly, further research is needed to examine cumulative effects of stress in a more diverse sample. This national dataset was limited to a largely homogenous sample. Caucasian adolescents living in middle- or upper-SES households made up the majority of the sample. Given that ethnic minorities and low-income pediatric populations are at greater risk for obesity and may be more likely to be exposed to cumulative stress (e.g. financial stress, neighborhood
violence), future research should examine accumulated risk factors in these populations (Burke et al., 2011; Ogden et al., 2014).

Conclusion

This study showed that accumulated stress from various domains of development may contribute to greater weight status in adolescents. Specifically in a population already at risk for weight gain due to rapid physical and social changes (Adair, 2008), adolescents may be at greater risk for stress impacting their health. For example, low-SES might represent an important stressor posing barriers to maintaining healthy lifestyles. This study suggests that not only does a greater “stress-pile up” impact poorer weight outcomes, but specific risk factors, including lower SES and depressive symptoms, may be notable vulnerabilities linked to adiposity. Although cumulative stress should be further examined in context of other strong contributing factors of obesity, examining the role of cumulative stress presents implications for adolescent weight control interventions as well. Identifying adolescents who most likely experience accumulated stress and understanding which stressors most strongly potentiate obesity risk may help guide future interventions in adolescent weight-control treatment and prevention.
References


Lohman, B. J., Stewart, S., Gundersen, C., Garasky, S., & Eisenmann, J. C. (2009). Adolescent overweight and obesity: Links to food insecurity and individual, maternal, and family


Appendix A. Approval from University Institutional Review Board

KENT STATE UNIVERSITY INSTITUTIONAL REVIEW BOARD
APPLICATION FOR APPROVAL TO USE HUMAN RESEARCH SUBJECTS

Fillable document with information about Institutional Review Board approval for research.

Principal Investigator
Name: Dr. Amy Sato, Ph.D
Address: 219 Kent Hall Addition
Phone: (330) 672-4888

Project: Faculty Research

KSU Faculty Co-Investigator(s) (Use additional sheets if necessary)
Name: Dr. Christopher Flessner, Ph.D.
Address: 325 Kent Hall Addition
Phone: (330) 672-2236

Estimated Project Duration:
Starting Date: 11/01/2012, Upon IRB Approval (But not before approval is obtained)
Ending Date: 11/01/2015

IRB Reviewer Determination
Level I – Exempt, Category 4
Level III – Full Board Review
Disapproved
Primary Reviewer: William E. Mears
Date: 10/25/12

IRB Administration Action
Approved Level I – Exempt, Category 4
Administrator, IRB: Robert Winkler
Date: 11/11/12

Full Board Review Action
Approved
Meeting Date: N/A

AGENDA Date
N/A

Correspondence
E-mail approval
Date
11/5/12

E-mail notice of annual review
Date

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Part I: Please answer the following questions by checking the correct response.

☐ Yes ☒ No 1. Will participants be identifiable to anyone other than the researchers through records, responses, or identifiers linked to the participants?

☐ Yes ☒ No 2. Could participants be at risk of criminal or civil liability, damage to employability or to financial standing, or undue embarrassment, if responses became known outside this research project?

☒ Yes ☐ No 3. Does research deal with sensitive aspects of participants' behavior, such as illegal conduct, drug use, sexual behavior, use of alcohol, or potential harm to self or others?

☒ Yes ☐ No 4. Does research involve the study of existing data? (If yes, please specify.)
   ☒ Documents, archives, and/or records ☐ Biological specimens

   4.a. Is the database, archives, or record collection publicly available? ☐ Yes ☒ No
   4.b. Are the subjects who provided the data individually identifiable? ☀ Yes ☒ No
   4.c. Will any identifying information that may link your data to individuals be included in your research records? ☐ Yes ☒ No

☒ Yes ☐ No 5. Does the research involve audio, video, digital, or image recordings of participants? (If yes, please specify.)
   ☒ Video-taped ☐ Audio-taped ☕ Photographed ☐ Other: (Specify: )

☐ Yes ☒ N/A 6. Are participants free to withdraw at any time without penalty?

☐ Yes ☒ No 7. Is there deception of participants? (If so, answer questions in Part VII, #35-44)

☒ Yes ☐ No 8. Does the research deal with participants under the age of 18?

☐ Yes ☒ No 9. Will identifiable medical information be collected?

10. Does the research deal with any of the following vulnerable populations:

☐ Yes ☒ No Legally incompetent adults ☒ Yes ☒ No Traumatized or Comatose

☐ Yes ☒ No Cognitively/Mentally impaired ☒ Yes ☒ No Economically Disadvantaged

☐ Yes ☒ No Physically challenged ☒ Yes ☒ No Terminally ill

☐ Yes ☒ No Pregnant women ☒ Yes ☒ No Prisoners

11. Does the project involve: (If yes, also answer question #20 on page 4).

☐ Yes ☒ No Administering drugs ☒ Yes ☒ No Medical devices

☒ Yes ☐ No Administering alcohol ☒ Yes ☒ No Invasive procedures

☐ Yes ☒ No Administering nutritional supplements ☒ Yes ☒ No Drawing blood

☑ Yes ☒ No Taking tissue samples ☒ Yes ☒ No Giving injections

12. Are you collecting any portion of your data on-line? ☐ Yes ☒ No

13. Are you requesting a waiver of any elements of the consent process? ☐ Yes ☒ No
   (If yes, answer questions in Part VIII, #43-44.)

Part II: Summary of Research

14.) Describe the purpose and significance of the proposed research; include sufficient background information and the specific objectives of the study. Summarize the major hypotheses. (Use non-technical language that can be understood by someone outside the discipline.)

A primary goal of this study is to examine mental health and physical health influences on child and family health and functioning. Pediatric obesity is a public health epidemic. Approximately 32% of US children and adolescents are currently either overweight or obese, and low-income youth are at even greater risk (Ogden et al., 2010). Adolescent obesity increases the risk for chronic diseases (e.g., cardiovascular disease and metabolic problems) and poor
psychosocial outcomes (e.g., poor quality of life and low self-esteem) (Israel & Ivanova, 2002; Weiss et al., 2004). Furthermore, obesity may reduce overall adult life expectancy (Fontaine et al., 2003). Examining contributing factors relating to child and adolescent obesity is essential to gain a greater understanding of this national epidemic. Research in this area will potentially identify specific risk factors and may lead to prevention and intervention strategies to implement in overweight and obese youth populations.

The National Institute of Child Health and Human Development (NICHD) conducted a large-scale longitudinal study known as the Study of Early Child Care and Youth Development (SECCYD) examining various factors (e.g., health behavior and school performance) related child care arrangements in children and their families. Using this existing data set of four phases from birth and 15 years old, the present study will examine these various contributors to children’s health, specifically relating to childhood obesity. The purpose of this study is to use this existing data from the 12 year longitudinal SECCYD study to examine anthropometric measures (i.e. recorded weight, height, and body mass index) as well as psychosocial factors (including stressful life events, mental health [e.g., parent and child anxiety], home observations, eating patterns, peer relations, social support, parenting, physical activity, and family characteristics) predicting weight status (e.g., overweight, obese) and weight trajectories from childhood to adolescence. Health disparities such as socio-economic status of families will also be explored relating to obesity risk factors.

The NICHD data set is available for purchasing by affiliated researchers. Upon IRB approval, we plan to apply for the purchase of this data set. These data will be used solely for statistical analyses and no identifying participant information is associated with the data set. All of the participants already consented to participate in this study. Participants were asked to complete child and parent questionnaires, demographic information, and child and parent behavior was observed. No person except for the principal investigator and the research staff identified in this application (please see below) will have access to the data. We will comply by the policies of the investigator, research staff, IRB, and the receiving institution required from the NICHD.

Principal investigator and research staff: Dr. Amy Sato (PI), Dr. Christopher Flessner (co-I) and Amy Fahrenkamp. Other graduate students may be involved in this project.

15.) Describe the study design, research methods and procedures. (Please append copies of the consent form and all measures, including interview questions and self-report questionnaires, to this form.) What are the qualifications of the individual(s) who will be collecting the data?

This study will use information from a data set with four phases of data collected from children and families. The data set consists of information from commonly used research methods including child and parent questionnaires, anthropometric measurements, demographic information, and parent and child observations. All of the data used for the present study have already been collected.

Part III: Research Participants

16.) Briefly describe the characteristics of your population(s). Describe the ethnic background, sex, age, state of health, and the criteria for inclusion or exclusion of participants. (Include rationale for use of special classes of participants such as pregnant women, children, institutionalized mentally disabled, prisoners, or those whose ability to give voluntary informed consent may be in question.) If your population is all one gender or ethnic group, please explain.

The NICHD research team conducted a large scale longitudinal study involving over 1300 participants, including children and their families. Participants were recruited from 10 locations across the U.S. Gender was fairly equal across conditions and about 80% of the sample were Caucasian. Data was collected from the children in four phases (Phase I: ages 0-3 n = 1364, Phase II: 3 years old through 1st grade n = 1226, Phase III: 2nd grade through 6th grade n = 1061, and Phase IV: 7th grade through 9th grade n = 1009). The exact frequencies of the sample regarding ethnicity, health disparities, and age will be known after purchasing and receiving the data set from NICHD. After we receive the dataset, we will know the socioeconomic status for the participant pool. At this point we are unaware of disadvantaged economic status.

17.) Indicate the anticipated sample size.

The NICHD data set consists of 1364 participants from 10 locations in the U.S.

18.) Explain the recruitment process. State how potential participants will be identified and who will make the initial contact. Explain how you will ensure that recruitment and selection of participants is equitable. (Please include all recruitment materials, including scripts, flyers, and advertisements as attachments to this form.)

The data have already been collected.
Part IV: Risks/Benefits

19.) Identify any expected or potential risks or discomforts (including physical, psychological, social, or legal) to which participants may be exposed as a result of participation in the research project (beyond those encountered in everyday life).

There are no known risks or benefits regarding the participants of this study. The data has already been collected, so the only known potential risk would be a violation of confidentiality.

a.) What safeguards will you use to protect the participants from these risks, as well as to protect their rights, welfare, and privacy? (Must provide a response; never answer "N/A").

All data will be kept confidential. The data will be stored on password-protected lab computers that one the researchers will have access to. Along with the specific computer log in information needed, the files containing the data sets will also be password protected and only accessible to the research team. No backup, except anything provided by NICHD, will be available. Any backup information will be stored on the password-protected computers with an additional password to protect the back up files, again to only be accessible to the researchers. Any hard copies of data set information provided by the NICHD will be kept in locked file cabinets in our locked research lab, accessible only to the research team. The data will be destroyed upon completion date of the study (November 1, 2015), unless a revision to the IRB is required and approved for further data analyses. If temporary files for analyses were created, then they will be deleted upon study completion as well. We will comply with any specific requirements regarding participant protection set forth by the NICHD.

20.) Describe the anticipated benefits to individual subjects and to society expected to be gained from this project. (This should include any direct benefits to the participants as well as any generalized gain in knowledge. If there are not direct benefits to individual subjects, state that.)

Individuals have already participated in this study. Without the cost of further participation from the sample, the data sets offer opportunities for additional research and publications to increase the benefit of this project. There are no known direct benefits to participants of this study. Society may benefit from this study by gaining a further understanding of various issues related to childhood obesity and health disparities in the U.S.

21.) Describe the qualifications of the person administering drugs, alcohol, or nutritional supplements, or drawing blood, taking tissue samples, or giving injections.

Please note:
   i. Persons doing venipuncture must provide a copy of their certification to draw blood and proof that they completed a blood-borne pathogens training course.
   ii. Indwelling venous catheters and lines can only be inserted and accessed by licensed/registered/certified medical personnel such as physicians, RNs, and EMTs. Proof of certification is required.
   iii. Arterial blood sampling can only be carried out in an appropriate medical facility such as a hospital, clinic, or the KSU Health Center. The procedure can only be carried out by qualified personnel under the direct supervision of a licensed physician.

N/A

22.) Describe any form of compensation to participants. (i.e., money, extra credit, etc. If money, extra credit, or grade is given to students who participate in the project, what opportunity for extra credit or grade is provided to students who choose not to participate?)

Please note:
   a. If the research participation affects the course grade (e.g., extra credit), then alternative opportunity for course credit is needed.
   b. For multi-phase projects, compensation should not be contingent upon completion of the whole project. Rather, some compensation should be given for each phase of the project. The nature of the compensation should be stated in the consent form.

All participants granted informed consent.

23.) Research participants will be informed of the risks and benefits through:
   - Consent form (Include with application)
   - Verbal Script (Include with application)
   - Parental Consent form for parents/guardians (required for children 18 of age and younger)
   - Assent form (in addition to Parental Consent form for children 12 years of age and younger)

Part V: Informed Consent (You must include a copy of the informed consent document with application materials.
Visit the IRB website for more information about informed consent documents)

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24.) Describe the consent process. Explain when and where consent will be obtained and identify who will be obtaining informed consent. 
All data has already been collected.

25.) If you will be using children under 18, explain in detail how you will obtain parental consent and assent (for children under 12) or consent (for children 12 to 18). If assent/consent will be obtained orally, supply a script of what you will say and how you will give the children the opportunity to agree to participate or decline.
All data has already been collected.

26.) Explain how the possibility of coercion or undue influence will be minimized in the consent process (e.g., if employer is approaching employees, instructors are approaching students, physicians are approaching patients, if compensation is involved, etc.).
All data has already been collected.

Part VI: Privacy and Confidentiality of Records

27.) Will this study use or disclose protected health information from a covered entity (a covered entity is a Doctor, Clinic, Dentist, Pharmacy, Health Clinic etc... that sends transactions electronically) as defined in the Health Insurance Portability and Accountability Act (HIPAA)?

☐ Not Applicable
☐ Applicant will use a HIPAA Authorization [specify type below]
☐ Form provided by covered entity ☐ Form created by applicant
☐ Applicant requests IRB waiver of Authorization

28.) Where will the signed consent forms be kept? (Consent forms must be kept in a secured location on campus, not in a private home or office.) If the study does not involve consent forms, answer “N/A”.
The NICHD keeps the participants’ consent forms.

29.) Describe specifically how you will maintain the confidentiality of the data.
All data will be kept confidential. The data will be stored on password protected lab computers that one the researchers will have access to. Along with the specific computer log in information provided, the files containing the data sets will also be password protected and only accessible to the research team. No backup, except anything provided by NICHD, will be available. Any backup information will be stored on the password-protected computers with an additional password to protect the back up files, again to only be accessible to the researchers. Any hard copies of data set information provided by the NICHD will be kept in locked file cabinets in our locked research lab, accessible only to the research team. The data will be destroyed upon completion date of the study (November 1, 2015), unless a revision to the IRB is required and approved for further data analyses. If temporary files for analyses were created, then they will be deleted upon study completion as well. We will comply with any specific requirements regarding participant protection set forth by the NICHD.

30.) How will the data/results of the research be disseminated?
☐ Thesis ☑ Publication
☐ Dissertations ☐ Course Requirement: Course #:
☐ Public presentation
☐ Other: Specify:

31.) How will the data be stored after study completion? Please be specific as to the retention or destruction of audio/video data or cell lines.
The data will be deleted upon study completion, along with any temporary files that are created while analyzing data.

32.) a). If the participants’ personal files (school, medical, etc.) will be read, where are the files kept (name the place, e.g. doctor’s office, hospital, clinic, etc.) and who will gather the information?

b). Has permission been obtained to gather this information? (Attach documentation)
c). Do the participants (and/or their parents or guardians) know that these files will be read? If no, explain.

33). a). Will individual results or other data be disseminated to the participants (and/or their parents or guardians)?

b). If so, explain the qualifications of the person(s) interpreting the results.

34.) Does the proposed study involve deception? ☑ No ☐ Yes (Please complete Part VII)

Part VII: Projects Involving Deception

35.) Describe the type of deception being used. Consider in your answer both deception by omission (an important aspect of the research is withheld from the subject) and deception by commission (the subject is misled about the true purpose of the research).

36.) Why is deception a necessary and unavoidable component of the experimental design? (Does the deception improve the internal or external validity of the study?)

37.) Has this research protocol (involving deception) been previously used? If "Yes," please provide information on any actual harms to the participants and reactions of the participants to the use of deception in this research.

38.) What alternative procedures were considered that did not involve deception and why were these alternatives rejected?

39.) Since deception precludes informed consent by the subject prior to participation:

a.) How will participants be debriefed?

b.) Who will debrief them?

c.) Will the debriefing of participants be:

☐ Immediate (immediately following the experimental session in which deception occurs)
☐ Delayed
☐ Full (all deceptive aspects of the study will be revealed)
☐ Partial (some deceptive aspects of the study will remain unexplained)

40.) If debriefing is delayed, why is delayed debriefing necessary and when will debriefing occur?

41.) If debriefing is partial, why is the partial debriefing necessary? Why is unexplained deception necessary? Would the subject be harmed in any way by full debriefing?

41.) Even if the subject is partially debriefed during the study, will full debriefing occur later?

42.) Does the presence of deception increase the risk of harm to the subject?

43.) Is the respondent free to withdraw his/her data after being fully debriefed? (e.g., form like audio/video taping).
Part VIII: Request for Waiver of Elements of Informed Consent

43.) Are you requesting a waiver of the documented informed consent form for each participant? ☐ Yes ☒ No

Please indicate the justification for requesting this waiver:

☐ The only record linking the subject to the research would be the signed consent document and the principal risk of the research would be breach of confidentiality.

☐ The research involves only minimal risk to the subjects and involves no procedures for which written consent is normally required outside of the research context (e.g. anonymous surveys of adults).

Note: Participants must still be provided with a written statement regarding the research that contains the required elements of informed consent. Refer to the Informed Consent Template on our website for more information.

44.) Are you requesting a waiver or alteration of any of the other required elements of informed consent? ☐ Yes ☒ No (An IRB may, on occasion, approve a consent process that omits some or all of the required elements of informed consent or waive the requirement for informed consent. The following criteria must be met: 1) the research involves no more than minimal risk, 2) waiver or alteration will not adversely affect the rights and welfare of subjects, 3) the research could not practically be carried out without waiver or alteration, and 4) when appropriate, the subjects will be provided with additional pertinent information after participation.)

a.) Provide justification for the waiver.

b.) Indicate why the proposed research presents no more than minimal risk to participants.

c.) Explain whether or not a waiver of written informed consent would adversely affect the rights and welfare of participants.

d.) Explain why it would be impracticable to carry out the research without a waiver or alteration of informed consent.

e.) How will pertinent information be provided to participants, if appropriate, at a later date?

Part IX: Conflict of Interest

45.) Do the researchers conducting this protocol have any potential conflicts of interest? Conflicts of interest may include financial or personal interest, or any condition in which the investigator’s judgment regarding a primary interest may be biased by a secondary interest. Examples include speaking and consultation fees, travel expenses, stock options, royalties, company ownership or equity, etc.)

☒ No ☐ Yes (If yes, conflict of interest must be disclosed)
Investigator Assurance

I certify that the information provided in this application is complete and correct. I understand that as Principal Investigator, I have ultimate responsibility for the protection of the rights and welfare of human research subjects, the conduct of the study, and the ethical performance of the project.

I agree to comply with all Kent State University policies and procedures on research involving human subjects (KSU policy #3342-3-03.2), as well as with all applicable federal, state, and local laws regarding the protection of human subjects in research. I agree that:

- The project will be performed by qualified personnel, according to the KSU approved protocol.
- Approval from the Institutional Review Board will be obtained prior to implementing any changes to the protocol.
- If the project involves approval/permission from other institutions, the research will not begin until permission has been obtained from these institutions.
- Legally effective informed consent will be obtained from human subjects if applicable, and documentation of informed consent will be retained in a secure environment for three years after termination of the project.
- Injuries, adverse events, and/or unanticipated problems involving risks to subjects or others will be reported in writing to the Kent State University IRB promptly, and no later than within 5 working days of the occurrence.
- A Continuing Review and Progress Report will be completed and submitted before the review deadline, as determined by the IRB appropriate to the degree of risk (but not less than once per year). All protocols are approved for a maximum period of one year. Research must stop at the end of the approval period unless the protocol is re-approved for another term.
- All research staff, employees, and students assisting in the conduct of the research will be informed of their obligations and responsibilities in the above commitments.

I further certify that the proposed research will not begin until approval has been obtained. A signed approval letter from the Office of Research Safety and Compliance communicates IRB approval.

[Signature]
10/25/12

Signature of Principal Investigator       Date

[Signature]
10/22/12

Signature of Co-Investigator       Date

Faculty Advisor Assurance:

I have reviewed and approved the research project described in this application. I agree to meet with the student on a regular basis to monitor study progress and assure that the well-being of subjects is adequately safeguarded. I agree to be available to assist the student investigator should any problems arise in the study.

[Signature]

Signature of Faculty Advisor       Date
Appendix B. Marital Status Demographic Questionnaire Item

What is your current relationship status with [HUSBAND]? Would you say you are separated, divorced, or widowed?
- Separated
- Divorced
- Widowed
- Don’t know
- Refused to answer
Appendix C. Adolescent Adiposity Outcome Measures

Today’s Date: ___ ___ / ___ ___ / ___ ___ ___ RA ID: ______

M     D     Y

1. HEIGHT (to 1/8”) ______ inches and ______/8th inch

2. WEIGHT (lbs., oz) ______ lbs _____ ounces

3. WAIST (to 0.1 CM) (a) __ __ __ . __ (b) __ __ __ . __ (c) __ __ __ . __ (1st) (2nd) (3rd, if necess.)

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Appendix D. Awaking Cortisol Record Form

Day 1 Date: _________________
   a. Time you collected saliva _____________
   b. Time you woke up this morning _________
   c. Time you went to bed last night _________

Day 2 Date: _________________
   a. Time you collected saliva _____________
   b. Time you woke up this morning _________
   c. Time you went to bed last night _________

Day 3 Date: _________________
   a. Time you collected saliva _____________
   b. Time you woke up this morning _________
   c. Time you went to bed last night _________
Appendix E. Child Depression Inventory – Short Form

Adolescents chose the response that best described how they felt in the past two weeks.

Responses
1. I am sad once in a while
   I am sad many times
   I am sad all the time
2. Nothing will ever work out for me
   Not sure things will work out for me
   Things will work out for me OK
3. I do most things OK
   I do many things wrong
   I do everything wrong
4. I hate myself
   I do not like myself
   I like myself
5. I feel like crying every day
   I feel like crying many days
   I feel like crying once in a while
6. Things bother me all the time
   Things bother me many times
   Things bother me once in a while
7. I look OK
   There are some bad things about my looks
   I look ugly
8. I do not feel alone
   I feel alone many times
   I feel alone all of the time
9. I have plenty of friends
   I have some friends but I wish I had more
   I do not have any friends
10. Nobody really loves me
    I am not sure if anybody loves me
    I am sure that somebody loves me
Appendix F: Center for Epidemiological Studies – Depression

These statements describe how people sometimes feel about themselves. Please answer all questions. THERE ARE NO RIGHT OR WRONG ANSWERS. Give you honest opinions and feelings Please circle the answer that comes closes to describing how often you have felt this way during the past week.

Responses:
- Rarely or none of the time (less than once a week)
- Some or little of the time (1-2 days a week)
- Occasionally or a moderate amount of time (3-4 days a week)
- Most or all of the time (5-7 days a week)

1. I was bothered by things that usually don’t bother me
2. I felt that everything I did was an effort
3. I felt I was as good as other people
4. I had trouble keeping my mind on what I was doing
5. I felt sad
6. I felt fearful
7. I felt lonely
8. I had crying spells
9. I talked less than usual
10. My sleep was restless
11. I enjoyed life
12. I felt that I couldn’t no shake off the blues even with the help of my family/friends
13. I thought my life had been a failure
14. I was happy
15. I couldn’t not get “going”
16. I felt hopeful about the future
17. People were unfriendly to me
18. I did not feel like eating; my appetite was poor
19. I felt depressed
20. I felt that people dislike me
Appendix G: Early Adolescent Home Observation Measurement of the Environment (H.O.M.E.)

Early Adolescent H.O.M.E. Inventory was modified to include 44 binary-choice items (5 subscales) from the original 60 item inventory (7 subscales). Sum of the total items computes the total score, with higher scores indicating higher levels of stimulation and support in adolescent’s home environment.

Subscale #1: Physical Elements

1. Adolescent’s room has at least two pictures or decorations appealing to an adolescent.
2. House or apartment has not potentially dangerous structural or health hazards
3. Home has at least 100 square feet of living space per person.
4. Home and immediate surroundings are not overly noisy.
5. House or apartment is clean.
6. The interior of the house or apartment is not dark or perceptually monotonous.
7. Immediate external environment is esthetically pleasing and contains no obvious health or safety hazards.

Subscale #2: Learning Materials

8. Adolescent has access to materials for arts and crafts and/or collections.
9. Adolescent has library card or name on library list.
10. Adolescent has access to at least 20 developmentally appropriate books.
11. Home has at least 2 types of reference materials (dictionary, encyclopedia)
12. Adolescent has access to a musical instrument.
13. Adolescent has access to desk or other suitable place for reading or studying.
14. Adolescent has access to home computer.
15. Adolescent has access to at least 2 appropriate board games.
16. Adolescent has access to at least 2 pieces of appropriate equipment for physical development or organized sport activities.
17. At least one full shelf of books is visible in home.

Subscale #3: Variety of Experiences

18. Family member has taken adolescent, or arranged for child to go, to a scientific, historical, or art museum during the past year.
19. Family member has taken adolescent, or arranged for adolescent, to attend some type of live musical or theater performance during the past year.
20. Family member has taken adolescent, or arranged for adolescent to go, on a trip of more than 50 miles from home during the past year.
21. Father regularly engages in outdoor activity with the adolescent at least one time every two weeks.
22. Adolescent spends some time with father (or father figure) 4 days of week.
23. Adolescent eats at least one more per day, on most days, with mother and father. (One parent families receive automatic “No.”)
24. Family visits or receives visits from relatives or friends at least once a month.
25. Family member has taken adolescent to a live organized athletic or sporting event during the past year.
Subscale #4: Acceptance and Responsivity

26. Parent mentions a particular skill, strength, or accomplishment of adolescent during interview.
27. Parent shows some positive emotional responses to praise of adolescent by visitor.
28. Parent does not refer to the adolescent in a derogatory manner or ridicule the adolescent or express hostility toward the adolescent in any way during the interview.
29. Parent talks to adolescent during visit (beyond correction, and introduction, or commands to answer visitor’s questions).
30. During the visit, when speaking of or to the adolescent, the parents’ voice conveys positive feeling.
31. Parent allows adolescent to have some privacy.
32. Parent encourages adolescent to contribute to the conversation during visit.
33. Parent responds appropriately, positively to adolescent’s questions or comments during the visit.
34. Adolescent can have a disagreement with parent without harsh reprisals.

Subscale #5: Regulatory Activities

35. Family has a TV, and it is used judiciously, not left on continuously.
36. Parent periodically discusses the hazards of alcohol and drug abuse with adolescent.
37. Parent has provided guidance or advice to adolescent during the past year concerning responsible sexuality and physical hygiene.
38. Adolescent has weekly routine household responsibilities.
39. Family has a fairly regular and predictable daily schedule.
40. Parent requires adolescent to sleep at home on school nights.
41. When parent is not available to adolescent at home, reasonable procedures have been established for check in with parents, or their designee, on weekends and after school.
42. Parent establishes rules for adolescent’s behavior with peers and asks questions to determine whether the rules are being followed.
43. Parent has had contact with at least two of the adolescents’ friends in the last month.
44. Parent knows signs of drug usage and remains alert to possible experimentation or abuse.
Appendix H. Family Education and Income Questionnaire

1. a. About how much total income, before taxes, did you family receive in the last year? Please include income from all sources listed in the questions above. By family, we mean you, your husband/partner, and your children, if living in your home.

Circle the income range that your annual income falls within. For example, if your total family annual income for last year was $22,500, you should circle “5. 20,001 - $25,000.”

**Annual Family Total Income Before Taxes**

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<tr>
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<tr>
<td>More than $1,000,000</td>
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</tr>
</tbody>
</table>

b. Please tell us how many people were supported by this income?
   1. Number of adults _______
   2. Number of children _______

Income-to-Needs Ratio Based on Pre-Tax Income

Computed by dividing the total family pre-tax income by the poverty threshold for a household, which were obtained from the U.S. Census Bureau, Current Population Survey.
Appendix I. Self-care Checklist – Neighborhood Safety Subscale

These items are about your neighborhood. Decide how true the sentence is for you. Responses:

1 = Not true at all
2 = A little true
3 = Somewhat true
4 = Mostly true
5 = Really true

1. It is safe to walk around my neighborhood.
2. It is safe in my neighborhood.
3. I am scared of some of the people in my neighborhood.
4. There are people in my neighborhood who might hurt me.