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Relationship Between Responses to Stress, Child Health Behaviors and Obesity: A Systematic Review and Meta-Analysis

A Dissertation Defense

Presented in

Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

By

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June 30th, 2023

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Abstract

INTRODUCTION: Childhood obesity is a risk factor for long term heath consequences such as diabetes, asthma, high cholesterol, and heart disease. However, causes for pediatric obesity are complex and include many variables such as calorie-dense diets, sedentary behavior, and short sleep duration. In addition to these variables, variances in homeostasis, can also impact obesity risk in pediatric populations. One of these variances of interest in the obesity and health literature is stress response. Relationships among these variables are not fully understood. Therefore, the purpose of this study was to systematically review the literature exploring predictive relationships between objective measures of stress response (e.g., cortisol) and obesity/health behaviors (e.g., sleep, diet, physical activity, and sedentary behavior). A meta-analysis was conducted to estimate the magnitude of these relationships, along with an exploratory analysis of moderators associated with these pathways. **METHOD:** Articles were retrieved from three databases based on exclusionary (e.g., eating disorder population, non-experimental studies) and inclusionary (e.g., ages 2-18, written in English) criteria. Articles were assessed via abstract and full-text review. Effect sizes were extracted from articles for analysis. **ANALYSIS:** Effect sizes calculated using Cohen's *d* and were assessed via a multilevel approach to meta-analysis to determine dependency among effect sizes. Three levels of analysis were used to assess the variance of the model including sampling variance of all extracted effect sizes (Level 1), variance between effect sizes within studies (Level 2), and variance between effect sizes among studies (Level 3). Omnibus testing was used to explore moderating effects. Publications were assessed for bias. **RESULTS:** Of 2,488 studies screened, 10 studies met criteria for the systematic review and meta-analysis. Three studies assessed sleep as primary outcome; the remaining 7 studies assessed body mass index (BMI) percentile or z-score as the primary health

outcome. Broadly, qualitative findings indicated that stress responses have variable impacts on sleep and obesity outcomes. These outcomes indicated both over- and under-functioning hypothalamic pituitary axis functioning are associated with higher weight status and poorer sleep outcomes. The overall meta-analysis did not indicate a significant effect of stress responses on health behaviors. A significant small effect was found among sleep-related studies. No significant moderators were found.

CONCLUSIONS: Qualitative literature indicates relationships among stress responses and health behaviors in children and adolescents; quantitative outcomes did not find a significant relationship between stress responses and health behaviors. There was a small significant effect between stress responses and sleep-related studies, and no significant effect among BMI/Obesity related studies. However, more research is needed to better understand directionality and mechanisms associated with these relationships to further strengthen intervention and prevention strategies. Additional research in this area of study may better optimize stress responses in children to improve health outcomes.

Relationship Between Responses to Stress, Child Health Behaviors and Obesity: A Systematic Review and Meta-Analysis

Across the United States, pediatric obesity rates have been rising steadily over the last four decades (Anderson et al., 2019). As of 2017 obesity rates for childhood populations ages 2-19 in the United States are 18.5% (Hales et al., 2017). Higher rates of overweight and obesity in pediatric populations can place children and adolescents at risk for several health and medical complications, including diabetes, asthma, high cholesterol, menstrual abnormalities, and heart disease (Sahoo et al., 2015). The causes of childhood obesity are complex and are often influenced by systemic injustice such as oppression, racism and/or poverty. For example, individuals and families who experience these injustices also often experience inaccessibility to necessary resources such as healthful foods and safe places to exercise, alongside stressors in the environment such as adverse childhood experiences, pollution, gun violence, chaotic households, and neighborhood safety (Cureton, 2011; Dougherty et al., 2020; Elsenburg et al., 2017; Kyler et al., 2021; Schwartz & Brownell, 2007; and Schiff et al., 2021). These stressors are distal factors associated with higher rates of overweight and obesity, which can ultimately lead to more proximal variables impacting weight gain such as consumption of high energy density foods, low levels of activity, sedentary behaviors, and poor sleep hygiene (Brewis, 2014; Schulz & Northridge, 2004).

The current literature on the relationship between stress, obesity, and related health behaviors is complex and findings are equivocal. Further, many studies use varying measures and definitions of stress which complicates the interpretation of the available findings. Therefore, it is important to review the literature on objective measures of stress responses (e.g., cortisol), obesity, and associated health behaviors systematically to better understand these findings and

the magnitude of these relationships across studies. Additionally, identifying moderators of the stress and health behavior relationship is important as additional variables may better explain these relationships and inform future research to mitigate the impact of stress on obesity and obesity-related outcomes. The current study aims to systematically review literature exploring relations between biological responses to stress response and obesity, diet, physical activity, sedentary behaviors, sleep, and detect potential moderators of these relationships. A meta-analysis will also be conducted to estimate the magnitude of these relationships, and to identify moderators that provide additional insight into relationships.

Conceptual Framework

Miller and Lumeng (2018) developed a conceptual model (Figure 1) which outlined the multiple pathways associated with stress, biomarkers, environment, health behaviors and BMI outcomes. This model draws from the extant literature and posits that stress, particularly early life stress exposure, is a distal predictor that impacts child health behaviors and that these behaviors ultimately predict weight status. This model also posits that several factors, including self-regulation, parental factors, and child biological factors such as the hypothalamic-pituitary-adrenal (HPA) axis mediate the relationship between stressors and health behaviors. The current study aims to systematically assess a pathway from Miller and Lumeng (2018) to determine whether the magnitude of the relationship(s) between the stress response, obesity and related-health behaviors differs depending on which variable is being investigated (See Figure 2). Further, we aim to explore possible moderators of the stress/obesity/health behavior relationship to determine if the directionality or magnitude of these relationships changes based on an outside (moderating) variable. The literature review below provides a summary of the extant research related to each pathway in the proposed model starting with proximal predictors (e.g., health

behaviors) of the primary outcome (weight status) and then reviewing the research on the relationship between stress and obesity and stress and health behaviors (e.g., diet, PA and sedentary behaviors and sleep).

Pathways to Weight Status

Dietary Intake, Physical Activity, Sleep, and Relationships to Weight Status

Decreasing consumption of energy-dense foods has been a target research area due to direct relationships between body mass index (BMI) z-scores and calorie consumption. For example, consistent findings over the last decade indicate that consumption of excessive sugar-sweetened beverages (SSB) contributes to weight gain in children and adolescents (Luger et al., 2017; Malik et al., 2013; Marshall et al., 2019), and research indicates that limiting SSB in childhood improves children's health (Bleich & Vercammen, 2018). Intake of foods high in calories, fat, and/or sugar are generally associated with excess weight gain (Dong et al., 2015; Emond et al., 2020) and more consumption of such foods are associated with higher rates of obesity in pediatric populations (Bray & Popkin, 1998; Jones et al., 2021; Luque et al., 2018).

Conversely, high-quality diets are associated with lower weight status in children and adolescents (Varnarelli et al., 2011). The United States Department of Agriculture (USDA) and the United States Department of Health and Human Services (HHS) recommend that children over the age of two and adults consume a balanced intake of fruits, vegetables, whole grains, lean proteins, and healthy fats such as oil and nuts while limiting foods with fat and sugar (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2020). Research supports this notion, such that intaking nutrient-rich food consumption has a positive effect on health, including reducing the risk of diseases and having overweight or obesity (Liberali et al., 2020).

In addition to reducing consumption of high-calorie density foods and increasing nutrient-rich consumption, increasing physical activity, and decreasing sedentary behavior has been found to be an important element of a healthy lifestyle (Elmesmari et al., 2018; Mitchell & Byun, 2014). Literature that suggests long-term engagement in physical activity may be a singular predictor of weight in childhood populations (Biddle et a., 2019; Howie et al., 2020; Kwon et al., 2015). For example, studies have found that youth who are active at young ages but decrease activity during adolescence are more at risk to have obesity than those who stay active (Howie et al., 2020; Kwon et al., 2015). Studies have also found that engagement in exercise improves psychosocial factors including higher levels of self-esteem and decreased depressive episodes (Biddle et al., 2019; Howie et al., 2020). Sedentary behavior also plays a role in long-term trajectories of weight. Specifically, research indicates that more screen time (e.g., watching television, playing video games, etc.) and engagement in sedentary activities at younger ages are associated with an increased risk of a higher BMI z-score (Daly et al., 2017; Hill et al., 2011; Jackson & Cunningham, 2017).

In addition to diet, physical and sedentary activity, sleep is another important factor in childhood BMI. Sleep can be measured in various ways, including sleep efficiency (the percentage of sleep achieved between sleep onset and wake onset), sleep duration, sleep quality (measure of time spent in sleep cycles) and sleep timing (Buysse, 2014). Sleep duration, which measures the length of sleep time, is most widely used in the literature to assess for sleep patterns in children. Broadly, the literature has primarily assessed the relationship between weight status and sleep using sleep duration. Research also indicates that shorter sleep duration promotes decreased physical activity, increased sedentary behavior, alters hormone production, and increases calorie intake (Hart et al., 2011; Magee et al., 2014 Morrissey et al., 2020).

Outcomes and Systemic Factors Related to Weight Status

Taken together, much of the observational research indicates that best practice for reduction in BMI or prevention of having pediatric overweight or obesity is engagement in a combination of health behaviors such as a nutrient-rich dietary intake, high levels of activity, and adequate sleep (Psaltopoulou et al., 2019). There is rich literature available regarding diet, physical activity, sedentary and sleep behavior change studies and their impact on pediatric obesity (Brown et al., 2019; Colquitt et al., 1996; Farooq et al., 2020); however, these studies have mixed outcomes with some reporting weight change and others reporting no changes in weight status. Additionally, studies with successful outcomes may only be beneficial at creating small, short-term health changes or reductions in weight status (Kamath et al., 2008; Mead et al., 2017; Wolfenden et al., 2020; Yoon et al., 2023).

This presents an important contextual dilemma in which a broader depth of research is needed to better understand other factors related to health behaviors, and how they affect BMI status in children, particularly ones that directly influence the most proximal predictors of obesity. Obesity is especially prevalent in children who experience systemic oppression and complex psychosocial stressors. The exact pathways from stress to weight outcomes is less understood in child and adolescent populations than adult populations (Miller & Lumeng, 2018), and even less understood in pediatric populations most at risk for the development of overweight or obesity (Browne et al., 2022; Tester et al., 2020; Valrie et al., 2020; Wang, 2011).

Stress and Obesity

Neuroendocrinology of Stress

Stress is a complex construct that attempts to explain individual's relationships to their environment. Previous conceptualizations of stress have indicated that stress occurs when an

individual appraises their environment to extend beyond their capacity to maintain their well-being, and that individuals perceive stress through cognitive appraisals (Lazarus & Folkman, 1984). However, within youth literature and research, it is important to challenge this conceptualization as cognitive appraisals in infants, children, and adolescents are not as developed as they are in adulthood (Grant et al., 2003). Therefore, more recent literature has defined stress in the context of environmental events or chronic conditions that objectively threaten well-being including psychological and physical health.

Individuals find various ways to manage stress either through adaptive (deep breathing, relaxation) or maladaptive (substance use, disengagement, risk taking) techniques. However, when faced with stress, biological responses to stressors also play a critical role in managing and maintaining homeostasis, which is the body's ability to respond internally to external stimuli (Tarullo & Gunnar, 2006). Specifically, the HPA axis is part of a neurobiological system that coordinates stress responses. During a stressful event, the hypothalamus increases hormone production and releases hormones into the pituitary gland, which is then triggered to produce the adrenocorticotropic hormone (ACTH). ACTH is responsible for promoting the production of cortisol, the body's stress response hormone. Cortisol is important for human survival, and functions as the dominate hormone in "fight or flight" scenarios. During these events, cortisol sends signals to the body to shut down areas of functioning that are unnecessary for survival.

Early and frequent exposure to stressful events and environments can permanently alter the HPA axis functioning and promote neuroendocrine abnormalities (Tyrka et al., 2006), specifically increased cortisol responses (Tarullo & Gunnar, 2006). After regular exposure to high levels of cortisol, the nervous system has a much harder time differentiating between psychologically threatening and nonthreatening events (Van Der Kolk, 2003; Weiss, 2007),

making individuals hyperalert to their surroundings even when it is not necessary. During these times other biological stress responses, such as cardiovascular and respiratory, become elevated and are also indicators of chronic stress outcomes (Condon, 2018).

In addition to increased vigilance, disruptions in HPA axis functioning and cortisol production childhood and adolescences can lend themselves to long-term health consequences into adulthood. Specifically, prolonged stress exposure that exacerbates atypical cortisol production has been linked to weight gain in pediatric populations through metabolic changes, overeating, and sedentary behaviors (Gundersen et al., 2011; Marniemi et al., 2002; Siervo et al., 2009). It is hypothesized that cortisol levels affect leptin production, a hormone involved in regulating the body's ability to measure satiety (Gunderson et al., 2011), which, in turn, increases appetite and dietary intake (Michels et al., 2017; Pervanidou & Chrousos, 2012). Stress can also impact sleep quality, and short sleep duration can also impact hormonal changes associated with dietary intake and intake regulation, such as impacting leptin production and promoting higher cortisol levels (Miller et al., 2015; Raikkonen et al., 2010).

Cortisol and Weight Status

The extant literature documents statistical evidence of various relationships between cortisol and weight status. However, the evidence is equivocal. A systematic review of the literature in an adult population indicates strong relationships among increased cortisol concentrations and obesity (Rodriguez et al., 2015). In pediatric populations, available evidence also suggests a relationship between higher of cortisol concentration with obesity in children. In a sample of 4–5-year-old children, researchers found that average daily cortisol levels were higher in children with overweight (Chu et al., 2017). Another study explored the relationship among hormone profiles before and after a weight loss program in 40 prepubescent children,

with initial baseline levels of cortisol measuring significantly higher in children with overweight or obesity compared to children without overweight or obesity (Geinehr et al., 2013). Researchers found a significant decrease in cortisol production after weight loss. Other research found similar relationships among cortisol and higher weight status (Wirix et al., 2017; Yu et al., 2020).

Despite evidence linking higher levels of cortisol to obesity in childhood, research relationship which indicate lower cortisol levels among children with overweight or obesity status or finds no significance between cortisol and weight status in children. For example, Kjolheded and colleagues (2014) found that cortisol was suppressed in children ages six through twelve with overweight or obesity after collecting salivary samples three times a day for four consecutive days. Another study found differences among sex, with significant findings indicating that girls with overweight, as compared to girls who did not have overweight status, had hypocortisol levels in the morning (Lumeng et al., 2014). A similar trend was found in boys but was not significant. To further implicate inconsistent findings with cortisol and weight status, a study collecting biospecimen throughout the day found no significance between truncal fat mass and a measure of cortisol in children ages six through thirteen years old children with obesity (Barat et al., 2007). Similarly, Hill (2010) found that, across a similar age-range group, no significant correlations or relationships existed among a measure of truncal fat mass at baseline and a collection of morning cortisol sample. This study also indicated that baseline levels of cortisol were not predictive or associated with changes to BMI status longitudinally. Given the vastly different cortisol levels that produce similar weight outcomes, it is important to further understand these mechanisms that may contribute to over- or under-HPA axis functioning.

Challenges of Measuring Physiological Stress Response

It is critical to understand and consider how we measure stress responses. Stress response via cortisol is often measured in the form of saliva, blood, urine, or hair (Levine et al., 2007), and often requires researchers to ask participants to provide multiple samples of the specimen over time. Researchers can also account for stress responses via pulmonary measures such as heart rate and blood pressure. There are various ways in which we can simulate what impacts of cortisol and other stress responses. For example, researchers who want to understand the impact of hormonal responses to stress throughout a typical day would assess for the diurnal cortisol slope or the cortisol awakening response. In this example, researchers are seeking to may collect saliva or blood specimens from their sample to further understanding observational relationships to environmental or sample-specific stress and hormones. Other researchers may want to measure stress responses following a protocol such as the Trier Social Stress Test (Birkett, 2011) or the Maastricht Acute Stress Test (Smeets et al., 2012). The goal of these tests is to strategically induce the human response to stress to measure differences in internal responsiveness. Measured outcomes from these tests may include participants answering questions about perceived stress or providing biological (cortisol) and physiological (heart rate) data. It is important to note that despite similar ways of measuring stress in these studies, outcomes may provide significant differences due to how the participant is experiencing stress.

Despite physiological stress being researched for many years, no singular biological marker or response of stress has been identified. Reasons for this include challenges with differentiating non-stress related arousal and stress-related arousal. For example, excitement and exercising both ignites similar biological pathways to stress responses systems such as increased production of cortisol and higher heart rate (Crosswell & Lockwood, 2020; Dickerson &

Kemeny, 2004). However, research also indicates that these measures are still strong sources of data for measuring stress as they play an important role in pathways to disease outcomes (Crosswell & Lockwood, 2020). Consideration of the types of stress responses that are measured and the population and context in which they are measured in are important to fully understand outcomes.

Stress Responses and Health Behaviors

Currently, the literature available on objective measures of stress responses and its relationship to obesity and health behaviors (dietary intake, PA, sedentary behavior, and sleep) in pediatric populations is limited. Much of the available literature does suggest that objective measures of stress responses may directly impact diet, PA, sedentary behavior, and sleep and obesity while other findings are mixed. Below, the extant literature on the cortisol/health behavior relationship are described. A brief discussion of what is known about potential moderators that may impact this relationship concludes this section.

Objective Measures of Stress Response on Diet

A recent systematic review and meta-analysis assessed the relationship between perceived stress and eating behavior in children ages 8-18 (Hill et al., 2018). Their findings indicated a relationship between stress and eating behavior (either nutrient-dense or calorie-dense), moderated by age. The analysis found that increased stress levels was associated with greater intake of calorie-dense foods in children across all ages. Stress was also associated with lower nutrient-dense food intake in older children, but no associations were found in nutrient-dense food intake and stress in younger children. While this analysis is an important contribution to the literature, the analysis was not limited to stress response measures but rather included subjective and self-report experiences. It is important to further expand on these findings

alongside other psychological and behavioral implications that may relate to stress and eating outcomes. Exploration of this relationship through objective measures of stress responses (e.g., cortisol, other biomarkers) may help us to better understand biological implications that impact long-term health outcomes, specifically eating behaviors.

Stress Responses and Physical Activity Levels

Currently, the associations between stress responses and physical activity are not well understood. Broadly, physical activity can improve HPA axis functioning, and research has shown change in cortisol patterns after physical activity in adults (Hackney, 2008). These relationships have also been found in children and adolescents. For example, positive relationships have been shown between cortisol and increased levels of physical activity in eight-year-old females (DuBose & McKune, 2014). A separate study also found increased cortisol awakening response in a non-clinical sample of children ages nine and ten who were exposed to 10-week exercise training program as compared to a control group (Wegner et al., 2019). Other studies show similar outcomes, in which higher levels of activity increase levels of cortisol (Kertes & Gunnar, 2004).

However, separate studies also found a decrease in cortisol response when regular exercise is established (Nabkasom et al., 2006). Others report no differences in physical activity and cortisol patterns, or that cortisol plays a mediating role when it comes to physical activity (Michels et al., 2015). The variability in outcomes across a multitude of research indicates further need to assess the relationship between cortisol and physical activity, particularly in a systematic and analytic context.

Sedentary behavior is also associated with biological responses and may be related to pathways associated with the proposed model (see Figure 2). Additionally, sedentary behavior is

reinforced as many children and adolescents have increased access to televisions, personal technology devices, and social media. Access to and use of these technologies can also increase perceived and objective levels of stress (Martinez-Gomez et al., 2009). It is important to consider these variables as contributors to objective measures of stress, which lend themselves to sedentary behavior.

Measures of Stress on Sleep

While the literature provides insight into stress and sleep outcomes, sleep outcomes associated with objective measures of stress in children have been under researched. For example, only a few studies have examined the relationship between cortisol and sleep duration. El-Sheikh and colleagues (2008) found that higher cortisol levels were associated with shorter sleep duration and poor sleep quality. Others have shown that shorter sleep duration lends itself to increased cortisol production in the morning (Fernandez-Mendoza et al., 2014; Lemola et al., 2015; Raikkonen et al., 2010). It is important to consider these and other findings associated with HPA axis functioning, along with stress responses and sleep outcomes analytically to fully understand relationships among stress and sleep patterns.

Moderators Associated with Health Behavior Relationship

The relationship between objective measures of stress and weight/health behaviors may be better understood by investigating moderating variables. It is possible that the pathway from cortisol to diet, physical activity and sleep is impacted by other factors (See Figure 1). Research has found that contributing factors to outcomes for these variables in pediatric populations likely include mental health, parental supports, and neuropsychological outcomes (Dockray et al., 2009; Mitchels et al., 2015). For example, stress may impact the severity of depression, which can, in turn, also impact satiety and huger cues (Michels et al., 2012; Reeves et al., 2008).

Additionally, increased stress exposure has been associated with executive functioning deficits (Evans et al., 2021; Williams et al., 2009). Thus, it is important to review moderating relationships in addition to the direct relationship between cortisol and weight/health behaviors to identify what variables may change the strength of these relationships.

The Current Study

Although there is a proliferation of literature on the relationship between physiological stress responses, obesity, and health behaviors in childhood, these relationships have, to our knowledge, never been examined collectively in a systematic or meta-analytic review. Additionally, no systematic review or meta-analysis has been conducted focusing specifically on objective measures of stress and health behaviors. Further, the current study will extend the work of Miller and Lumeng (2018) by measuring the magnitude of the cortisol/health behavior relationships across studies to determine which relationships are the strongest and, therefore, may have implications for the development of intervention and prevention programs. Finally, given some equivocal findings in the literature, it is important to better understand moderators that impact the strength of these stress/health behavior relationships. Therefore, the current systematic review and meta-analysis aimed to: 1) examine the effect of stress responses, as measured by an objective measure such as salivary, blood, or hair cortisol, on obesity and related health behaviors (diet, physical activity, sedentary behavior and sleep) in children and adolescents; 2) explore other potential variables (i.e., executive functioning) that may moderate the relationship between objective measures of stress and health behaviors (diet, physical activity, sedentary behavior, and sleep) in children and adolescents; and 3) determine the magnitude of the effect across studies for these relationships. Hypotheses for our initial aim were that measures indicating typical levels of stress responses (i.e., higher curve of diurnal slope,

average cortisol diurnal, average cortisol awakening responses, etc.) would be associated with positive health behavior outcomes, while stress measures that indicate poorly functioning HPA-axis functioning will be associated with poorer health outcomes. Based on the literature, our hypothesis for the second aim was that the stress response/eating behavior relationship would have the largest effect size as compared to physical activity, sedentary behavior, and sleep duration. Additionally, as an exploratory aim, we attempted to review moderators that impacted the stress/health behavior relationship. It is important to understand these relationships systematically and through a meta-analysis to better serve pediatric populations at highest risk for having overweight or obesity, as stress is a chronic and distal factor that contributes to proximal variables related to weight status.

Methods

Eligibility Criteria

This study aimed to include studies that evaluated relationships among stress responses and obesity/health behaviors. As part of the inclusionary criteria, studies that were identified as relevant included empirical, peer-reviewed studies targeting children and adolescents ages two through 18-years-old. Studies included a measure of obesity or at least one health behavior such as diet, sleep, physical activity, or sedentary behavior. Stress response measures included in studies were objective such as cortisol collection, blood pressure, or heart rate. Studies included were performed in the United States of America, written in English, and published between 2014 and 2022. Exclusionary criteria included adult populations and studies that did not identify stress-related relationships among interested variables (i.e., studies that included heart rate as part of physical fitness assessment or outcomes). Additional exclusion criteria included 1) studies that did not assess objective measure of stress response; 2) studies without obesity

measures or health behavior outcome; 3) studies that did not include a measure(s) of stress response; and 4) eating disorder populations (e.g., binge eating disorder, avoidant restrictive food intake disorder, anorexia nervosa or bulimia). See Table 1 for more details regarding final search strategy.

Search Methods

The study followed the guidelines and checklist materials from the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2021). We searched PubMed, CINAHL, and PsychINFO for eligible data sources. Keywords included are presented in Table 1. Search was limited to empirical and peer-reviewed studies comparing objective measures of stress response and obesity/health behaviors in children ages two through 18. Covidence, a web-based collaboration software program that streamlines literature review, was used to upload all searched articles, remove duplicates, code articles, and extract data and bias information for eligible studies (Covidence Systematic Review Software, 2023). Reviewers referred to the study Code Book (See Appendix) for their assessment of inclusionary, exclusionary, and extraction criteria. Articles were first independently reviewed for eligibility through title and abstract. Two coders assessed each article for eligibility characteristics, with a third coder to settle discrepancies. Following this initial review, full-text review of approved articles occurred with two coders per article and third coder for consensus, along with additional consensus meetings to determine final eligibility when necessary.

Of the 2,488 unique records, 69 studies were assessed for eligibility. Of those, 33 were not U.S. studies, 19 had no objective measure of stress response, three were adult populations, three were wrong study design, and one had no measures of obesity and/or health behaviors.

Ultimately, 10 studies were eligible for extraction in this review. See Table 2 for study selection flow diagram.

Data Extraction and Management

Articles were deemed eligible for extraction. Reviewers were provided a coding guide for data extraction to find relevant criteria for the systematic review and meta-analysis. Data extracted included authors, study design, participant description, outcome statistics and summary of main outcomes. Articles were also assessed for bias through a quality assessment tool via the National Institutes of Health. Details of the bias assessment are provided in Table 3. Extraction and bias assessment also included two reviewers with a third for consensus when necessary.

Outcome Measures and Moderators

For each article, the primary outcome(s) was identified, and effect sizes were calculated (see Statistical Analysis for calculations). Stress responses were considered the predictor variable; health behaviors/obesity were considered the outcome variable. Given the potential nuances for outcomes that may vary based on the health variables, age range was identified as a potential moderator. The ages 10 and under and 11 and over were used as a split for the categorical variable as the Center for Disease Control defines childhood as under 11 and adolescence as over 11 (Center for Disease Control and Prevention). Additionally, it may be important to consider how development may play a role in health-related outcomes, particularly in populations that experience significant stressors (Leech et al., 2014; Suglia, et al., 2018).

Statistical Analyses

Meta-analysis data was analyzed using R version 2023.06.0+421 (R. Development Core Team, 2009) and R package *metafor* (Viechtbauer, 2010) with syntax from Assink & Wibbelink

(2016). Given the opportunity for interdependence of effect sizes within and between studies, a three-level structure was applied to the study analysis to assess for three different variance mechanisms (Cheung, 2014; Hox, 2010; Van den Noortgate et al., 2013). This process allows a statistical advantage to look at with-in study heterogeneity as well as between-study heterogeneity, which traditional univariate meta-analysis does not allow for. The first level assessed for the sampling variance of each effect size collected for all studies. The second level assessed the variance between effect sizes within each study. The third and final level assessed for variance among all effect sizes across all studies. Analyses were conducted using a random effects model to account for potential differential effect of stress response on each study.

Study characteristics including sample size, sample group means, standard deviations, correlations, regression coefficients, and odds ratios were identified within each article. Effect sizes of regression coefficients were initially calculated into Cohen's *d*, Odds Ratios, or Fishers Z_r and, when necessary, transformed into Cohen *d* using a validated web-based calculator (Higgens et al., 2019) to measure the magnitude of outcomes of within and between each study (Cohen, 1988). Before analyzing the effect sizes, all articles, effect sizes, variance, and moderators were combined into one data file to facilitate the *metafor* R package. Articles were assigned unique identifiers. Effect sizes and associated variances were grouped with their parent study and provided unique identifiers. Moderators were transposed into categorical variables and were assigned a dummy variable of either 0 or 1.

The Restricted Maximum Likelihood estimation method (REML) was used to estimate the model parameters. To reduce Type I error, test coefficients were analyzed with the *t* distribution (number of effect sizes – total number of coefficients in the model). Log-likelihood-ratio tests were performed to determine the heterogeneity of effect sizes in level 2 (within-study

variance) and level 3 (between-study variance). The final step of multilevel comparisons was to examine total variance distribution across all levels of the model. Forest plots were created from outcome data to demonstrate heterogeneity and/or homogeneity of study outcomes. Moderator variables were analyzed via omnibus testing based on the *F* distribution. Publications were assessed for bias via the National Institutes of Health Quality Assessment Tool (see Table 5).

Results

Qualitative Synthesis of All Results

Of the 2,488 studies that were initially screened, 10 studies were selected as meeting criteria for the systematic review and meta-analysis. All studies screened were from the United States since 2014. Of the 10 studies, three studies analyzed sleep as their primary health behavior. The remaining seven studies analyzed BMI or BMI z-score and their relationship to stress responses. No studies within our criteria measured physical activity, sedentary behavior, or dietary recall and their relationships to objective stress response measures. All studies that were analyzed showed significant findings in relationships between stress response and health outcomes. Broadly, qualitative findings found relationships among stress responses, obesity, and sleep indicate higher or lower than average cortisol secretion was associated with poor health outcomes (i.e., less sleep and higher weight status). See Table 4 for characteristics for included articles.

Articles were assessed for bias using the National Institutes of Health (NIH) quality assessment tool for observational studies (see Table 5 for detailed questions). Articles indicated mostly low to medium risk of bias across all questions. However, sample size justification, blinded exposure to participant status, and participant retention were moderate to high risk of bias.

Qualitative Characteristics and Results of Sleep Specific Studies

Three studies specifically looked at sleep variables in relationship to stress responses. All studies were cohort longitudinal studies with relatively diverse samples except for Kiel et al. (2015) which consisted of predominately white, well-educated dyads (mothers and children). A separate study sample included adolescent girls in low-income communities across the United States (Rocha et al., 2022). The third study had an economically diverse sample of participants (Trude et al., 2002). Sleep was measured using actigraphy and assessed for various constructs in each study such as sleep onset, wake after sleep onset (WASO), sleep efficiency, and sleep duration (Trude et al., 2022); and sleep duration, latency, and variability (Rocha et al., 2022). Kiel, Hummel, and Luebbe (2015) used a parent-reported measure to predict toddler changes in sleep patterns. Two studies utilized cortisol collection to assess for stress responses (Rocha et al., 2022; Kiel et al., 2015). Rocha and colleagues (2022) collected five samples across three days; Kiel and colleagues (2015) collected three samples throughout the day on two consecutive days at two separate time points.

Broadly, there was a negative relationship among sleep and stress responses, indicating that biological responses to stress such as lower blood pressure or average levels of cortisol as compared to peers showed improvement or better outcomes in various domains of sleep. The study determined sleep trajectories between time one and time three of worsened, irregular, improved, and regular for each sleep variable assessed. Improved sleep duration and sleep efficiency trajectories were associated with a lower diastolic percentile at time three. Rocha (2022) found that a flatter diurnal slope, which is associated with chronic stress (Miller et al., 2002; Young et al., 2019), mediated the relationship between parental education and sleep duration, indicating that high parent education may impact HPA-axis functioning resulting poorer sleep duration. This study also reported a negative correlational relationship between

sleep latency and cortisol awakening response, and sleep latency and diurnal slope, indicating that healthier sleep patterns (i.e., falling asleep after 10-20 minutes) are associated with typical HPA-functioning (Rocha et al., 2022).

In a longitudinal study by Kiel and colleagues (2015), researchers found a predictive relationship of cortisol on sleep, indicating that complex relationship between parenting, toddler stress biology, and sleep. The study showed that children between the ages of two and three showed blunted changes in cortisol secretion throughout the day and into the night when parents were using more critical control as a parenting strategy. As a result, this predicted toddler sleep challenges as perceived by the mother.

Qualitative Characteristics and Results of Weight Specific Studies

Seven studies analyzed stress responses with weight-related variables. Three of these studies included both cross-sectional and longitudinal data (Dai, et al., 2021; Distel et al., 2019; Doom et al., 2020). Two studies included only longitudinal data (Black et al., 2018, Francis et al., 2020; O'Connor et al., 2020) and one study included only cross-sectional data (Lumeng et al., 2014). Most studies had a diverse racial and ethnic sample except for Dai (2021) and Black et al. (2018) who included primarily non-Hispanic white children and adolescents. Age ranges for these studies were approximately six-years-old through 11-years-old. All studies were child participants; none of the studies reported on dyad samples. Most of the studies utilized diurnal or morning awakening cortisol to assess for stress responses (Black et al., 2018, Dai, et al., 2021; Distel et al., 2019; Doom et al., 2020; Francis et al., 2020; and Lumeng et al., 2014). Collectively, these studies indicated various atypical HPA-functioning with higher and lower than average cortisol levels associated with higher weight outcomes.

Four studies (Dai et al, 2021; Doom et al., 2020; Francis et al., 2020; and Lumeng et al., 2014) indicated that blunted or lowered activity among variables such as morning cortisol,

cortisol reactivity, flatter diurnal cortisol slope, and lower sAA slope had associations with health outcomes. Dai and colleagues (2021) found relationships between higher blood pressure and increased body fat and their association with early morning lower diurnal cortisol levels. Findings from Doom and colleagues (2020) also indicated predictive relationships among sAA activity and overweight/obesity, indicating that disruption in sAA activity was predictive of having a higher likelihood of overweight/obesity in middle school. Lumeng and colleagues (2014) showed a relationship pattern among girls who experienced significant home chaos that a flatter diurnal pattern of cortisol predicted a higher likelihood of having overweight status. This pattern, however, was not seen in boys from same study sample. Finally, in another study where negative relationships among BMI and cortisol were found, Francis et al. (2020) indicated that youth in a severe obesity trajectory had lower awakening cortisol than youth in a nonoverweight trajectory. Collectively these studies suggest lowered cortisol activity (as compared to average or high cortisol activity) was associated with poor weight outcomes.

Two studies (Black, et al., 2018; Distel et al., 2019) indicated disrupted HPA-functioning broadly found higher levels of cortisol in relation to health outcomes. Distel and colleagues (2019) reported higher BMI status was associated with higher levels of cortisol found in a hair sample even after controlling for age. Of note, hair cortisol also moderated the relationship between food insecurity and BMI status, indicating the importance of HPA-functioning between environmental stressors and weight outcomes.

Similarly, in a population of children where stressful life events were assessed researchers looked at relationships between and among hormone relationships that are impacted by stress and their relationship to health outcomes (Black 2018). Specifically, the study assessed neuroendocrine coupling, or how well hormones are correlated. The investigators looked at

dehydroepiandrosterone (DHEA), which plays a significant role in neuroprotection when the brain is vulnerable to environmental stressors and demands (Campbell, 2011), and interacts with cortisol to protect brain functioning during times of stress (Dismukes et al., 2016). Findings from this study indicated that children with tighter hormone coupling between cortisol and DHEA had higher BMI status than children with lower BMI status. However, the study did not find differences in outcomes among children who experienced significant life stressors versus those who did not.

Finally, in a longitudinal design study looking at caregiver stress, immune functioning in adolescence and health behaviors, O'Connor and colleagues (2019) assessed for early stress exposures via inflammatory markers such as C-reactive protein (CRP) and glucocorticoid resistance, which can become elevated when exposed to stress (Baumeister et al., 2016; Frodl et al., 2012). This research indicated positive correlational concurrent relationships among obesity and measured CRP. Other findings indicated positive relationships among BMI and interleukin and Tumor Necrosis Factor (TNF)-alpha, both of which regulate inflammatory responses and, when elevated, can lead to cardiovascular disease and decrease the body's ability to respond to stress.

Meta-Analytic Outcomes between Stress Responses and Obesity/Health Behavior Outcomes

This meta-analysis examined the relationship between stress responses and health behaviors. It contained 10 independent studies (*k*) reporting 90 effect sizes (#*ES*) with a total sample of 4,398 participants (See Table 6 for outcomes). Within the overall model, estimates at Level 1did not indicate a significant effect of stress responses on obesity and health behaviors ($d = 0.122$, $p = 0.342$). Additionally, results of heterogeneity among all effect sizes were significant

(Q(df = 99) = 808.848), p <0.001), indicating significant variability among effect sizes at the population level (Level 1).

Given the three-level meta-analytic model used in this study, heterogeneity of the within-study variance (Level 2) and the between-study variance (Level 3) were assessed. Results from two separate log-likelihood-ratio tests, one of which compared model fit of the original three-level model to the fit of a two-level model in which within-study variance was no longer modeled. The second log-likelihood-ratio tests compared fit of the model where Level 2 (within study variance) is freely estimated and the variance at level 3 is fixed at 0. In the overall model, results indicated significant within-study variance at Level 2 and between-study variance at Level 3. This indicates greater variability in effect sizes than in the sampling variance on its own. Determining the collective variance within each of the three levels of the model was also analyzed. Findings from this analysis indicated total model variance attributions of 4.05 percent to within-study sampling variance (Level 1), 32.53 percent to effect sizes within studies (Level 2), and 37.60 percent to effect sizes between studies (Level 3).

Given the difference in health outcomes, the same series of analyses were performed with studies of sleep- and obesity-related outcomes in separate models (See Table 6). Of the three studies found to measure sleep, overall sleep model estimates (Level 1) indicated a small overall effect between stress responses and sleep behaviors (d = -0.091, p = 0.07). Results of heterogeneity among all effect sizes were significant (Q(df = 46) = 402.237, p-val < .001) indicating significant variance at the individual level. We also found that, at level 2 (between study variance) the null hypothesis was rejected indicating that the original fit of the model of three levels is a better statistical fit than the reduced model. This outcome suggests significant variability of effect sizes within studies but no significant variance in outcomes between studies.

Seven studies were found with health outcomes associated with weight status. The overall model results from these studies did not show a significant effect between stress responses and obesity (Q (df = 41) = 823.856, p-val < .001). Both log-likelihood-ratio tests results reported significant within-study variance at Level 2 and between-study variance at Level 3.

Moderator Analyses

Given then significance of the likelihood ratio tests, moderator analyses were performed to the overall model. However, due to varied moderators across each study, age was a common potential moderator variable. Additionally, the samples of each study varied in age of participants. Age was transposed to a dummy variable 0 or 1 for child participants under 10-years-old and over 10-years-old to support the three-level structure of our meta-analytic plan. Findings did not indicate a significant moderating effect ($F(1,87) = 0.005$, $p = 0.945$).

Discussion

Stress plays a critical role in the health development of children, and there is evidence that supports the need to reduce stress in children and adolescents to inform and improve future health outcomes. Related research has examined physiological stress responses and their relationship to outcomes such as mood (Dockray et al., 2009), internalized or externalized behaviors (O'Connor, Gartland, & O'Connor, 2020; Ouellet-Morin et al., 2011; Shirtcliff et al., 2005), parenting stress and/or interactions (Brummelte et al., 2011; Essex et al,, 2002; Seltzer, 2010). Other studies have examined adverse childhood events (ACEs) (Brindle et al., 2022) and direct relationships to cortisol activity. Additional research has assessed broad-based health outcomes with self-report measures and relationships to cortisol (Adam et al., 2017). Broadly,

these studies have found relationships among stress responses indicating the potential to better understand biological functioning as it relates to environmental and social demands.

However, despite the wide range of research that emphasizes relationships among stress and health outcomes (Adam et al., 2017; Brummelte et al., 2011; Dockray, et al., 2009; Essex et al,, 2002; O'Connor, Gartland, & O'Connor, 2020; Ouellet-Morin et al., 2011; Seltzer, 2010; Shirtcliff et al., 2005) very few studies have looked at relationships using biological stress responses measures. To our knowledge, this is the first systematic review and meta-analysis that looks at direct relationships between objective measures of stress responses and health outcomes amongst children and adolescent populations. The lack of studies that use biological stress responses measures in this domain highlights the need to better understand biomechanisms that can re-wire brain functioning which causes long-standing impairments in health decision-making in our youth. By understanding fundamental changes in our biochemistry, we can build a stronger foundation on which to create preventative health intervention programs at schools and in communities, and to inform future policy to address and resolve systemic environmental stressors that negatively impact accessibility to adequate diet, sleep, and exercise.

Qualitatively, findings from our systematic review and meta-analysis suggest relationships between dysregulated HPA-functioning and health behaviors (Black et al., 2017; Dai et al., 2021; Distel et al., 2019; Doom et al., 2020; Francis et al., 2020; Kiel et al., 2015; Lumeng et al., 2016; O'Connor et al., 2019; Rocha et al., 2022; Trude et al., 2022). We were only able to report on sleep and weight related outcomes as dietary intake, sedentary behavior, and physical activity were not found in our review of the literature. Reasons for rule out of this literature included inclusion of only perceived measures of stress and the study performed in another country, or having an adult-only population.

Sleep findings were broadly consistent to other literature assessing general stress and sleep relationships (Bassett et al., 2015; McManimen et al., 2022; Ordway et al., 2021). Collectively, the studies in our review indicated a negative relationship among sleep and stress responses, suggesting that high stress responses were associated with less optimal sleeping patterns. Alternatively, better sleep was associated with more typical HPA functioning such as typical or average cortisol awakening responses and more curvature of the cortisol diurnal slope. We found important relationships associated with environmental stressors that have indirect impacts to health behaviors. For example, the relationship between higher parent education and poor sleep was mediated by a flatter diurnal slope (Rocha et al., 2022), which might indicate that stressors associated with parenting practices and expectations are important indicators of biological functioning. Additionally, outcomes that were associated with better sleep had a lower risk of obesity over time, which indicates that functional biological responses to stress and/or reduction of environmental stressors can have multiple positive impacts on health outcomes. Collectively, we see that typical functionality of cortisol within the HPA-axis serves as a protective mechanism for better sleep quality in children and adolescents, while atypical functioning likely promotes poorer sleep outcomes. Given the limited availability of studies associated with this relationship, more research needs to be completed to better understand this association.

Regarding weight/BMI and stress response relationships, broadly irregular patterns of HPA-axis functioning were found. Specifically, both high and low cortisol outcomes (as compared to peers) were associated with higher BMI and weight status. In a sample of rural, low-income families, O'Connor and colleagues (2020) found a significant relationship between weight outcomes and inflammatory markers, indicating that children who experience significant

stress may present with additional weight gain as early as adolescence. O'Connor et al. (2020) describe this finding as significant as children may experience negative impacts of stress on their health much earlier than adulthood, which is what much of the literature currently suggests. Additionally, like parent-related patterns described in sleep, high parental-reported stress also significantly impacted higher weight status in children and adolescents (O'Connor et al., 2020).

Researchers also found positive relationships among cortisol and weight status in children and adolescents (Black et al., 2018; Distel et al., 2019). In a population of Mexican American youth with low-income status, Distel and colleagues (2019) found striking relationships among food insecurity, high cortisol levels, and BMI status. Despite nonsignificant findings of biological indicators of stress (hair cortisol) and parent-reported chronic stress, hair cortisol was positively associated with BMI status in children. Simple slopes tests also revealed food insecurity and BMI were greatest among the children with the highest levels of hair cortisol; however, this relationship was not significant in children with average or low levels of cortisol. These outcomes also indicate the potential for biomarkers of stress to be a better measure than perceived or self-reported stress tests.

To add to the complexity of these findings, Black and colleagues (2018) found that tighter coupling of DHEA and cortisol was associated with higher BMI, which may ultimately indicate slower metabolic functioning that increases weight status. This is an important finding as it may suggest that decreased access to food due to food insecurity serves as an environmental stressor which ultimately impacts children's metabolism by decreasing fat burning abilities. This is particularly problematic if only high calorie, unhealthful foods are more readily available. Collectively, these findings from these studies suggest a complex relationship between food availability, stress, and weight status, indicating that accessibility to food may have a distal yet

critical impact on weight outcomes (Black et al., 2018; Distel et al., 2019). Additional research should be explored to determine the long-term implications of these variables.

Alternatively, other studies in our review found negative relationships associated with cortisol and weight outcomes (Dai et al., 2021; Doom et al., 2020). In a longitudinal study, Doom and colleagues (2020) found patterns associated with lower levels of stress biology and likelihood of overweight or obesity from preschool into middle school. However, these findings showed predictive characteristics of weight status that lead to disrupted HPA-axis functioning rather than stress predicting weight gain longitudinally. Regardless of the direction of the relationship, the significant findings of these analyses remain critical and relevant to long-term outcomes associated with stress and health as it indicates these variables are tightly associated. In a large one-year longitudinal study, Dai and colleagues (2021) found that lower latent trait cortisol (calculated using a single trait-multistate model of three-day loadings of cortisol waking and 30-mins post waking) was associated with high blood pressure and body fat composition. In a low-income, preschool-aged sample, hypocortisol (lower morning cortisol levels) and higher weight status were significantly associated in young girls (Lumeng, 2014). While not significant, the boys showed a trend in the same relationship. This pattern was interesting given it was the only one of the studies that assessed for sex differences in health-related outcomes. Future studies should assess for sex differences in cortisol patterns given that there may be significant variability in stress response and weight-related outcomes.

Collectively, HPA-axis dysfunction (i.e., atypical levels of cortisol and other elevated biomarkers of stress) was associated with poor weight outcomes. However, the direction of relationship remains unclear, as findings show both higher and lower cortisol responses are associated with increased weight gain. It will be important to understand additional mechanisms

that are associated with these relationships to determine specific factors that contribute to outcomes. For example, more information on food choice, selection, consumption may be necessary to understand the nuances of these associations. Future research in managing weight outcomes in pediatric and childhood populations should have a strong emphasis on stress responses by seeking to add cortisol measurement and stress tests to further understand this relationship. It would also be beneficial for these studies to have a strong understanding of environmental stressors in order to address mechanisms that may better explain nuances associated with these outcomes.

Quantitatively, and to our surprise given the myriad of relationships found amongst our included research studies, our meta-analysis did not find a significant effect on the overall relationship between stress responses and health outcomes. This appears contradictory as there is evidence to suggest this relationship exists (Adam et al., 2017; Karlen et al., 2015; Papafotiou, et al., 2017; Rosemalen, et al., 2005). We also found that there was substantial proportion of overall variance from both the within-study (Level 2) and between-study (Level 3) findings. Qualitative and quantitative findings are complex in that varied physiological responses to stress (i.e., elevated and lower than normal cortisol awakening) are associated with poor health outcomes, which may confound our results. Additionally, the studies in this review included a variety of ways to measure stress responses including diurnal cortisol, cortisol awakening response, inflammation markers, and pulmonary measures. The literature in this area remains equivocal as other studies have also indicated challenges in comparison of various stress response measures (Dines, 2019; Kramer et al., 2012; Seddon et al., 2020). Future research may attempt to further understand how to adequately compare and assess these modalities of measuring stress responses.

Given the nuances of stress response measurements, this variability may contribute to our insignificant findings as it is challenging to determine if the effect sizes among each measurement can be fully compared to one another.

Additionally, it was challenging to determine other domains that may have strengthened our understanding of the relationship between overall stress responses and health behavior outcomes. There was a multitude of mechanisms that were not able to be assessed via moderator analyses that may be unique contributors to our explored relationships such as environmental, financial, access to healthful foods, education level, and parental stress due to insufficient ability to compare these variables across studies. Finally, an additional contributor to non-significant findings may be sample size. Despite the wide number of effect sizes found across studies, eligible studies and individual participant numbers were limited which may contribute to the significant heterogeneity in the variance of our findings. Future research will benefit from more targeted interventions and assessment of moderators to better understand how why this overall relationship was not significant.

Our findings from the sleep model indicate a small effect between stress responses and sleep outcomes. Specifically, our findings showed significant variance within studies but no significant variability between studies. Reasons for this may include within-study sampling error, differences in ages and populations, variability in how studies were conducted. Sleep measures varied from each study. For example, some studies were interested in sleep duration and wake after sleep onset (WASO) with actigraphy data, with a separate study reporting on their toddler's sleep. Variability in this level of the analysis may also be due to small sample sizes. This finding is an important contribution to the literature as future studies should consider the type of sleep

measurements used in the context of stress responses to hopefully establish consistent patterns of findings.

Limitations

While the strength of this study and its contributions to the literature are significant, there were also limitations to our findings. As suggested, this analysis includes a small number of studies with limited sample sizes. Even though several of our studies included longitudinal data with large sample sizes, some of these studies had significantly poor retention rates that contributed to bias and may have impacted outcomes. While our studies included diverse samples, the limited number of studies contributes to lack of broad generalizability to larger populations. None of the studies included were part of a randomized controlled trial with a control sample, which also may further undermine our understanding of the magnitude of these relationships or provide a strong comparison of stress responses and health outcomes. There was also significant variability in terms of control variables across all studies.

Studies from this review focused on singular health behaviors or obesity outcomes. Literature suggests that gold-standard treatment for obesity prevention and intervention strategies that address multiple domains of health is most efficacious. For example, a program might provide better outcomes programs if it targets both increased intake of nutrient dense food and physical activity. Given this information, studies from this review may not be fully assessing the complex relationships between cortisol secretion and health behaviors. An additional confounding factor to the complexity of these relationships is that we were unable to establish a strong basis for moderator analyses outside of age. Given that each study addressed different factors that may or may not have contributed as a moderating variable (i.e., parenting, mental health status, income, education, etc.) it was challenging to compare outcomes in this domain. Other studies have found relationships in these areas (Hoffman et al., 2022; Kidwell et al., 2015;

Quittner et al., 1990). Future studies and should consider increasing the scope of this literature by also measuring various potential mechanisms associated with the stress response and health behavior outcomes such as executive functioning, parenting styles, and even additional health behaviors (i.e., sleep as a moderator for relationship between stress responses and BMI outcomes).

Finally, our findings were limited to the health outcome of obesity and the health behavior of sleep. None of the studies included measures of sedentary and physical activity, and food intake in the context of objective stress responses. Reasons for this were multiple including incorrect population (adults), or studies that were performed in another country. Additionally, because our study only included objective measures of stress responses, we excluded studies that only assessed for perceived stress.

Implications and Future Directions

Our findings emphasize the complexity associated with stress responses and health behavior outcomes, particularly when it comes to weight status. Implications of these findings qualitatively suggest the need for future prevention and interventions youth programs designed to improve sleep and/or decrease risk for obesity to include stress management skills that regulate HPA-axis functioning (Williams et al., 2022). Current studies have begun to address skills that may be most beneficial to HPA-axis regulation including meditation and breathing (Kappes et al., 2023). Kappes (2023) also used these skills within the context of trauma informed care, indicating the need better understand if similar interventions are effective within groups who have experienced significant stressors.

Other interventions that will benefit from this research are ones that assess multiple health behaviors that lead to reduction of weight status while also better understanding stress responses in children. Adult literature has established theoretical models on how physical

activity may improve brain health following experiences of early life adversities (Donofy et al., 2021), but models for children are still under investigation and need more data to better support how these pathways operate. Given the need for better understanding of complex relationships, interventions that assess two or more health behaviors (physical activity, diet, sedentary behavior, and/or weight outcomes), along with measures of stress responses in a population that has experienced significant adversity, may provide rich data to better understand long-term stress response impacts of using multiple health behaviors.

At the systems level, health care providers should consider the role that stress responses play in children and adolescent health outcomes. For example, children who are at increased risk for exposure to stress may present to their healthcare teams in poor health while also managing systemic challenges such as parental stress, poor health literacy, financial challenges, and environmental concerns. Knowing that these are related variables should reinforce the importance of integrated healthcare teams who can address multiple layers of patient care. This includes not only opportunities for psychoeducation around stress and health outcomes but also providing families with access to social workers and therapists to address gaps in services and provision of resources to decrease stress within families and among communities.

Additional implications of this study include creating polices to improve systemic environmental stressors associated with our included study findings, particularly policies addressing economic disparities that impact children and adolescents' abilities to engage in adaptive health behaviors. From this study, we can see a qualitative trend which suggests a level of significance related to stress responses and health outcomes. If children and adolescents are unable to participate in physical activity due to their neighborhood environment or do not have regular access to nutrient-rich foods, we would expect that their environment would be, by

default, a stressful one. This cycle of stress response and poor access to engagement in health behaviors has a significant impact on communities, particularly in communities who have remained in a state of stress for years. Policies from local and federal governments should emphasis opportunities for proximal factors related to decreasing stressors and improving health engagement such as increasing neighborhood safety to promote activity and access to affordable nutrient-rich food. Additionally, distally related policies to health behaviors but may improve stress responses to promote activity and nutrient-rich dietary intake include access to better education systems, affordable healthcare, and greater opportunities for financial security.

Despite broad literature indicating a relationship between these variables, there needs to be more research that better understands mechanisms associated with dysregulated stress responses, particularly in understanding why both over- and under-regulated HPA-axis functioning are associated with poor sleep and weight outcomes in children and adolescents. Studies in our review emphasized variables that contribute to the complexity broad stressors can have on children and adolescent biological functioning and health. Future studies should target a better understanding of mechanisms associated relationships among and between biological stress responses and food insecurity, parent-child relationships, executive functioning skills, parental stress, and sex.

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Figure 1.

Theoretical pathways of association from stress to obesity in early childhood

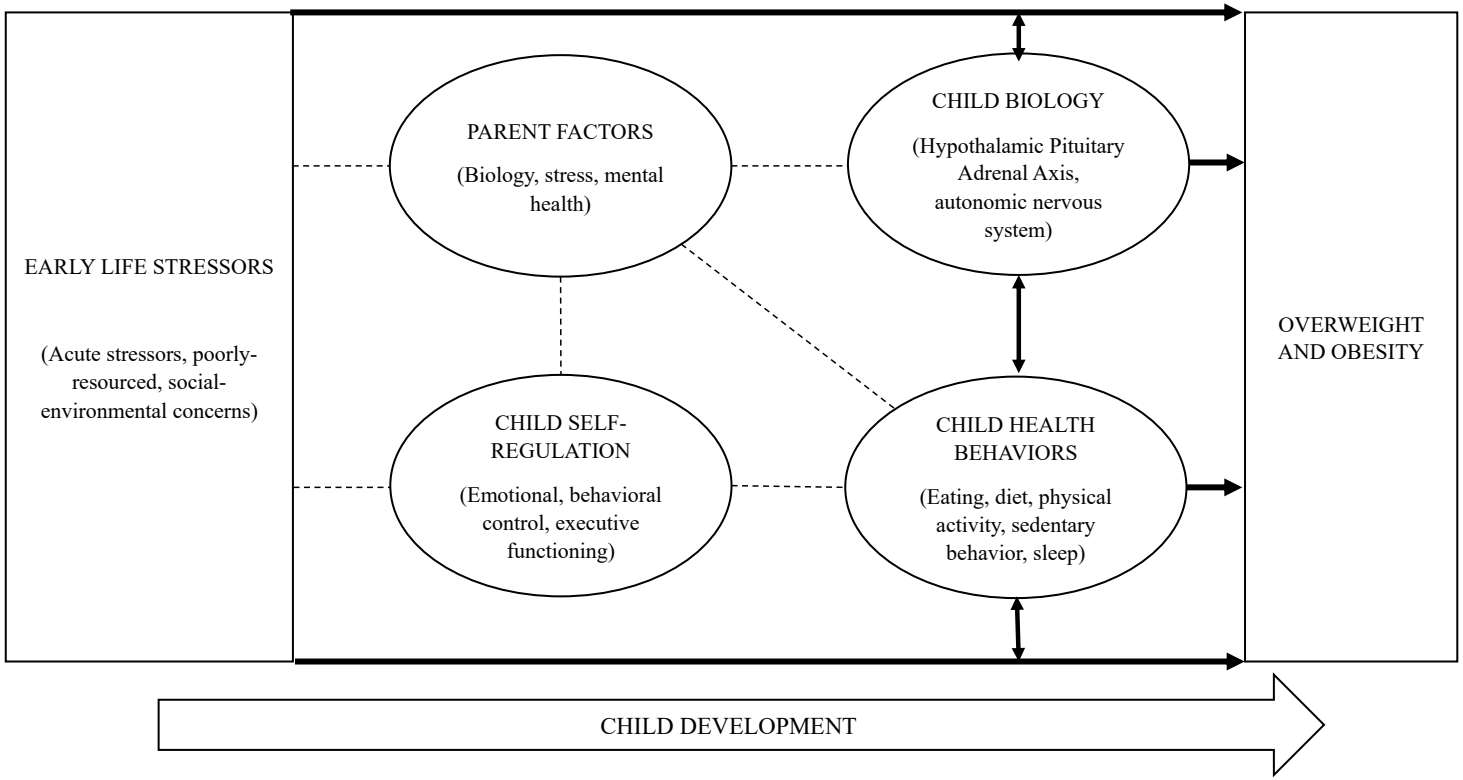
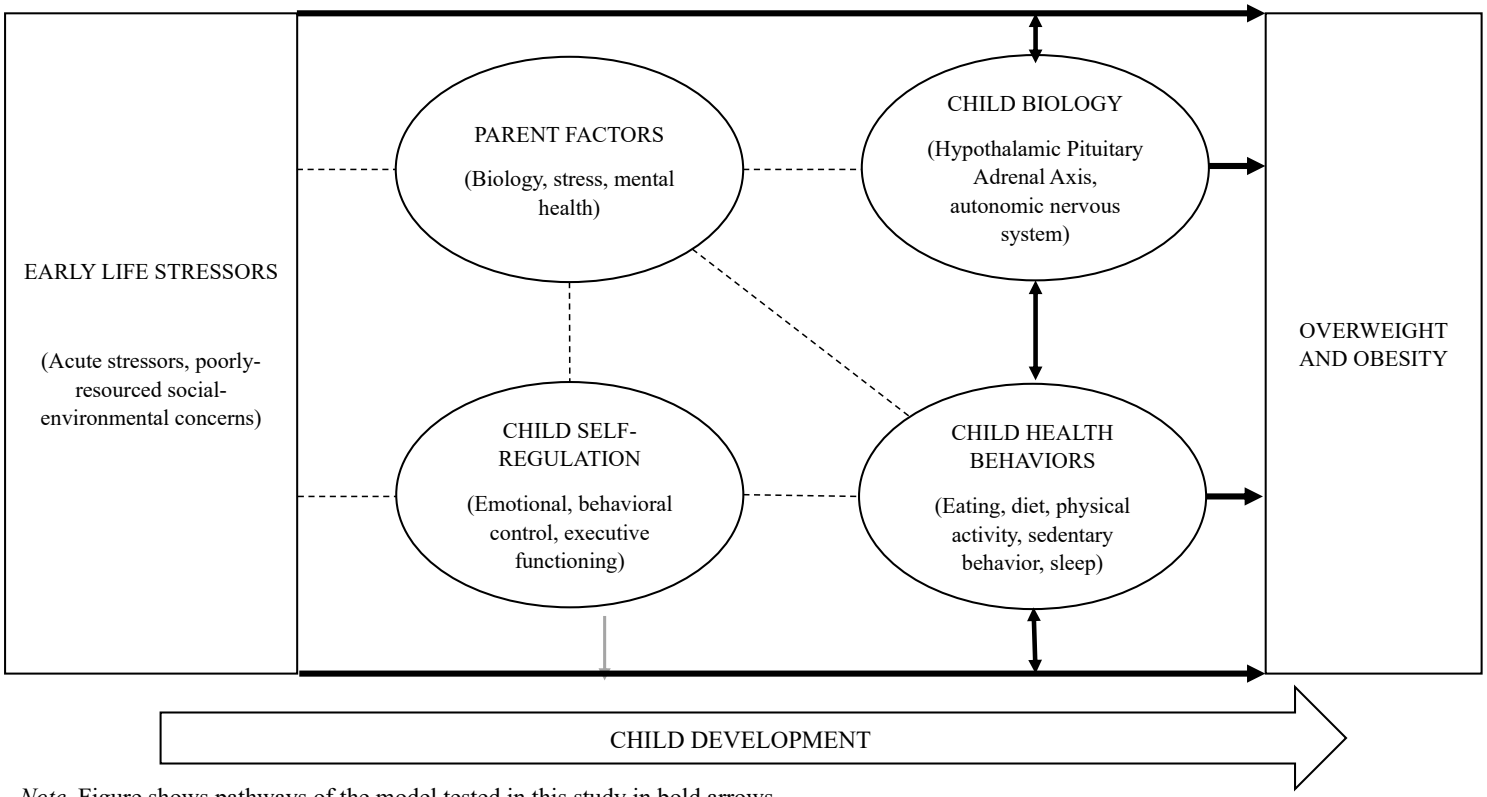


Figure 2.

Hypothesized pathways of association from stress to obesity in early childhood



Note. Figure shows pathways of the model tested in this study in bold arrows.

Table 1.

Search Terms

Category	Words & Phrases
Stress	Cortisol, hair cortisol, urine cortisol, salvia cortisol, blood pressure, heart rate, cardiovascular, pulmonary, hypothalamic pituitary adrenal axis, norepinephrine, epinephrine, adrenaline, HPA axis
Health Behaviors	BMI z-score, BMI, Body mass index, body fat, adiposity, weight, obese, obesity, overweight, waist circumference, anthropoemtrics, junk food, fruits, vegetables, my plate, dietary intake, dietary recall, physical activity, exercise, MVPA, sedentary behavior, sleep, actigraphy, poor sleep, sleep hygiene, sugar, sweets, sugar sweetened beverages
Study Type	Empirical Study, Longitudinal Study, Quantitative Study, Follow-up Study, Prospective Study, Systematic Review, Treatment Outcome, Brain Image, MetaAnalysis, Meta Analysis, Meta-Analysis, Twin Study, Experimental Replication, Field Study, Randomized Controlled Trial, Mixed Methods"
Age[a]	**PsycINFO:** Preschool age (2-5 yrs), school age (6-12 yrs), adolescence (13-17 yrs) **CINAHL:** adolescent: 13-18 years, child: 6-12 years, child, preschool: 2-5 years

[a] age field necessary to select in these databases

Table 2.

PRISMA Study Selection Flow Diagram

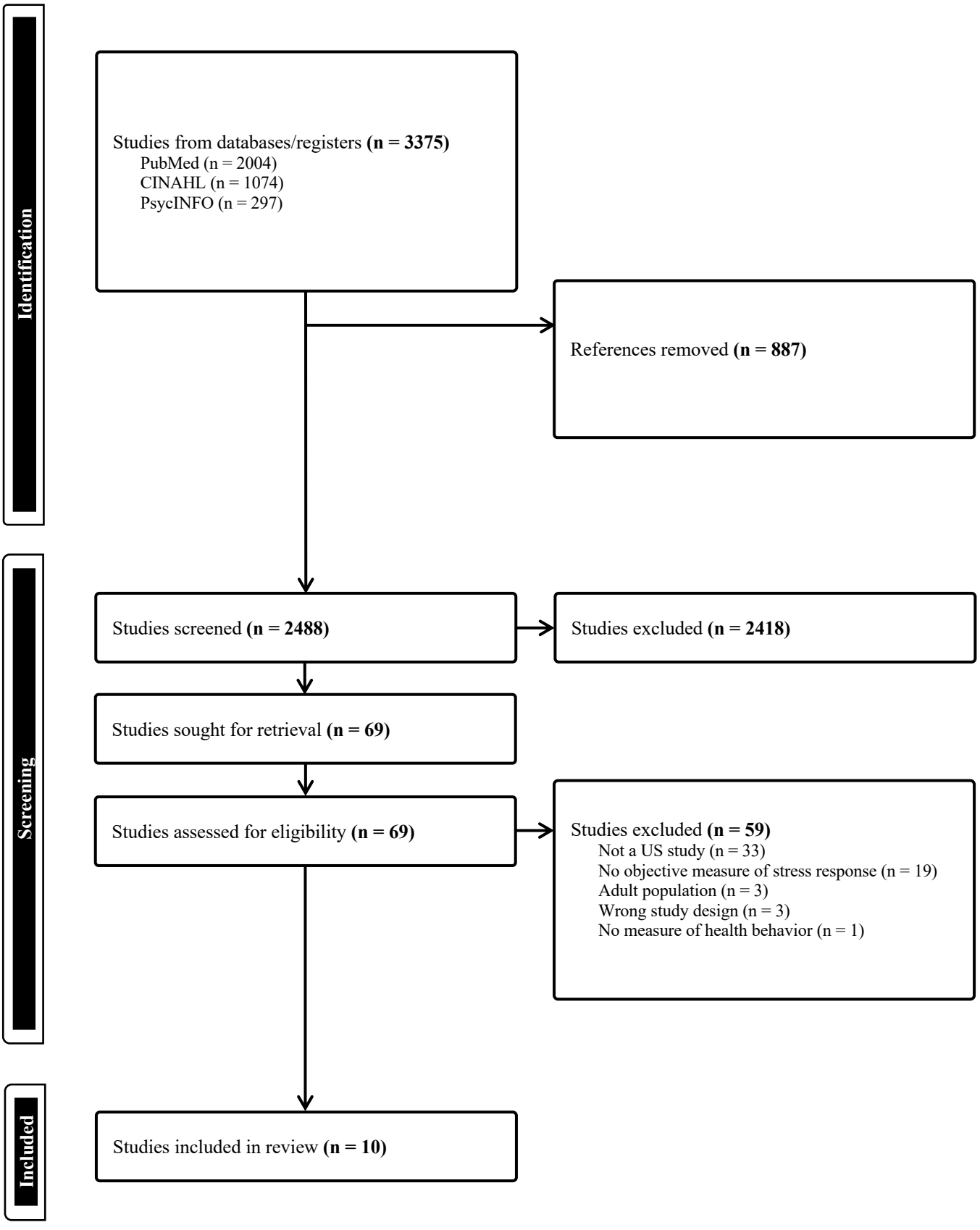


Table 3.

Risk-of-bias assessment for included studies.

Author (year)	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Black (2018)														
Dai (2021))														
Distel (2019)														
Doom (2020)														
Francis (2020)														
Kiel (2015)														
Lumeng (2014)														
O'Connor (2020)														
Rocha (2022)														
Trude (2022)														

Note. Green = low risk of bias, yellow = medium risk of bias, red = high risk of bias.

Risk-of-bias questions corresponding to the column numbers can be found in Table 5.

Table 4.

Systematic Review Characteristics of Included Studies

Author (year)	Sample size Age Sex	Race/ ethnicity	Design	Stress Response(s)	Health Behavior(s)	Main finding(s)
Trude, et al., (2022)	T1 *n* = 464 Age = 12.1 yrs T2 *n* = 348 Age = 12.6 yrs T3 *n* =276 Age = 13.2 yrs	Black/African American = 470 (100%)	Cohort Longitudinal	Systolic/Diastolic Blood Pressure Systolic/Diastolic Percentage	Sleep (duration, latency, variability, wake time) BMI z-score and weight category	In young black girls with predominately low-income background, longer sleep duration and better sleep efficiency were associated with lower risk of overweight/obesity over time. Improved sleep trajectory (duration and efficiency) was associated with a lower diastolic percentile at T3.
Francis et al., (2020)	*n* = 1,077 12 mos – 15 yrs 50% males	Black/African American = 129 (12%) Non-Hispanic White = 876 (82%) Other race/ethnicity = 72 (6%)	Cohort Longitudinal	Awakening Cortisol via Saliva	Sleep (Children's Sleep Habits Questionnaire) BMI, BMI trajectories;	Youth in severe obesity trajectory exhibited lower awakening cortisol at age 15 compared to participants in the non-overweight trajectory indicating HPA axis dysregulation in adolescents with higher BMI status. Youth in the severe and overweight/obesity trajectories had higher levels of disordered eating attitudes as compared to youth in non-overweight trajectories.
Rocha et al., (2022)	T1 *n* = 311 T2 *n* = 223 T3 *n* = 159 Average age across time points: 16.40 yrs 57% female	Asian/Pacific Islander = 77 (22%) Hispanic/Latinx = 147 (42%) Non-Hispanic White = 105 (30%) Other Race/Ethniciy = 21 (6%)	Cohort Longitudinal	Salivary Cortisol	Sleep (duration, latency, variability)	Flatter diurnal cortisol slope was a mediator in association between parental education and sleep duration, potentially indicating that chronic stress associated with higher levels of parental education indirectly affect sleep through dysregulation of the HPA axis. At T1, significant negative correlations among sleep latency and cortisol awakening response, and sleep latency and diurnal slope. Significant positive correlations between sleep variability and diurnal slope.
Kiel et al., (2015)	T1 *n* = 88 Age = 18.96 mos T2 *n* = 46 Age = 24 mos T3 *n* = 38 Age = 36 mos 50.9% female	European American = 42 (82.4%) Latinx = 1 (2%) Biracial, any race = 6 (11.8%)	Cohort Longitudinal	Salivary Cortisol	Infant-Toddler Social and Emotional Assessment	When mothers reported high critical control parenting patterns, children with low variability and high morning values of cortisol predicted increased sleep problems.
Black et al., (2017)	*n* = 405 Mean age = 9.28 yrs 51.3% male	White Non-Hispanic = 363 (89.6%) African American = 31 (7.4%) Asian = 11 (.2%)	Cohort Longitudinal	Salivary Cortisol, DHEA, and Testosterone	BMI	In a population of children who experienced stressful life events, children with higher BMI had tighter hormone coupling (Cortisol-DHEA) than children with lower BMI. DHEA and testosterone levels were higher in children who had overweight/obesity.

O'Connor et al., (2019)	*n* = 337 Mean age = 11.0 44.5% female	Black/African American = 120 (35%) Non-Hispanic White = 217 (65%)	Cross-sectional study	Blood serum sample to assess inflammatory markers	BMI	BMI was significantly and positively associated with c-reactive protein (CRP), an inflammatory marker associated with stress responses. BMI was positively correlated with CRP, interleukin, TNF-alpha, and glucocorticoid resistance.
Distel et al., (2019)	*n* = 52 Average age = 8.39 61% female	Mexican-origin = 100%	Longitudinal & Cross Sectional	Hair cortisol	BMI	Greater BMI at T3 was associated with higher levels of cortisol at T3. When controlling for age, T3 hair cortisol was significantly associated with T3 BMI. When controlling for age, hair cortisol moderated the relationship between food insecurity and T3 BMI. Simple slopes test indicated positive relationship between food insecurity and BMI for children with the highest levels of hair cortisol. Relationship was not significant for children with average or below average levels of hair cortisol.
Doom et al., (2020)	*n* = 257 Average age = 8.0 66.1% male	Non-Hispanic White = 122 (52.9%) African American = 38 (16.7%) Hispanic/Latinx = 23 (10.1%) American Indian = 1 (0.4) Asian/Pacific Islander = 2 (0.8%) Multiracial = 44 (19.1%)	Longitudinal & Cross Sectional	Diurnal cortisol Salivary alpha amylase (sAA)	BMI	Overweight/obesity predicted greater changes in stress biology over time. Overweight/obesity in preschool predicted future (at middle school) lower morning cortisol and sAA levels, blunted cortisol reactivity and lower sAA slope across the day. Blunted sAA reactivity to stress in preschool predicted higher likelihood of overweight/obesity in middle school.
Dai et al., (2021)	*N* = 1336 4th grade *n* = 689 5th grade *n* = 647 4th grade average age = 9.20 years 5th grade average age = 10.53 years 53% female (4th grade) 55% female (5th grade)	4th grade Non-Hispanic White = 613 (89%) 5th grade Non-Hispanic white = 530 (82%)	Longitudinal & Cross-sectional	Salivary cortisol Blood Pressure	BMI Waist-to-hip ratio Percent fat	Lower morning latent trait cortisol (LTC) in 4th grade was predictive of 5th grade blood pressure and higher body fat composition. Lower morning LTC in 4th grade was predictive of higher blood pressure in 5th grade. Negative relationship was found between cortisol and body composition. Lower morning cortisol was associated with increased body fat.
Lumeng et al., 2014	*N* = 331 3- through 4-years-old 49.6% males	Not reported	Cross-Sectional	Salivary Cortisol	BMI Children's Eating Behavior Questionnaire	Hypercortisolism pattern was associated with higher likelihood of having overweight among girls. Boys showed similar patterns but was not significant.

Table 5.

NIH Risk-of-Bias Assessment

1.	Was the research question or objective in this paper clearly stated?
2.	Was the study population clearly specified and defined?
3.	Was the participation rate of eligible persons at least 50%?
4.	Were all the subjects selected or recruited from the same or similar populations (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants
5.	Was a sample size justification, power description, or variance and effect estimates provided?
6.	For the analyses in this paper, were the exposure(s) of interest measured prior to the outcome(s) being measured?
7.	Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?
8.	For exposures that can vary in amount or level, did the study examine different levels of the exposure as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)?
9.	Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?
10.	Was the exposure(s) assessed more than once over time?
11.	Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?
12.	Were the outcome assessors blinded to the exposure status of participants?
13.	Was loss to follow-up after baseline 20% or less?
14.	Were key potential confounding variables measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)?

Table 6.

Meta-Analysis Outcomes

	K	***#ES***	**Mean *d***	**95% CI**	***p***	$\sigma^2_{level\ 2}$	$\sigma^2_{level\ 3}$	**Level 1 Variance**	**Level 2 Variance**	**Level 3 Variance**
Overall Model	10	90	0.127	[−0.132, 0.376]	0.342	0.076***	0.147***	4.18	32.60	37.92
Sleep Model	3	47	-0.091	[-0.190 - .008]	0.07*	0.091***	0.000	11.81	88.19	10.34
BMI/Obesity Model	7	42	0.023	[-0.123 – 0.538]	0.096	0.052***	.195***	3.16	20.45	45.06

Note. ***k*** = number of unique studies; ***#ES*** = number of effect sizes; mean *d* = mean effect size (*d*); CI = confidence interval; $\sigma^2_{level\ 2}$ = variance between effect sizes within the same study; $\sigma^2_{level\ 3}$ = variance between studies.

*p< .1

****p* < .001

Figure 3.

Forest Plot of Stress Response Relationship with BMI/Obesity and Sleep

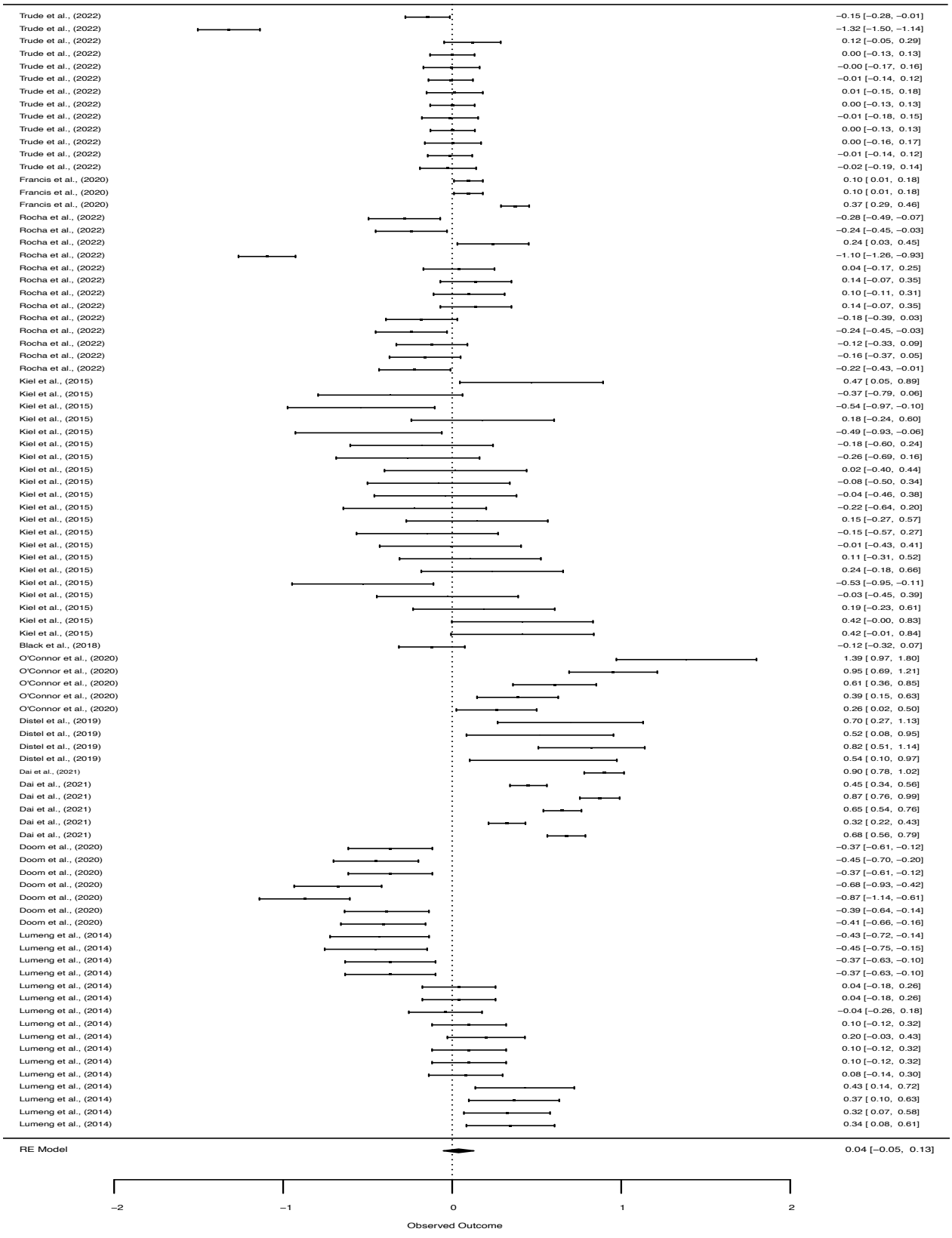


Figure 4.

Forest Plot of Effect Sizes for Stress Response Relationship with BMI/Obesity

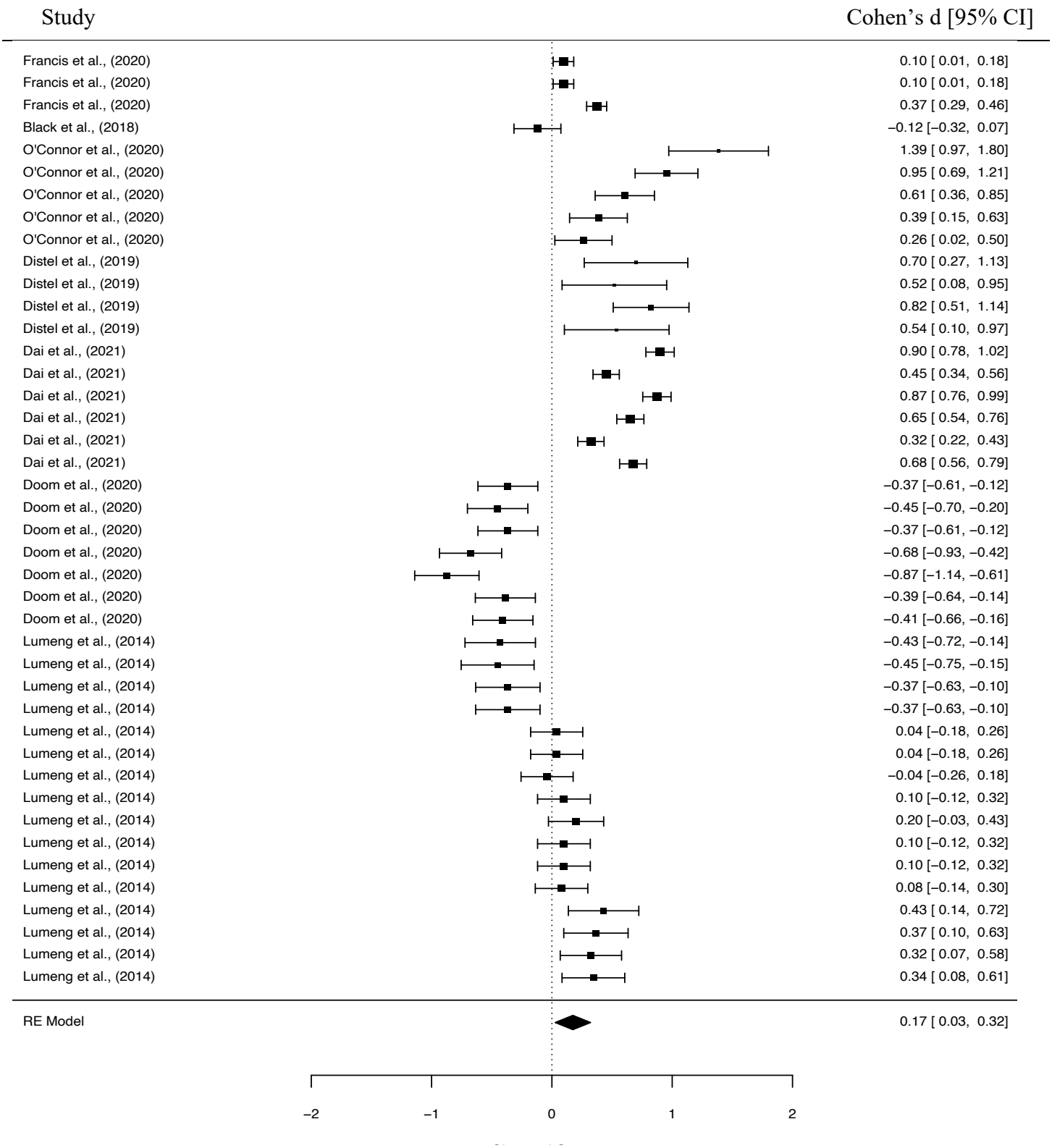
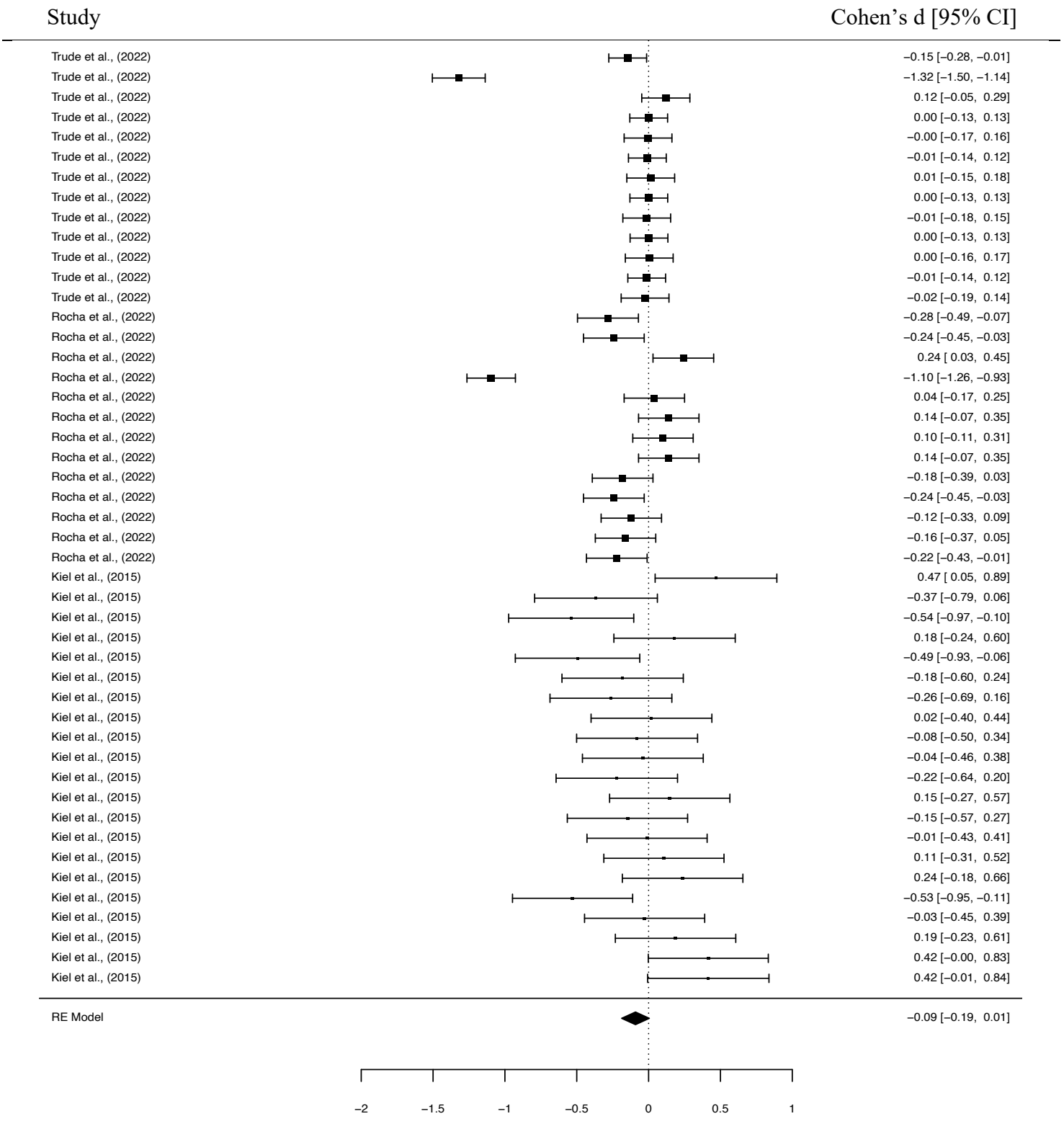


Figure 5.

Forest Plot of Effect Sizes for Stress Response Relationship with Sleep



Appendix

Data Screening and Extraction Form

General Information

Study ID:	
Study Title:	
Year of study:	
Date of Initial Screening:	
Date of Extraction:	
Person who screened:	Person who extracted data:
Citation:	

Publication Information

Publication type: Journal Article ☐ Book chapter ☐ Other (specify e.g., manual) _____	
Country of study:	Language of the article:

Study Eligibility

Study Characteristics *Even if a study does not meet the inclusion criteria, all study characteristics will be documented*			Page/ Para/ Figure #
Aim of study	To examine the effect of objective measures of stress (i.e., cortisol) on health behaviors (diet, physical activity, sedentary behavior and sleep) in children and adolescents	☐ Yes ☐ No →Exclude ☐ Unclear	
Participants	Does the study primarily include children ages 2-17?	☐ Yes ☐ No →Exclude ☐ Unclear	
	Does the study include a sample of only adults (18+)?	☐ Yes→Exclude ☐ No ☐ Unclear	
	Does the study include an eating disorder sample (e.g., anorexia nervosa, bulimia, binge eating disorder)	☐ Yes→Exclude ☐ No ☐ Unclear	
Sample size	What is the total sample size of children participating?	Sample size: _____	
	Does the study include adults? If yes, what is the total adult sample size?	Sample size: _____	
	Does the study encompass a dyad structure (i.e., participants must be child and guardian)?	☐ Yes ☐ No ☐ Unclear	

	If the study includes a dyad structure, what is the sample size of the dyads?	Sample size: _____	
Type of study	☐ Original, peer reviewed and empirical articles ☐ Intervention pilot/feasibility studies ☐ Intervention evaluation studies ☐ Randomized Controlled Trial (RCT) ☐ Cluster Randomized Controlled Trial (cluster RCT) ☐ Quasi experiemental ☐ Cohort Logitudinal Studies ☐ Pre-post single group comparison ☐ Others: _____	☐ Yes ☐ No →Exclude ☐ Unclear	
	☐ Systematic review/meta-analyses ☐ Non-peer reviewed articles ☐ Others: _____	Yes →Exclude	
Methodology	Does the study provide quantitative outcomes?	☐ Yes ☐ No →Exclude ☐ Unclear	
	Does the study compare against a control group? *(Not an exclusionary criteria)*	☐ Yes ☐ No ☐ Unclear	
Publication Date	No limits on publication date; may have specific exclusionary reasons once articles are reviewed	☐ Yes ☐ No →Exclude for specific reasons	
Language	Is the article written in English?	☐ Yes ☐ No →Exclude	
Intervention description	Does the study include a description of the intervention studied or tested?	☐ Yes ☐ No →Email author ☐ Unclear	

Summary of Assessment for Inclusion

Include in review ☐ Exclude from review ☐	
Independently assessed, and then compared? Yes ☐ No ☐	Differences resolved Yes ☐ No ☐
Request further details? Yes ☐ No ☐	Contact details of authors: (if further details needed)
Notes: (i.e. What details are missing?)	

DO NOT PROCEED IF PAPER EXCLUDED FROM REVIEW

Study details

Dimensions	Descriptions as stated in the report/paper	Page/ Para/ Figure #
Aim of study	*What was the study designed to assess? Are these clearly stated?*	
Name of intervention (if applicable)	*What was the name of the intervention?*	
Aim of intervention (if applicable)	*What was the problem that this intervention was designed to address?*	
# of groups	*How many groups were there? If more than one, name control and intervention group (s).*	
Total study duration		
Setting	*Where did the study take place? (e.g., academic medical center, university teaching hospitals, rural, metropolitan, school, workplace, community, GP clinic)*	
Providers	*Who were the providers? (e.g., number, profession, education/training, ethnicity)*	
Participants	*Where were participants recruited from?*	
	Mean/range of participants' age: _____	
	Gender composition of participants sample: *Males (n):_____ Females (n):_____* *Males (%):_____ Females (%):_____*	
	Number of dyad sample (when applicable): *Dyad: (n):_____*	
	Ethnicity breakdown *(n):_____ (%):_____*	
	Other participants' characteristics: (e.g. SES, chronic medical condition, etc.)	
Study numbers	*Eligible for inclusion: _____*	
	Excluded: _____	
	Refused to take part: _____	
	Randomized to intervention group(s): _____	

	Randomized to control group(s): _____	
	Excluded post randomization (for each group; with reasons if relevant):	
	Withdrawn (for each group; with reasons if relevant): _____	
	Lost to follow up (for each group; with reasons): _____	
	Included in the analysis (for each group; for each outcome):	
	How often did the intervention take place (if applicable)?	
	How long did the intervention last if (applicable)?	
	If there were follow-up sessions/activities post interventions, what were they and how long did they last?	
Delivery	*How was the intervention delivered? (Check all that apply)* ☐ Face-to-face ☐ Telephone ☐ Website ☐ Mobile apps ☐ Media (i.e. radio, TV, pamphlet) ☐ Others: _____	
Structure	*How was the intervention structured? (Check all that apply)* ☐ One-on-one ☐ Dyads ☐ School-based ☐ Groups ☐ Family	
Frequency	*How often did the intervention/study take place?*	
Duration	*How long did the intervention/study last?*	
	How long was each session?	
Follow up	*If there were follow-up sessions post intervention or study activities, what were they and how long did they last?*	
Cultural adaptations (if applicable)	*Was there cultural tailoring to the intervention?* Yes ☐ No ☐ Unclear ☐	
	How was the intervention culturally tailored? ☐ Tailored to specific ethnic/cultural identities ☐ Tailored broadly for ethnic/cultural minorities ☐ Tailored for other cultural reasons	

Unit of Analysis	*What was the unit of analysis?* ☐ Individual ☐ Group ☐ Community	
Dependent Variable Measures	*How were outcomes measured?* ☐ Saliva ☐ AM ☐ PM ☐ Other notes: ☐ Urine ☐ AM ☐ PM ☐ Other notes: ☐ Blood ☐ AM ☐ PM ☐ Other notes: ☐ Blood pressure ☐ Heart Rate ☐ Other:	
Outcome variables	*What was used to measure PA outcomes?* *What was used to measure sedentary outcomes?* *What was used to measure dietary intake outcomes?* *What was used to measure sleep outcomes?*	
Risk of Bias	*Selection bias:* *Was there true randomization?* ☐ Yes ☐ No ☐ Unclear	
	Was the intervention standardized? ☐ Yes ☐ No ☐ Unclear	
	Performance bias: *Were intervention conditions known to:* ☐ No one ☐ Participants ☐ Providers ☐ Data collectors ☐ Others	
	Detection bias: Was there blinding of outcome assessment? ☐ Yes ☐ No ☐ Unclear	
	Attrition bias:	

	Did they study explain participant attrition and exclusion from analyses? ☐ Yes ☐ No ☐ Unclear	
	Reporting bias: ☐ Self-report ☐ Biochemical verification ☐ Saliva ☐ Blood ☐ Urine ☐ Both ☐ Other: _____	

Data Extraction Table

Manuscript	Sample Characteristics, Study Design	Method for Measuring Stress	Construct and measure used for diet, physical activity, sedentary behavior and/or sleep	Findings

Summary of Data Extraction

Completed data extraction ☐ Request further details? Yes ☐ No ☐	
Verified by second coder? Yes ☐ No ☐	Second coder: _____
Verification completed on: _____	Differences resolved Yes ☐ No ☐
Notes:	

Asian/Pacific Islander (n) (%)
Black/African American (n) (%)
Hispanic or Latinx (n) (%)
Multiracial (n) (%)
Native American (n) (%)
Non-Hispanic White (n) (%)
Other Race/Ethnicity (n) (%)

Health Behavior Outcome 1 (M,SD *and/or* %)
Health Behavior Outcome 2

Health Behavior Outcome 3
Health Behavior Outcome 4

Covariate(s)
List any covariates/control variables included in analyses

Effect Size(s) of Stress Measures on Health Behavior Outcomes

Please note: Effect sizes can include correlations (r), linear regression (b or beta), logistic regression (odds ratio), mean differences (t, F, d).

Please include all effect sizes for this relationship reported by authors.