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PEDIATRIC REVIEW

Parental influence on children's early eating environments and obesity risk: implications for prevention

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Most childhood obesity prevention efforts have focused on school-age children and adolescents and have had limited success. We argue that the first years of life, including the prenatal period, the postnatal suckling period and the transition to the modified adult diet, may provide opportunities for preventive interventions. These early periods are characterized by high plasticity and rapid transitions, and parents have a high degree of control over children's environments and experiences. Observational and experimental evidence reveal persistent effects of early environments on eating behavior and obesity risk, suggesting that interventions should be tested during these early periods. The central task parents have in early development points to their potential as key targets and agents of change in early preventive interventions. In this paper, we review evidence of early environmental effects on children's eating and obesity risk, highlighting ways that parental feeding practices and parents' own behaviors impact these outcomes and calling for further experimental research to elucidate whether these factors are indeed promising targets for childhood obesity preventive interventions.
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Introduction

In a recent review in this journal, Gluckman and Hanson[1] described how obesogenic environments affect epigenetic processes in early development, producing individual differences in obesity risk. In early development, plasticity is high and genetic potential can be adjusted to result in differing phenotypes, depending on environmental features.[1] Despite evidence that early environments may have powerful and persistent long-term effects on obesity risk, there have been few attempts to begin obesity prevention early in development. Most prevention efforts have focused on older children and adolescents and have been met with limited success.[2] In this paper, we focus on evidence for the impact of early environments on the development of ingestive behavior and obesity risk in the first few years of life.

Genetic contributions to individual differences in obesity risk are well documented,[3,4] but given our focus on identifying opportunities for early prevention, we focus on potentially modifiable aspects of early environments that may moderate obesity risk. Because human evidence is limited and largely derived from observational studies, we include relevant experimental data from animal research on other mammalian species. Our focus is on the first years of life, a period characterized by high plasticity, dramatic transitions in ingestive behavior and the potential for early experience to have persistent effects on subsequent obesity risk. In addition, parents have a relatively high degree of control over children's eating environments during this early period. This period of development includes the prenatal period, infancy and the transition to 'table foods,' which is relatively complete by the time children begin formal schooling.

Parental influence on eating and weight status in early life

The primary function of parenting is to meet the child's basic needs, including sustenance. Early in development, children are dependent on parents to provide food needed to sustain growth and health. In addition to providing genetic potential, parents shape the early environments in which

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children's genetic potential is expressed. Initially, *in utero*, the mother is the proximal environment, with an array of maternal factors affecting fetal and infant development. At birth, parents begin making decisions about breast-feeding or formula feeding, and later, about introducing solid and table foods. Although we use the term 'parents' throughout, we acknowledge that other caregivers also have critical tasks in shaping early eating environments; we use the term parents to refer to caregivers who are regularly responsible for the child's well-being in early life. Parental influence persists into middle childhood and adolescence, but as children develop, their ecologies widen to include additional extra-familial contexts and influences. The extended period of parental nurturance and offspring dependence is uniquely human, and parents' attitudes and behaviors are influenced by many other uniquely human factors, including culture, cuisine, economics, ethnicity and education.

Parents' own food preferences, intake patterns and eating behaviors influence the foods available to young children, and parents also serve as models for children's behavior, affecting early learning of food preferences and eating behaviors.[5] Parents shape the flavors that become familiar to children before birth and the foods available postnatally, and repeated exposure to flavors during the prenatal and postnatal periods affects children's subsequent acceptance of foods and flavors.[6–8] Humans do not have to learn to like sweet and salty tastes; thus, children will like and readily consume foods and beverages high in sugar and salt, which are often high in energy density. However, children's diets should be high in nutrient density but of moderate energy density, including a variety of foods that are not high in sugar or energy, such as vegetables. In the current environment, the availability of inexpensive palatable foods high in sugar, salt and fat can limit children's opportunities to learn to like and accept healthy foods, resulting in diets high in added sugar, fat and salt, and low in fruits and vegetables, complex carbohydrates and low-fat meat and dairy products,[9] dietary patterns that are linked to obesity risk. Vegetables are initially rejected by young children; early experience with these foods could potentially offset such neophobia and encourage childhood diets that are healthier and lower in energy density. Parents' feeding practices shape children's early experience with food; these practices are influenced by the broader environment and by parental perceptions of environmental threats to children's health.[10] Many traditional child-feeding practices used today evolved in response to environmental threats of food scarcity, which prevailed until recently. In these environments, food availability was unpredictable, and diets were plant-based, lower in palatability and energy density.[11,12]

Despite growing awareness that childhood obesity has become a major health threat, and that children's current environments are often characterized by food surfeit not scarcity, traditional feeding practices and the perception that 'a chubby baby is a healthy baby' persist, particularly among low-income and ethnic groups at highest risk for obesity.[13] These traditional practices include feeding frequently in response to any sign of child distress, offering preferred foods and using coercion to promote children's intake. Currently, these practices can exacerbate the effects of obesogenic environments, promoting eating in the absence of hunger, excessive energy intake, rapid weight gain and obesity.[5,14] Traditions, by definition, are slow to change, and altering them will be a major challenge.

In countries where adult obesity rates are high and children may live with one or two overweight parents,[15] children may be at increased risk due to genetics, environmental factors and gene-by-environment interactions and correlations. There is evidence that obese adults create 'obesogenic' family environments, which can exacerbate the impact of the larger obesogenic environment.[16] Developmental systems theories highlight periods of instability or transition as opportune times to promote change;[17] the prenatal period, the postnatal suckling period and the postnatal transition to the adult diet show promise for early prevention.

Prenatal period

The availability of nutrients and flavors in the uterine environment affect fetal growth and flavor experience and begin to lay the foundation for individual differences later in development. Both prenatal environments of maternal undernutrition and maternal overnutrition are linked with later obesity risk and increase future susceptibility to obesity in animal models through alterations in neural, metabolic and behavioral processes.[18,19] Levin and colleagues[20] have shown that pre- and postnatal dietary manipulations trigger reorganization of neural pathways related to energy balance in rat pups. In addition, maternal dietary intake during pregnancy influences flavor experience of the fetus *in utero*, which affects acceptance of foods later in development.[7,8] In the following section, we present evidence that the prenatal environment shapes neural, metabolic and behavioral systems to affect obesity risk.

Gluckman and colleagues[1,21] have described pathways from prenatal undernutrition to subsequent obesity as one example of predictive adaptive responses gone awry: the presence of an undernourished prenatal environment triggers physiological adaptations that prepare the fetus to store energy and survive in postnatal environments of food scarcity. These adaptations become problematic when the individual's postnatal environment is characterized by plenty, rather than scarcity. Numerous epidemiological and observational studies in humans have revealed associations between prenatal undernutrition and subsequent child health outcomes[22] in cases where environments experienced later in development are not characterized by food scarcity. In their early studies, Barker and colleagues[23–25] reported that lower birth weights were associated with increased likelihood of cardiovascular disease and higher systolic blood

pressure in adulthood; more recently, they found that those with lower birth weights were more likely to store fat centrally, a factor that has been associated with type 2 diabetes and cardiovascular disease. In these studies, low birth weight is considered a marker for fetal undernutrition and restricted fetal growth.[24,26–29] Observational studies reveal that individuals born under conditions where their parents did not have adequate access to food, such as famine[30,31] or poverty,[32] were more likely to be obese as adults.

Although most of the data on the impact of early nutrition on human growth and subsequent obesity risk have focused on fetal undernutrition, maternal and fetal overnutrition is an increasingly common problem; chronic positive energy balance affects the majority of the population. Experimental studies in humans are rare, but observational studies in humans have shown that gestational diabetes or excessive gestational weight gain is associated with fetal overnutrition and elevated birth weight, which predict later offspring obesity; elevated maternal pre-pregnancy weight status is also positively associated with higher birth weight and later obesity risk.[33–37] Cole and colleagues[38] describe two ways to end up with an overweight phenotype: by starting out large or by growing fast. Links between fetal overnutrition and obesity risk take on particular significance in countries where obesity is prevalent among adult women of childbearing age, as in the United States, where two-thirds of adults are overweight or obese,[39] and gestational weight gain guidelines are frequently exceeded.[34] In a study linking greater gestational weight gain to higher body mass index z-scores, greater adiposity and higher blood pressure at age 3, Oken *et al.*[34] highlighted this problem. Although previous guidelines for weight gain during pregnancy focused on reducing the risk of low birth weight, the Institute of Medicine has recently revised gestational weight gain guidelines, acknowledging childhood obesity risk as a potential adverse health outcome of excessive gestational weight gain.[40] The revisions include a modification of the recommended weight gains by maternal pre-pregnancy weight status and the inclusion of an obese category, for which the least weight gain is recommended.

Experimental research with animal models provides greater insight into the mechanisms linking maternal overnutrition during gestation to offspring obesity and comorbidities.[41–45] The offspring of pregnant dams fed excess calories gained more weight and had more body fat when fed a postnatal high-fat diet, compared to rats fed the same postnatal diet without the experience of excess calories during gestation.[45] Interactive effects of prenatal and postnatal overnutrition were revealed when rats were exposed to a 'junk food diet' either prenatally, postnatally, both or never. Rats with prenatal-only exposure were fatter than those without any exposure, but rats that were exposed to junk food throughout the study had the riskiest outcomes, including the highest levels of adiposity and hyperinsulinemia.[46] In a similar 2×2 experimental design, offspring of dams fed high-fat chow during pregnancy had higher adult body weights and blood pressure than offspring who experienced a standard maternal diet *in utero*, but rats experiencing effects of high-fat diets *in utero* and during suckling showed the greatest body mass and highest blood pressure, as well as hyperinsulinism and hyperleptinemia as adults. The epigenetic processes that begin with maternal obesity have a greater impact on those who are already genetically susceptible to obesity.[47,48] When rats predisposed (DIO) and resistant (DR) to diet-induced obesity were exposed to maternal obesity during gestation, the DIO offspring were fatter postnatally.[48]

The maternal diet during pregnancy can also influence offspring eating behaviors and weight through effects on the development of food and flavor preferences in humans. The infant brings a set of predispositions to early feeding interactions that evolved in response to a limited and unpredictable food environment:[6] a preference for sweet and salty tastes (tastes that could predict the presence of nutrients), a tendency to reject bitter and sour tastes (which could be toxic), a neophobic rejection of novel foods and flavors (which might be dangerous) and a predisposition to learn to prefer energy dense foods (which would be adaptive in contexts where food is scarce). The food industry has provided a wide range of inexpensive foods that are palatable because they are attuned to these predispositions. Current contexts, in combination with children's predispositions and traditional feeding practices, promote diets too high in added sugar, fat and salt, and total energy, while discouraging the consumption of complex carbohydrates, fruits and vegetables.[49]

Because flavor preferences are learned, children can learn to like healthy foods if given the opportunity. Research by Mennella *et al.*[7] has revealed that the fetus experiences flavors from the maternal diet as amniotic fluid is swallowed *in utero*. This experience influences infants' food and flavor preferences. Mothers who drank carrot juice during pregnancy had infants who showed greater acceptance of carrot-flavored cereal at weaning compared to infants without this familiarization.[7] Whether the fetus first becomes familiar with the flavors of French fries or carrots will depend on the mother's dietary choices during pregnancy. Early sensory experience during gestation and lactation can provide a 'flavor bridge',[8] facilitating the acceptance of foods consumed by the mother and promoting the transition to table foods.

Postnatal suckling period

Associations between prenatal undernutrition and adult obesity and health have been interpreted as evidence that prenatal influence is deterministic, but more recently, researchers have acknowledged that postnatal environments can modify prenatal effects. Waterland[50] described epigenetic processes that continue after birth in mice, especially

during periods of transition. Levin[48] reported that individual differences in intake and weight patterns persist after the second week of life if, during subsequent development, rats remain in stable dietary environments. Many humans who are at genetic risk for obesity are also born into obesogenic family environments and remain in these environments, masking the plasticity in their trajectories toward obesity. Although individuals tend to stay in broadly stable environments, it is important to consider that in the case of human development, there is potential for environmental change and variability, and that individuals experience rapid and drastic transitions in their experiences with food, particularly during the first years of life.

Lucas *et al.*[51] argued that early postnatal experience is central to the prediction of developmental trajectories, asserting that it is not the prenatal environment that is directly responsible for obesity and its comorbidities later in life, but rather what happens between birth and the emergence of the adult phenotype: in particular, early postnatal growth. Rapid growth during the first few months of life is a robust predictor of elevated obesity risk later in life,[52–55] and this association is also observed among infants with normal birth weights, in whom rapid growth does not reflect 'catch-up' growth. This relationship persists across studies: those in developed and developing countries, using various time points and indices of growth,[46] and adjusting for other factors linked to the prenatal environment, such as smoking and gestational weight gain.[56]

One factor that has been linked to early growth is the first decision parents make about feeding their infant: whether to breastfeed or formula feed. During the early months after birth, the growth rate of breastfed infants slows relative to formula-fed infants, and their trajectories tend to track at lower levels during later infancy. These differences are evident when comparing the recently released WHO growth charts, based on the growth of healthy breastfed infants, and the CDC growth charts, which are based predominately on formula-fed infants.[57] Growth of obese mothers' infants can be affected because obese mothers have more problems breastfeeding. This is in part due to a diminished prolactin response to infant suckling,[58] which leads to delayed onset of lactation and breastfeeding failure.[59] In the United States, mothers from lower socioeconomic, minority or underserved groups are also less likely to initiate breastfeeding and are more likely to formula feed their infants,[60] perhaps contributing to the elevated obesity risk in their children.

Meta-analyses[61,62] and epidemiological data[63,64] provide evidence that breastfeeding may be protective against obesity in childhood and beyond.[65–69] Breastfeeding also has positive physiological effects on brain development, glucose metabolism and gut and immune functioning, all of which may affect obesity risk. Potential mechanisms explaining these associations include physiological and metabolic effects of breastfeeding, effects on growth rate, facilitated learning of self-regulation in breastfed infants and confounding by sociodemographic correlates of both breastfeeding and healthy weight outcomes.[61,70] Using nationally representative US data, Grummer-Strawn and Mei[63] showed a dose–response, protective association between breastfeeding duration and overweight risk at age 4 in non-Hispanic White children but not among other groups. In two systematic reviews, Owen and colleagues[62,68] found an overall small protective effect of breastfeeding, acknowledging the potential role of confounding in observational studies, given the larger effects found in smaller studies and the attenuation of effects when adjusting for demographic factors. Overall, the protective effects of breastfeeding reported from observational studies are reduced after adjustments for a myriad of confounding factors such as maternal weight status, race/ethnicity and socioeconomic status.[62]

Two experimental studies have assessed the impact of breastfeeding on growth and obesity risk in humans, although this was not the primary focus of either study. Lucas and colleagues used an experimental design to assess the effects of early postnatal diets on premature infants' growth and development by randomizing infants to receive banked breast milk, fortified preterm formula or standard formula. Breast milk consumption was associated with decreased blood pressure in adolescence,[71] and preterm formula was associated with higher intelligence quotients[72] and with more rapid neonatal growth, but among these preterm infants, early postnatal diet was not associated with differences in growth measures at either 9 or 18 months or 7.5–8 years.[73] In a second study using an experimental design, Kramer *et al.*[74] conducted a large cluster-randomized intervention trial. Hospitals in Belarus ($N=34$) were randomly assigned to continue their standard care or to adopt the procedures of the Baby Friendly Hospital Initiative to promote longer duration and exclusivity of breastfeeding. In addition to breastfeeding outcomes, measures of infant health and growth were obtained. Although the experimental and control groups differed in breastfeeding duration and exclusivity, there were no differences in weight status at 6.5 years between groups.[75] However, it is important to note that treatment and control groups both included participants who were breast-feeding and formula feeding. In addition, the findings from these studies are limited in the first case to premature infants[71–73] and in the second to individuals living in Belarus.[74,75] Kramer *et al.*[75] acknowledged that the prevalence of obesity in Belarus is lower than in the United States and much of western Europe, limiting the generalizability of the findings and suggesting that in Belarus, maternal diets may be healthier, breast milk composition may be different and children's subsequent environments may be less obesogenic. However, these findings suggest that confounding factors may be partially responsible for breastfeeding's protective effect.

Although the effects of breastfeeding on obesity risk can be disputed, breastfeeding has other benefits that can promote healthier diets, perhaps reducing obesity risk. Experimental research with humans indicates that healthy

food preferences can be learned during the suckling period, consistent with classic animal studies showing flavor transmission during weaning and its effects on rat pups' flavor preferences.[76] Infants whose mothers had regularly consumed carrot juice during lactation had fewer negative reactions to carrot-flavored cereal 4 weeks after the introduction of solids, compared to infants who had no previous experience with carrot flavors.[7] In addition, in a study of 4- to 6-month-old infants, repeated exposure to pureed vegetables led to increased intake of those vegetables, and these effects were greater among breastfed than among formula-fed infants[77] indicating that feeding mode moderated the effects of exposure and also providing support for the interaction of early and later environmental effects. Forestell and Mennella[78] assert that the initial advantages conferred by breastfeeding must be followed by continued repeated exposure to fruits and vegetables after solids are introduced. Overall, human and animal research shows plasticity during the postnatal suckling period, and the human research highlights potential advantages of breastfeeding when mothers eat a healthy and varied diet.

Transition to a modified adult diet

The plasticity that characterizes early human development persists throughout the transition to a modified adult diet, a period characterized by dramatic, rapid changes in the foods offered to children as 'table foods' are introduced. During this period, extensive learning about food and eating occurs; food preferences are learned by familiarization and associative conditioning and by observing the eating behavior of others. As young omnivores, children are prepared to learn food and flavor preferences, and the transition from the exclusive milk diet to 'table foods' proceeds rapidly, beginning during the second half of the first year of life. Data from the United States reveal that by 18 months of age, about 80% of children's energy intake comes from 'table foods'.[79] A key question is: What foods are these young children consuming? The family's cultural, ethnic and socioeconomic characteristics will influence what foods are on the family table, influencing children's early food preferences and intake patterns. Unfortunately, the current adult diet that most children transition to is obesogenic.[80] Parents' feeding strategies are used to influence what and how much children eat and can moderate broader obesogenic influences during this key transition period.[5]

In general, we learn to prefer what becomes familiar,[81] and parents have a critical task in facilitating repeated experiences with new foods. Such experiences influence children's preferences for foods and flavors during the transition to the adult diet, when many new foods are introduced in the home environment.[82,83] Research reveals that infants who were repeatedly exposed to a variety of solid foods during infancy showed greater acceptance of fruits and vegetables in childhood,[84] and experience with some fruits and vegetables during infancy resulted in acceptance of similar foods in early life.[85,86] Unfortunately many parents are unaware that repeated exposure is necessary to promote liking and do not persist in presenting new foods if they are initially rejected.

Observational learning also affects children's intake; observing others consuming healthy foods can promote children's acceptance of these foods. Because children usually eat in social contexts, there are many opportunities for parents, peers and siblings to model healthy (or unhealthy) eating behaviors. Mothers who drank more milk had daughters who drank more milk, were more likely to meet dietary recommendations for dairy-related nutrients and had higher bone density.[87] Adult models can be also effective at increasing children's willingness to try novel foods,[88–90] especially when the models eat enthusiastically[91] and when both the models and the children are eating the same foods.[88]

Traditional feeding practices are often used to promote children's intake of healthy foods or to limit consumption of unhealthy foods, but these tactics can be counterproductive. However, when children were pressured or coerced to eat (for example, 'Finish your soup') or were given rewards for eating a food (for example, 'If you finish your peas, then you can watch TV'), preference and intake of the healthy foods decreased.[82,92] Corroborating these experimental findings, in a retrospective study, young adults reported that the foods they disliked as adults were those they had been coerced to eat as young children.[93] To limit consumption of 'junk' foods, parents may restrict these foods believing that this will decrease children's consumption. Again, these tactics may be counterproductive; restricting access to a food tends to promote children's interest in and consumption of that food when it is subsequently available.[94–96] Attempts at controlling children's intake, which can include prompts to eat in the absence of hunger (for example, 'It's time to eat') or to eat beyond satiety (for example, 'Finish your peas') can also increase children's responsiveness to external, contextual cues, which they can learn to use to control intake rather than internal cues signaling hunger and fullness. There is limited experimental evidence that these practices may contribute to children's diminished abilities to self-regulate their energy intake,[95,97,98] by increasing responsiveness to palatable food and to portion size as a determinant of meal size.

During early childhood, children are also learning how much to eat, and parents can determine the portions and energy density of the foods served to children. Experimental evidence shows that children eat more when they are served larger portions, and portion size effects on energy intake have been observed among children as young as 2 years.[99,100] More recently, it has been shown that the effects of larger portions extend beyond single meals, promoting increased energy intake over a 2-day period.[101] Recent work from our laboratory revealed that portion size

effects are moderated by child weight status: overweight children showed greater increases in intake with increasing portion size.[102]

Under some conditions, infants and young children are sensitive to the energy density of foods in the diet and can increase their consumption when the energy density of the diet is reduced.[103,104] These findings are consistent with experimental animal model research showing adjustments in energy intake in response to changes in energy density.[105] Although children may respond by consuming more when the energy density of foods in the diet is reduced, they may not adjust their intake in response to increases in energy density, consistent with findings that adults tend to be more responsive to energy deficits than to energy surfeit.[106] Children consumed more calories when served higher energy density meals,[107–109] and recent research reveals that effects of higher energy density on energy intake persist across meals and have a cumulative impact on energy intake, producing significant increases in total energy intake over a 2-day period.[107] Chronic consumption of diets high in energy density is positively associated with childhood obesity and predictive of later adiposity in both industrialized and developing countries.[110–116] The combined effects of portion size and energy density can promote excessive energy intake by children, resulting in a significant accumulation of excess calories over multiple meals.[117,118]

The broader obesogenic environment becomes more influential during the transition to the adult diet, but parents can still retain relatively high levels of control over their children's environments and thus have the potential to moderate obesogenic influences during this period of rapid transitions and learning. Research shows that children learn to like and eat what others in their environment eat and offer to them. Although creating possibilities for the promotion of healthy eating, observing the eating of others can also contribute to unhealthy phenotypes in young children if others are making unhealthy choices. For example, French fries are the most frequently consumed vegetable among adults[119] and also among 15-month olds.[80]

Implications for childhood obesity prevention

Observational studies support the hypothesis that these early periods of rapid transition and development show promise as targets for childhood obesity prevention research. Research using experimental and quasi-experimental designs is needed to provide additional evidence regarding the efficacy of early interventions to prevent obesity. Additional experimental evidence is needed in some of these areas, as well as pilot tests of interventions focusing on these factors and interactions with genetic factors. Below, we list some potential targets of future research toward the development of preventive interventions, based on an integration of the evidence for early environmental and epigenetic effects on eating and weight status in the prenatal period, the postnatal suckling period and the transition to the modified adult diet. Research is also needed to provide evidence on how to effect change in traditional feeding practices that may exacerbate children's obesity risk in today's environment, particularly among low-income and minority groups at highest risk:

- Target parents and other caregivers, especially those who are overweight
- Promote healthy maternal weight before pregnancy
- Prevent excessive gestational weight gain
- Promote healthy and varied diets during pregnancy
- Provide guidance on early feeding practices:
  - Promote exclusive, long duration of breastfeeding
  - Help parents learn to discriminate hunger from other signs of distress
  - Teach parents alternative soothing strategies besides feeding
  - Teach parents feeding approaches to promote longer sleep duration in infancy
  - Provide guidance on the introduction to solids
  - Provide guidance about alternative, child-centered feeding practices, avoiding traditional, controlling feeding practices
- Provide information on appropriate portion sizes
- Provide information on what 'table' foods to introduce and strategies to promote acceptance (for example, repeated exposure, modeling)
- Continue these efforts throughout early development (that is, prenatal period, infancy, early childhood) to maximize potential effects during multiple periods of developmental instability and transition

The probabilistic nature of development suggests that these efforts are useful even when prenatal conditions were less than ideal. Animal model research corroborates this idea, including evidence that it is possible to offset effects of prenatal environments by certain postnatal experiences, through the provision of leptin[120] or exercise,[121] and by handling pups during the postnatal period.[122] Further exploration of these areas will provide an evidence base for early preventive interventions that could set the stage for healthy trajectories (although it is also important to note that early prevention efforts will not 'inoculate' children against the effects of broader obesogenic environments). Parents and caregivers have the opportunity to be agents of change throughout this ongoing process because they have the ability and the responsibility for shaping infants and children's early food environments at a point in development when these environments are likely to impact children's food preferences and eating behaviors. It is likely that effects would be most robust in contexts where the epidemic is worst (for example, in low-income families[13]), highlighting a need for additional research in these contexts to facilitate appropriate tailoring of interventions.

Conclusion: a developmental approach to obesity prevention and maintaining healthy trajectories

Although research has shown effects of perinatal and early childhood environments on child eating and weight, most childhood obesity prevention efforts have focused on periods of the life span that follow these early, highly plastic developmental periods, after children's initial growth trajectories and eating habits have already been established.[2] In early life, children's ecologies are narrow, providing opportunities to establish healthy trajectories and habits before children's worlds become more complex. In addition, developmental systems theories argue that periods of transition and instability, such as those in early life, offer opportunities for systemic reorganization, intervention and change.[17] Although outside the scope of this paper, healthy practices in other contexts such as schools and efforts to alter the broader obesogenic environment are additional urgent goals. In the meantime, the efforts described herein could potentially have robust effects on early physiology, metabolism, growth and eating behaviors, increasing the probability of adaptive ontogeny into childhood and beyond.

Conflict of interest

The authors declare no conflict of interest.

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