Review
PEDIATRIC OBESITY

Obesity

Pathways of Association from Stress to Obesity in Early Childhood

Alison L. Miller [1,2] *and Julie C. Lumeng* [2,3,4]

Objective: The objective of this study is to critically review the literature on early life stress in relation to obesity in humans, including the multiple biological and behavioral mechanisms through which early life stress exposure (birth to the age of 5 years) may associate with obesity risk during childhood.

Methods: A review of the literature was conducted to identify studies on associations between early childhood stress and risk for obesity and the mechanisms of association. Multiple databases (PubMed, PsycInfo, Google Scholar) were used in the search as well as a "snowball" search strategy. All study designs were included.

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 the stress examined (e.g., chronic vs. acute), sample characteristics, and study designs. Longitudinal studies are needed, as the effects of early life stress exposure may not emerge until later in the life-span. 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 biological and behavioral pathways in children that may increase risk for later obesity. Little work has detailed the 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.

Obesity (2018) **26**, 1117–1124. doi:10.1002/oby.22155

Introduction

Early life stress and adverse childhood experiences are associated with obesity and overweight in adults and adolescents (1-3). 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 the age of 5 years may contribute to obesity risk (childhood overweight, obesity, or excessive weight gain), focusing on children in the United States. 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 life-span can exert a powerful influence on the developing child (3-7). 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 among 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.

[1] Department of Health Behavior and Health Education, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA. Correspondence: Alison L. Miller (alimill@umich.edu) [2] Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan, USA [3] Department of Pediatrics, University of Michigan Medical School, University of Michigan, Ann Arbor, Michigan, USA [4] Department of Nutritional Sciences, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.

Funding agencies: This work was supported by the Robert Wood Johnson Foundation Healthy Eating Research Program.
Disclosure: The authors declared no conflict of interest.
Received: 25 April 2016; **Accepted:** 22 January 2018; **Published online 14 April 2018. doi:10.1002/oby.22155**

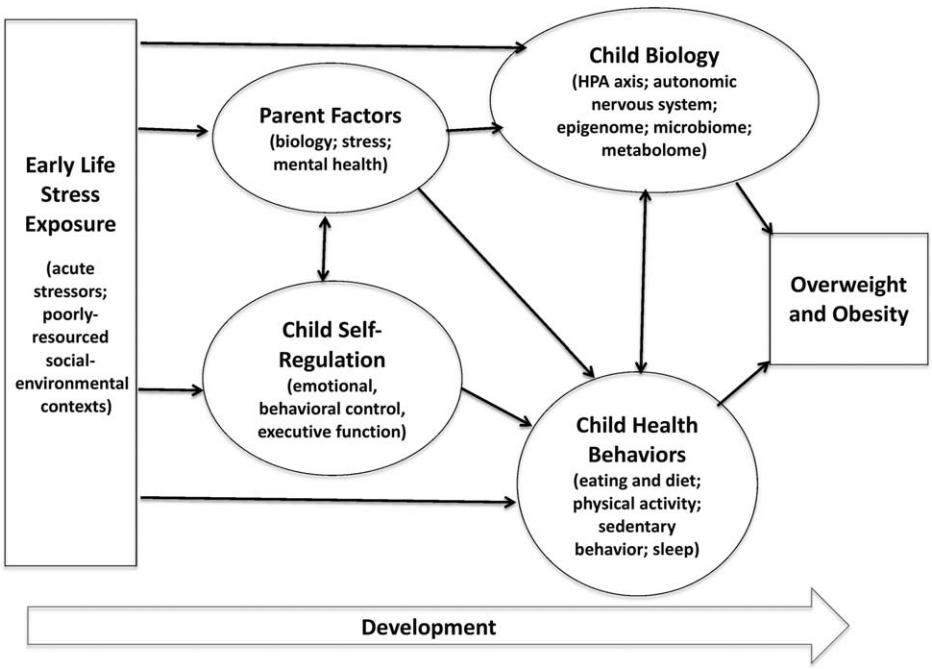

Figure 1 Hypothesized pathways of association from stress to obesity in early childhood.

Early life stress exposure can set off a cascade that has far-reaching negative neurobiological, cognitive, social-emotional, behavioral, and physical health effects (3-5,7). Stress biology is increasingly understood as influencing the formation and organization of neuroendocrine, metabolic, and stress regulation systems that develop rapidly early in life (5,6) and shape later health (7). Further, children may 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) (8). 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 biological pathways (7) and prenatal exposures (9), as well as behavioral pathways (10-12). Such associations are not always straightforward and may vary across development and by social context (2,13). For example, a recent study of more than 35,000 Canadian children (13) found that an 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 the 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 United States 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 (2). Such social-contextual stressors have been associated with an increased risk for obesity in children (1,12). Associations between poverty and increased body weight emerge in early childhood (14), and longitudinal work has shown that chronic and increasing poverty during early life associates with increased adiposity in adolescence (15) in US samples. Stress may in part explain such associations. For example, food insecurity may drive child comfort food consumption (16) and obesity-promoting feeding practices (17). Over time, such patterns may result in greater child weight. Yet, not all children living in poverty are at equal risk (2,12), and childhood stress exposure (e.g., maltreatment (3,18), negative life events (1,2)) 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 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 here.

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 and/or caregiving threats (19), and the autonomic or sympathetic nervous system, which regulates cardiovascular functioning (20). 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 (5). Over time and repeated activation, wear and tear (or the allostatic load) builds up, and the risk for poor health outcomes increases (5,20).

Although childhood stress exposure is hypothesized to shape later health outcomes through such biological mechanisms (5,20), the role of stress biology in obesity risk has been studied primarily in adults (21) or animal models (22,23). Furthermore, most studies have considered acute stress exposure (see reference 22 for an animal model of chronic social stress). Correlational studies have found associations among childhood maltreatment, blunted cortisol, and higher visceral fat mass (21), although such studies have mostly relied 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 associations (24,25) or no associations between salivary cortisol and greater weight (26). Others have found positive associations between hair and urinary cortisol and obesity (27,28). Cortisol was also found to moderate stress-weight associations (29). Studies of low-income preschool-aged children have identified concurrent associations among blunted salivary cortisol (30,31), blunted salivary alpha amylase (an indicator of sympathetic nervous system functioning) (32), and overweight, suggesting that associations may emerge early in the life-span. Findings may be inconsistent because of the different methods used and the different populations studied (e.g., US (24,30-32) vs. European (25-29) samples). Furthermore, early life 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 (e.g., through endocrine-disrupting chemicals (33) or diet (34)). Epigenetic changes are heritable and thus may be in part responsible for intergenerational transmission of stress effects and obesity risk from mother to child (9) and even across generations (35).

Microbiome

The microbiome, or "gut-brain axis," is another biological system that has received attention as potentially important in regulating both energy balance (36) and stress (37,38). 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 (36) and possibly by maternal stress (39). Initial evidence suggests that gut microbiota differ between children with obesity or overweight and children with normal weight (40) and that certain characteristics of infant gut microbiota predict early weight gain (41).

Metabolome

Finally, researchers have also begun to examine the metabolome, or the detailed profile of metabolites in the body, in relation to stress in animals (42) and, to some degree, in humans, with some evidence for metabolome differences related to psychosocial stress (43) and obesity (44). The very few studies to consider this in children have found some differences between children with obesity and lean children (e.g., amino acid patterns (45)) but also yielded mixed results (46,47). 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.

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 (37), but few of these systems have been examined in the same study in humans, and almost none of these systems have been examined 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 is released, growth is inhibited and visceral fat increases (48). 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 an 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 (37). 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 not only to associate with the primary biological stress systems and central nervous system functioning but also to shape subsequent gastrointestinal and immune system functioning (38), which are both centrally involved in obesity (36). In addition, hormones such as leptin and ghrelin that are known to regulate both stress and appetite (49) have recently been shown to affect the composition of the gut microbiota in mice (50). In sum, articulating how each of these systems interact and may affect stress-obesity associations longitudinally is a critical gap in the science; because 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., the capacity to engage in goal-directed behavior) (4,51), which may in part shape child health behaviors such as eating (52), physical activity and sedentary behavior (53), and sleep (Figure 1) (10).

Unpacking the mechanisms through which 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 (4)), articulating the directional nature of associations is also important. In this section, we review evidence for parenting and self-regulation as potential influences on child health behaviors and obesity risk (primarily among US 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 (7,35), and prenatal stress and nutrition exposures can shape child metabolism (9) as well as food preferences (54). Postnatal parent factors may influence health-related parenting behaviors including feeding, modeling (or not) healthy eating, setting limits on screen time, and encouraging physical activity (55,56). For example, maternal stress has been associated with controlling feeding styles (57), uninvolved but not indulgent or authoritarian feeding styles (11), and with mother and child emotional eating (58). Certain feeding styles (e.g., restrictive) have been associated with greater child weight (10), although associations are not consistent across studies (57), and associations may be bidirectional (59). Parenting stress (60) and parent-child interactions characterized by less sensitivity (61) have been associated with greater child weight in some studies, although others (62) have found no association. Parenting stress has been associated with fewer TV time limits and less physical activity in low-income families with preschoolers (55,62). Overall, there is more evidence for associations between parent stress and reduced physical activity and/or increased sedentary behavior in children than for associations between parent stress and child diet (see reference 56 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 (63), 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 and/or TV time (64). Stressed or depressed parents may have difficulty managing their own or their child's negative emotions or with establishing regular schedules for mealtimes, bedtimes, screen time, and/or physical activity (for the parent or child). Stressed parents are likely a source of stress for children as well. Thus, stress may pose unique 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 have utilized 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 (65) but is much less understood in children in relation to the 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 can also determine how an individual responds to stress and challenge (4). Chaotic household functioning can undermine the development of self-regulation (4), and poorer self-regulation has been associated with longitudinal risk for obesity (66), particularly among young children exposed to early life stress (51). Self-regulation may function as a mediator of the association between stress exposure and child health behaviors that promote obesity risk (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 and/or executive functioning. Self-control of emotions and behavior has often (58,67,68), though not always (68), 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 the cognitive aspects of self-regulation, with some suggestion that poor executive functioning skills are associated with consuming palatable food, whereas better executive functioning (69) is associated with fruit and vegetable consumption. Cognitive deficits have also been identified among children with overweight (70). 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 (71). 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 (72), and associations between eating in the absence of hunger and weight gain were greatest for children with low inhibitory control (73). Thus, the literature on poor self-regulation and overweight in children is somewhat mixed, the direction of effects is debated (68,70), 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 (Figure 1). Such behaviors include eating, activity, and sleep.

Obesogenic eating: eating behavior and dietary intake

Both how (eating behavior style) and what (dietary intake) a child eats 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 (74), and observed eating in the absence of hunger (75) 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 (52,76). Family stress has been associated with obesogenic eating behaviors (30) as well as unhealthy diets (77), suggesting that 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 (23) and that early life stress associates with obesogenic eating in adults (8). In school-aged children, higher waking cortisol was associated with the intake of sweet foods (78), and experimental work in children has shown that acute stress increases the consumption of palatable foods (79). Even as early as preschool, child stress biology associates with more obesogenic eating behavior and higher child weight (30,80). Thus, associations between obesity-promoting eating behaviors and stress exposure seem to emerge early in the life-span. 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, 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 and 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 are not as 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 (62), and among school-aged children, chronic and acute stressors were associated with less physical activity and more sedentary behavior (53,81). Experimental work with children has shown that stress exposure can reduce physical activity and increase sedentary behavior (81). In turn, engaging in physical activity can reduce the biological stress response (82). In the context of acute social stress, engaging in physical activity also reduces the intake of palatable foods (83). Sedentary behaviors and, to a somewhat lesser degree, physical activity can be altered through intervention (84). 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 (53). Low-income status (85) and family conflict (86) 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 routines may contribute to poor sleep health (85). Short sleep duration consistently associates with an increased risk for obesity, particularly in younger children (10,87). Additional aspects of sleep health, including timing (i.e., when in the night one sleeps (88)), regularity from weekday to weekend (88), and quality (85), have also been found to associate with obesity risk, but not as consistently. Inadequate sleep may promote obesity through many pathways, including biological (e.g., leptin/ghrelin production (89)) and behavioral pathways (e.g., late night snacking, food choices (90), emotional eating in response to stress (23), 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 (91). 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. However, 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 the fact that there are 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 BMI, to include more direct and precise measures of adiposity, are also important. For example, it has been suggested that physical and sexual abuse may pose a specific risk for severe obesity (18), and more fine-grained analysis could allow careful examination of risk pathways. Future work should also consider differences in how the timing and nature of specific stress exposures may shape obesity risk (e.g., would risk differ for infants compared with preschoolers who experience maltreatment or for children who experience community violence vs. 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 the effects of early life stress exposure on risk for obesity in children (6). Related intervention implications are outlined below and in Table 1.

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 developed 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 the 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) (92). Although developmental models have long highlighted the transactional nature of parent-child interactions, bidirectional processes (especially child contributions) are

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.	Reduce physiological effects of stress exposure (e.g., using stress-reduction techniques) with children.
Early life stress can shape obesity risk through multiple behavioral pathways.	Engage novel behavioral intervention targets such as sleep, self-regulation, and overall parenting support.
There are sensitive periods for both stress exposure and for response to intervention.	Deliver interventions at developmentally sensitive periods 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.	Identify individual child/parent characteristics that may moderate the association between early life stress and later obesity risk and use them to tailor the intervention approach (e.g., structure the intervention based on whether or not the child has high food reward sensitivity and whether or not the parent has depression); consider biologically sensitive intervention approaches.
Bidirectional associations between parent and child behaviors may shape obesity risk.	Address bidirectional effects: 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.	Support parents in their efforts 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.	Use behavior change approaches with parents 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.	Connect intervention efforts across social-contextual settings (e.g., home, school, community); combine broad approaches with efforts tailored to address individual child and family needs.

just beginning to be elucidated in the childhood obesity literature (59,61). 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 (93), and trials are under way to test whether engaging parents alone is effective (94). Although few obesity prevention interventions have focused on general parenting, some have shown promise (95,96). 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 causes, individual differences in child biological propensity for obesity, and difficulty engaging parents if they do not believe their children are at risk (97). Thus, supporting parents by first addressing their needs (e.g., using mindfulness to reduce stress) may be important for obesity-prevention parenting. Technology and e-health 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. In addition, dual-generation approaches that address both child and parent needs (e.g., colocating 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 the 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 a broad impact.

Conclusion

Obesity is a multifaceted problem that requires multilevel solutions. Stress exposure during childhood can convey greater risk than stress exposure later in the life-span, 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 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.

References

1. Lumeng JC, Wendorf K, Pesch MH, et al. Overweight adolescents and life events in childhood. *Pediatrics* 2013;132:e1506-e1512.
2. Gundersen C, Lohman BJ, Garasky S, Stewart S, Eisenmann J. Food security, maternal stressors, and overweight among low-income US children: results from the National Health and Nutrition Examination Survey (1999-2002). *Pediatrics* 2008; 122:e529-e540.
3. Danese A, Tan M. Childhood maltreatment and obesity: systematic review and meta-analysis. *Mol Psychiatry* 2014;19:544-554.
4. Blair C, Raver CC. Child development in the context of adversity: experiential canalization of brain and behavior. *Am Psychol* 2012;67:309-318.
5. Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychol Bull* 2011;137:959. doi:10.1037/a0024768
6. Wachs TD, Georgieff M, Cusick S, McEwen BS. Issues in the timing of integrated early interventions: contributions from nutrition, neuroscience, and psychological research. *Ann N Y Acad Sci* 2014;1308:89-106.
7. Entringer S, Buss C, Wadhwa PD. Prenatal stress, development, health and disease risk: a psychobiological perspective—2015 Curt Richter Award Paper. *Psychoneuroendocrinology* 2015;62:366-375.
8. Greenfield EA, Marks NF. Violence from parents in childhood and obesity in adulthood: using food in response to stress as a mediator of risk. *Soc Sci Med* 2009; 68:791-798.
9. Entringer S, Buss C, Swanson JM, et al. Fetal programming of body composition, obesity, and metabolic function: the role of intrauterine stress and stress biology. *J Nutr Metab* 2012;2012:32548. doi:10.1155/2012/632548
10. Dev DA, McBride BA, Fiese BH, Jones BL, Cho H. Risk factors for overweight/obesity in preschool children: an ecological approach. *Child Obes* 2013;9:399-408.
11. Hughes SO, Power TG, Liu Y, Sharp C, Nicklas TA. Parent emotional distress and feeding styles in low-income families. The role of parent depression and parenting stress. *Appetite* 2015;92:337-342.
12. McCurdy K, Gorman KS, Kisler T, Metallinos-Katsaras E. Associations between family food behaviors, maternal depression, and child weight among low-income children. *Appetite* 2014;79:97-105.
13. Kakinami L, Barnett TA, Séguin L, Paradis G. Parenting style and obesity risk in children. *Prev Med* 2015;75:18-22.
14. Oddo VM, Jones-Smith JC. Gains in income during early childhood are associated with decreases in BMI *z* scores among children in the United States. *Am J Clin Nutr* 2015;101:1225-1231.
15. Kendzor DE, Caughy MO, Owen MT. Family income trajectory during childhood is associated with adiposity in adolescence: a latent class growth analysis. *BMC Public Health* 2012;12:611. doi:10.1186/1471-2458-12-611
16. Sharkey JR, Nalty C, Johnson CM, Dean WR. Children's very low food security is associated with increased dietary intakes in energy, fat, and added sugar among Mexican-origin children (6-11 y) in Texas border Colonias. *BMC Pediatr* 2012;12: 16. doi:10.1186/1471-2431-12-16
17. Bauer KW, MacLehose R, Loth KA, Fisher JO, Larson NI, Neumark-Sztainer D. Eating- and weight-related parenting of adolescents in the context of food insecurity. *J Acad Nutr Diet* 2015;115:1408-1416.
18. Richardson AS, Dietz WH, Gordon-Larsen P. The association between childhood sexual and physical abuse with incident adult severe obesity across 13 years of the National Longitudinal Study of Adolescent Health. *Pediatr Obes* 2014;9:351-361.
19. Gunnar MR. Quality of early care and buffering of neuroendocrine stress reactions: potential effects on the developing human brain. *Prev Med* 1998;27:208-211.
20. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav* 2012;106:29-39.
21. Maniam J, Antoniadis C, Morris MJ. Early-life stress, HPA axis adaptation, and mechanisms contributing to later health outcomes. *Front Endocrinol (Lausanne)* 2014;5:73. doi:10.3389/fendo.2014.00073
22. Scott KA, Melhorn SJ, Sakai RR. Effects of chronic social stress on obesity. *Curr Obes Rep* 2012;1:16-25.
23. Dallman MF, Pecoraro N, Akana SF, et al. Chronic stress and obesity: a new view of "comfort food." *Proc Natl Acad Sci U S A* 2003;100:11696-11701.
24. Hillman JB, Dorn LD, Loucks TL, Berga SL. Obesity and the hypothalamic-pituitary-adrenal axis in adolescent girls. *Metabolism* 2012;61:341-348.
25. Kjölhede EA, Gustafsson PE, Gustafsson P, Nelson N. Overweight and obese children have lower cortisol levels than normal weight children. *Acta Paediatr* 2014;103:295-299.
26. Vanaelst B, Michels N, Clays E, et al. The association between childhood stress and body composition, and the role of stress-related lifestyle factors—cross-sectional findings from the baseline ChiBSD survey. *Int J Behav Med* 2014;21:292-301.
27. Reinehr T, Kulle A, Wolters B, et al. Relationships between 24-hour urinary free cortisol concentrations and metabolic syndrome in obese children. *J Clin Endocrinol Metab* 2014;99:2391-2399.
28. Veldhorst MA, Noppe G, Jongejan MH, et al. Increased scalp hair cortisol concentrations in obese children. *J Clin Endocrinol Metab* 2013;99:285-290.
29. Michels N, Sioen I, Boone L, et al. Cross-lagged associations between children's stress and adiposity: the Children's Body Composition and Stress study. *Psychosom Med* 2015;77:50-58.
30. Lumeng JC, Miller A, Peterson KE, et al. Diurnal cortisol pattern, eating behaviors and overweight in low-income preschool-aged children. *Appetite* 2014;73:65-72.
31. Miller AL, Clifford C, Sturza J, et al. Blunted cortisol response to stress is associated with higher body mass index in low-income preschool-aged children. *Psychoneuroendocrinology* 2013;38:2611-2617.
32. Miller AL, Sturza J, Rosenblum K, Vazquez DM, Kaciroti N, Lumeng JC. Salivary alpha amylase diurnal pattern and stress response are associated with body mass index in low-income preschool-aged children. *Psychoneuroendocrinology* 2015;53: 40-48.
33. Stel J, Legler J. The role of epigenetics in the latent effects of early life exposure to obesogenic endocrine disrupting chemicals. *Endocrinology* 2015;156:3466-3472.
34. Milagro FI, Mansego ML, De Miguel C, Martínez JA. Dietary factors, epigenetic modifications and obesity outcomes: progresses and perspectives. *Mol Aspects Med* 2013;34:782-812.
35. Crews D, Gillette R, Scarpino SV, Manikkam M, Savenkova MI, Skinner MK. Epigenetic transgenerational inheritance of altered stress responses. *Proc Natl Acad Sci U S A* 2012;109:9143-9148.
36. Mueller NT. The gut microbiome and childhood obesity: connecting the dots. *Child Obes* 2015;11:227-230.
37. Sudo N, Chida Y, Aiba Y, et al. Postnatal microbial colonization programs the hypothalamic-pituitary-adrenal system for stress response in mice. *J Physiol* 2004; 558:263-275.
38. Foster JA, McVey Neufeld K-A. Gut-brain axis: how the microbiome influences anxiety and depression. *Trends Neurosci* 2013;36:305-312.
39. Jašarević E, Howerton CL, Howard CD, Bale TL. Alterations in the vaginal microbiome by maternal stress are associated with metabolic reprogramming of the offspring gut and brain. *Endocrinology* 2015;156:3265-3276.
40. Karlsson CL, Onnerfalt J, Xu J, Molin G, Ahrne S, Thorngren-Jerneck K. The microbiota of the gut in preschool children with normal and excessive body weight. *Obesity (Silver Spring)* 2012;20:2257-2261.
41. Scheepers LE, Penders J, Mbakwa CA, Thijs C, Mommers M, Arts IC. The intestinal microbiota composition and weight development in children: the KOALA Birth Cohort study. *Int J Obes (Lond)* 2015;39:16-25.
42. Shao WH, Chen JJ, Fan SH, et al. Combined metabolomics and proteomics analysis of major depression in an animal model: perturbed energy metabolism in the chronic mild stressed rat cerebellum. *OMICS* 2015;19:383-392.
43. Boyle SH, Matson WR, Velazquez EJ, et al. Metabolomics analysis reveals insights into biochemical mechanisms of mental stress-induced left ventricular dysfunction. *Metabolomics* 2015;11:571-582.
44. Zhang A, Sun H, Wang X. Power of metabolomics in biomarker discovery and mining mechanisms of obesity. *Obes Rev* 2013;14:344-349.
45. Perng W, Gillman MW, Fleisch AF, et al. Metabolomic profiles and childhood obesity. *Obesity (Silver Spring)* 2014;22:2570-2578.
46. Mihalik SJ, Michaliszyn SF, de las Heras J, et al. Metabolomic profiling of fatty acid and amino acid metabolism in youth with obesity and type 2 diabetes: evidence for enhanced mitochondrial oxidation. *Diabetes Care* 2012;35:605-611.
47. Wahl S, Yu Z, Kleber M, et al. Childhood obesity is associated with changes in the serum metabolite profile. *Obes Facts* 2012;5:660-670.

48. Stratakis CA, Chrousos GP. Neuroendocrinology and pathophysiology of the stress system. *Ann N Y Acad Sci* 1995;771:1-18.
49. Kyrou I, Tsigos C. Stress hormones: physiological stress and regulation of metabolism. *Curr Opin Pharmacol* 2009;9:787-793.
50. Rajala MW, Patterson CM, Opp JS, Foltin SK, Young VB, Myers MG. Leptin acts independently of food intake to modulate gut microbial vomposition in male mice. *Endocrinology* 2014;155:748-757.
51. Evans GW, Fuller-Rowell TE, Doan SN. Childhood cumulative risk and obesity: the mediating role of self-regulatory ability. *Pediatrics* 2012;129:2010-3647.
52. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. Energy-dense, low-fiber, high-fat dietary pattern is associated with increased fatness in childhood. *Am J Clin Nutr* 2008;87:846-854.
53. Lundahl A, Nelson TD, Van Dyk TR, West T. Psychosocial stressors and health behaviors: examining sleep, sedentary behaviors, and physical activity in a low-income pediatric sample. *Clin Pediatr* 2013;52:721-729.
54. Bischoff AR, DalleMolle R, Silveira PP. Fetal programming of food preferences and feeding behavior. In: Rajendram R, Preedy VR, Patel VB, eds. *Diet, Nutrition, and Fetal Programming*. Cham, Switzerland: Springer; 2017:453-470.
55. Lampard AM, Jurkowski JM, Lawson HA, Davison KK. Family ecological predictors of physical activity parenting in low-income families. *Behav Med* 2013; 39:97-103.
56. O'Connor SG, Maher JP, Belcher BR, et al. Associations of maternal stress with children's weight-related behaviours: a systematic literature review. *Obes Rev* 2017; 18:514-525.
57. Gemmill AW, Worotniuk T, Holt CJ, Skouteris H, Milgrom J. Maternal psychological factors and controlled child feeding practices in relation to child body mass index. *Child Obes* 2013;9:326-337.
58. Tan CC, Holub SC. Emotion regulation feeding practices link parents' emotional eating to children's emotional eating: a moderated mediation study. *J Pediatr Psychol* 2015;40:657-663.
59. Derks IP, Tiemeier H, Sijbrands EJ, et al. Testing the direction of effects between child body composition and restrictive feeding practices: results from a population-based cohort. *Am J Clin Nutr* 2017;106:783-790.
60. Tate EB, Wood W, Liao Y, Dunton GF. Do stressed mothers have heavier children? A meta-analysis on the relationship between maternal stress and child body mass index. *Obes Rev* 2015;16:351-361.
61. Skouteris H, McCabe M, Ricciardelli LA, et al. Parent-child interactions and obesity prevention: a systematic review of the literature. *Early Child Dev Care* 2011;182:153-174.
62. Walton K, Simpson JR, Darlington G, Haines J. Parenting stress: a cross-sectional analysis of associations with childhood obesity, physical activity, and TV viewing. *BMC Pediatr* 2014;14:244. doi:10.1186/1471-2431-14-244
63. Lampard AM, Franckle RL, Davison KK. Maternal depression and childhood obesity: a systematic review. *Prev Med* 2014;59:60-67.
64. Gross RS, Velazco NK, Briggs RD, Racine AD. Maternal depressive symptoms and child obesity in low-income urban families. *Acad Pediatr* 2013;13:356-363.
65. Teixeira PJ, Carraça EV, Marques MM, et al. Successful behavior change in obesity interventions in adults: a systematic review of self-regulation mediators. *BMC Med* 2015;13:84. doi:10.1186/s12916-015-0323-6
66. Anderson SE, Sacker A, Whitaker RC, Kelly Y. Self-regulation and household routines at age three and obesity at age eleven: longitudinal analysis of the UK Millennium Cohort study. *Int J Obes (Lond)* 2017;41:1459-1466.
67. Graziano PA, Calkins SD, Keane SP. Toddler self-regulation skills predict risk for pediatric obesity. *Int J Obes (Lond)* 2010;34:633-641.
68. Thamotharan S, Lange K, Zale EL, Huffhines L, Fields S. The role of impulsivity in pediatric obesity and weight status: a meta-analytic review. *Clin Psychol Rev* 2013;33:253-262.
69. Riggs NR, Spruijt-Metz D, Chou CP, Pentz MA. Relationships between executive cognitive function and lifetime substance use and obesity-related behaviors in fourth grade youth. *Child Neuropsychol* 2012;18:1-11.
70. Liang J, Matheson BE, Kaye WH, Boutelle KN. Neurocognitive correlates of obesity and obesity-related behaviors in children and adolescents. *Int J Obes (Lond)* 2014;38:494-506.
71. Tomiyama AJ, Dallman MF, Epel ES. Comfort food is comforting to those most stressed: evidence of the chronic stress response network in high stress women. *Psychoneuroendocrinology* 2011;36:1513-1519.
72. Pieper JR, Laugero KD. Preschool children with lower executive function may be more vulnerable to emotional-based eating in the absence of hunger. *Appetite* 2013; 62:103-109.
73. Rollins BY, Loken E, Savage JS, Birch LL. Effects of restriction on children's intake differ by child temperament, food reinforcement, and parent's chronic use of restriction. *Appetite* 2014;73:31-39.
74. Domoff SE, Miller AL, Kaciroti N, Lumeng JC. Validation of the Children's Eating Behaviour Questionnaire in a low-income preschool-aged sample in the United States. *Appetite* 2015;95:415-420.
75. Asta K, Miller AL, Retzloff L, Rosenblum K, Kaciroti NA, Lumeng JC. Eating in the absence of hunger as a predictor of weight gain in low-income toddlers. *Pediatrics* 2016;137:e20153786. doi:10.1542/peds.2015-3786
76. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition* 2007; 23:557-563.
77. Renzaho AM, Dau A, Cyril S, Ayala GX. The influence of family functioning on the consumption of unhealthy foods and beverages among 1- to 12-y-old children in Victoria, Australia. *Nutrition* 2014;30:1028-1033.
78. Michels N, Sioen I, Braet C, et al. Relation between salivary cortisol as stress biomarker and dietary pattern in children. *Psychoneuroendocrinology* 2013;38:1512-1520.
79. Balantekin KN, Roemmich JN. Children's coping after psychological stress. Choices among food, physical activity, and television. *Appetite* 2012;59:298-304.
80. Francis LA, Granger DA, Susman EJ. Adrenocortical regulation, eating in the absence of hunger and BMI in young children. *Appetite* 2013;64:32-38.
81. Roemmich JN, Gurgol CM, Epstein LH. Influence of an interpersonal laboratory stressor on youths' choice to be physically active. *Obes Res* 2003;11:1080-1087.
82. Roemmich JN, Lambiase M, Salvy SJ, Horvath PJ. Protective effect of interval exercise on psychophysiological stress reactivity in children. *Psychophysiology* 2009;46:852-861.
83. Horsch A, Wobmann M, Kriemler S, et al. Impact of physical activity on energy balance, food intake and choice in normal weight and obese children in the setting of acute social stress: a randomized controlled trial. *BMC Pediatr* 2015;15:12. doi: 10.1186/s12887-015-0326-7
84. Metcalf B, Henley W, Wilkin T. Effectiveness of intervention on physical activity of children: systematic review and meta-analysis of controlled trials with objectively measured outcomes (EarlyBird 54). *BMJ* 2012;345:e5888. doi:10.1136/bmj.e5888
85. El-Sheikh M, Kelly RJ, Buckhalt JA, Benjamin Hinnant J. Children's sleep and adjustment over time: the role of socioeconomic context. *Child Dev* 2010;81:870-883.
86. El-Sheikh M, Buckhalt JA, Mize J, Acebo C. Marital conflict and disruption of children's sleep. *Child Dev* 2006;77:31-43.
87. Cappuccio FP, Taggart FM, Kandala N-B, Currie A. Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* 2008;31:619-626.
88. Miller AL, Kaciroti N, LeBourgeois MK, Chen YP, Sturza J, Lumeng JC. Sleep timing moderates the concurrent sleep duration–body mass index association in low-income preschool-age children. *Acad Pediatr* 2014;14:207-213.
89. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med* 2004; 141:846-850.
90. Lundahl A, Nelson TD. Sleep and food intake: a multisystem review of mechanisms in children and adults. *J Health Psychol* 2015;20:794-805.
91. Burt J, Dube L, Thibault L, Gruber R. Sleep and eating in childhood: a potential behavioral mechanism underlying the relationship between poor sleep and obesity. *Sleep Med* 2014;15:71-75.
92. Altman M, Wilfley DE. Evidence Update on the treatment of overweight and obesity in children and adolescents. *J Clin Child Adolesc Psychol* 2015;44:521-537.
93. Quattrin T, Roemmich JN, Paluch R, Yu J, Epstein LH, Ecker MA. Treatment outcomes of overweight children and parents in the medical home. *Pediatrics* 2014; 134:290-297.
94. Boutelle KN, Braden A, Douglas JM, et al. Design of the FRESH study: a randomized controlled trial of a parent-only and parent-child family-based treatment for childhood obesity. *Contemp Clin Trials* 2015;45:364-370.
95. Slusser W, Frankel F, Robison K, Fischer H, Cumberland WG, Neumann C. Pediatric overweight prevention through a parent training program for 2-4 year old Latino children. *Child Obes* 2012;8:52-59.
96. Smith JD, Montano Z, Dishion TJ, Shaw DS, Wilson MN. Preventing weight gain and obesity: indirect effects of the family check-up in early childhood. *Prev Sci* 2015;16:408-419.
97. Skouteris H, McCabe M, Swinburn B, Newgreen V, Sacher P, Chadwick P. Parental influence and obesity prevention in pre-schoolers: a systematic review of interventions. *Obes Rev* 2011;12:315-328.