

**TITLE:** Pathways of Association from Stress to Obesity in Early Childhood

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RUNNING HEAD: Early Life Stress and Childhood Obesity

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**What is already known about this subject?**

- Early-life stress exposure and adverse childhood experiences are associated with obesity and overweight in adults and adolescents.
- Early life stress is proposed to “get under the skin” and increase risk for obesity through multiple interconnected biologic and behavioral pathways.
- These pathways are not well-understood in young children due to study design limitations and lack of a developmental framework.

**What does this review add?**

- This review summarizes the evidence linking sources of early life stress during early childhood (birth to age 5 years) to obesity risk.
- We critically review the evidence for connections among behavioral and biological mechanisms that operate during early childhood and may confer obesity risk.
- Directions for future research are presented, with implications for intervention and clinical obesity prevention.

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**Objective:** To critically review the literature on early life stress in relation to obesity in humans including multiple biological and behavioral mechanisms through which early-life stress exposure (birth to age 5 years) may associate with obesity risk during childhood.

**Method:** Review

**Results:** Early life stress and adverse childhood experiences are associated with obesity and overweight in adults. Evidence is less consistent in children. Studies vary in the nature of stress examined (e.g., chronic vs. acute), sample characteristics, and study designs. Longitudinal studies are needed as effects of early-life stress exposure may not emerge until later in the lifespan. Early-life stress exposure is associated with biological and behavioral pathways that may increase risk for childhood obesity.

**Conclusions:** There is evidence that early life stress is associated with multiple biologic and behavioral pathways in children that may increase risk for later obesity. Little work has detailed interconnections among these mechanisms across development or identified potential moderators of the association. Mapping the mechanisms connecting early-life stress exposure to obesity risk in young children longitudinally should be a priority for obesity researchers. Recommendations for developmentally-sensitive approaches to research that can inform obesity prevention strategies are presented.

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### **Pathways of Association from Stress to Obesity in Early Childhood**

Early life stress and adverse childhood experiences are associated with obesity and overweight in adults and adolescents.<sup>1-3</sup> Yet, the mechanisms through which early childhood stress exposure may confer risk for obesity are not well-articulated. We posit that this gap is due to differences in the types of stress exposure studied (e.g., chronic vs. acute stressors), little examination of potential moderators or consideration of how different stress processes interact, and limited use of developmentally-sensitive, longitudinal study designs. We review here how stressors experienced from birth to age 5 years may contribute to obesity risk (childhood overweight, obesity, or excessive weight gain), focusing on children in the United States (U.S.). Specifically, we detail how exposure to stress during early life may shape specific biological and behavioral processes that may promote obesity risk during childhood and across development and critique the literature on stress-obesity associations in young children.

#### **Developmental Framework: How Might Stress Exposure Promote Obesity Risk?**

Early life stressors experienced by young children can include maltreatment (e.g., abuse/neglect); chaotic home environments; and exposure to other negative life events, such as witnessing violence or trauma or experiencing material deprivation. Although different stressors may activate different pathways, experiencing chronic or severe acute stress early in the lifespan can exert a powerful influence on the developing child.<sup>3-7</sup> Little research has articulated how early-life stress affects the multiple interacting biological and behavioral pathways that may elevate risk for obesity during childhood and beyond. It is beyond the scope of this review to detail links between different stressor types, timing of exposure, and risk for obesity. However, applying a developmental framework to review pathways through which stress can “get under the skin” during early life is an important step in guiding scientific understanding and intervention development.

Early-life stress exposure can set off a cascade that has far-reaching negative neurobiological, cognitive, social-emotional, behavioral, and physical health effects.<sup>3-5,7</sup> Stress biology is increasingly understood as influencing the formation and organization of neuroendocrine, metabolic, and stress-regulation systems that develop rapidly early in life<sup>5,6</sup> and shape later health.<sup>7</sup> Further, children may

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develop behavioral habits in response to stress exposure that are adaptive for their immediate context but harmful in the long-term (e.g., eating comfort foods to reduce stress).<sup>8</sup> Finally, early relationships are critical to a developmental approach. Parents can help children cope with stress. They also influence risk for obesity in children through biologic pathways<sup>7</sup> and prenatal exposures<sup>9</sup> as well as behavioral pathways.<sup>10-12</sup> Such associations are not always straightforward, and may vary across development and by social context.<sup>2,13</sup> For example, a recent study of over 35,000 Canadian children<sup>13</sup> found that authoritarian parenting style was associated with obesity in school-aged children (ages 6-10 years). However, in younger children (ages 2-5 years) the association was present only for children who were not living in poverty, suggesting that pathways other than parenting style may influence obesity risk for young children living in poverty. Articulating the mechanisms that connect early-life stress exposures and obesity risk can give us new, developmentally-sensitive tools for intervention. In order to detail the complex pathways through which early-life stress exposure may promote obesity, however, we require more sophisticated research designs that integrate a developmental perspective.

Many young children in the U.S. grow up in poverty, which is often characterized by early-life stress exposure both to acute (e.g., violence exposure) and chronic stressors (e.g., worry over limited resources), as well as food insecurity.<sup>2</sup> Such social-contextual stressors have been associated with increased risk for obesity in children.<sup>1,12</sup> Associations between poverty and increased body weight emerge in early childhood,<sup>14</sup> and longitudinal work has shown that chronic and increasing poverty during early life associates with increased adiposity in adolescence<sup>15</sup> in U.S. samples. Stress may in part explain such associations. For example, food insecurity may drive child comfort food consumption<sup>16</sup> and obesity-promoting feeding practices.<sup>17</sup> Over time, such patterns may result in greater child weight. Yet, not all children living in poverty are at equal risk<sup>2,12</sup> and childhood stress exposure (e.g., maltreatment;<sup>3,18</sup> negative life events<sup>1,2</sup>) has been shown to associate with obesity across populations. Characterizing the complex nature of the associations between early-life stress exposure and risk for obesity, and identifying potential moderators of association, is essential in order to inform interventions to prevent childhood

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obesity. Figure 1 outlines the hypothesized pathways through which stress may affect behavior and biology and confer risk for obesity during childhood; evidence for each pathway is reviewed below.

**Evidence for Biological Mechanisms Connecting Early Life Stress to Obesity in Children**

**Stress Biology.** Primary biological systems involved in stress regulation and associated with obesity include the hypothalamic-pituitary-adrenal (HPA) axis, which activates cortisol output and regulates neuroendocrine responses to social/caregiving threats,<sup>19</sup> and the autonomic, or sympathetic nervous system which regulates cardiovascular functioning.<sup>20</sup> A well-functioning biological stress regulation system activates during stress so that the organism can respond, and returns to a baseline state once the stress has passed. Under chronic stress exposure, the system may not recover adequately as the stress never truly “passes”. Such prolonged exposure can routinely overactivate biological stress processes such that they become less-effective regulators of stress.<sup>5</sup> Over time and repeated activation, wear-and-tear, or allostatic load, builds up and risk for poor health outcomes increases.<sup>5,20</sup>

Although childhood stress exposure is hypothesized to shape later health outcomes through such biological mechanisms,<sup>5,20</sup> the role of stress biology in obesity risk has been studied primarily in adults<sup>21</sup> or animal models.<sup>22,23</sup> Furthermore, most studies have considered acute stress exposure (see<sup>22</sup> for an animal model of chronic social stress). Correlational studies have found associations between childhood maltreatment, blunted cortisol and higher visceral fat mass,<sup>21</sup> although such studies mostly rely on retrospective reporting of adversity, which can be unreliable. Prospective studies of children are few. Some observational studies examining HPA axis functioning (cortisol output) and concurrent weight in school-aged children and adolescents have found inverse<sup>24,25</sup> or no association between salivary cortisol and greater weight.<sup>26</sup> Others found positive associations between hair and urinary cortisol and obesity.<sup>27,28</sup> Cortisol was also found to moderate stress-weight associations.<sup>29</sup> Studies of low-income preschool-aged children identified concurrent associations between blunted salivary cortisol,<sup>30,31</sup> blunted salivary alpha amylase (an indicator of sympathetic nervous system functioning)<sup>32</sup> and overweight, suggesting associations may emerge early in the lifespan. Findings may be inconsistent due to the different methods used and different populations studied (e.g., U.S.<sup>24,30-32</sup> vs. European<sup>25-29</sup> samples). Furthermore, early life

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stress likely shapes risk for later obesity through more than one biological process. Recent work has highlighted additional biological systems that may lie in the pathway from early life (and prenatal) stress exposure to obesity and/or unhealthy fat deposition patterns either through interacting with biological stress-regulation systems or through novel pathways.

**Epigenetics.** One such system is the epigenome, which acts to regulate gene expression through DNA methylation and histone modification processes. It is proposed as a central mechanism through which environmental exposures and stress may promote obesity for example through endocrine-disrupting chemicals<sup>33</sup> or diet.<sup>34</sup> Epigenetic changes are heritable and thus may be in part responsible for intergenerational transmission of stress effects and obesity risk from mother to child<sup>9</sup> and even across generations.<sup>35</sup>

**Microbiome.** The microbiome, or “gut-brain axis”, is another biological system that has received attention as potentially important in regulating both energy balance<sup>36</sup> and stress.<sup>37,38</sup> The microbiome refers to the genetic makeup of the community of microbes that inhabit the human body, is directly transmitted from mother to child, and is likely influenced by diet<sup>36</sup> and possibly maternal stress.<sup>39</sup> Initial evidence suggests that gut microbiota differ between obese/overweight and normal weight children<sup>40</sup> and that certain characteristics of infant gut microbiota predict early weight gain.<sup>41</sup>

**Metabolome.** Finally, researchers have also begun to examine the metabolome, or the detailed profile or metabolites in the body, in relation to stress in animals<sup>42</sup> and to some degree in humans, with some evidence for metabolome differences related to psychosocial stress<sup>43</sup> and obesity.<sup>44</sup> The very few studies to consider this in children found some differences between obese and lean children (e.g., amino acid patterns<sup>45</sup>), but also yielded mixed results.<sup>46,47</sup> As analytic techniques and methods evolve, metabolomic approaches may allow identification of previously unrecognized biological mechanisms linking psychosocial stress exposure and obesity, enabling researchers to take a more systems-wide approach. Articulating how such biological pathways interact and operate in children is a new and potentially high-impact area for research.



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**Interactions Across Biological Systems.** Our understanding of how stress exposure may dysregulate these biological systems and promote risk for obesity is just emerging, as relatively little research has examined this issue. Experimental work in animals continues to yield new insights regarding cross-system processes,<sup>37</sup> but few of these systems have been examined in the same study in humans, and almost none in children. Prior mechanistic work has shown how dysregulation of biological stress-response systems can directly disrupt metabolic functioning; for example, when the HPA axis system is activated and cortisol released, growth is inhibited and visceral fat increases.<sup>48</sup> In the case of the gut-brain axis, elegant experimental work in animals has shown that postnatal microbial colonization of the gut is necessary for appropriate HPA axis response to a stressor; without such exposure, germ-free mice showed excessive biological stress responses, and stress responses were normalized only when the mice were exposed to microbes postnatally, not later in development.<sup>37</sup> Birth and very early infancy are critical periods for bacterial colonization of the infant gut by the mother, and early variations in the gut microbiota are proposed to associate not only with the primary biological stress systems and central nervous system functioning, but also shape subsequent gastrointestinal and immune system functioning,<sup>38</sup> which are centrally involved in obesity.<sup>36</sup> As well, hormones such as leptin and ghrelin that are known to regulate both stress and appetite<sup>49</sup> have recently been shown to affect the composition of the gut microbiota in mice.<sup>50</sup> In sum, articulating how each of these systems interact and may affect stress-obesity associations longitudinally is a critical gap in the science; as little research has examined these factors in children, unpacking this complexity is an exciting direction for future work. Modeling how biological stress systems interact over time, and consideration of moderators, can enhance understanding of the complex biological effects of early stress exposure on children's concurrent and later risk for obesity.

### **Evidence for Parenting and Child Self-Regulation Factors Connecting Early Life Stress to Obesity in Children**

Early life stress may also affect parenting and child self-regulation (i.e., capacity to engage in goal-directed behavior),<sup>4,51</sup> which may in part shape child health behaviors such as eating,<sup>52</sup> physical activity and sedentary behavior,<sup>53</sup> and sleep (see Figure 1).<sup>10</sup> Unpacking the mechanisms through which

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stress may affect both parenting and child self-regulation and in turn shape child health behaviors may inform behavioral interventions. As there are likely bidirectional associations (e.g., between parenting and child self-regulation<sup>4</sup>), articulating the directional nature of associations is also important. We review evidence for parenting and self-regulation as potential influences on child health behaviors and obesity risk (primarily among U.S. samples).

**Parenting.** Parents contribute to child risk for obesity in complex ways. For example, a parent's stress biology can shape child stress biology through multiple mechanisms,<sup>7,35</sup> and prenatal stress and nutrition exposures can shape child metabolism<sup>9</sup> as well as food preferences.<sup>54</sup> Post-natal parent factors may influence health-related parenting behaviors including feeding, modeling (or not) healthy eating, setting limits on screen time, and encouraging physical activity.<sup>55,56</sup> For example, maternal stress has been associated with controlling feeding styles;<sup>57</sup> uninvolved but not indulgent or authoritarian feeding styles;<sup>11</sup> and with mother and child emotional eating.<sup>58</sup> Certain feeding styles (e.g., restrictive) have been associated with greater child weight,<sup>10</sup> although associations are not consistent across studies<sup>57</sup> and associations may be bidirectional.<sup>59</sup> Parenting stress<sup>60</sup> and parent-child interactions characterized by less sensitivity<sup>61</sup> have been associated with greater child weight in some studies, although others<sup>62</sup> have found no association. Parenting stress has been associated with fewer TV time limits and less physical activity in low-income families with preschoolers.<sup>55,62</sup> Overall, there is more evidence for associations between parent stress and reduced physical activity/increased sedentary behavior in children than between parent stress and child diet (see <sup>56</sup> for review).

Maternal mental health difficulties have been studied as a barrier to engaging in health-promoting behaviors. Evidence for the association of maternal depression and childhood obesity is mixed,<sup>63</sup> but among low-income mothers of young children, maternal mental health symptoms were associated with shorter child sleep duration, obesity-promoting feeding practices, and more sedentary/TV time.<sup>64</sup> Stressed or depressed parents may have difficulty managing their own or their child's negative emotions, or establishing regular schedules for mealtimes, bedtimes, screen time, and/or physical activity (for parent or child). Stressed parents are likely a source of stress for children as well. Thus, stress may pose unique

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challenges to engaging in obesity prevention-focused parenting, but findings are mixed and research on how stress may shape health-related parenting behaviors is warranted. Importantly, most studies of health-related parenting utilize parent-report measures of stress as well as behaviors; objective measurement may help clarify whether health-related parenting behaviors are behavioral mechanisms through which stress-obesity associations become established in children.

**Child Self-Regulation.** Self-regulation is central to adult weight management<sup>65</sup> but much less well-understood in children in relation to risk for obesity. Self-regulation, or the ability to control one's thoughts, feelings and behaviors to achieve a goal, can be shaped by stress and also determine how an individual responds to stress and challenge.<sup>4</sup> Chaotic household functioning can undermine the development of self-regulation,<sup>4</sup> and poorer self-regulation has been associated with longitudinal risk for obesity<sup>66</sup> particularly among young children exposed to early life stress.<sup>51</sup> Self-regulation may function as a mediator of the association between stress exposure and child health behaviors that promote obesity risk (see Figure 1). Obesity research related to self-regulation in children has generally examined two aspects of self-regulation: 1) self-control of emotions and behavior and 2) cognitive capacity/executive functioning. Self-control of emotions and behavior have often,<sup>58,67,68</sup> though not always<sup>68</sup> been associated with overweight and obesity during early childhood, but the pathways of association have not been well-articulated. There is a smaller literature on cognitive aspects of self-regulation, with some suggestion that poor executive functioning skills were associated with consuming palatable food<sup>69</sup> whereas better executive functioning<sup>69</sup> was associated with fruit and vegetable consumption. Cognitive deficits have also been identified among children with overweight.<sup>70</sup> It is also possible that self-regulation moderates associations of stress and obesity-promoting behaviors. For example, studies of adults have shown that individuals who are highly food-responsive eat more when stressed than do their less food-responsive peers, particularly if they report that they do not cope well with stress.<sup>71</sup> Some studies have tested self-regulation as a moderator of associations between stress exposure and obesity risk behaviors (primarily eating behaviors) in children. For example, children ate in an absence of hunger paradigm when stressed only if they had low executive control skills,<sup>72</sup> and associations between eating in the absence of hunger

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and weight gain were greatest for children with low inhibitory control.<sup>73</sup> Thus, the literature on poor self-regulation and overweight in children is somewhat mixed, the direction of effects is debated,<sup>68,70</sup> and the mechanisms of association are not yet clear. It will therefore be important for future research to consider how best to model the construct of self-regulation in children as it relates to stress exposure, health behaviors, and obesity risk.

### **Evidence for Child Health Behaviors Connecting Early Life Stress to Obesity in Children: Eating, Activity, and Sleep**

In addition to the indirect effects of stress on child obesity risk that may operate through either parenting and/or self-regulation, stress may also affect child health behaviors directly (see Figure 1). Such behaviors include eating, activity, and sleep.

**Obesogenic Eating: Eating Behavior and Dietary Intake.** Both how (eating behavior style) and what a child eats (dietary intake) have been associated with chronic and acute stress. Parent-reported eating behavior styles including high food enjoyment, food responsiveness, eating in response to external cues (vs. hunger), eating in response to emotional distress, and low satiety responsiveness<sup>74</sup> and observed eating in the absence of hunger<sup>75</sup> are also associated with obesity in early childhood. Dietary patterns associated with obesity in children include consumption of high-sugar, energy-dense, low-fiber, high-fat foods.<sup>52,76</sup> Family stress has been associated with obesogenic eating behaviors<sup>30</sup> as well as unhealthy diets,<sup>77</sup> suggesting children may engage in obesogenic “stress-eating” as a behavioral response to stress. This is consistent with prior work showing that chronic stress exposure increases consumption of palatable, high-fat foods in animals<sup>23</sup> and that early life stress associates with obesogenic eating in adults.<sup>8</sup> In school-aged children, higher waking cortisol was associated with intake of sweet foods<sup>78</sup> and experimental work in children showed that acute stress increased consumption of palatable foods.<sup>79</sup> Even as early as preschool, child stress biology associates with more obesogenic eating behavior and higher child weight.<sup>30,80</sup> Thus, associations between obesity-promoting eating behaviors and stress exposure seem to emerge early in the lifespan. Much of this work is cross-sectional. Thus, longitudinal research is essential in order to determine whether early-emerging eating behaviors promote obesity over time; if so,

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developing interventions to reduce such eating behaviors may be warranted. Furthermore, understanding individual differences in children's eating-related responses to stress is a promising research direction with implications for tailoring interventions to address child factors. For example, identifying how high food responsiveness in children may interact with both chronic environmental stress as well as acute stress in the moment may allow us to address stress-eating pathways to prevent obesity for young children with this high-risk profile who grow up under stressful conditions.

**Physical Activity and Sedentary Behavior.** Physical activity and sedentary behavior are implicated in adult stress-obesity pathways but less well-studied in children. Physical activity and sedentary behavior can independently associate with obesity, but we do not know how stress may affect each pathway. In preschool-aged children, family stress was associated with less physical activity<sup>62</sup> and among school-aged children, chronic and acute stressors were associated with less physical activity and more sedentary behavior.<sup>53,81</sup> Experimental work with children has shown that stress exposure can reduce physical activity and increase sedentary behavior.<sup>81</sup> In turn, engaging in physical activity can reduce biological stress response.<sup>82</sup> In the context of acute social stress, engaging in physical activity also reduced intake of palatable foods.<sup>83</sup> Sedentary behaviors and to a somewhat lesser degree, physical activity can be altered through intervention.<sup>84</sup> In sum, emerging evidence suggests a connection between stress and activity level in children, and could have implications for intervention (e.g., increasing opportunity for physical activity in schools).

**Sleep.** Finally, child sleep is increasingly recognized as important for obesity prevention and as negatively affected by stress.<sup>53</sup> Low-income status<sup>85</sup> and family conflict<sup>86</sup> have been associated with shorter child sleep duration and poor sleep quality. Home sleep environments with high levels of noise and chaos, insufficient locations for child sleep, or lack of bedtime routine may contribute to poor sleep health.<sup>85</sup> Short sleep duration consistently associates with increased risk for obesity, particularly in younger children.<sup>10,87</sup> Additional aspects of sleep health, including timing (i.e., when in the night one sleeps<sup>88</sup>), regularity from weekday to weekend,<sup>88</sup> and quality<sup>85</sup> have also been found to associate with obesity risk, but not as consistently. Inadequate sleep may promote obesity through many pathways,

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including biological (e.g., leptin/ghrelin production<sup>89</sup>) and behavioral (e.g., late night snacking, food choices<sup>90</sup>, or emotional eating in response to stress,<sup>23</sup> as sleep is also important for stress regulation). Such pathways have mostly been studied in adults, and support for certain pathways is emerging in children.<sup>91</sup> As sleep is modifiable, it is an important behavioral mechanism to test in interventions.

### **Research Summary and Future Directions**

We have highlighted biologically- and behaviorally-mediated pathways that may connect early childhood stress exposure to risk for obesity during childhood and beyond. These pathways likely interact and some may be modifiable through intervention. Yet, the basic mechanisms of association have yet to be precisely specified, patterns of association are complex (and bidirectional), and moderators should be considered. Gaps in the science are due to study design limitations, specifically few longitudinal studies that incorporate careful, objective measurement of both biological and behavioral mechanisms and examine how such pathways interact. Prospective studies examining chronic and acute stress exposures in relation to weight outcomes across development are rare. More sophisticated measurement and analysis of weight status beyond just body mass index, to include more direct and precise measures of adiposity is also important. For example, it has been suggested that physical and sexual abuse may pose a specific risk for severe obesity,<sup>18</sup> and more fine-grained analysis could allow careful examination of risk pathways. Future work should also consider how differences in how the timing and nature of specific stress exposures may shape obesity risk (e.g., would risk differ for infants compared to preschoolers who experience maltreatment, or for children who experience community violence versus family conflict?).

### **Implications for Intervention**

Despite the potentially formative role of early-life stress exposure, young humans are remarkably adaptive. This review has identified potentially novel intervention targets (e.g., stress biology; self-regulation; sleep) that may counteract effects of early-life stress exposure on risk for obesity in children.<sup>6</sup> Related intervention implications are outlined below and in Table 1.

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**Use Models that Incorporate Development, Individual Factors, and Bidirectional Effects.**

Just as early stress biology can shape the brain's later response to stress, behavioral habits developing during childhood can shape later behavior. Novel behavioral intervention targets could have broad impact if addressed during developmentally-sensitive periods. For example, interventions that focus on sleep routines during the first year of life may promote both child and parent capacity to cope with stress, laying the foundation for self-regulation development. Interventions to reduce the physiological effects of stress during early childhood could reduce impact on obesity-promoting biological pathways.

Interventions that address multiple relationship contexts (parents, peers) across development may be important, as obesity is a stressor that may in turn affect peer relationships. Determining how parents and children affect each other over time is critical in developing more effective relationship-focused interventions (e.g., around early feeding).<sup>92</sup> Although developmental models have long-highlighted the transactional nature of parent-child interactions, bidirectional processes—especially child contributions—are just beginning to be elucidated in the childhood obesity literature.<sup>59,61</sup> Finally, individual child (e.g., self-regulation; reward sensitivity) and/or parent characteristics (e.g., depression) may moderate the effectiveness of interventions. Tailoring interventions to address such factors may yield powerful effects.

**Support Parents.** A key aspect of a developmental approach is to incorporate the critical role of early relationships and focus on parents' capacity to engage in health-promoting behaviors and to help young children cope effectively with stress. Helping parents foster these activities may be critically important in order for obesity prevention efforts to be successful. Behavioral interventions that engage both parents and children have been more successful in treating obesity than those that focus on children only,<sup>93</sup> and trials are underway to test whether engaging parents alone is effective.<sup>94</sup> Although few obesity prevention interventions focus on general parenting, some have shown promise.<sup>95,96</sup> Supporting parents is essential in reducing the impact of early-life stress exposure on behaviors that can increase child risk for obesity. For example, even relatively straightforward strategies (e.g., limiting sugar-sweetened beverages) may not work if parents are too taxed to engage in behavior change. Furthermore, obesity prevention when children do not yet have obesity raises unique challenges due to complex root

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causes, individual differences in child biological propensity for obesity, and difficulty engaging parents if they do not believe their children are at risk.<sup>97</sup> Thus, supporting parents by first addressing their needs (e.g., using mindfulness to reduce stress) may be important for obesity-prevention parenting. Technology and ehealth methods (e.g., text reminders about TV limits or bedtime) may help parents engage in recommended practices. Involving multiple family members (e.g., fathers, siblings) may be important in maintaining change at home. As well, dual-generation approaches that address both child and parent needs (e.g., co-locating high-quality child care with job-training resources) can support parents to reduce the effects of their own stress exposure on their children.

**Connect Social Contexts and Policies.** Finally, it is essential to address the multiple contexts in which children live by engaging different sectors that shape both child development and parenting (e.g., health care, education, policy). Community-level changes that address neighborhood safety, food access, and physical activity opportunities may have the potential to reduce obesity risk for both adults and children. Furthermore, policy changes related to workforce development, health care, occupational and environmental health, child care and education may promote positive health outcomes for children, as programming is most effective when components are systemically connected across home, school, and community. For families experiencing a high degree of stress, combining contextual-level interventions with individual or family-level programming could have powerful effects with broad impact.

### **Conclusion**

Obesity is a multifaceted problem that requires multi-level solutions. Stress exposure during childhood can convey greater risk than stress exposure later in the lifespan, as early alterations in biological stress systems and behaviors can become embedded and shape developmental health trajectories over time. Given the plasticity in early development, intervening to target specific mechanisms that promote risk for obesity during childhood may be highly effective. To prevent obesity in children experiencing stress, we must embrace a developmental, contextually-sensitive perspective on research that can inform intervention strategies. Understanding the multiple sources of stress that young



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families can experience and how they affect biological and behavioral pathways to health is a critical first step toward reshaping these pathways and giving young children a healthy start in life.

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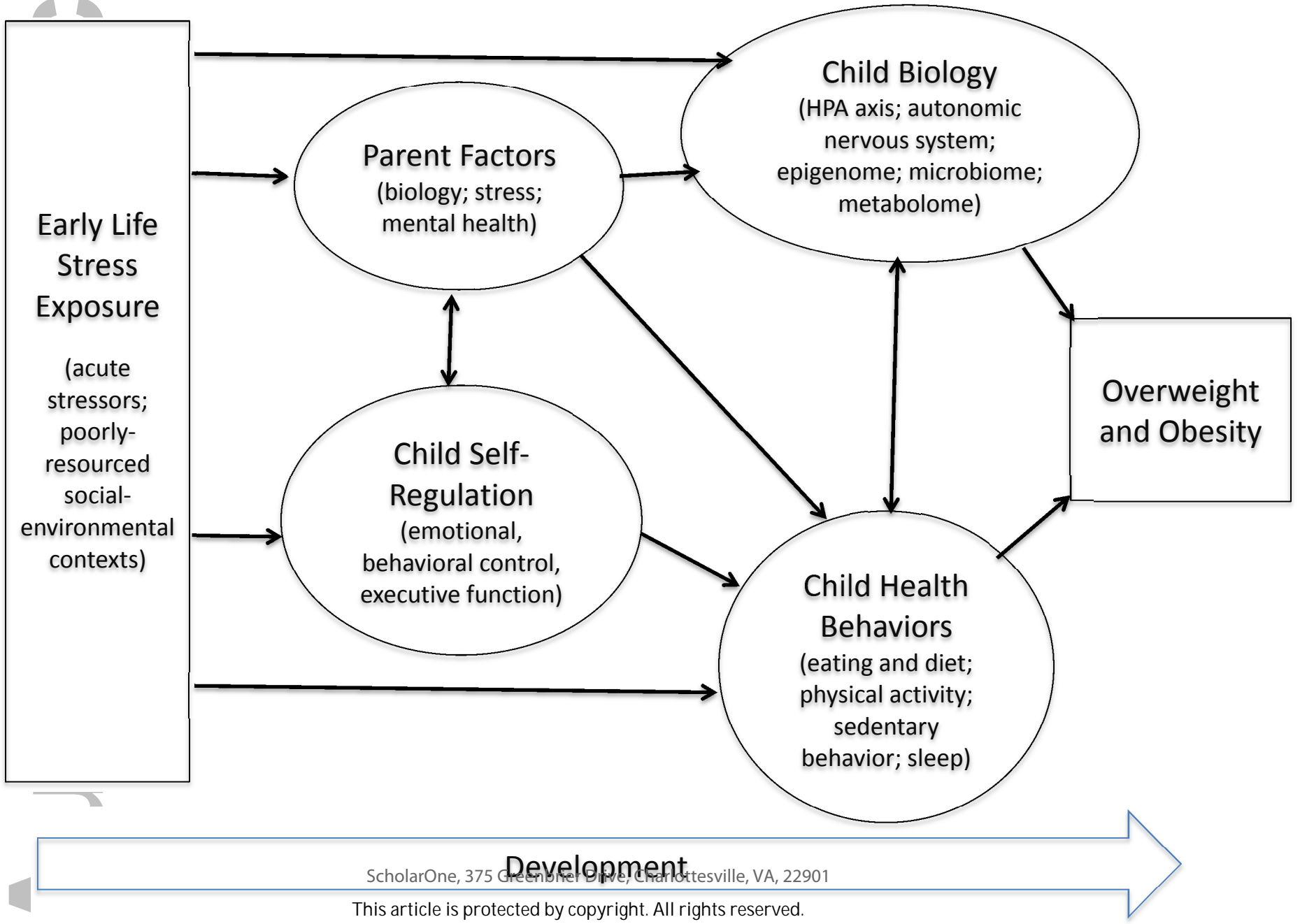
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**Figure 1. Hypothesized Pathways of Association from Stress to Obesity in Early Childhood**



**Table 1. Research on Early Life Stress and Childhood Obesity and Recommendations for Intervention**

Research Finding	Intervention Implication
Early life stress can disrupt stress biology systems which in turn may promote risk for obesity	<u>Reduce physiological effects</u> of stress exposure (e.g., using stress-reduction techniques) with children
Early life stress can shape obesity risk through multiple behavioral pathways	<u>Engage novel behavioral intervention targets</u> such as sleep, self-regulation, and overall parenting support
There are sensitive periods for both stress exposure and for response to intervention	<u>Deliver interventions at developmentally-sensitive periods</u> to address relevant biological and behavioral risk factors at different points in development (e.g., parent-child relationship focus during early development; peer-context focus during later childhood)
Individual parent and child factors may moderate intervention effects	<u>Identify individual child/parent characteristics that may moderate</u> association between early life stress and later obesity risk and use to tailor intervention approach (e.g., structure intervention based on whether or not child has high food reward sensitivity; whether or not parent has depression); consider biologically-sensitive intervention approaches
Bidirectional associations between parent and child behaviors may shape obesity risk	<u>Address bidirectional effects:</u> Note the child's contribution and how it may shape parent responses (e.g., during early feeding interactions) when designing interventions
Parenting can shape child biological and behavioral responses to stress, yet parents are stressed themselves	<u>Support parents in their efforts</u> and give them tools to help themselves manage their own stressors or other challenges so that they can support their child
Parents are difficult to engage in obesity prevention, but if they engage, programs are more likely to result in health behavior change	<u>Use behavior change approaches with parents</u> to address barriers and increase parent engagement and readiness to change (e.g., motivational interviewing, goal setting); assess parents' beliefs about obesity risk; engage additional family members
Obesity has many root causes; broad contextual changes could help many children	<u>Connect intervention efforts across social-contextual settings</u> (e.g., home, school, community); combine broad approaches with efforts tailored to address individual child and family needs