Stress in Early Life and Childhood Obesity Risk

Healthy Eating Research
Building evidence to prevent childhood obesity

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Abstract

The association between stress in early life and obesity and overweight in adulthood is well established. There is also increasing evidence of a link between stress exposure in childhood (or in utero) and child and adolescent obesity. Major sources of early life stress include adverse childhood experiences (e.g., abuse), poverty, food insecurity, and poor relationships with primary caregivers. Exposure to chronic and acute early life stressors can disrupt the biological stress regulation system, change the structure of regions of the brain responsible for emotion regulation and other important tasks, and promote obesogenic eating behavior and dietary patterns, as well as lifestyle factors (e.g., poor sleep, low physical activity) that may increase obesity risk. This research review summarizes and provides examples from the scientific literature on the association between early life stress exposure and childhood obesity risk. The review finds that there are multiple, highly intertwined biological, behavioral, and cross-cutting pathways that are altered by acute and chronic stress exposure in ways that contribute to heightened obesity risk. Developing a better understanding of the mechanisms that link early life stress exposures with childhood obesity risk will be particularly important for developing future childhood obesity prevention interventions that seek to reduce health disparities. Given that once obesity develops it is difficult to treat and very likely to persist into adulthood, prevention in childhood is essential. Targeting early childhood, when biological systems, stress regulation, diet, and activity patterns are forming, has particular prevention potential, rather than waiting until later childhood or adulthood when such patterns are well-established.

Introduction

Early life stressors, such as experiencing chronic early-life poverty or adverse childhood experiences (ACEs) such as abuse or domestic violence exposure, are associated with obesity and overweight in adults,[1,2] and increasing evidence has been found for associations in adolescents[3,4] and children.[5-10] In particular, early life stress can have a powerful influence on the developing child. Overstimulation of biological stress responses can have profound negative effects on structure and function of the brain and other biological systems, which are developing rapidly in the early years.[11,12] In addition to changes in biological systems, stress exposure in early life can affect children's dietary, physical activity, and other health behaviors, increasing their risk of overweight and obesity. Furthermore, these biological and behavioral pathways often interact in ways that increase obesity risk and may have lifelong impacts on children's diet, weight, and health.[13-15] Children living in low-income households are more likely to both experience early life stress and be at increased risk for obesity for a myriad of reasons.

Pathways of association between early life stress and later life obesity have been extensively researched; however, in young children our understanding of these pathways is currently limited. Interpreting how early life stress shapes health outcomes requires a developmental and life course approach, as chronic exposure can have lasting effects in multiple domains of development, including negative neurobiological, cognitive, social-emotional, behavioral, and physical health.[16,17] A conceptual model for the interacting pathways between early life stress exposure and obesity risk in children is presented in Figure 1. Timing of stress exposure in childhood is also critical as it has been linked to specific brain functions associated with cognitive and affective development.[16,18] Thus, it is important to introduce interventions focused on reducing stress exposure and the related effects early in life. This research review describes key behavioral and biological mechanisms through which acute and chronic exposure to early life stress can lead to obesity, policy implications of these findings, and future research needs.

Several important sources of early life stress and implications for obesity risk are described below.

Sources of Stress in Early Childhood

Stress in early life can come from adverse childhood experiences, socioeconomic factors (poverty and food insecurity), and relationships with parents and primary caregivers.

- **Adverse Childhood Experiences:** Risk factors known as ACEs, which include, but are not limited to, abuse (physical, sexual, or emotional), domestic violence, perceived discrimination, death or incarceration of a parent, or mental illness of a household member, can cause toxic or prolonged stress and result in negative effects on a child's developing brain.[11,19] ACEs have been associated with adverse weight and health outcomes in children and adults.[1-9, 20-26]

- **Socioeconomic-Related Factors**

  - **Poverty:** Many risk factors that contribute to early life stress are related to poverty. Living in poverty is often characterized by high levels of adversity and stress, and by limited opportunities to buffer the effects of stress. For example, poverty increases children's and pregnant mothers' risk of exposure to neighborhood violence, residential instability and/or poor housing quality, and environmental chaos, including disorder and high noise levels. In addition, high-poverty neighborhoods have built-environment disadvantages such as few safe outdoor play spaces and healthy food sources, which have been associated with childhood obesity.[27,28] Associations between poverty/lower socioeconomic status and increased body weight emerge in early childhood[29] and continue through adolescence.[30,31]

  - **Food Insecurity:** Low-income families may not have the resources to ensure consistent access to food or a balanced diet.[32] While the relationship between food insecurity and childhood obesity is not necessarily direct and somewhat inconsistent,[33] food insecurity may be associated with obesity-promoting factors such as unhealthy in-home and community food environments;[34,35] poor diet quality;[36-38] and obesogenic feeding practices.[39] Food insecurity is also associated with household chaos and lack of mealtime planning[40] and maternal stress,[41] which are also risk factors associated with risk for obesity.

- **Parenting and Primary Caregiver Relationship:** The parent-child relationship is a central mechanism though which early life stress exposure may shape obesity risk in positive and negative ways. Loving and stable relationships with a primary caregiver can buffer the effects of early life stress exposure, whereas unpredictable child-caregiver relationships can negatively affect a child's stress responses and may increase a child's risk for obesity through altered biological or behavioral pathways.[42,43] In addition, parent stress and mental health can directly shape parenting behaviors that may promote childhood obesity through feeding or other health behavior routines.[44-47]

Definitions of Stress

EARLY LIFE STRESS: Refers to acute or chronic stress exposure (including socioeconomic stressors, adverse childhood experiences, and relationship stressors) occurring prenatally to early school age. The studies presented in this review use different metrics for stress, which are noted in each evidence bullet.

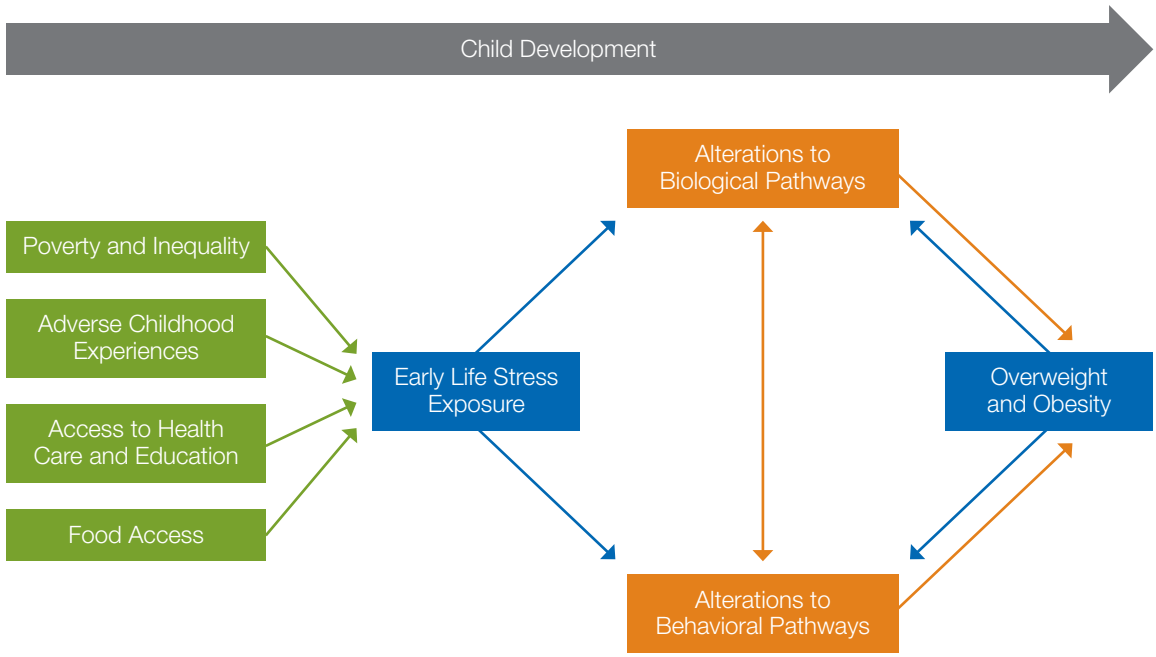
CHRONIC STRESS: Stress that is recurring, constant, or lasts for a long period of time.

ADVERSE CHILDHOOD EXPERIENCES (ACES): Risk factors that include, but are not limited to, abuse (physical, sexual, or emotional), domestic violence, perceived discrimination, death or incarceration of a parent, or mental illness of a household member. ACEs can be a single, acute event, or can be chronic and occur throughout childhood.

Methods

A review of the literature was conducted to identify studies on associations between early childhood stress, adverse experiences, and obesity risk. Three databases (PubMed, PsycInfo, and Google Scholar) were used in the search. Search terms used in these databases included, but were not limited to: early life stress, adverse events, stress, trauma, abuse, neglect, poverty, low income, child, development, infancy, preschool, obesity, overweight, BMI, weight gain, physical activity, sedentary behavior, eating behavior, dietary intake, sleep, parenting, feeding, self-regulation, executive functioning, stress biology, intervention, prevention. All study designs were included. Article titles and abstracts were reviewed and relevant articles were included. A "snowball" search strategy was used to find additional relevant articles. The reference sections of included papers as well as the "related studies" section of PubMed were systematically reviewed to find pertinent studies. Predominantly peer-reviewed empirical articles and review papers were included; however, relevant position papers or white papers from leading organizations such as the American Academy of Pediatrics were also reviewed and included.

Figure 1: Conceptual Model of Pathways Between Early Life Stress and Child Obesity Risk



Key Research Results

Results are grouped into pathways that are primarily biological and primarily behavioral in nature, for ease of presentation. However, we note that the pathways interact and are mutually influential (see Figure 1). Figure 2 highlights the biological aspects of these complex, bidirectional, and transactional relationships as they relate to obesity risk in children.

Alterations in Biological Pathways

- Chronic stress and adversity early in life can negatively impact brain development, particularly areas that govern executive function and reward systems. Executive functions include working memory, behavioral inhibition, and cognitive flexibility. Reward systems regulate wants and desires related to pleasurable activities, such as consuming food. Disruptions in both the reward systems and the capacity for self-control through diminished executive function have implications for obesity and poor eating behaviors. The connections between chronic stress; eating and reward systems; executive function and brain development; physical activity; and obesity are complex and multidirectional.

- Chronic stress exposure can disrupt the functioning of the hypothalamic-pituitary-adrenal (HPA) axis, a central biological stress-regulation system that generates cortisol (a hormone released in response to stress) as an end-product. Such disruptions have the potential to increase risk for obesity, yet this pathway has not often been examined in children.

- There are few studies examining the relationship between increases in activity in the sympathetic nervous system (SNS) and obesity risk in children, and their findings are conflicting. Yet, exposure to chronic early life stress has been shown to lead to stimulation of the SNS, which results in the release of stress hormones through several channels. Similar to the effects of the HPA axis, this may influence eating behaviors as well as patterns related to body fat accumulation and thus increases obesity risk.

- Maternal psychological stress prior to and during pregnancy has been associated with increased risk of overweight and obesity for the developing child. Prenatal stress exposure may impact programming of the primary pathways that contribute to body composition, metabolic function, and obesity risk, and therefore increase a child's susceptibility to overweight and obesity throughout childhood.

- Early life stress exposure may disrupt hormones that regulate appetite, metabolism, and fat storage, and thus increase risk for obesity, unhealthy eating behaviors, and/or unhealthy fat deposition patterns. Few studies have examined pathways between obesity-related hormones, eating, and obesity risk in children, and data are mixed.

Alterations in Behavioral Pathways

- Starting as early as infancy, both how and what a child eats can shape obesity risk. Dietary intake and related feeding practices can be affected by a variety of sources of stress in childhood. Factors that impact feeding and eating behavior ranging from the individual child level (e.g., food preferences, appetitive drive) to the caregiver (e.g., feeding practices, family meals, provision of healthy vs. unhealthy foods) and community or structural level (e.g., healthy food access, poverty-related food insecurity) have been shown to impact obesity risk in children.

- Chronic stress during childhood is often associated with decreased levels of physical activity and increased levels of sedentary activity, which may increase obesity risk. Several factors such as the built environment, household stress and absence of routines, and parental mental health may influence this relationship.

- Children in low-income or resource-constrained households are more likely to experience disrupted sleep or shorter sleep duration due to a variety of factors such as household chaos, screen time, and lack of bedtime routines. Decreased sleep duration, particularly among children, is associated with dysfunction of hormones that control appetite, obesity promoting behaviors, and increased odds of overweight and obesity.

Studies Supporting Research Results

Alterations in Biological Pathways

Chronic stress and adversity early in life can negatively impact brain development, particularly areas that govern executive function and reward systems. Executive functions include working memory, behavioral inhibition, and cognitive flexibility. Reward systems regulate wants and desires related to pleasurable activities, such as consuming food. Disruptions in both the reward systems and the capacity for self-control through diminished executive function have implications for obesity and poor eating behaviors. The connections between chronic stress; executive function and brain development; physical activity; and obesity are complex and multidirectional.

- The prefrontal cortex (PFC) is responsible for executive functions, which enable an individual to engage in goal-directed activities, such as restraint and regulation of behavior.[48] The PFC is highly intertwined with biological stress response, and stress arousal causes PFC activity to decrease. This has negative implications for executive function skills and learning,[19] and can affect a child's reactivity to stressful situations and capacity to make healthy adaptations.[17]

  - A prospective, longitudinal study found that children who were exposed to chronic stressors in early childhood had both lower self-regulatory abilities, measured by an "ability to delay gratification," and larger Body Mass Index (BMI) gains over four years from age 9 to 13.[49]

  - Studies have also examined links between executive function and eating behaviors that promote excessive weight gain in children. One study found that better executive function skills were positively associated with fruit and vegetable intake and negatively associated with consumption of high-calorie snack foods among more than 1,500 fourth grade children.[50] Another study of 3- to 6-year-old children found that children with lower executive functioning skills, as measured through behavioral tasks, parent questionnaires, and teacher reports, consumed more calories during an "eating in the absence of hunger" task.[51] Related work in younger, toddler-aged children suggests that poor self-regulation,[52] particularly for food,[53] is associated with overweight.

  - A recent review found that obesity was consistently associated with lower executive function in children, though there is some debate about the direction of the association.[54] For example, stress negatively impacts executive function and size of the hippocampus in children, which has implications for self-regulation and obesity, but these neural changes can be improved through physical activity.[54] However, children who are overweight may be less likely to engage in physical activity, which has implications for unhealthy weight and poor neural development. In addition, studies of adults suggest that obesity can exert biological changes that negatively impact cognition,[55] although this has rarely been studied in children.[56,57]

- There is evidence that early life stress also alters the development and functioning of brain regions that control responses to natural rewards, such as food, and can promote intake of highly palatable foods, high in sugar or fat.[58-61]

  - Little research has examined this pathway in children. A study examining healthy college students ages 21 to 30 found that students with self-reported lower quality maternal care, a stressor in early life, showed increased dopamine release in the nucleus accumbens in response to a stressful task and cortisol release compared to students who reported higher quality maternal care.[58] Dopamine release can in turn lead to greater reward-seeking behavior, including consumption of naturally rewarding, highly palatable food, which has the potential to become a learned response to stress.[62]

  - Other research has suggested that early life stress may reduce responsiveness to reward.[59-61] For example, a longitudinal study found that cumulative stress during childhood and adolescence, and specifically from kindergarten to grade 3, was associated at age 26 with reduced activity in the ventral striatum, an area of the brain that is central to reward responsiveness.[59] Stress was measured annually from kindergarten through grade 12 via a parent report that asked about major life stressors, such as divorce, medical problems, or death of someone close.

  - Diminished reward-related brain activity is significant because having low reward sensitivity can lead an individual to seek out highly palatable food to temporarily boost dopamine levels and stimulate their reward system.[63] Repeated stimulation and conditioning of the reward system through consumption of highly palatable foods can also promote overeating.[64] This association is complicated, however, and likely goes in both directions.

Chronic stress exposure can disrupt the functioning of the hypothalamic-pituitary-adrenal (HPA) axis, a central biological stress-regulation system that generates cortisol (a hormone released in response to stress) as an end-product. Such disruptions have the potential to increase risk for obesity, yet this pathway has not often been examined in children.

- Early life stress or childhood abuse can lead to impaired functioning of the HPA axis. The HPA axis is critical for responding effectively to stress, and under optimum conditions is activated in response to acute stress to produce cortisol. In the case of acute stress, feedback loops are in place such that the HPA axis is signaled to stop production of cortisol once the stress has passed.[65,66] Chronic stress exposure can result in excessive cortisol production at first, but over time can lead to lower-than-typical cortisol levels due to adaptations in the brain that impair such feedback loops.[67,68]

- Persistent stimulation of the HPA axis that leads to over- or under-secretion of cortisol may increase obesity risk.[65-67] The direction of the association between obesity and cortisol production is still unclear, however, as many studies are cross-sectional. Obesity has been associated with the body's inability to identify when to stop production of cortisol,[65] for example. When paired with low levels of growth and sex hormones, which also occur during chronic stress, excess cortisol can lead to increases in internal body fat.[65-67] Cortisol can also increase the activity of an enzyme that is important in fat storage.[66,69]

- Experimental evidence in animals suggests excess cortisol release may also increase appetite and cravings for "comfort food."[70,71] In humans, experimental work showed that administration of cortisol led to increased energy intake and decreased sensitivity to leptin, which suppresses appetite.[72] Over time, repeated exposure to stress may blunt cortisol responses as described above, but poor eating habits in response to stress may remain, independent of biological cues, resulting in obesity.[73] Research has also suggested that neuropeptide Y (NPY), an appetite stimulant, may be secreted in response to cortisol,[66,67] further enhancing the possible risk of overweight/obesity.

- Associations between cortisol production and overweight/obesity have been studied less often in children, and the direction of association has been inconsistent. There is some evidence that suggests that salivary cortisol levels may be lower[74] and HPA axis activity altered[75] in overweight or obese, compared to non-overweight, children and adolescents.

- Among younger children, similar associations have also been found between stress exposure, blunted salivary cortisol, and higher weight in low-income preschoolers.[76] They are also seen between blunted daily cortisol and emotional overeating in children this age.[73]

- One case control study found that hair cortisol was higher in 8- to 12-year-olds with higher BMI z-scores and waist circumference.[77] Hair cortisol is thought to be a more reliable indicator of chronic cortisol exposure than salivary, serum, or urinary cortisol as it captures exposure over longer periods of time and is less susceptible to daily variation and sleep patterns.[65,77]

- Ultimately, the activity of the HPA axis and the levels of cortisol present are likely contingent upon a variety of factors such as the type of stress or maltreatment experienced, the proximity of that stress to the time of measurement, the child's age at maltreatment, et cetera.[68] Given the hypothesized changes in cortisol output patterns in response to chronic stress exposure over time[68] and the complex interactions across bodily systems, more mechanistic and longitudinal research using measures of multiple physiologic systems is needed to better understand this relationship in children and its association with obesity risk.

There are few studies examining the relationship between increases in activity in the sympathetic nervous system (SNS) and obesity risk in children, and their findings are conflicting. Yet, exposure to chronic early life stress has been shown to lead to stimulation of the SNS, which results in the release of stress hormones through several channels. Similar to the effects of the HPA axis, this may influence eating behaviors as well as patterns related to body fat accumulation and thus increases obesity risk.

- The SNS is another key biological pathway through which chronic stress may increase obesity risk. It is well understood that stress increases SNS activity. One recent experimental study in animals demonstrated that, in addition to the action of the HPA-axis, increased baseline SNS activity further increases levels of serum cortisol. This evidence suggests that the SNS may have a significantly greater role in increasing cortisol levels, and thereby shaping obesity risk, than was previously understood.[78]

- Additionally, one study in animals demonstrated that chronic stress exposure leads to secretion of NPY from sympathetic nerves, which can lead to increases in abdominal fat.[79] In individuals exposed to chronic stress, the release of the same appetite stimulant may lead to the increased intake of high carbohydrate and high fat food.[69]

- Recent studies have demonstrated that lower levels of salivary alpha-amylase (sAA)—a marker of SNS activity—in the morning and lower sAA reactivity to a stressor were associated with higher BMI z-scores in low-income, preschool children and overweight among toddlers.[80,81] Similar to the HPA-axis, chronic stress may lead to altered or blunted SNS activity over time.[82,83]

- Furthermore, blunted sAA release in response to stress has also been linked to several behaviors that may be related to obesity risk such as impulsivity and inability to delay gratification.[84,85]

Maternal psychological stress prior to and during pregnancy has been associated with increased risk of overweight and obesity for the developing child. Prenatal stress exposure may impact programming of the primary pathways that contribute to body composition, metabolic function, and obesity risk, and therefore increase a child's susceptibility to overweight and obesity throughout childhood.

- Studies have found an association between maternal self-report of stress during pregnancy and higher odds of child overweight.
  - One study compared health and physiological markers of disease risk in young adults born to mothers who either experienced a major stressful life event during pregnancy (e.g., death or severe illness of someone close, relationship conflicts, severe financial problems) or did not experience major stress during pregnancy. The study found that children of subjects in the prenatal stress group had higher BMI and percentage body fat.[86]
  - One population-based cohort study,[87] which measured stress in the form of maternal bereavement due to the death of a close relative in the year prior to or during pregnancy, found an association between maternal bereavement and increased risk of child overweight.
  - Another study examined whether prenatal maternal stress due to a natural disaster had an influence on child obesity risk.[88] Women who were pregnant during or conceived in the three months following the January 1998 Québec Ice Storm completed several surveys to measure stress due to the storm both objectively, in terms of exposure to the ice storm and resulting damages, and subjectively, in terms of how the women reacted. A follow-up when their children were 5½ years old found that higher levels of maternal stress were associated with higher child obesity risk.
- A growing body of evidence suggests that prenatal stress exposure has an impact on length of gestation, birth weight, and fetal growth through numerous biological pathways.[89] A recent review of several prospective, population-based studies, for example, found that women experiencing high levels of stress during pregnancy had significantly higher risks for preterm delivery, low birth weight babies, and lower rates of fetal growth, independent of other risk factors.[89] This is significant because low birth weight and fetal growth restriction are characteristics that have an established relationship with later obesity risk[90-92] due to their association with higher infant body fat percentage and higher cortisol levels in adults.
- One way maternal stress may impact fetal growth, length of gestation, and later obesity risk, is through prenatal overexposure to stress hormones, such as corticotropin-releasing hormone (CRH). CRH is the main regulating hormone of the body's biological stress-response system; it is also produced in the placenta and predicts pregnancy duration, as levels naturally change over the course of the pregnancy and rise towards the time of delivery.[93] Placental CRH levels also increase, however, in response to maternal stress, with higher maternal stress levels in the second trimester correlating with higher than typical CRH levels in the third trimester.[93] Several studies have also found that higher placental or blood cord CRH levels are associated with preterm labor or fetal growth restriction.[93] One longitudinal study found that women with the highest placental CRH concentrations at 33 weeks gestation had a higher relative risk for preterm delivery and fetal growth restriction, suggesting that placental CRH could play a role in fetal development and prematurity.[93]
- Further examining the potential impact of prenatal elevated CRH exposure, for which maternal stress is a risk factor, a prospective cohort study of pregnant women and their children measured maternal CRH levels in the second trimester and child body fat at a 3-year follow-up. It found that higher CRH concentrations were positively associated with central adiposity in 3-year-old children.[94] This supports the notion that prenatal exposure to elevated stress hormones may impact child obesity risk.

Early life stress exposure may disrupt hormones that regulate appetite, metabolism, and fat storage, and thus increase risk for obesity, unhealthy eating behaviors, and/or unhealthy fat deposition patterns. Few studies have examined pathways between obesity-related hormones, eating, and obesity risk in children, and data are mixed.

- Leptin is a hormone secreted by fat cells, which helps to regulate energy balance by inhibiting hunger and increasing energy expenditure. Though it is a satiety hormone, elevated leptin levels are associated with obesity in adults and children.[56] A state known as "leptin resistance," which is associated with obesity, occurs when a dysfunction in the hypothalamus prevents leptin from reaching the brain and suppressing hunger hormones. This results in more leptin secretion, fat storage, and increased levels of hunger hormones, which can lead to obesity.[56,95]

- Leptin is also secreted in response to stress.[96] Leptin production in response to acute stress has been shown to reduce stress-related eating behaviors in adults such as comfort food consumption;[96] but the data on eating and leptin in children are mixed.[97] Overstimulation through chronic stress, however, can lead to excess leptin levels and leptin resistance (as described above), which is associated with obesity.[98] One cross-sectional study evaluated leptin levels as a potential mechanism driving the link between early life adversity and obesity.[98] The retrospective study examined adults who reported early life adversity, defined as physical, emotional, or sexual abuse and neglect before age 18, and gave each participant a score based on the number, severity, and chronicity of adversity. Higher adversity scores were positively associated with higher leptin levels, which suggests that leptin could lie in the pathway from early life stress to obesity. The mechanisms linking excess leptin levels and obesity have not been fully explained by the literature, and more studies are needed that focus on children.

- There is evidence that NPY is secreted in response to elevated cortisol levels resulting from stress.[66,67] Typically, NPY levels decrease when leptin is secreted and the hunger-feeding cycle comes to an end, but this does not occur when leptin receptors are dysfunctional.[95] Elevated NPY secretion in response to stress can therefore increase food intake.

Alterations in Behavioral Pathways

While not discussed in depth in this review, it is important to acknowledge the potential impact of affective disorders resulting from early life stress on childhood obesity risk. It is well understood that chronic stress in early childhood can and often does lead to affective disorders such as anxiety, posttraumatic stress disorder, and depression.[17, 99] There are specific neurobiological changes that occur in response to persistent stress that increase a child's vulnerability to depression and other mental health problems.[99] These disorders may be associated with future risk of obesity in children and adolescents and represent another means through which early life stress may impact a child's future weight and health.[100,10] Similar to others described in this review, this relationship is likely cyclical in nature as children with overweight are more likely to experience bullying which may negatively affect mental health.[102]

Starting as early as infancy, both how and what a child eats can shape obesity risk. Dietary intake and related feeding practices can be affected by a variety of sources of stress in childhood. Factors that impact feeding and eating behavior ranging from the individual child level (e.g., food preferences, appetitive drive) to the caregiver (e.g., feeding practices, family meals, provision of healthy vs. unhealthy foods) and community or structural level (e.g., healthy food access, poverty-related food insecurity) have been shown to impact obesity risk in children.

- One study among low-income preschool children found that living in a chaotic home environment increased children's risk for obesogenic eating behaviors.[103] For example, children may engage in "stress-eating" as a behavioral response to chronic early life stress. This study also found that surgency, defined by high pleasure seeking and impulsivity, increased the odds of obesogenic behaviors such as overeating in response to external cues, eating in the absence of hunger, and having a frequent desire to eat.[103] Other studies have also suggested that temperamental characteristics, such as surgency, may affect feeding styles that increase obesity risk in infants and young children.[104]

- In a cross-sectional study of 4,320 school-age children, higher levels of self-reported stress were associated with obesogenic eating behaviors such as low consumption of fruits and vegetables, high consumption of high fat foods, and increased snacking.[105] The strongest association was between stress and high fat food consumption.

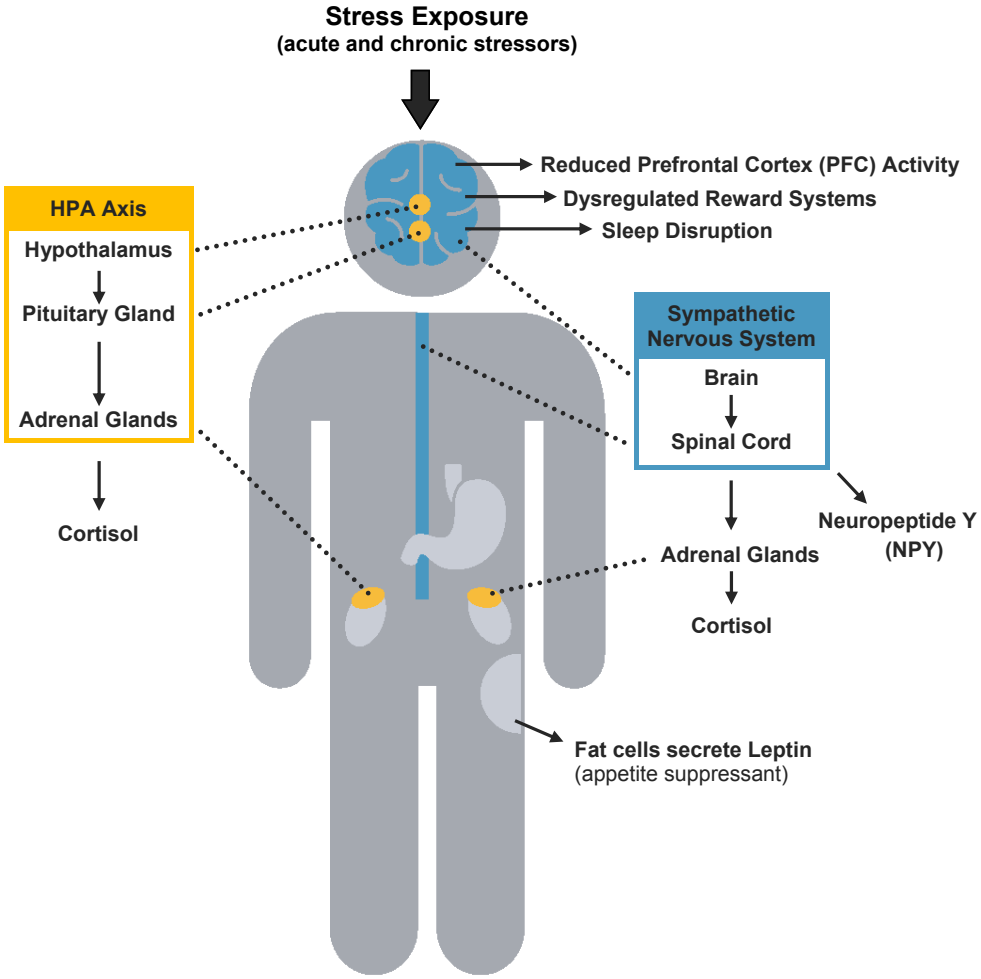
- Specific forms of early childhood stress such as poverty and food security may lead to specific obesity-promoting dietary practices or feeding patterns. For instance, consider complimentary food introduction among infants living in households experiencing poverty and family-level stress. When being offered unfamiliar foods, such as fruits and vegetables, young children often refuse them between eight and 15 times before they are willing to eat them.[106] One study among low-income mothers showed that low-income families often do not have the resources for the food waste created in this process and are more likely to purchase energy-dense, nutrient-poor foods that will be accepted by their children.[106] However, research has demonstrated there may not be a direct relationship between food insecurity and obesity and that many factors likely influence this relationship.[32-41]

- Other family-level factors such as lack of time for food preparation and regular family meals can influence a child's risk for obesity. In one cross-sectional study with a large sample of preschool-aged children from Philadelphia, an increase in the number of stressors reported by parents was associated with increased child fast food consumption.[107]

- Research has also shown that consistent family meals may be protective against obesity, particularly in older children. Most families, regardless of household income or education level, face barriers to sharing family meals consistently.[108,109] However, resource constrained, high stress households may be even less likely to regularly eat meals together for a variety of reasons.

Figure 2: Individual Biological Stress Responses Relevant for Obesity Risk

This simplified diagram, while not exhaustive, depicts several biological pathways impacted by stress exposure. More information on each one of the specific pathways depicted and how they intersect can be found in the review.



Biological Changes and Their Implications for Childhood Obesity Risk

Cortisol (elevated levels short-term, blunted response long-term)	→	Increased: visceral fat, energy intake, NPY release Decreased: sensitivity to leptin, PFC activity
Reduced PFC Activity	→	Increased: eating in the absence of hunger Decreased: executive function skills, ability to regulate and respond to stress
Dysregulated Reward Systems	→	Increased: dopamine release and reward-seeking, consumption of highly palatable food Decreased: reward responsiveness and dopamine levels over time
Changes in Appetite Hormones	→	Leptin: levels increase or decrease over time, brain responsiveness to leptin decreases NPY: levels increase, increased appetite, intake of high carb/fat food, abdominal fat
Reduced Sleep Quality/Duration	→	Increased: response to food stimuli Decreased: leptin levels

For instance, some low-income parents may not be able to prepare and serve dinner due to job structure such as working second or third shifts.[110-113] Some research has shown that another barrier to family meals among low-income mothers is the amount of time involved in establishing order among family members and preparing meals.[114]

- Finally, maternal depression, a source of toxic stress in early childhood,[17] is associated with obesity-promoting feeding practices and styles and child overweight and obesity. For instance, several studies of low-income mothers of preschool-aged children have demonstrated that maternal depression is significantly associated with lower likelihood of being present or involved at meals. These studies have also shown that the higher likelihood of using verbal pressure to get children to eat and other negative mealtime practices are linked to overweight and obesity risk.[41,115,116] Similarly, a study of low-income mothers of 5-year-old children found that mothers with depressive symptoms, when compared to non-depressed mothers, were more likely to have children who consumed sugary drinks daily and ate out in restaurants three or more times per week. They were also less likely to set limits around eating and model healthy eating behaviors. This study also found that mothers with moderate to severe depressive symptoms were more likely to have an overweight or obese child.[117]

Chronic stress during childhood is often associated with decreased levels of physical activity and increased levels of sedentary activity, which may increase obesity risk. Several factors such as the built environment, household stress and absence of routines, and parental mental health may influence this relationship.

- Exposure to acute, interpersonal stress has been shown to decrease physical activity in children in a laboratory setting.[118] In this experimental study, children were given the choice to engage in physical or sedentary activity following a stressful condition. Children that were more reactive to stress, as measured by change in heart rate, were less active after exposure to stress than the control group.[118]

- Several cross-sectional studies have also demonstrated that early childhood stress is associated with increased levels of sedentary activity and decreased levels of physical activity among children.[119,120] There are many factors that may interact with or influence this observed relationship between stress exposure and activity levels in children, such as the built environment, neighborhood safety, and household rules. Effects can also vary by child sex and age, confirming the importance of taking a developmental and life course approach.[121]

- The built environment has a significant impact on children's ability to engage in physical activity.[122,123] A recent review demonstrated that low-income or resource-constrained neighborhoods are less likely to have parks or other recreation resources where children can participate in physical activity.[122] Furthermore, this review showed that children and families in low-income neighborhoods are more likely to experience barriers to physical activity, such as higher levels of crime, unsafe traffic levels and patterns, and a lack of sidewalks.[122] Additionally, many low-income homes are smaller in size and often overcrowded, which presents barriers to being active indoors.[124]

- Chronic family-level stress can also influence children's levels of physical activity and obesity risk. A cross-sectional study including 110 parent-child pairs explored associations between parent stress and child obesity risk factors of physical inactivity and television watching (measured via parent report).[125] This study found that high levels of parenting stress were associated with less physical activity and fewer limits on time spent watching television among preschool-aged children.

- Another cross-sectional study, which surveyed low-income mothers of preschool-aged children, found that children whose mothers had depressive symptoms watched 23 more minutes of television daily compared to children whose mothers were not depressed.[126] Research has demonstrated that children experiencing more time engaged with screens are at higher risk of overweight and obesity.

Children in low-income or resource-constrained households are more likely to experience disrupted sleep or shorter sleep duration due to a variety of factors such as household chaos, screen time, and lack of bedtime routines. Decreased sleep duration, particularly among children, is associated with dysfunction of hormones that control appetite, obesity promoting behaviors, and increased odds of overweight and obesity.

- Home sleep environments characterized by noise, chaos, irregular or insufficient child sleep locations, and a lack of bedtime routine may contribute to a child sleeping for shorter periods, on a less-regular schedule, or not obtaining restful sleep. Low-income status and family conflict have each been associated with poor sleep health in children.[127-129] Insufficient sleep, specifically short duration, is consistently associated with increased risk of obesity during childhood.[130,131]

- A recent systematic review and meta-analysis of 22 longitudinal studies examining the association between sleep duration and BMI in childhood or adolescence found that children with shorter sleep duration had roughly twice the odds of being overweight or obese compared to their peers with longer sleep duration.[132]

- Two additional meta-analyses that included both children and adults demonstrated that shorter sleep duration was associated with increased odds of obesity.[130, 133] Interestingly, both of these studies showed that the relationship between sleep and obesity was stronger among children than adults. This may be due to the rapid brain development that occurs during childhood and the critical role adequate sleep plays in brain development. Decreased sleep duration and quality may lead to specific alterations in regions of the brain involved in energy balance.[134]

- Several biological and behavioral mechanisms have been proposed to explain this relationship. Leptin and ghrelin are two hormones that play a key role in appetite regulation.[134] When leptin is released, it acts as a satiety signal, decreasing energy intake. Ghrelin has the opposite role, stimulating hunger upon its release. Research has generally shown that sleep deprivation leads to increased ghrelin and decreased leptin levels;[135-138] however, several studies have demonstrated little or no change in these hormones in response to shorter sleep duration.[139-142] Some of these studies were conducted in adults only.

- In addition to the role of leptin and ghrelin, pleasure centers in the brain may also play a role in the relationship between sleep deprivation and obesity. One study in adults found that restricted sleep (4 hours per night) compared with regular sleep (9 hours per night) increased brain activity, measured by functional magnetic resonance imaging (fMRI), in regions of the brain associated with reward in response to food stimuli.[143]

- Additionally, results from an experimental study in children suggest that relatively small changes in sleep duration can have a significant impact on weight, energy intake, and fasting leptin levels.[144] These results support the important role of adequate sleep in obesity prevention.

- Finally, this relationship—similar to many others described in this review—may be more cyclical than unidirectional. It is well understood that obesity is associated with increased risk for sleep apnea, causing disrupted sleep. Furthermore, sleep itself is critically important for coping with stress.[145] Therefore, it is possible that this is a vicious cycle through which disrupted sleep leads to obesity and inability to cope with stress, which in turn exacerbates the disrupted sleep patterns.

Conclusions

Early life stress is associated with childhood and later life obesity. Several important sources of early life stress include ACEs, poverty, food insecurity, and parenting or relationship with the primary caregiver. These experiences can be stressful for children through their physical settings, disruptions in routine, and lack of resources, and can set off a cascade that has far-reaching negative neurobiological, cognitive, social-emotional, behavioral, and physical health effects. There are multiple behavioral as well as biological pathways that link persistent stress in childhood to obesity risk, and many of these interact in cyclical or yet to be determined directions. Based on the evidence presented in this review we conclude that:

- Stress exposure in early childhood can disrupt healthy functioning, including stress biology and stress-related behaviors.

- Associations between unhealthy weight and stress-related biology and behaviors may be bidirectional and also interact over time.

- Associations between early life stress and obesity in children are currently inconsistent. This may be due to the fact that the effects of early life stress on weight can emerge over time. In addition, many studies examining these associations are cross-sectional and only show correlations. Longitudinal pathways between early stress and later obesity risk have not been clearly articulated.

This review, while not exhaustive, presented an overview of developmental processes and contexts that contribute to early life stress and their effect on biological and behavioral risk factors for obesity. Understanding the multiple sources of chronic stress that children can experience and how they can affect biological and behavioral pathways to weight and health is a critical first step toward developing effective prevention and intervention programs and policies that modify these pathways to eliminate risk for young children and change health trajectories for already overweight or obese preschoolers.

Policy Implications

Early childhood stress and ACEs result from dysfunction at various levels (individual, community, public policy) and in various sectors (education, policy, welfare, health care). Therefore, the policies and programs that aim to resolve this issue must be similarly multidimensional. It is critically important to utilize a two-generation approach in programs that address the effect of ACEs on obesity and healthy eating, beginning at pregnancy. Without programs such as adult education, job training, and targeted home visiting that support parents' efforts to create a stable and healthy home environment, efforts to counteract the effects of early childhood stress on weight and health may be of limited utility.

Family Level

- **Scale up successful group prenatal care programs such as Centering Pregnancy Plus.** These programs provide low-income pregnant women with group sessions that include self-management activities, prenatal care, and facilitated discussions, all of which follow evidence-based guidelines. Research shows that these programs lead to better maternal psychosocial outcomes, improved maternal physical health, and better birth outcomes, all of which could decrease childhood obesity risk.[146,147]

- **Support two-generation programs that deliver adult education and parenting support for low-income families that can help counteract the effects of stress.** Adult education and job training should be a priority as these efforts can have a positive impact on parental employment, income, and child development.[148,149] In addition to general adult education, targeted home visitation can help parents, particularly those experiencing resource constraints, improve parenting skills and make positive changes in the home environment.[149] It is important for home visitation programs to include healthy eating and obesity prevention messages.[150-152] These programs can connect families to important social services, such as federal food assistance programs.

- **Involve the whole family unit.** It is critical that interventions engage the entire family, particularly fathers and extended family members, in making changes to create healthy home environments.[153] Research on child health and obesity prevention interventions has largely neglected to include fathers despite their notable impact on children's health and development.[154,155]

Community Level

- **Ensure access to safe, healthy, culturally-appropriate, and affordable food in all communities.** Healthy food financing initiatives that provide access to healthy food in underserved communities play an important role. However, in order for these efforts to impact obesity prevalence, it is also important to continue to conduct research on interventions such as modifying the in-store environment or altering prices of specific food and beverages to promote healthy food purchases.

- **Support improvements to the built environment in low-income communities.** These changes could range from access to safe places for recreational physical activity to zoning for green space or healthy food outlets.[17] Low-income or resource constrained neighborhoods are more likely to experience barriers to physical activity such as high levels of crime and a lack of recreation spaces. Increasing the number of locations where children could safely and regularly engage in physical activity could have broad impact on population-level obesity risk over time.[122,123]

Public and Private Policy Level

- **Ensure access to high quality child care for all children, regardless of income, geography, or race.** Programs that improve the nutrition and physical environment in child-care facilities, such as the Child and Adult Care Food Program (CACFP), play a critical role in mitigating the effects of early childhood stress on childhood health by providing access to high quality, nutritious foods and beverages and opportunities for physical activity that children may not otherwise receive. It has been shown that attending Head Start, which follows such guidelines, is associated with healthier weight in children.[156] It may be particularly important to ensure that the nutrition and physical activity environments in family child-care homes and friend, family, and neighbor care, where many low-income children are enrolled, also follow these guidelines. Child-care subsidies also play a critical role in making quality child care affordable for all families.

- **Provide access to affordable health care for low-income children and their families, particularly for mental health services**. Policy changes such as Medicaid expansion or changes to insurance reimbursement could decrease out-of-pocket health care costs and may decrease the prevalence of maternal depression.[17,148,149] Additionally, community health workers have been demonstrated to be an effective means through which access to health care can be improved and health disparities can be diminished.

- **Continue support for and optimization of federal food assistance programs including the Supplemental Nutrition Assistance Program (SNAP), National School Lunch Program (NSLP), School Breakfast Program (SBP), CACFP, Summer Food Service Program (SFSP), and Special Supplemental Nutrition Program for Women, Infants, and Children (WIC).** Research has continuously demonstrated the critical role these programs play in providing much needed, quality nutrition to food insecure children. In some cases, research has demonstrated that the current level of benefits for these programs is too low and needs to be increased to ensure that low-income families can purchase high quality, nutrient-dense foods. Across all food assistance programs, there are many children and families who are eligible but not currently enrolled. Strategies such as categorical eligibility, which is automatic eligibility for a child to receive free school meals due to their receipt of other assistance programs, and the community eligibility provision, which allows schools with a high percentage of low-income students to serve free breakfast and lunch to all students, ensure that children and families in need are enrolled in these programs and receive nutritious food.

- **Continue to support income and housing support programs**. Programs such as Temporary Assistance for Needy Families, the Earned Income Tax Credit, and child-care subsidies provide a critical safety net for low-income families. Without access to these supports, families may experience increased levels of stress and children may be at increased risk for ACEs and, consequently, childhood obesity and other adverse health outcomes.

Future Research Needs

The mechanisms through which early life stress exposure affects stress biology, behavior patterns, and obesity are multifaceted and complex. Specific effects of biological stress on obesity have mostly been studied in animals and adults, and are not yet fully understood in childhood. Therefore, mapping these pathways in detail should be a priority for childhood obesity and child development researchers, and future prevention efforts. Understanding how stress can shape child behaviors (e.g., diet and eating, activity levels, and sleep) from an early age is also important. Further research in the following areas would increase our knowledge about the mechanisms that link early life stress with childhood obesity.

- More longitudinal and experimental studies are needed that examine pathways between early life stress, biological and behavioral stress regulation, and obesity risk in children. To date most research has focused on adults and relied on cross-sectional study designs or retrospective reporting of adversity, which can be unreliable. Longitudinal studies that model these associations starting prenatally are also important because the associations between early-life stress exposure or stress biology and stress-related behaviors may not be apparent during early childhood, but may emerge later in development.

- Future studies should focus on how different sources of stress shape pathways to obesity risk, and consider how differences in the timing of stress exposure may result in different obesity risk profiles.

- More research is needed to clarify the direction of complex associations between stress-related behavioral and biological pathways and obesity in children. For example, physical activity can reduce risk for obesity and reduce the body's stress response, but children who are overweight or stressed may be reluctant to exercise.[121] As well, eating comfort foods buffers HPA response to stress,[157] but obesity can also alter HPA axis activity;[158] in sum, associations are complex.

- Further research on biological systems that may influence either obesogenic behaviors or fat deposition is ongoing, and may contribute to a better understanding of how stress can affect these systems. In addition to those mentioned in this review, biological systems that merit further investigation include brain regions outside the hypothalamus, the metabolome, which consists of all small molecule chemicals in the body, the microbiome, which consists of bacteria that colonize our bodies and help to shape the immune system, and the epigenome, which is involved in gene expression.

- Future studies should also examine whether targeting behavioral factors such as eating, physical activity, and sleep behaviors in the context of early childhood prevention and intervention programs has a protective effect against childhood obesity risk over time, in the face of stress.

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About Healthy Eating Research

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