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THE IMPACT OF CHRONIC STRESS ON CHILDHOOD OBESITY AND THE

PROTECTIVE EFFECTS OF PARENTAL WARMTH

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THE FACULTY OF THE GRADUATE SCHOOL

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BY

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ABSTRACT

Mexican American children have the highest rates of obesity in the U.S. Mexican American children may experience chronic stress, which has been linked to child obesity. Hair cortisol accumulation has been implicated as a mechanism for this association. Highly sensitive parenting may buffer the harmful effects of chronic stress and hair cortisol on obesity. Thus, the aims of this project were to 1) identify the direct and indirect effects of chronic stress and hair cortisol on children's zBMI and 2) examine the protective effects of parental warmth. This study examined hair cortisol levels, zBMI and chronic stress of children ages 6-10 from low-income Mexican-origin families. Parental warmth was assessed through video-taped family interactions and parent self-report. Chronic stress and hair cortisol levels were not associated, however, hair cortisol was positively related to child zBMI. Parental warmth moderated the relation between chronic stress and zBMI. Clinical and research implications are discussed.

CHAPTER ONE

INTRODUCTION

Latino children in the U.S. have some of the highest rates of childhood obesity in the country (Ogden et al., 2016), and even with major public health efforts to halt the growing epidemic, there has been little impact on the obesity rates for Latino youth (Center for Disease Control and Prevention [CDC], 2011). Low-income Latino youth face an especially stark picture in terms of weight management due to environments with fewer outdoor spaces and stores with healthy food options (Lobstein, Baur, & Uauy, 2004; Singh, Siahpush, & Kogan, 2010). In fact, health disparities in the U.S. between social class and ethnicity have been found to be increasing (Singh, Siahpush, & Kogan, 2010). Of special interest is the Mexican-origin population, who face particularly high rates of childhood obesity (Fernandez, Redden, Pietrobelli, & Allison, 2004). In a nationally representative study of childhood waist circumference, a measure of obesity, researchers found that Mexican-origin children had the highest waist circumferences as compared to non-Latino white children and African American children (Fernández et al., 2004). Differences in Body Mass Indices (BMI) for children from low-income and ethnic minority backgrounds has led researchers to focus on additional psychosocial factors that may affect obesity rates. One major factor that may play a role in this health disparity is exposure to chronic stress. Children from low-income Latino families experience disproportionate stress

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(American Psychological Association [APA], 2011; Djuric et al, 2008; National Institutes of Health [NIH], 2011). Mexican-origin youth, whose parents emigrated from Mexico to the U.S. face uniquely stressful environments: almost half of undocumented immigrants living in the U.S. are Mexican-origin (Krogstad & Passel, 2015), and undocumented immigrant children and U.S. born children of undocumented immigrants experience the chronic fear of deportation that has been shown to have a serious negative impact on mental health (Yoshikawa, Suárez-Orozco, & Gonzales, 2016). In addition, Mexican-origin youth with documented legal status also face multiple stressors, including ethnic discrimination (Pérez, Fortuna, & Alegría, 2008), cultural adaptation stress (Gonzales, Germán, & Fabrett, 2012) and overrepresentation in low-income neighborhoods (South, Crowder, & Chavez, 2005).

Although the relation between stress and obesity in adulthood has been studied thoroughly, research is somewhat more limited for children. Obesity and overweight in the school-aged years (6-10) are particularly important to examine because of its strong association with adult obesity and other chronic illnesses (Simmonds, Llewellyn, Owen, & Woolacott, 2016). Recent literature using cross-sectional data has found that children who experience more chronic stress have higher BMIs (Sweeting, Wright, & Minnis, 2005; Yin et al., 2005), but research using longitudinal data has yet to fully explain the mechanisms through which stress affects weight (van Jaarsveld et al., 2009). This study hypothesizes that chronic stress increases BMI through the dysregulation of the body's stress response system. One way of measuring the body's response to chronic stress is through the accumulation of cortisol, a key hormone in the regulation of the body's fight or flight response. Research has found that chronic stress is

positively associated with hair cortisol levels (Van Uum et al. 2008; Kirschbaum et al. 2009; Thomson et al. 2010; Groeneveld et al., 2013), and in turn, increased cortisol levels have also been found to be present in children who are overweight or obese (Veldhorst et al., 2014). This study aims to further understand the relation between chronic stress and childhood obesity, specifically by examining the accumulation of cortisol as a mechanism through which chronic stress impacts BMI.

This study also aims to examine how parental warmth may buffer the relation between chronic stress and childhood obesity. Parenting has been a target for childhood obesity interventions, and some studies have found that warmth is a particularly important factor in weight management programs (Rhee et al., 2016), but others have found the inverse relation (Olvera & Power, 2010). How parental warmth affects childhood obesity within the context of chronic stress has yet to be studied, and therefore this study will test whether parental warmth moderates the relation between chronic stress and BMI. In addition, as it is hypothesized that hair cortisol will mediate the relation between chronic stress and BMI, this study will also test whether parental warmth buffers the relation between chronic stress and accumulation of hair cortisol and/or buffers the relation between accumulation of hair cortisol and obesity. This study aims to examine the effects of chronic stress in the context of a highly stressed population: Mexican-origin children living in the U.S. Though chronic stress may negatively affect the home environment, highly sensitive parents may dampen the effects of persistent and protracted stress on children's physical health through warm and attentive parenting.

CHAPTER TWO

REVIEW OF RELEVANT LITERATURE

Weight and Health Disparities for Low-Income Latino Children

One of the major health issues facing the US is childhood obesity. Overweight and obese children have been found to have an increased risk of developing metabolic syndrome, Type 2 Diabetes, respiratory, and cardiovascular illness (Duncan, Li, & Zhou, 2004; Okosun et al., 2010; Visness et al., 2010). In the most recent NHANES study of weight to recumbent length in the United States, Latino children have been found to have higher BMIs compared to non-Latino white and black children (Ogden et al., 2016). In the nationwide study, researchers found that 25% of Hispanic/Latino children between the ages of 6 and 10 in the U.S. were obese (having a BMI above the 95th percentile) (Ogden et al., 2016). Comparatively, the nationwide average of obesity among this age group is 17.5% (Ogden et al., 2016). Some research has found a decrease in childhood obesity since efforts to increase exercise and healthy eating behaviors began in 2000s, though others have not found this trend (Ogden et al., 2016; CDC, 2011). In a 2011 report of BMI among New York City public school children, researchers found an overall decline in obesity rates, but found that the smallest decrease was among Latino children (CDC, 2011). Research on individuals from different Latin American backgrounds found that Mexican American youth may be at a particularly high risk for obesity (Martorell, Khan, Hughes, & Grummer-Strawn, 1998). In addition to ethnic differences in obesity, children from low-income homes are more likely to become overweight (Lobstein, Baur & Uauy, 2004). Some argue that

this disparity in healthy body weights may be due to fewer opportunities for physical exercise and limited access to nutritious foods (Lobstein, Baur, & Uauy, 2004), though others argue that exposure to chronically stressful environments and parenting play important roles as well (Kitzmann, Dalton, & Buscemi, 2008). These factors may have a role in placing low-income Mexican-origin children at an increased risk for becoming overweight and obese.

Impact of Stress for Low-Income Mexican-origin Children

One quarter of Mexican American children in the United States between 2007 and 2011 were living in poverty (Macartney, Bishaw, & Fontenot, 2013). Children living in poverty experience a variety of stressors including more family conflict, greater exposure to violence and environmental toxins (Conger & Donnellan, 2007; Attar, Guerra, & Tolan, 1994; Sullivan, Kung, & Farrell, 2004; Margolin & Gordis, 2000; Chakraborty & Zandbergen 2007; Chang et al. 2009; Schreier & Chen 2013). Children who grow up in poverty are also more likely to experience frequent moves (Simons et al., 2002) which can lead to additional stress in the home environment. In addition to the increased likelihood of individual stressors, growing up in poverty has been hypothesized to create a "context of stress" in which stressors build upon each other incrementally (McLoyd, 1998). Mexican-origin children living in poverty may also experience unique stressors such as discrimination stress and the stress of acculturating to white Anglo-Saxon culture in the US (Polo & Lopez, 2009; Wight, Aneshensel, Botticello, & Sepulveda, 2005). Children from families immigrating to the U.S. from Mexico experience prolonged, or *chronic*, stress from multiple sources; Immigrant families are more likely to be exposed to violence and economic strain (Paral, 2009), and, within the current context of anti-

immigrant sentiment in the U.S., immigrants are more likely to experience discrimination (Cervantes, Goldbach, & Padilla, 2012).

Though children in the U.S. who grow up in poverty may have fewer healthy food options, food accessibility cannot fully explain the disproportionate levels of obesity in these populations. Researchers have focused on alternative hypotheses to understand the psychosocial factors that may be at play in the development of obesity among youth from low-income backgrounds. There is a large literature on the relation between increased chronic stress and adult obesity (Block, He, Zaslavsky, Ding, & Ayanian, 2009; Kivimaki et al., 2006), and a growing body of literature on the relation between stress and obesity in childhood (Wilson & Sato, 2014). Research has found that the experience of multiple stressors within the family environment can place children at risk for becoming overweight or obese (e.g. Garasky et al., 2009; Gunderson et al., 2010; Koch et al., 2008). In addition, research on perceived parental stress has found a positive association between parental stress and childhood BMI (Parks et al., 2012; Stenhammar et al., 2010; Zeller et al., 2007). In two cross-sectional studies, children who self-reported higher levels of chronic stress had higher BMIs (Sweeting et al., 2005; Yin et al., 2005). Longitudinal research on school aged children (ages 11-16) found that children reporting higher chronic stress had higher BMIs at baseline than those with low stress, and this difference persisted, but did not increase, over 5 years (van Jaarsveld et al., 2009). The relation between stress and obesity may be one factor influencing the disproportionate rates of obesity among Latino youth in the U.S.

Understanding the mechanisms through which chronic stress leads to becoming overweight in childhood is crucial for developing effective public health initiatives. Chronic stress has been hypothesized to affect body weight maintenance through multiple mechanisms,

as stress has been found to have both direct and indirect pathways to the development of obesity (Wardle, Chida, Gibson, Whitaker, & Steptoe, 2011; Tsigos & Chrousos, 2006). Children who have higher levels of stress have been found to consume more sweets as well as be more likely to partake in "emotional eating" of high calorie and low nutrient foods (Balantekin & Roemmich, 2012; Shimai et al., 2000). One-time stress in the absence of nutrient dense foods has been associated with reduced BMI, though chronic stress has been associated with an increased appetite and intake of high calorie, fat and sugar foods (Torres & Nowson, 2007). It has been hypothesized that stress is associated with obesity only within the context of highly nutrient dense foods, which are easily available for low-income families living in the U.S. (Torres & Nowson, 2007). Mexican-origin youth may experience both environments of chronic stress (as related to immigrant legal status, poverty, and discrimination) as well as environments with highly nutrient dense foods, and therefore may be at a particularly high risk for becoming overweight or obese in childhood.

Stress and the Accumulation of Hair Cortisol

One way chronic stress is thought to affect BMI is through the body's hormonal stress response (Rosmond, 2005). Many researchers have focused on a physiological indicator of accumulated stress on the body's functioning: *allostatic load* (Evans & Kim, 2013). Allostatic load is the measurement of the "wear and tear" of multiple systems in the body including the hypothalamic pituitary adrenal axis (HPA), sympathetic-adrenal-medullary axis (SAM), metabolic functioning and inflammation (Evans & Schamberg, 2009, Goodman et al., 2005). Over time, increased allostatic load has been associated with numerous health problems including higher rates of obesity (Vincennati et al., 2009). Researchers have theorized that

chronic stress directly affects children's biological regulatory systems in a way that causes long-term negative effects (McEwen & Seeman, 1999; Evans & Kim, 2013). The relation between chronic stress in childhood and health in adulthood has led some researchers to hypothesize that early experiences of stress leave a permanent mark on physiological functioning (Cohen, Janicki-Deverts, Chen, & Matthews, 2010).

A main path through which stress affects the body is the hypothalamic–pituitary–adrenal (HPA) axis (Tarullo & Gunnar, 2006). In humans and other mammals, the hypothalamus responds to a stressor by increasing its release of corticotropin releasing hormone (CRH) and arginine vasopressin (AVP) into the pituitary gland. The pituitary gland then produces and releases adrenocorticotropic hormone (ACTH), an activating hormone that stimulates the adrenal gland to produce cortisol (Tarullo & Gunnar, 2006). Cortisol is a steroid hormone that targets brain activity and is a key hormone for fight or flight survival in mammals (Tarullo & Gunnar, 2006). Though the HPA axis is essential for confronting stressors, chronic elevation of the HPA axis can produce deleterious effects (Sapolsky et al., 2000). Specifically, research has suggested that increased levels of cortisol are associated with visceral fat disposition as well as increased appetite (Dalman, Pecoraro & le Fleur, 2005). Chronically stressful environments during childhood, such as those experienced by the population of this study, may affect the body's production and regulation of cortisol (Gunnar & Donzella, 2001; Evans & Kim, 2013).

One way of studying chronic HPA axis activity is through sampling levels of cortisol in hair. Cortisol levels accumulate in hair growth through sebum, blood and sweat from the scalp (Russell, Koren, Rieder, &Van Uum, 2012). This process leads to a sample of accumulated cortisol levels, giving a unique look at the effects of stress on the body over time. As hair grows

at about 1cm per month, hair sampling can provide information about cortisol levels for the past 3-6 months (Vanalest et al., 2012). Cortisol is crucial in the regulation of the body, including metabolism, blood pressure and immune responses (Marieb & Hoehn, 2007). Under stressful situations, cortisol is released in higher doses, and cortisol is modulated when coping with stressful experiences (de Kloet et al, 2005). Those who experience chronic stressors have been found to have dysregulated cortisol expulsion, leading the hormone to build up within the body (McEwen, 2007). Chronic stress has been found to be related to hair cortisol accumulation in both adults and children (Van Uum et al. 2008; Kirschbaum et al. 2009; Thomson et al. 2010; Groeneveld et al., 2013). In a review of previous literature, research using hair cortisol has found a positive relation between hair cortisol levels and health outcomes such as myocardial infarctions, chronic pain levels, hospitalized neonates, chronic stress exposure during pregnancy, spontaneous preterm birth, and Cushing's Syndrome (Gow et al., 2010; Kramer et al., 2009; Pereg et al., 2011; Thompson et al., 2009; Yamada et al., 2007; Van Uum et al., 2008). Though hair sampling is relatively new in assessing cortisol levels in children, the field has moved towards the use this technique because of its low burden and ability to sample build-up over time (Vanalest et al, 2012).

Because childhood obesity is part of a spectrum of cardio-metabolic disorders that have been associated with high levels of cortisol, or hypercortisolism, researchers have studied the effects of increased cortisol levels on obesity. Researchers studying the relation between obesity and saliva, urine and sebum cortisol levels have found mixed results (Veldhorst et al., 2014). Some have hypothesized that these measurements may not be an accurate reflection of the chronic accumulation of cortisol that is hypothesized to be related to metabolic syndrome

(Veldhorst et al., 2014). Researchers have therefore turned to hair cortisol measurement to study the relation between cortisol and obesity (e.g. Jackson, Kirschbaum, & Steptoe, 2017; Wester et al., 2014; Veldhorst et al., 2014). In a study of adults, Stadler and colleagues (2012) found a positive relation between hair cortisol levels and BMI. In addition, another study, Manenschijn and colleagues (2011) found a positive relation between waist circumference and waist-hip ratio and hair cortisol levels among normal-weight adults. Fewer studies have used hair cortisol to study stress and obesity among children, however recent literature in this field has been expanding rapidly (e.g. Michels & Huybrechts, 2016; Vliegenthart et al, 2016; Olstad et al., 2016; Larsen, Fahrenkrug, Olsen, & Heitmann, 2016). One study of 8-12 year olds found that obese children have significantly higher levels of hair cortisol than their normal weight peers (Veldhorst et al., 2014). In this study, the researchers examined hair cortisol levels among 20 obese children and 20 normal weighted peers, and found that obese children had significantly higher levels of cortisol in their hair samples. Another study found that parent's hair cortisol levels significantly predicted children's status of being overweight (Chen et al., 2007). Thus, recent research has begun exploring how the accumulation of hair cortisol affects childhood weight status, however, prospective data with psychosocial reports of stress and family environment have been absent so far and are important in understanding the directionality of the association.

The accumulation of cortisol in the body (as measured by hair cortisol levels) could be an important pathway through which chronic stress affects body weight. With the mounting evidence that chronic stress is associated with higher BMI and that increased hair cortisol levels is related to both high levels of stress and obesity, it is essential to understand the direction of

these relations and how they relate to one another. Though the directionality of the relation

between hair cortisol and obesity has not been fully teased out, researchers hypothesize that,

similarly to processes that have been well studied in Cushing's syndrome, chronic stress impacts

the body and then leads to the increase in fat storage (Michels & Huybrechts, 2016; Manenschijn

et al., 2011; Stalder et al., 2012). This study aims to create greater understanding of the direct

relation between chronic stress and childhood obesity as well as identify a possible mechanism

(hair cortisol accumulation) that underlies this relation.

Parental Warmth

Given increased risk for the development of obesity in the context of chronic stress, it is

important to determine protective factors. One hypothesized protective factor is parental warmth.

Parental warmth (also referred to as responsivity or sensitivity) is a construct reflecting a parent's

general tendency to attend to a child's needs in a supportive and affectionate way (Zhou et al.,

2002). Warm parents also tend to express approval and show direct positive emotions and

behaviors towards their child (Zhou et al., 2002). Parental warmth has been studied as a

protective factor in numerous outcomes associated with highly stressful environments including

depression, educational achievement, and externalizing behaviors (e.g. McLeod, Weisz, &

Wood, 2007; Wagner, Cohen & Brook, 1996; Simpkins, Weiss, McCartney, Kreider, & Dearing,

2006; Deater-Deckard, Ivy, & Petrill, 2006). Traditionally, parental warmth has been studied as

part of the attachment literature, and researchers have hypothesized that high parental warmth

protects children from the effects of stress by encouraging a secure attachment among children

and parents, increasing the child's sense of control over his/her environment and promoting a

sense of security within his/her relation to his/her caregiver (Zhou et al., 2002; Ainsworth, Blehar, Waters, & Wall, 1978; Radke-Yarrow, Zahn-Waxler, & Chapman, 1983; Staub, 1979).

Given that parental warmth buffers the effects of chronic stress for a number of deleterious outcomes, it may also moderate the association between stress and childhood obesity. Though little research has focused on parental warmth as a moderator for this relation, research has examined parental warmth in the context of obesity. Parents have been the target of many interventions on childhood weight management because they often control children's eating behaviors and physical activity. In a study by Agras and collegues (2004), no association between parenting styles and childhood BMI was found. Other studies have found that children with parents with overindulgent (high nurturance and low control) as well as uninvolved (low nurturance and low control) parenting styles were at the highest risk for obesity (Wake et al., 2007). In another study on parenting styles, Rhee and colleagues (2006) found that children with authoritarian parents (strict disciplinarians with low parental warmth) were at the highest risk for becoming overweight. The researchers found that those with authoritarian parents were at a five-fold risk of becoming overweight by first grade as compared to children with authoritative parents (firm but with high warmth and acceptance). In a 2016 study, Rhee and colleagues examined the impact of specific parenting elements as opposed to parenting styles on children's eating behaviors and BMI over time. The researchers coded parenting behaviors during a parent-child dyad interaction though eating a meal. After a 16-week behavioral weight control program, parental warmth was the strongest predictor of reduced or stable weight after the 16-week program. The mixed findings on the relation between parental warmth and childhood BMI may be due to an underlying interaction between stress and parental warmth. In a context of high

chronic stress, children with highly warm parents may feel more secure and have a sense of control over their environment, including their food choices, potentially buffering the effects of stress on childhood BMI. This relation between parental warmth and childhood BMI may not be salient for children from low stress situations.

Though this study hypothesizes that parental warmth will moderate the direct effect between chronic stress and childhood BMI, it is also hypothesized that hair cortisol is the mechanism through which stress affects childhood BMI. Therefore, it is also hypothesized that parental warmth may moderate the indirect pathways between stress and hair cortisol and/or hair cortisol and BMI. Because this model has not been tested before, this study will test all pathways on which parental warmth may influence the relation between stress and childhood BMI.

Recent literature suggests that parental warmth may also buffer the effects of chronic stress on physiological measures of stress. In a retrospective study, Carroll and colleagues (2013) found that individuals who experienced high amounts of chronic stress during childhood had greater physiological indicators of stress as adults, but this association was moderated by parental sensitivity and warmth during childhood. Other studies have found similar results; Chen and colleagues (2011) found that adults from low-income backgrounds with high maternal warmth had fewer biological markers of inflammation than those raised by less warm parents. The researchers studied adults who grew up in low SES environments and found that those who were raised by warmer mothers had less production of interleukin 6, an indicator of systemic inflammation associated a physiological dysregulation (Chen et al., 2011). In addition, Taylor and colleagues (2004) found that children who experience harsh parenting and insecure attachments have higher blood pressure responses to stressful cues than those who had secure

relations. In these cases, parental warmth may be buffering the effects of chronic stress on the body's physiological response (the HPA axis). Likewise, parental warmth moderates the relation between stressful life experiences and the accumulation of cortisol in children, (Hansen & Chen, 2010). Thus, it is possible that the link between chronic stress and accumulation of hair cortisol can be mitigated in a warm parenting context.

Alternatively, parental warmth may also moderate the impact of accumulation of cortisol on obesity. Previous research on parenting practices and childhood obesity point to parental warmth as a key factor, one which may be more influential when a child has accumulated high levels of cortisol. It is possible that even with increased levels of cortisol due to chronic stress, children benefit from highly sensitive caregivers. Highly warm parents may be better equipped to encourage healthy eating and exercise for children who have experienced chronic stress and who are more susceptible to becoming overweight or obese (Golan & Crow, 2004). Children who have chronically elevated levels of cortisol may be at an especially high risk for childhood obesity in the absence of a warm and sensitive family environment. Recent research has found that chaotic home environments exacerbate the effects of cortisol on emotional dysregulation for children (Miller et al., 2017). Forexample, highly sensitive parents of children with hypercortisolism may prevent emotional eating behaviors by promoting more effective coping strategies to manage stressors (Dallman, Pecoraro, & la Fleur, 2005; Davies & Cummings, 1994). This study aims to understand how parental warmth may play a protective role in a highly stressed population, as creating a warm home environment may affect children with high levels of cortisol differently than those with low levels.

Limitations of Previous Research

Though it is clear that health disparities are present along ethnic and socioeconomic lines in the U.S., it is still unclear which psychosocial factors contribute to this inequality. This study aims to clarify the relation between highly stressful environments and childhood obesity. In addition, though there is a growing literature on the link between stressful environments and BMI, more research is needed on the physiological mechanisms involved in this process. This study aims to examine hair cortisol as a possible mechanism for the relation between stressful life experiences and childhood obesity.

Parental warmth may moderate the direct relation between chronic stress and obesity or the key pathways through which stress impacts weight management (i.e., the link between chronic stress and accumulation of cortisol and/or the link between cortisol and obesity). Research on the moderating role of parental warmth has been limited to retrospective accounts of warmth and measurements of cortisol in adulthood. This study aims to understand the relation between parental warmth and accumulation of cortisol prospectively. In addition, many studies have looked at adult physiological health as the outcome for early childhood stress. The present study aims to examine the more proximal effects of highly stressful environments on children's physiological health and wellbeing. Previous research has used singular methods to measure parental warmth including child reported surveys or video observation of meals. The present study will use a multi-method approach by analyzing both parental self-report of warmth as well as observational data from tasks unrelated to eating. Measuring parental warmth outside the context of meals may provide a more generalizable measurement of the parent-child relation.

Finally, this study will examine parental warmth as an overall context rather than simply "overindulgence" to which it has previously been equated.

Specific Aims and Hypotheses

      Specific Aim 1: The first aim of this study is to understand the relation between stress and BMI. There is currently little direct evidence linking childhood chronic stress with childhood obesity (Michels & Huybrechts, 2016; Kitzmann, Dalton & Buscemi, 2008). Though there is indirect evidence of this link, including an association between low SES and increased childhood weight (Vieweg, Johnston, Lanier, Fernandez, & Pandurangi, 2007; Germann, Kirschenbaum, & Rich, 2007; Howe et al., 2011), there remains to be a study on the direct effects of stressful life circumstances on childhood obesity. This study aims to explore a highly stressed population using culturally sensitive measures to understand the full spectrum of life stressors on the child (see Figure 1 for aims and hypotheses).

      Figure 1. Aims and Hypotheses
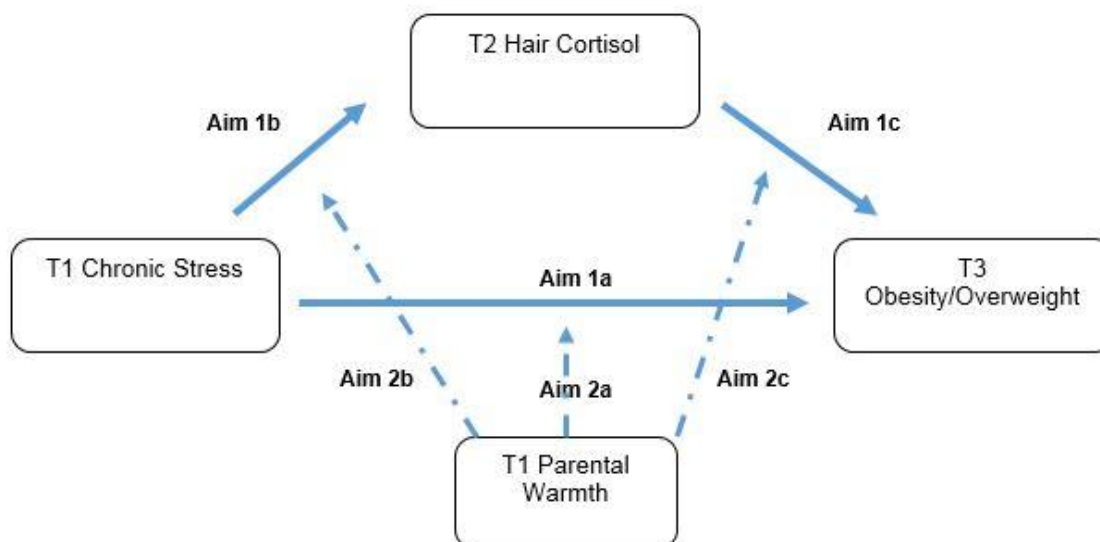
Hypothesis 1a: Children's high levels of chronic stress as reported by their parents using the Hispanic Stress Inventory (HSI) (Cervantes et al., 1991) at time 1 (T1) will be related to higher zBMI scores at time 3 (T3, one year later).

Hypothesis 1b: Higher levels of chronic stress at T1 will be related to high levels of hair cortisol accumulation as measured to reflect cortisol accumulation between 7-10 months later.

Hypothesis 1c: Higher levels of cortisol accumulation in the hair follicle will predict higher zBMI scores at T3.

Hypothesis 1d: Hair cortisol levels will mediate the effect of chronic stress on zBMI. The causal link between children's T1 stress and T3 zBMI will be statistically accounted for by children's elevations in cortisol concentrations in hair samples.

Specific Aim 2: The second aim of this study is to understand how parental warmth impacts this process**.** This study aims to examine parental warmth through both self-report and coded, video-taped interactions. Because parental warmth has been found to have a unique role for children experiencing stress and may impact childhood weight management, this study will examine whether parental warmth will moderate the direct effect of chronic stress on childhood BMI (**Aim 2a**). However, since this study aims to explore a new mechanism through which stress affects childhood BMI, hair cortisol, it is possible that parental warmth attenuates the indirect relations linking stress to BMI. To test this, this study aims to explore whether parental warmth buffers the effects of stress on the body at the biological level, and, therefore, examine whether parental warmth dampens the accumulation of cortisol in the body for children experiencing high chronic stress (Chen et al., 2011; Carroll et al., 2013) (**Aim 2b**). On the other hand, it is possible that parental warmth is interacting with the relation between accumulation of

cortisol and childhood zBMI (**Aim 2c**). Children with high levels of hair cortisol and parents who are sensitive to his/her needs may be less likely to become overweight or obese. This study aims to explore both hypotheses to test which relation is moderated by parental warmth.

Hypothesis 2a: Coded and parent reported parental warmth will buffer the direct relation between chronic stress and zBMI. For children with high chronic stress at T1, low parental warmth will predict high zBMI at T3.

Hypothesis 2b: Coded and parent reported parental warmth will buffer the relation between chronic stress and hair cortisol. For children with high chronic stress at T1, low parental warmth will predict high hair cortisol.

Hypothesis 2c: Coded and parent reported parental warmth will buffer the relation between hair cortisol and zBMI. For children with high levels of hair cortisol, low parental warmth will predict high zBMI at T3.

This research was approved and conducted in compliance with the Loyola University Chicago's Institutional Review Board and the school district's research review committee.

Participants

Mexican-origin immigrant families with at least one child between the ages of 6 and 10 years were recruited for this study. Eligible families had at least one Mexican-origin immigrant parent and one child between 6-10 years of age. Only one parent was required to be a Mexican-origin immigrant for the family to participate, but both parents were encouraged to participate in the study. Grandparents or aunts/uncles living in the home and providing care to the child were also included as secondary caregivers if no father was present in the home. Single parent families were also eligible to participate in the study. Children were both native and foreign-born. Family income was at or below 150% of the federal poverty line at the time of consent. Out of 162 interested participants, 58 did not meet eligibility criteria, were no longer interested, or were not able to be contacted or scheduled, yielding a final sample of 104 families. Of these, 60 participants have valid hair cortisol measurements. Please see measures section for more information on sample limitations.

Procedures

Data collection consisted of three, three-hour home visits, spaced six months apart. After initial eligibility screening, the family was scheduled for a home visit to complete audio- and video-recorded observational interaction tasks, parent questionnaires, and child questionnaires.

Bilingual research assistants administered the questionnaires verbally to both parents and

children to overcome any language or literacy barriers. Parents completed a parent-report

questionnaire packet, children completed a child-report questionnaire packet with a research

assistant. At the end of the visit, children had their height and weight measured if they had

assented to this and their parents consented. BMI measurement was conducted in the privacy of

the home, and children were given the option to step into a room away from the other family

members with the research assistants if they preferred (some children preferred to stay with their

parent). At T3, children had a small sample of hair collected to analyze for cortisol. Because hair

cortisol is a measure of the accumulation of stress from the previous 3-6 months, this is

discussed as a measure of T2 physiological stress activation.

Interaction task. Families (mother and/or father, youth) completed a set of audio- and

video-taped interaction tasks designed to generate family discussion and interaction regarding a

variety of topics related to the experience of being a Mexican immigrant to the U.S. The

vignettes involved hypothetical problem situations relevant to low-income immigrant families

and appropriate for discussion with young children, in which families were asked to discuss how

they would manage or respond to the situation for 5 minutes/vignette (e.g., The family's TV is

broken and they cannot afford a new one at this time (economic stress); There is a big event

coming up (celebration, birthday) and a close relative in Mexico will miss it (family separation);

The family receives a letter in English from the child's school and the family does not

understand it (acculturation stress); A family member needs to move into the home and there is

limited space (family transition/overcrowded housing). See Measures section for information on coding of this task.

Measures

      Demographics. Parents reported on demographic information, including parent and child age, gender, and race/ethnicity. Parents also provided information about their education, job, and income, which was combined as an index for socioeconomic status (SES; Hollingshead, 1975).

      Child stress. Caregivers completed the Hispanic Stress Inventory (HSI), a culturally informed measure of psychosocial stress among immigrant and non-immigrant Latinos (Cervantes, Padilla, & Salgado de Snyder, 1991). The HSI comprises 73 items organized into 5 subscales: Immigration Stress (e.g., "I felt pressured to learn English."), Family / Culture Stress (e.g., "I had serious arguments with family members."), Parental Stress (e.g., "My children haven't respected my authority as they should."), Marital Stress (e.g., "Both my spouse and I have had to work."), and Occupational / Economic Stress (e.g., "Since I'm Latino, I'm expected to work harder."). For each item, parents indicated whether their family had experienced the particular stressor in the previous three months. They then rated the perceived stressfulness of each endorsed item on a five-point scale (1 = not at all, 2 = somewhat, 3 = moderately, 4 = very, 5 = extremely). Previous versions of the HSI have achieved high levels of internal consistency (alphas ranging from .77 to .91) and adequate test-retest reliability (.61 to .86). This study used the total sum stress score, which includes the total number of stressors and stressfulness ratings of each stressor. Reliability was adequate: Primary caregivers' Cronbach's alphas ranged from

.79 (parental stress) to .86 (immigration stress and secondary caregivers' Crobach's alphas ranged from .66 (parental stress) to .85 (occupational/economic stress).

Child stress was assessed through parent-report on modified items from the Hispanic Stress Inventory (Cervantes et al., 1991). Hispanic Stress Inventory (HSI) was designed to assess the unique stressors related to being a Latino American. The measure includes an immigrant and non-immigrant versions. The items were modified to assess for child's experiences of these stressors. Examples of stressors include "members of the family had problems with immigration papers" and "the family's money problems interfered with school." The total number of stressors checked was used to assess child chronic stress. Reliability was adequate on this measure: Primary caregivers' Cronbach's alpha = .68 and secondary caregivers' =.67.

Parental warmth subjective measure. Subjective report of parental warmth was assessed through parent-report on the warmth subscale of the Child's Report of Parental Behavior Inventory (CRPBI-Parent Report Version; Schludermann & Schludermann, 1998; Schwarz, Barton-Henry, & Pruzinsky, 1985). The CRPBI was developed to assess parental behaviors including warmth and control. The eight items on the warmth scale include items such as: "I smile at my child very often" and "I let my child know that I am proud of the things they do." Parents rate these items on how much the statement is representative of their own experience on a three-point scale (1 = not like, 2 = somewhat like, 3 = like). The inventory has been used in other pediatric weight management studies (Rhee et al., 2016; Stein et al., 2005). This study used the parent self-report version of the questionnaire, which has been shown to be consistent with the child report (Schwarz, Barton-Henry, & Pruzinsky, 1985; Tein, Roosa, &

Michaels, 1994). Reliability was adequate: Primary caregivers' Cronbach's alpha PC = .67 and secondary caregivers' Cronbach's alpha = .75.

Parental warmth objective measure. The parental warmth between primary caregiver and child were coded from the video-recorded interaction task. Coded parental warmth scores for each of the four vignettes were analyzed to assess for overall warmth. Observational data were coded using a global-coding method called the Family Interaction Macro-coding System (FIMS; Holmbeck, Zebracki, Johnson, Belvedere, Hommeyer, 2007). FIMS has established reliability and construct validity with various samples of youth and their families (e.g., Coakley et al., 2002; Kaugars et al., 2011; Greenley, Holmbeck, & Rose, 2006). The parental warmth code is defined in the FIMS coding manual as "signs of positive connection in relation" as seen in verbal and nonverbal behaviors (Holmbeck et al., 2007). Verbal behaviors can include statements reflecting love, care and interests towards other's feelings and nonverbal behaviors can include tone of voice, touching and smiling (Holmbeck et al., 2007). Coders rated primary caregivers on a scale from 1 Not at All ("Subject is not at all caring and is not considerate of the other individual. Never smiles in a caring or loving manner. No real signs of friendliness, may be highly engaged with task (or not) but not with the other person") to 5 Very Often ("Frequently expresses care, love and consideration. Frequently smiles in a way that demonstrates caring and love. Engaged/friendly behaviors toward other person include finishing sentences, making the other person feel good, attended to."). Videos were coded by trained, Spanish-English bilingual research assistants. All research assistants followed the same coding methodology and were trained by the same study coordinator. In addition, videos were double coded to assess for

reliability. Interrater reliability was good: primary caregiver's Cronbach's alpha = .85 and secondary caregivers' Cronbach's alpha = .88.

Body Mass Indices. Children were weighed and measured for their height during an initial home visit (T1), 6 months later (T2) and follow-up visit 1 year later (T3). The following anthropometric measurements were collected at each time point: weight, measured to the nearest 0.1 kg (Seca 770, Hamburg, Germany); and height, measured to the nearest 0.1 cm (e.g. Invicta Stadiometer, Invicta, London, UK) and used to calculate BMI (kg/m2). Anthropometric measurements were used to calculate child zBMI based on CDC growth charts (Kuczmarski et al., 2002). At T1, 11 children declined to participate in the anthropometric measurements, and at T3, 12 children declined to participate or were unable to be reached for follow-up.

Hair cortisol. At the T3 home visit, children ($n = 92$ out of 97 families participating at T3) provided a hair sample to be assayed for cortisol. Given that hair cortisol concentrations reflect the preceding months' accumulation of cortisol, the samples collected at T3 home visits reflect T2 HPA axis activity. All samples were assayed by the Behavioral Immunology and Endocrinology Laboratory at the University of Colorado Denver Anschutz Medical Campus, in collaboration with Mark Laudenslager, Ph.D. Some hair samples ($n = 8$) were problematic, in that the hair was so tangled that the scalp end was undetectable, or too few strands of hair were collected, so they could not be processed. The remaining hair samples ($n = 84$) were processed per best practices.

For long hair samples ($n = 20$), the most proximal 3cm hair segment to the scalp was cut and used to measure the past three months of cortisol accumulation (Russell et al., 2012). Given

that some long hair samples were of insufficient weight for processing ($n = 34$), the most

proximal 6cm hair segment to the scalp was cut in order to include those samples in the assay.

For samples less than 3cm in length ($n = 30$), Dr. Laudenslager's laboratory processed the entire

sample. The length of all hair samples were evaluated as a potential covariate in all analyses.

Each hair sample was placed in a pre-weighed 2 ml cryovial (Wheaton, Millville, NJ,

USA) and washed three times in 100% isopropanol and dried as previously described (D'Anna-

Hernandez, Ross, Natvig, & Laudenslager, 2011). After washing, drying, and re-weighing on a

high sensitivity electronic balance (Mettler Toledo Model MS105, Greifense, Switzerland), the

hair was ground in the same tube using a ball mill (Retsch, Haan, Germany) with a single 3/16

inch (~4.8mm) stainless steel ball bearing. Specially milled aluminum cassettes were designed to

hold three of these cryovials for grinding. The cassettes containing the cryovials were submerged

in liquid nitrogen for approximately 3 minutes before grinding to freeze hair samples rendering

them brittle for easier grinding. Samples were ground for 4-5 minutes. The powdered hair (2-

36mg based on weights after washing) was extracted in the same cryovial in 1000μl HPLC grade

methanol for 24 hours at room temperature on a side-to-side shaker platform. This self-contained

process ensured no loss of hair in the processing by confining initial extraction steps to the same

cryovial. Following the methanol extraction, the cryovial was spun for three minutes in a

centrifuge at 1700g to pellet the hair and the supernatant was removed, placed into a

microcentrifuge tube, and dried under a stream of nitrogen under a fume hood in a drying rack.

The extracts were then reconstituted with assay diluent based on amount of hair and extraction

volume. Cortisol levels were determined using a commercial high sensitivity EIA kit

(Salimetrics LLC, State College, PA, USA) per manufacturer's protocol as described previously (D'Anna-Hernandez et al., 2011). Hair cortisol concentrations are reported in the metric pg/mg (picogram per milligram), indicating the amount of cortisol in picograms per milligram of hair.

To assess the repeatability of results, intra- and inter-assay coefficients of variation (CV) were computed. A pooled control sample of previously ground hair was extracted as above and included on each EIA plate in duplicate for determination of inter-assay coefficients of variation. Inter-assay CV for the control hair pool was 9.2%, and intra-assay CV was 2.8%. Hair samples with weights less than 5mg ($n = 31$ in the present study) have an increased assay error (15-20%). Of note, due to difficulty in processing very low-weight samples, only $n = 9$ samples with this higher assay error are included in the present sample of hair cortisol concentration data.

During hair sample ($n = 84$) processing, some samples ($n = 22$) were found to be of insufficient weight to yield a hair cortisol concentration. Further, some samples ($n = 2$) yielded nondetectible hair cortisol concentrations, resulting in a final sample of assayed samples of $n = 60$. Within this sample of hair cortisol concentrations, some values ($n = 8$) fell below the limit of sensitivity for assay. Thus, all analyses were run first with the full sample of hair cortisol data ($n = 60$), then with a subsample excluding values below the limit of sensitivity for assay ($n = 52$).

Food insecurity. Primary caregivers rated 1-item measure of food insecurity that is utilized in annual USDA food surveys and the NHANES (Keenan, Olson, Hersey, & Parmer, 2001). Caregivers could rate their food as 1"enough of the kinds of food we want to eat", 2 "enough but not always the kinds of food we want to eat" 3 "sometimes not enough to eat" and 4 "often not enough to eat."

Analytic Strategy

        Preliminary data analyses. Prior to hypotheses testing, the psychometric properties of

all measures were evaluated. To reduce the number of potential analyses, Pearson correlation

coefficients were computed to assess associations between parent and child stress. If data was

highly correlated ($r \geq .4$, $p < .05$) composite scores were created. However, if there was not

significant agreement, analyses were conducted separately. In addition, descriptive statistics

were computed for all outcome measures to determine basic distributional properties. Data

transformation and reduction techniques were used when appropriate. Other variables such as

gender, socioeconomic status, and age were considered as control variables. Descriptive analyses

of weight, cortisol levels, and stress levels were conducted using frequency measures and

Pearson's bivariate correlations.

        Analytic plan for Aim 1. Longitudinal hierarchical multiple (HM) regression analyses

were conducted to determine the direct effect of **Aim 1a.** stress at T1 on youth zBMI at T3.

Simple multiple regression analyses were conducted to determine the direct effect of **Aim 1b.**

stress at T1 on accumulation of hair cortisol at T2. Longitudinal hierarchical multiple regression

analyses were conducted to determine the impact of **Aim 1c.** hair cortisol accumulation at T2 on

zBMI at T3. Finally, **Aim 1d.** the relation of chronic stress as mediated by hair cortisol

accumulation was tested using bootstrapping. Such analyses were based on methods outlined by

Cohen & Cohen (1983), Baron and Kenny (1986), Holmbeck (2002), Preacher and Hayes

(2008). Power analysis using G*Power indicated that this study had the power to predict a small

to medium effect for Aim 1a, a medium to large effect for Aim 1b and 1c,and a large effect for Aim 1d.

        Analytic plan for Aim 2. To test the **Aim 2a**. interaction between parental warmth and stress on childhood zBMI, multiple regression moderation analysis were conducted of both the observed and subjective parental warmth. To test the hypothesis for **Aim 2b**. interaction between parental warmth and chronic stress on the accumulation of hair cortisol, multiple regression moderation analysis were conducted of both observed and subjective parental warmth. To test the competing hypothesis **Aim 2c.** that parental warmth buffers the effect of hair cortisol accumulation on zBMI, a multiple regression moderation analysis was conducted to test the interaction both observed and subjective parental warmth on the effect of hair cortisol on zBMI. Such analyses were based on methods outlined by Cohen & Cohen (1983), Baron and Kenny (1986), Holmbeck (2002), Preacher and Hayes (2008). Power analysis using G*Power indicated that this study had the power to predict a medium to large effect for the analyses in Aims 2a., 2b., and 2c.

CHAPTER FOUR

RESULTS

Preliminary Analyses

All independent and dependent variables were tested for skewness. Results indicated that

chronic stress, zBMI, and parental warmth were not highly skewed. Specifically, skewness

values of child stress, parental stress, zBMI, parental self-reported warmth and parental coded

warmth ranged from -1.54 to 2.52. Thus, it was not necessary to transform these variables in

order to conduct analyses. All variables were also tested for kurtosis. Results revealed that the

chronic stress, zBMI, and parental warmth variables were not highly platykurtic or leptokurtic.

Kurtosis values ranged from -.94 to 9.62.

As is typical in hair cortisol research, these data were positively skewed and significantly

deviated from normality ($p < .001$, Kolmogorov-Smirnov test). Data handling procedures were

based on prior research examining hair cortisol concentrations in children (Groeneveld et al.,

2013). First, two children were excluded because of extremely high cortisol values (>3 SDs

above the mean). Then, after $\log_{10}$ transformation, hair cortisol concentrations did not deviate

from normality ($p = .20$).

Descriptive statistics and correlations for variables included in this study's primary

baseline analyses can be found in Table 1. Preliminary analyses included an examination of the

degree of association between parents' self-reported stress and parents' report of child stress in

order to determine if composite variables were appropriate. Primary caregiver and secondary caregiver reports of chronic stress parent were significantly correlated with one another ($r = 0.52$, $p<.001$). Therefore, a composite score was created for parental stress. Although primary caregivers' reports of child stress were significantly correlated with reports of their own stress, ($r = 0.28$, $p = .01$), a composite was not created because this correlation coefficient was not high enough to meet the pre-determined cutoff ($r = 0.40$). Secondary caregivers' self-reported stress and report of child stress were not significantly correlated ($r = .22$, $p = .09$).

Further analyses examined the degree of association between child gender and age with other variables included in the analyses. Child age was significantly correlated to hair cortisol with the low sensitivity samples ($r = -.312$, $p = .02$), marginally correlated with parent stress ($r = -.186$, $p =.063$) and hair cortisol without the low sensitivity samples ($r = -.265$, $p = .063$). Gender was significantly related to hair cortisol with low sensitivity samples ($r = -.395$, p .002) and marginally correlated with hair cortisol without the low sensitivity samples ($r = -.240$, $p = .093$). Thus, all primary analyses and models which included hair cortisol as a dependent variable include both age and gender as covariates.

Children's T3 zBMI scores ranged from -3.01 to 3.12 with a mean of .97. One child was underweight, 44% were normal weight, 17% were overweight (BMI $> 85^{th}$ percentile) and 38% were obese (BMI $> 95^{th}$ percentile). There were no significant differences between boys' and girls' zBMI scores ($M_{males} = 1.17$, $SD = .95$, $M_{females} = .84$, $SD = 1.16$, $t = 1.45$, $p = .15$). Forty-three percent of primary caregivers reported having enough of the kinds of foods they want to eat at home, 44.2% reported having enough food but not always the kinds they want to eat and 8.2%

reported sometimes not having enough food to eat. Food insecurity was significantly related to primary caregiver's report of child stress ($r = .34$, $p = .001$) and to combined self-reported parent stress ($r = .22$, $p = .033$). Hair cortisol levels before log transformations ranged from .53 to 369.60 pg/mg ($SD = 63.44$). After transforming the data for normality, hair cortisol levels ranged from -.27 to 2.10 pg/mg ($SD = .47$). When including low-sensitivity samples, boys had significantly higher hair cortisol levels than girls ($t = 3.22$, $p = .002$). However, when low-sensitivity samples were excluded, there were no significant differences between hair cortisol for boys and girls ($t = 1.72$, $p = .09$).

Table 1. Descriptive Statistics and Correlations for Study Variables

	n	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age	104	8.39	1.33														
2. Gender	104	**	**	0.017	_												
3. Time 1 zBMI	93	1	1.06	-0.05	-0.2	_											
4. Time 3 zBMI	92	0.97	1.08	-0.08	-0.15	0.93**	_										
5. Hair Cortisol with Low Sensitivity Samples	58	0.69	0.47	-0.31*	-0.40*	0.08	0.20	_									
6. Hair Cortisol without Low Sensitivity Samples	50	0.8	0.4	-0.27	-0.24	0.19	0.29*	1.00**	_								
7. PC reported Child Stress	98	1.91	1.89	-0.06	-0.04	0.1	0.02	0.10	-0.01	_							
8. SC Reported Child Stress	69	1.12	1.53	-0.009	-0.05	0.13	0.04	0.037	0.02	0.13	_						
9. Parent Stress	103	106.59	32.14	-0.186	-0.15	0.03	0.01	-0.11	-0.22	0.37**	0.04	_					
10. PC Self-Reported Warmth	103	21.66	1.98	0.029	-0.07	0.05	0.09	0.05	-0.03	-0.07	-0.02	-0.20*	_				
11. PC Observed Warmth	100	3.32	0.53	0.002	0.03	-0.2	-0.19	-0.05	-0.06	-0.05	-0.03	-0.19	0.06	_			
12. SC Self-Reported Warmth	71	20.73	2.55	0.032	0.04	0.08	0.08	0.03	0.12	-0.07	-0.17	-0.1	0.21	-0.15	_		
13. SC Observed Warmth	68	3.04	0.61	-0.036	0.028	-0.29*	-0.29*	-0.06	-0.02	-0.25*	0.01	-0.27*	-0.05	0.66**	-0.12	_	
14. Food Insecurity	98	1.64	0.63	0.03	-0.13	0.05	-0.01	0.13	0.05	0.34**	0.07	0.22*	0.09	-0.09	-0.07	-0.23	

Notes: *p<.05, **p<.01, PC: Primary Caregiver, SC: Secondary Caregiver

Multiple Regressions (Hypothesis 1a, 1b, 1c).

Hierarchical multiple regressions were used to examine the relation between chronic stress at T1, hair cortisol at T2 and child zBMI at T3. Due to the short time period (one year), there was little change between T1 zBMI and T3 zBMI ($r = .93$, $p < .001$), and no predictor variables were significant once T1 zBMI was entered into the analyses. Thus, analyses conducted with T3 zBMI did not control for T1 zBMI. However, analyses were repeated utilizing T3 BMI (controlling for T1 BMI), which is more sensitive to change relative to zBMI. All analyses that included hair cortisol as the dependent variable were conducted both controlling for age and gender and without controlling for these variables. Differences between analyses with and without controlling for age and gender are noted below. Results are summarized in Tables 2-5. These analyses were conducted with both primary and secondary caregivers' reports of child stress and the combined report of primary and secondary caregivers' self-reported stress.

Table 2. HM Regressions Stress predicting T3 Body Mass

| | BMI | | | | | | | | | zBMI | | | | | | | | |
| | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | |
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	0.74 (2.80)	81	0.15	0.11 (0.07)	78	1.65	-0.02 (0.09)	53	-0.27	0.92 (0.39)	91	2.34*	1.03 (0.15)	88	6.84**	0.99 (0.19)	58	5.27***
T1 BMI	0.74 (0.11)	81	22.46***	0.92 (0.04)	78	23.26***	1.02 (0.05)	53	19.87***	-	-	-	-	-	-	-	-	-
Stress	-0.10 (0.13)	81	-0.29	-0.03 (0.20)	78	-1.34	-0.03 (0.03)	53	-0.82	0.00 (0.03)	91	0.13	0.01 (0.06)	88	0.18	0.03 (0.10)	58	0.32

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 3. HM Regressions Stress predicting T2 Hair Cortisol

Hypothesis 1b. HML Regressions Stress predicting T2 Hair Cortisol

| | Hair Cortisol with Low Sensitivity Samples | | | | | | | | | Hair Cortisol without Low Sensitivity Samples | | | | | | | | |
| | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | |
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	1.25 (0.19)	49	5.03***	1.96 (0.40)	54	4.86***	1.37 (0.26)	38	5.36***	1.20 (0.26)	49	4.55***	1.57 (0.40)	46	4.14***	0.85 (0.09)	31	9.05***
Gender	-0.33 (0.11)	49	-2.98**	-0.36 (0.12)	54	-3.00**	-0.43 (0.15)	38	-2.83**	-0.22	49	-2.24	-0.21	46	-1.05	-0.26	31	-1.50
Age	-0.11 (0.04)	49	2.56*	-0.09 (0.04)	54	-2.09*	0.01	38	0.40	-0.34	49	-0.35*	-0.09 (0.04)	46	-2.07***	-0.27	31	-1.43
Stress	0.00 (0.00)	49	-1.26	0.03 (0.03)	54	0.89	0.02 (.046)	38	-0.82	0.00 (0.00)	49	-1.55	-0.01 (0.03)	46	-0.35	0.00 (0.05)	31	0.08

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Hypothesis 1a, that higher levels of chronic stress at T1 would be associated with higher zBMI at T3 was tested using linear multiple regression analyses (see Table 2). Parent chronic stress was not found to be associated with child zBMI. Additionally, neither primary nor secondary caregivers' reports of child chronic stress at T1 were associated with zBMI at T3. Chronic stress was also not associated with T3 BMI though controlling for T1 BMI, age and gender.

Hypothesis 1b, that higher levels of chronic stress at T1 would be associated with higher levels of hair cortisol at T2, was also tested using linear multiple regression analyses (see Table 3). Parent chronic stress was not directly associated with hair cortisol. Additionally, child stress was not found to be associated with hair cortisol levels.

Hypothesis 1c, that higher levels of hair cortisol at T2 would be associated with greater zBMI at T3 was tested using linear multiple regression analyses (see Tables 4 and 5). Children's hair cortisol without the low sensitivity samples significantly predicted T3 zBMI ($\beta = .29$, $p = .04$). Hair cortisol also significantly predicted T3 BMI when controlling for T1 BMI, age and gender ($\beta = .14$, $p = .01$).

Table 4. HM Regressions T2 Hair Cortisol without Low Sensitivity Samples Predicting T3 Body Mass

	BMI				zBMI		
	Coefficient (SE)	df	t-ratio		Coefficient (SE)	df	t-ratio
Intercept	-2.18 (1.30)	43	-1.68	Intercept	0.17 (0.38)	49	0.44
Age	0.05	43	0.91	-	-	-	-
Gender	0.07	43	1.37	-	-	-	-
T1 BMI	1.10	43	17.11*	-	-	-	-
Hair Cortisol	1.76 (0.69)	43	2.56*	Hair Cortisol	0.88 (0.42)	49	2.08*

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 5. HM Regressions T2 Hair Cortisol with Low Sensitivity Samples Predicting T3 Body Mass

	BMI				zBMI		
	Coefficient (SE)	df	t-ratio		Coefficient (SE)	df	t-ratio
Intercept	-2.04 (1.18)	43	-1.42	Intercept	0.57 (0.27)	57	2.12*
Age	0.03	43	0.54	-	-	-	-
Gender	0.02	43	0.34	-	-	-	-
T1 BMI	1.10 (0.06)	43	19.18***	-	-	-	-
Hair Cortisol	1.52 (0.50)	43	3.05**	Hair Cortisol	0.48 (0.32)	57	1.50

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Mediational Bootstrapping (Hypothesis 1d)

Hypothesis 1d, that the relation between chronic stress and zBMI would be mediated by hair cortisol levels was not conducted because of the non-significant relations between chronic stress, hair cortisol, and zBMI.

Multiple Regression Moderational Analyses (Hypotheses 2a, 2b, & 2c)

To test the Aim 2a, that parental warmth would buffer the effects of chronic stress on child zBMI, multiple regression moderation analyses were conducted (see Tables 6-9). There was a significant interaction effect of primary caregivers' self-reported warmth and child chronic stress on child zBMI at T3. Children with higher chronic stress and lower reported parental warmth had the highest zBMIs ($\beta = .22$, $p = .047$), showing that parental warmth buffers the impact of stress on zBMI. Simple slopes for the association between chronic stress and zBMI were tested for low (-1 SD below the mean), moderate (mean), and high (+1 SD above the mean) zBMI. The slope tests revealed a marginal positive association between chronic stress and zBMI for children with the lowest levels of parental warmth ($\beta = -.26$, $b = .14$, $p = .07$). Figure 2 plots the simple slopes for the interaction at T3. The associations between chronic stress and zBMI at other levels of parental warmth were non-significant. Interactions between parent stress and self-reported warmth were non-significant. Additionally, interactions between parent stress and observed warmth were non-significant. Interactions between child stress and observed warmth were also non-significant. Finally, this hypothesis was examined using T3 BMI and controlling for T1 BMI, and all analyses were non-significant.

To test the hypothesis for Aim 2b, that parental warmth would buffer the association between chronic stress and hair cortisol, multiple regression moderation analyses were conducted of both observed and subjective parental warmth (see Tables 10-13). There was a marginal interaction between secondary caregivers' observed warmth during vignette tasks and parental stress on child hair cortisol with the low-sensitivity samples ($\beta = .01$, $p = .06$) and without the

low-sensitivity samples (β = -.01, p = .08). Secondary caregiver observed warmth buffered the impact of stress on hair cortisol (marginal effect). However, this result was no longer marginal when controlling for child age and gender. Analyses were also conducted to assess the interactions between child stress and observed parental warmth, child stress and self-reported parental warmth, and parental stress and self-reported warmth on hair cortisol levels. None of these interactions were significant.

To test the competing hypothesis Aim 2c that parental warmth buffers the effect of hair cortisol accumulation on zBMI, a multiple regression moderation analyses were conducted to test the interaction both observed and subjective parental warmth on the effect of hair cortisol on zBMI (see Tables 14 and 15). The interactions between hair cortisol (both with and without low-sensitivity samples) and self-reported and observed warmth on zBMI were all non-significant.

Table 6. Interaction between Stress and Primary Caregiver Self-Reported Warmth on zBMI

	Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	0.94 (0.11)		8.06***	1.03(0.10)		9.90	1.03 (0.15)		6.60***
PC SR Warmth	0.09 (0.11)		0.80	0.09 (0.10)		0.93	0.05 (0.15)		0.36
Stress	-0.06 (0.12)		-0.50	0.03 (0.10)		0.30	0.03(0.15)		0.15
PC SR Warmth x Stress	-0.17 (0.11)	88	-1.60	-0.22(0.11)	85	-2.02*	0.08(0.16)	55	0.49

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Figure 2. Primary Caregiver Self-Reported Warmth Buffers the Effect of Chronic Stress on Child zBMI at T3

Table 7. Interaction between Stress and Secondary Caregiver Self-Reported Warmth on zBMI

	Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	1.01(0.15)		6.52***	1.13 (0.14)		8.36***	0.99 (0.15)		6.42***
SC SR Warmth	0.15 (0.15)		0.99	0.14 (0.12)		1.08	0.12 (0.15)		0.77
Stress	-0.07 (0.20)		-0.34	-0.02 (0.13)		-0.18	-0.03 (0.17)		-0.16
SC SR Warmth x Stress	0.30 (0.20)	57	1.53	0.06 (0.13)	54	0.46	-0.21 (0.18)	55	-1.17

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 8. Interaction between Stress and Primary Caregiver Observed Warmth on zBMI

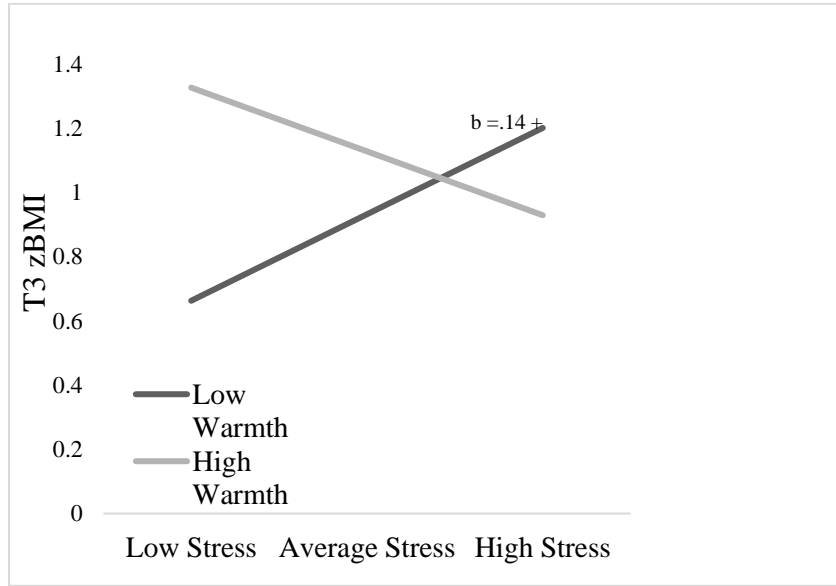|	Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	4.75 (2.43)		1.96+	1.48 (0.97)		1.52	2.58(1.08)		2.38*
PC OB Warmth	-1.16 (0.74)		-1.57	-0.15 (0.29)		-0.52	-0.48 (0.32)		-1.46
Stress	-0.02 (0.02)		-1.09	0.22 (0.30)		0.45	0.49 (0.70)		0.70
PC OB Warmth x Stress	0.01 (0.01)	85	1.09	-0.06 (0.10)	82	-0.68	--0.144 (0.22)	43	-0.65

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 9. Interaction between Stress and Secondary Caregiver Observed Warmth on zBMI

	Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	2.00 (2.57)		0.78	2.63 (0.95)		2.76**	2.19 (0.87)	21	2.51*
SC OB Warmth	-0.00 (0.90)		-0.00	-0.47 (0.30)		-1.57	-0.41 (0.29)	43	-1.41
Stress	0.01 (0.02)		0.35	0.02 (0.32)		0.07	0.45 (0.65)	21	0.70
SC OB Warmth x Stress	-0.01 (0.01)	55	-0.72	-0.02 (0.11)	52	-0.20	-0.14 (0.22)	43	-0.63

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 10. Interaction between Stress and Primary Caregiver Self-Reported Warmth on Hair Cortisol

| | Hair Cortisol with Low Sensitivity Samples | | | | | | | | | Hair Cortisol without Low Sensitivity Samples | | | | | | | | |
| | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | |
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	2.41 (2.188)		1.10	2.11 (0.96)		2.21*	1.65 (1.28)		1.29	4.61(3.87)		1.19	2.34 (0.95)		2.48*	1.64 (1.60)		1.05
Age	-0.11 (0.04)		-2.50*	-0.09 (0.04)		-2.01*	-0.10 (0.06)		-1.72+	-0.10 (0.03)		-2.48*	-0.07(0.04)		-1.73+	-0.06(0.06)		-1.11
Gender	-0.33 (0.11)		-2.88**	-0.35 (0.12)		-2.90*	-0.34 (0.16)		-2.13*	-0.15 (0.11)		-1.34	-0.15(0.12)		-1.27	-0.17(0.16)		-1.05
PC SR Warmth	-0.00 (.10)		-0.01	-0.01 (0.04)		-0.19	0.02 (0.06)		0.35	-0.10 (0.17)		-0.60	-0.03 (0.04)		-0.77	-0.00 (0.07)		-0.01
Stress	-0.00(.02)		-0.14	-.13 (0.44)		-0.29	0.25 (0.54)		0.46	-0.03(0.03)		-0.72	-0.45 (0.42)		-1.08	-0.00 (0.65)		-0.00
PC SR Warmth x Stress	0.00 (0.00)	44	0.00	-0.01(0.04)	49	0.36	-0.01 (0.02)	33	-0.44	0.00 (0.00)	44	0.58	-0.02 (0.04)	41	1.08	0.00 (0.02)	26	0.01

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 11. Interaction between Stress and Secondary Caregiver Self-Reported Warmth on Hair Cortisol

| | Hair Cortisol with Low Sensitivity Samples | | | | | | | | | Hair Cortisol without Low Sensitivity Samples | | | | | | | | |
| | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | |
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	4.40 (2.97)		1.48	2.30 (1.05)		2.20*	1.80 (0.98)		1.84+	4.97 (3.14)		1.57	1.30 (1.07)		1.20	0.76 (1.03)		0.74
Age	-0.11 (0.05)		-2.16*	-0.06 (0.06)		-0.84	-0.10 (0.05)		-1.70	-0.08(0.05)		-1.75	-0.06 (0.07)		-0.88	-0.06 (0.05)		-1.18
Gender	-0.31 (0.15)		-2.04*	-0.44(0.18)		-2.52*	-0.37(0.17)		-2.23*	-0.15(0.14)		-1.06	-0.21(0.18)		-1.13	-0.19(0.16)		-1.17
SC SR Warmth	-0.09 (0.13)		-0.70	-0.02 (0.05)		-0.45	0.01 (0.04)		0.35	-0.14 (0.15)		-0.94	0.02 (0.05)		0.29	0.04 (0.05)		1.00
Stress	-0.02 (0.03)		-0.90	-0.34 (0.42)		-0.80	-0.14 (0.57)		-0.25	-0.03 (0.03)		-1.19	-0.11 (0.42)		-0.25	0.13 (0.59)		0.23
SC SR Warmth x Stress	0.00(0.00)	35	0.81	0.02 (0.02)	32	0.93	0.01 (0.03)	33	0.28	0.00 (0.00)	28	1.06	0.01 (0.02)	25	0.31	-0.01 (0.03)	26	-0.21

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 12. Interaction between Stress and Primary Caregiver Observed Warmth on Hair Cortisol

| | Hair Cortisol with Low Sensitivity Samples | | | | | | | | | Hair Cortisol without Low Sensitivity Samples | | | | | | | | |
| | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | | Parent Combined Stress | | | Primary CG Report of Child Stress | | | Secondary CG Report of Child Stress | | |
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	4.66 (1.25)		3.73***	2.11 (0.73)		2.88**	3.12 (0.74)		4.24***	5.83 (1.92)		3.03**	1.65 (0.76)		2.15*	2.88 (0.81)		3.53**
Age	-0.12(0.04)		-2.84**	-0.09(0.05)		-1.92*	-0.10(0.05)		-1.70	-0.13(0.04)		-3.22**	-0.08(0.05)		-1.78	-0.07(0.05)		-1.26
Gender	-0.34(0.11)		-2.98**	-0.38(0.12)		-3.00**	-0.44(0.16)		-2.70*	-0.17(0.10)		-1.66	-0.19(0.13)		-1.48	-0.28(0.16)		-1.67
PC OB Warmth	-0.61(0.36)		-1.67	-0.03 (0.17)		-0.16	-0.29 (0.16)		-1.75+	-0.90 (0.55)		-1.63	0.05 (0.19)		0.25	-0.32 (0.19)		-1.73+
Stress	-0.02 (01)		-1.71+	0.10(0.18)		0.53	-0.62 (0.32)		-1.95+	-0.03 (0.02)		-1.78+	0.13 (0.18)		0.74	-0.59 (0.35)		-1.70
PC OB Warmth x Stress	0.00 (0.00)	50	1.25	-0.02 (0.06)	47	-0.40	0.21 (0.10)	31	1.99+	0.01 (0.01)	40	1.36	-0.05 (0.06)	39	-0.78	0.20 (0.11)	24	1.71

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 13. Interaction between Stress and Secondary Caregiver Observed Warmth on Hair Cortisol

	Hair Cortisol with Low Sensitivity Samples									Hair Cortisol without Low Sensitivity Samples								
	Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress			Parent Combined Stress			Primary CG Report of Child Stress			Secondary CG Report of Child Stress		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	4.40 (1.22)	21	3.61**	1.84(0.77)	21	2.37*	2.54 (0.65)	21	3.90***	5.79 (1.88)		3.10*	1.27 (0.90)		1.43	1.99 (0.67)	21	2.97*
Age	-0.13(0.05)		-2.40*	-0.09(0.06)		-1.50	-0.10(0.05)		-1.71	-0.12(0.05)		-2.34*	-0.07(0.06)		-1.17	-0.07(0.05)		-1.15
Gender	-0.25(0.14)		-1.65	-0.39(0.16)		-2.30*	-0.36(0.17)		-2.19*	-0.01(0.15)		-0.05	-0.17(0.17)		-1.00	-0.17(0.16)		-1.08
SC OB Warmth	-0.62 (0.41)	40	-1.51	0.05 (0.17)	40	0.30	-0.14 (0.14)	43	-0.98	-1.08 (0.60)		-1.79+	0.14 (0.21)		0.65	-0.12 (0.16)	43	-0.79
Stress	-0.02 (0.01)	21	-1.64	0.16 (0.18)	21	0.88	-0.33 (0.36)	21	-0.94	-0.03 (0.02)		-2.03+	0.16 (0.18)		0.88	-0.49 (0.39)	21	-1.24
SC OB Warmth x Stress	0.00 (0.00)	34	01.19	-0.04 (0.06)	40	-0.62	-0.13 (0.13)	31	0.97	0.01 (0.01)	27	1.63	-0.05 (0.06)	24	-0.72	0.18 (0.14)	24	1.25

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 14. Hair cortisol with the low sensitivity samples interaction with parental warmth on zBMI

	PC Self Report Warmth			SC Self Report Warmth			PC Observed Warmth			SC Observed Warmth		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	-0.90 (0.19)		5.89	0.92 (0.18)		4.91	2.16 (1.63)		1.32	0.92 (0.17)		4.96
Warmth	0.06 (0.07)		0.52	0.08 (0.07)		1.13	-0.52 (0.50)		-1.04	-0.61 (0.40)		-2.14*
Cortisol WS	0.46 (0.33)		1.41	0.54 (0.40)		1.40	-0.50(0.50)		-0.99	0.41(0.38)		-1.05
Warmth X Cortisol LS	-0.05 (0.23)	54	-0.25	-0.11 (0.18)	37	0-.63	0.30 (0.56)	52	-0.27	-0.27 (0.48)	36	-0.55

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Table 15. Hair cortisol without the low sensitivity samples interaction with parental warmth on zBMI

	PC Self Report Warmth			SC Self Report Warmth			PC Observed Warmth			SC Observed Warmth		
	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio	Coefficient (SE)	df	t-ratio
Intercept	-1.18 (4.30)		-0.28	-2.03 (4.66)		-0.44	3.31 (2.50)		1.32	3.04 (1.54)		1.98
Warmth	0.06 (0.20)		0.31	0.09 (0.22)		0.39	-0.99 (0.78)		-1.27	-1.16 (0.55)		-2.09*
Cortisol LS	1.40 (5.40)		0.25	1.97 (5.08)		0.39	-1.73 (2.52)		-0.69	-0.35 (1.65)		-0.21
Warmth X Cortisol LS	-0.02 (0..26)	52	-0.10	-0.03 (0.25)	30	-0.14	0.83 (0.80)	50	1.04	0.63 (0.63)	29	1.00

Notes: +p<.10, *p<.05, **p<.01, ***p<.001

Food Insecurity

Because of the unexpected null finding between stress and zBMI, food insecurity was examined as a possible moderator for the individual pathways of the model. Food insecurity did not moderate the pathway between chronic stress and hair cortisol nor the direct path between chronic stress and zBMI. However, food insecurity significantly moderated the relation between hair cortisol (with low sensitivity samples) and BMI when controlling for age and gender ($\beta =$ 1.38, p = .048). Simple slopes for the association between hair cortisol and BMI were tested for low (-1 SD below the mean), moderate (mean), and high (+1 SD above the mean) food insecurity. The slope tests revealed a significant positive association between hair cortisol and BMI for children with the highest levels of food insecurity (β = 2.06, b= 3.18, p = .02).Though hair cortisol was significantly related to BMI, this relation was exacerbated when children had higher levels of food insecurity. Figure 4 plots simple slopes for this interaction.

Figure 3. Food Insecurity Moderates the Relation between Hair Cortisol and BMI at T3.

b = 3.18
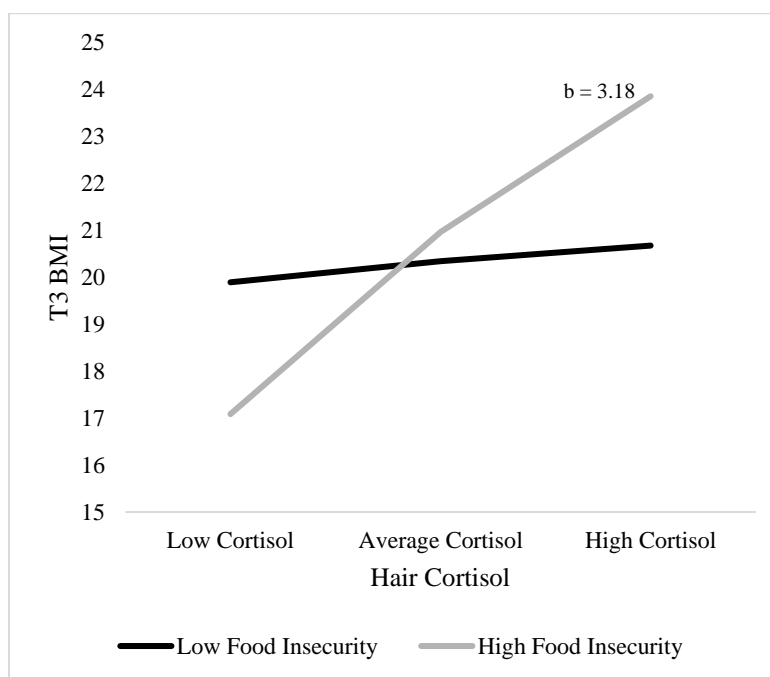
CHAPTER FIVE

DISCUSSION

The present study revealed several findings regarding associations among chronic stress hair cortisol and zBMI among low-income Mexican origin children. Higher levels of hair cortisol at T2 were associated with higher child BMI at T3, even when controlling for T1 BMI. Additionally, primary caregiver's self-reported warmth buffered the association between child chronic stress and zBMI. However, hair cortisol did not mediate the relation between chronic stress and zBMI. These results, as well as implications for clinical application, limitations, and future areas for research, are discussed.

Stress and Hair Cortisol

The current study did not find evidence to support the hypothesis that greater accumulation of psychological stress was related to higher levels of hair cortisol. This finding was unexpected given the number of studies finding a medium to large effect of chronic stress (as measured by several stress questionnaires) and hair cortisol levels among adults (Stalder & Kirschbaum, 2012) and more recent research finding associations among children (e.g. Vliegenthart et al., 2016). However, a number of studies have also found that hair cortisol was not associated with psychosocial measures of stress (e.g. Yamada et al., 2007; Van Uum et al., 2008; Stalder et al., 2010; Stalder et al, 2012). In fact, some studies have found that individuals with diagnosable anxiety and depression have *lower* levels of hair cortisol (Steudte et al., 2011a;

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2011b). Additionally, there is significantly less research on childhood hair cortisol (Stalder & Kirschbaum, 2012). One explanation for the present study's null finding is that several studies have found that childhood hair cortisol levels change over development, with levels lowering as children enter adolescence (Dettenborn et al., 2012; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013). Congruent with this literature, age was negatively correlated with hair cortisol levels among this sample. It is possible that the age range of children, six to ten years old, was too broad to capture the effect. With these mixed findings, it is unclear whether chronic stress, as related to mental health difficulties, is related to hyper or hypocortisolism (Gerber et al., 2011). Some researchers have explained this null association by proposing that hair cortisol provides a qualitatively distinct measurement of stress rather than a synonymous one to that which is measured by questionnaires (Stalder et al., 2012). Finally, since chronic stress was measured exclusively through parent report, it is possible that child chronic psychosocial stress was not completely captured. The present study's findings that psychosocial stress and hair cortisol had distinct relations to zBMI reveal the unique contributions both measurements may play in understanding the development of childhood obesity.

Hair Cortisol and zBMI

This study found a significant positive association between hair cortisol levels and child zBMI. The positive association between zBMI and hair cortisol, though based on concurrent data, indicates a relation between the accumulation of hair cortisol and childhood zBMI. Additionally, hair cortisol was positively related to BMI at T3, even when controlling for age, gender and T1 BMI. These results are consistent with the findings of Veldhorst and colleagues

(2014) in which researchers found that children with higher zBMI also had higher levels of hair cortisol. These findings are also consistent with new research that has supported a longitudinal association between hair cortisol and obesity among adults (Jackson, Kirschbaum, & Steptoe, 2017). Researchers retrospectively examined adults with diabetes and hair cortisol levels and found that hair cortisol levels were positively associated with the persistence of obesity over four years (Jackson, Kirschbaum, & Steptoe, 2017). This association contributes to the literature examining the dynamic relation among chronic stress, HPA-axis activity, and obesity. There is strong evidence linking the accumulation of hair cortisol and increased risk of metabolic syndrome and cardiovascular disease (e.g. Stalder et al., 2013; Manenschijn et al., 2013), and the current study's finding suggests that childhood obesity may be part of this spectrum of stress-related diseases. Hair cortisol concentrations may be an important biological marker to examine among pediatric obesity populations; when studying the complex relation between stress and childhood obesity, biological markers such as hair cortisol may provide insight into the accumulation of stress beyond that which can be measured in psychosocial questionnaires. Though research has been mixed on the direct relation between psychosocial stress and obesity (e.g. van Jaarsveld et al., 2009), chronic stress's impact on childhood obesity may be more insidious and better examined using physiological measures such as hair cortisol.

Parental Warmth as a Moderator

Primary caregivers' self-reported warmth moderated the direct relation between child stress and zBMI (Hypothesis 2a).Although chronic stress was not directly related to zBMI, there was an interaction between chronic stress at T1 and zBMI at T3. Simple slopes revealed a

marginal association between chronic stress and zBMI for children with the lowest parental warmth. The effect of chronic stress on zBMI was dependent on level of primary caregiver self-reported warmth. This finding underscores the importance of general parenting behaviors in the development and prevention of childhood obesity. Children experiencing chronic stress may partake in "emotional eating" of high calorie and low nutrient foods (Balantekin & Roemmich, 2012; Shimai et al., 2000; Torres & Nowson, 2007). However, in the presence of a warm primary caregiver, children may increasingly use non-food related coping strategies to deal with daily stressors. Primary caregivers may display sensitive parenting styles in day-to-day interactions, providing warm homes in the face of chronically stressful environments. Though research has been mixed in terms of direct associations between parental warmth and childhood weight management (e.g. Agras et al., 2004; Wake et al., 2007; Rhee et al., 2006), this finding highlights the unique buffering effect primary caregiver warmth may have on children within the context of chronic stress. Interventions targeting childhood weight management among low-income, highly stressed populations, such as Mexican-origin immigrant families, may benefit from targeting overall parental sensitivity using family systems based intervention techniques (e.g. Dalen, Brody, Staples & Sedillo, 2015).

Secondary caregivers' observed warmth was negatively associated with child zBMI and was also negatively related to parent stress. In this study, a majority of secondary caregivers were fathers. Thus, this finding highlights the importance of fathers in weight management of low-income Mexican-origin children. Previous research on parenting styles and childhood weight management, particularly among Mexican-origin immigrant children, has mainly focused

on mothers (e.g. Olvera & Power, 2010; Arredondo et al., 2006; Melgar-Quiñonez & Kaiser, 2004). Fathers play a unique role in Mexican-origin families: though fathers may not play as much of a role in the daily management of chronic stressors (e.g. lack of resources and cultural conflicts), their overall warmth, as observed in brief family interactions, may impact their children's overall sense of security. Mexican-origin families adhere to traditional gender roles for parenting than their European American counterparts (Crockett, Brown, Russell & Shen, 2007), and mothers may be more likely to prepare daily meals and schedule exercise time. However, the results from the present study indicate that paternal warmth may be directly linked to a reduced risk of childhood obesity. Stress also negatively impacted secondary caregivers' observed warmth. Though parental sensitivity may be protective of obesity among low income Mexican-origin children, chronic stress may negatively impact parents' ability to attend to children sensitively. As hypothesized in the ecological model of childhood weight management, parenting behaviors do not occur in isolation (Davison & Birch, 2001); highly stressful environments may make it difficult for low-income Mexican origin fathers to attend sensitively to their children's needs. Future research should examine the role of fathers in reducing the impact of chronic stress in low-income Mexican-origin families.

The current study revealed interesting methodological findings about measuring parental warmth with primary and secondary caregivers in this population. Secondary caregivers' observed warmth was directly associated with lower zBMI, however, their self-reported warmth was not. On the other hand, primary caregiver's self-reported warmth moderated the relation between stress and zBMI though their observed warmth did not. This unexpected finding

highlights how measurement of parental warmth may vary by caregiver. Parental warmth among Mexican-origin primary caregivers, who in this study were majority mothers, may not be measured as accurately using observational methods. Researchers have found that Mexican American teenagers perceived their mothers and fathers to be warm regardless of the content of their conversations, indicating that parents' instrumental support (e.g. providing financially, maintaining a clean and organized home) were examples of "warmth and loving" (Crockett, Brown, Russell & Shen, 2007). Visible and verbal affirmations of affection towards the child as measured by observational methods may not accurately reflect the instrumental sensitive parenting in which primary caregivers engage. Maintaining an orderly home, managing responsibilities and providing meals may be more potent signs of maternal warmth to Mexican-origin children experiencing high levels of chronic stress. As such, self-report measures may better capture this type of instrumental parental warmth and support.

The present study found a trend for the interaction between parental warmth and stress on childhood hair cortisol levels (Hypothesis 2b.). Secondary caregivers' observed warmth marginally buffered the relation between chronic stress and hair cortisol levels. Parents who reported the highest stress and were observed to have higher warmth had children with the lowest levels of hair cortisol. However, this finding was no longer marginal when controlling for age and gender. Previous research has supported the buffering effect of parental warmth on the accumulation of cortisol within the body (e.g. Carroll et al., 2013; Chen et al., 2011), however our relatively small sample size may have limited our ability to detect this effect. Future research

utilizing larger sample sizes are necessary for understanding how sensitive parenting may impact the accumulation of hair cortisol.

Results from the present study did not support the hypothesis (2c.) that parental warmth would buffer the relation between hair cortisol and zBMI. Higher levels of hair cortisol were associated with high zBMI regardless of parental warmth. This finding suggests that once stress has accumulated within the body, as measured by hair cortisol levels, parental sensitivity may play less of a role in preventing childhood obesity. Parenting and childhood obesity literature varies widely on the types of parenting styles that are most beneficial to preventing obesity among children (e.g. Agras et al., 2004; Wake et al., 2007; Kitzmann, Dalton & Buscemi, 2008). The finding that hair cortisol is association with childhood zBMI regardless of levels of parental warmth may indicate that other parenting styles may be useful among children with elevated levels of hair cortisol. Future research should examine the impact of alternative parenting styles, such as monitoring and consistency as possible buffers for the relation between hair cortisol levels and childhood weight gain.

Food Insecurity

Food security, the level to which food is considered stable and adequate in a home, is a growing problem in the United States. Somewhat paradoxically, research has found that children with greater food insecurity are more likely to become overweight or obese (Dietz, 1995; Casey et al., 2006). Researchers have hypothesized that this relation is driven by multiple mechanisms including metabolic changes (increasing the body's ability to store energy in the form of fat) and the high availability of low-cost, high calorie, low nutrient foods to individuals in poverty

(Alaimo, Olson & Frongillo, 2001; Drewnowski & Specter, 2004). Food insecurity may be especially influential among low-income minority groups in the development of obesity and other obesity related diseases (Winkleby, Robinson, Sundquist, & Kraemer, 1999; Casey et al., 2006.) As such, food insecurity was examined as a possible moderator of the relations among stress, hair cortisol and zBMI. The current study found that food insecurity moderated the relation between hair cortisol levels and zBMI such that children with the highest hair cortisol levels and the highest food insecurity had the highest zBMI. This finding supports previous research indicating that food insecurity is related to excess adipose among children. However, this finding expands upon previous findings: children with increased levels of hair cortisol due to chronic stress and food insecurity may be more likely to overconsume high calorie and low nutrient foods. For highly stressed low income Mexican-origin children, fearing that there will not be enough food to eat may accelerate the fat-storing biological processes responsible for obesity.

Limitations

Though the results from this study are novel in the use of hair cortisol as a measure of the accumulation of stress, there are several limitations. A major limitation of this study is sample size: due to unforeseen problems with the processing of the hair samples (see hair cortisol subsection in Measures), the full sample was only 58 participants. Therefore, it is possible that we did not detect an association between accumulation of stress and hair cortisol due to limited power. Additionally, as noted above, all measurements of stress were reported by parents. It is possible that children's self-report of chronic stress would be a more highly related to

physiological measurements of accumulation of stress. Another limitation was that hair cortisol was only sampled once, making it difficult to determine directionality of the association between zBMI and hair cortisol. Because of the complex system of hormonal responses involved in metabolism allostasis, it is possible that adiposity in fact increases overall levels of hair cortisol within the body (Rodriguez, Epel, White, Standen, Secki & Tomiyama, 2015). Next, recent research has found that stress might be more related to location of adipose on the body, with central adiposity being a higher risk factor for adult illnesses (Rodriguez et al., 2015). In this sample, only zBMI was measured. Additionally, though the examination of school-aged youth provided unique insights into a key developmental period for obesity, the present study did not measure pubertal status. Therefore, it was impossible to control for the onset of puberty in this sample. Food intake was also not measured in this study, limiting the study's ability to form conclusions about consumption of high calorie foods. Finally, because T1 and T3 were only spaced 12 months apart, there was little variability in zBMI scores (which are computed using age and gender norms), and therefore T1 zBMI accounted for almost all the variance in T3 zBMI when added to a regression model. Therefore, BMI was used in all longitudinal analyses, however, zBMI is a preferable measure for children (Law et al., 2014).

Implications and Future Directions

There are both clinical and research implications for the findings of the present study. Though the use of hair samples to detect accumulation of cortisol is relatively new (Russell et al., 2012), the results of the present study suggest that it might be an important tool in understanding how health disparities persist among low-income Mexican-origin youth. Though stress was not

directly related to zBMI among the current sample, hair cortisol was. This finding indicates that hair cortisol might be an important complementary measurement to use in understanding how chronic stress, due to poverty, immigration, discrimination and acculturation, may impact the health development of school-aged Mexican-origin youth. The findings of the present study highlight the impact of highly sensitive parenting on childhood obesity; children with the highest levels of stress and lowest levels of parental warmth had the highest zBMI. Clinical interventions for childhood obesity should focus on supporting overall parental sensitivity, not just management of parenting behaviors during meal times. Though these results affirmed child resilience through sensitive parenting, higher chronic stress was also directly associated with lower secondary caregiver warmth. Though parental warmth buffered the effect of reported stress on childhood obesity, it did not buffer the relation between hair cortisol and zBMI. Furthermore, food insecurity exacerbated the relation between hair cortisol and zBMI. Taken together, these findings underscore the need for economic and social policies that support low-income Mexican-origin families in reducing chronic stress.

Future studies should examine larger samples of hair cortisol assessed over multiple time points. In addition, future studies should include child self-reported chronic stress as well as parent reports. It would be beneficial to examine adipose distribution by measuring waist and hip ratios of children to better understand whether certain types of obesity are more highly associated with accumulation of hair cortisol. Additionally, it would be important to study zBMI over a longer period to understand the long-term effects of hair cortisol on childhood weight gain over time.

Future research should also examine the impact of cultural values on the relation between stress and childhood obesity among low-income Mexican-origin youth. Some research has shown that Mexican American parents are less likely to describe their children accurately as being overweight, and that both mothers and fathers of Mexican origin tend to prefer higher weighted children (Pasch et al., 2016). Future research should examine whether parental preference for higher weighted children affects whether parental warmth buffers the impact of stress on childhood obesity. Though the sample in the present study consists of children of Mexican-origin immigrants, families may differ on acculturation to the U.S. Research on generational differences in pediatric obesity rates has found that acculturation to the U.S. is associated with greater amounts of television watching and fast food intake (Gordon-Larsen, Harris, Ward & Popkin, 2003) as well as with more disordered eating behaviors (Chamorro & Flores-Ortiz, 2000). The relation of stress and obesity may be dependent on level of acculturation to the U.S. Future studies should aim to examine how acculturation may play a role in this health disparity.

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VITA

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