Perspective: Childhood Obesity Requires New Strategies for Prevention

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ABSTRACT

The prevalence of obesity among youth in the USA is currently >18% with projections that more than half of today's children will be obese as adults. The growth trajectory of children more likely to become obese is determined by weight in earliest childhood, and childhood body mass index (BMI) tracks through adolescence and adulthood. Childhood consequences of obesity include increased risk of asthma, type 2 diabetes mellitus, orthopedic disorders, and reduced academic performance. Health implications of obesity in adulthood include premature coronary artery disease, hypertension, type 2 diabetes, stroke, and certain cancers, contributing to the leading causes of adult mortality. Early childhood obesity is influenced by prenatal exposure to maternal obesity and environmental obesogens, and is associated with poverty, food insecurity, and poor nutritional quality. New strategies for primordial prevention of early childhood obesity require focusing attention on growth parameters during the first 2 y of life, with support for increasing the duration of breastfeeding, and improvements in dietary quality and availability, particularly the reduced consumption of added sugars. Reducing the prevalence of obesity among adolescent females and reducing exposure to environmental obesogens may reduce the prevalence of transgenerational obesity. The reduction of early childhood obesity could improve population health, quality of life, and longevity throughout the life course. *Adv Nutr* 2020;11:1071–1078.

Keywords: early childhood obesity, obesity/morbidity, body mass index, breastfeeding, population health

Introduction

Despite major national and state-level efforts, by 2016 the prevalence of obesity in the USA had increased to 39.8% among adults (compared with 33.7% in 2007–2008) and to 18.5% among youth <18 years of age (from 16.8% in 2007–2008) (1, 2). Based on 2016 levels of childhood obesity in the USA, simulated growth trajectories predict 57% of today's children will be obese at the age of 35 y (3). The consequences of obesity contribute to the leading causes of death and disability among adults: cardiovascular diseases including premature coronary artery disease, hypertension, atrial fibrillation and stroke, cancer, osteoarthritis, type 2 diabetes, and chronic kidney and liver diseases (4). In contrast to adults, mortality related to obesity among youth is rare (5), contributing to complacency regarding the health implications and morbidity of childhood obesity, including early onset of type 2 diabetes and hypertension. Obesity-related conditions progress through adulthood affecting all areas of adult well-being and life expectancy (6). Thus, it is imperative that public policy interventions be implemented to alter the development of obesity in childhood.

Growth trajectories for obesity are established in infancy and early childhood, and track into adulthood (3). Previous publications have recommended initiating screening for childhood overweight and obesity at ages 6–12 y, however, this may miss an important window during which obesity may be developing in many younger children (7–9). By waiting until age 6 y, these positions fall into the category of primary prevention of obesity-related health morbidities. This review summarizes contemporary data on lifelong health consequences of pediatric obesity, pediatric obesity growth trajectories, causes of childhood obesity, and new strategies for the prevention and reduction of early childhood obesity, including population-level, primordial efforts to

The authors reported no funding received for this work.
Author disclosures: The authors report no conflicts of interest.
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Abbreviations used: DM, diabetes mellitus; SNAP, Supplemental Nutritional Assistance Program.

Box 1: Key points

- Childhood obesity often begins in utero and early infancy. Maternal obesity, excessive weight gain during pregnancy, and rapid weight gain during the first 2 y of life are associated with childhood obesity.
- Children who are obese by age 5 y are more likely to be obese as adolescents, and adolescents with obesity are highly likely to be obese as adults.
- Obesity during childhood is associated with increased risk of asthma, type 2 diabetes, orthopedic disorders, and reduced academic performance.
- Obesity during pregnancy is associated with increased risk of miscarriage, birth defects among newborns, and adult obesity among offspring.
- Obesity during adulthood contributes to the major causes of adult mortality: premature coronary artery disease, hypertension, stroke, chronic kidney and liver disease, and many types of cancer.
- New strategies for primordial prevention of early childhood obesity include achievement of healthy maternal weight prior to pregnancy, widespread adoption of breastfeeding for the first 6 mo of life, careful monitoring and intervention for excessive weight gain during the first 2 y of life, reducing consumption of added sugars, such as juices, among children, improving dietary quality and availability for children, and reducing exposures to environmental obesogens.

reduce the risk factors predisposing to obesity, and the life course health consequences and costs of childhood obesity (10, 11) (**Box 1**).

Current Status of Knowledge

Childhood obesity: causes and growth trajectories

Extensive data now exist to help understand the etiology of obesity as a multigenerational disease that begins during fetal life, with multiple contributing causes as summarized in **Table 1** (12). Viewing obesity as a lifetime disease, with origins preconception, in utero, and during early infancy with intergenerational effects is essential to guide efforts to reduce obesity and cardiovascular disease of adulthood.

Prenatal causes.

Preconception maternal and paternal dietary quality, weight status, assisted reproductive technology involving embryo culture, and environmental exposures alter the developmental plasticity of gametes, and subsequent fetal programming, resulting in postnatal cardiometabolic disease risk (12, 13). Prenatal or early life exposure to endocrine disruptor chemicals (also known as obesogens), such as air pollutants or pesticides, at a critical time for differentiation of mesenchymal stem cells into either adipocytes or osteoblasts may result in enhanced adipocyte numbers, which is considered irreversible and may be transmitted across future generations (13).

A mother who begins pregnancy obese has a significantly higher risk of late childhood obesity in her offspring (OR = 4.47; 95% CI: 3.99, 5.23) (14). The prevalence of prepregnancy obesity in the USA in 2015 was ∼26%, a 2% absolute increase since 2011 (15). For the developing fetus in the setting of maternal obesity, exposure to increased concentrations of inflammatory cytokines, hypermethylation of DNA, and histone modification produce epigenetic changes associated with increased risk of obesity in both the child and subsequent generations (13). Estimates of the proportion of childhood overweight/obesity prevalence attributable to maternal overweight, obesity, and excessive gestational weight gain ranged from 10% to 22% in a recent meta-analysis (14).

Early childhood contributions to obesity.

Methodologic issues complicate the assessment of the effect of feeding infants formula versus breast-feeding for the first 6 mo of life on childhood obesity. A 2013 meta-analysis suggests a longer duration of breastfeeding results in a 13% lower prevalence of childhood obesity (16). Exclusive breastfeeding for the first 6 mo of life is recommended with a Healthy People 2020 target in the USA of 60%, compared with the current level of 25% (17, 18). Breastfeeding is lowest among families living under 200% of the USA poverty level, lower maternal educational attainment, younger maternal age, and among African Americans or Hispanics compared with Caucasians (19).

The health effects of a suboptimal diet and high BMI contributed to an estimated 11 million deaths among adults globally in 2017 (20). The rapid and fundamental changes in food and beverages has translated into the consumption of 80% of calories from packaged foods and beverages among Americans, with >70% considered ultraprocessed foods (21). The contribution of added sugars to poor cardiometabolic health, obesity, and type 2 diabetes mellitus (DM) is well-documented (22). Among US children ages 2 y and above, added sugars accounted for 14% of daily caloric intake in 2013–2018, versus the recommended intake <5–10% of total calories for children and adolescents (23). The consumption of sugar-sweetened beverages, accounting for more than half of dietary added sugars in the USA, begins in the first year of life, with 43% of infants and 72% of toddlers consuming ≥1 sugar-sweetened beverage or dessert daily in 2009 (24). Sugar-sweetened beverage consumption has been positively associated with higher BMI among children (24–26).

Socioeconomic factors and obesity.

Poverty, food insecurity, social stressors, rural environments, and lower educational attainment of parents are important associates of early childhood obesity (27–29). Young children with obesity are more likely to live in poverty and in

TABLE 1 Risk factors for the development of childhood obesity during prenatal, neonatal/infancy, and childhood/adolescent periods

Period	Risk factors (individual, family, and societal levels)
Prenatal	Maternal prepregnancy obesity or malnutrition
	Maternal smoking, diabetes, hypercholesterolemia
	Exposure to environmental toxins/endocrine disruptors
	Excessive weight gain during early pregnancy
	Paternal obesity
	Antibiotic exposure
	Genetic variants and syndromes
Neonatal and infancy	Preterm or caesarean delivery
	Birth weight >4 kg
	Formula feeding or short duration of breastfeeding <4 mo
	Rapid weight gain during infancy, especially first 6 mo
	Prolonged or repeated exposure to broad-spectrum antibiotics in first 23 mo
	Exposure to environmental toxins/endocrine disruptors
	Consumption of unhealthy diet: excess added sugars
Childhood and adolescence	Social: poverty, low educational attainment, food insecurity, family stressors
	Family: parental obesity; family history of hypertension, dyslipidemia, early cardiovascular disease, or stroke
	Physical: inadequate exercise, increased screen time, inadequate sleep, obstructive sleep apnea
	Poor nutritional intake: obesogenic diet including excessive intake of sugar-sweetened beverages and ultraprocessed/fast foods

households with lower educational attainment (28, 29). Food insecurity, defined as a lack of dependable, regular access to high-quality food, affects ≥12% of households and almost 17 million children in the USA who do not know when, or how adequate, their next meal will be (30). Obesity is present in 30% of Hispanic children living in households with food insecurity, with the highest rates of obesity (40%) among American Indians (31, 32). Episodic household food shortages are associated with the consumption of more energy-dense/nutrient-poor foods when available, with the average added sugar intake over 70 g daily, compared with the recommended level of 25 g or less daily (33).

Pediatric obesity growth trajectories.

The growth trajectory of children more likely to become obese is determined by weight in earliest childhood, and childhood BMI tracks through adolescence and adulthood (27, 34). Infants and toddlers with rapid postnatal growth, as evidenced by crossing weight-for-length percentiles in the first 3–6 mo of life or accelerating BMI between the ages of 2–6 y, were more likely to be obese by age 12–14 y (34, 35). Between 70 and 90% of children with obesity in kindergarten were obese through age 14 y, independent of sex, race, or socioeconomic backgrounds (34, 35). Approximately 70–80% of adolescents with overweight or obesity will be obese as adults (36, 37).

Clinical presentation and health implications of obesity in childhood

Health implications of obesity affect virtually every organ system, with some effects well-recognized in childhood, whereas other long-term effects manifest in adulthood.

Morbidity associated with obesity in childhood and adolescence.

In addition to well-known comorbidities of childhood obesity as summarized in **Table 2**, additional adverse health effects of obesity among children and adolescents include reactive airway disease and increasing prevalence of type 2 DM (38, 39). The risk of asthma among youth with obesity is almost twice that of normal weight children (38). Prediabetes with abnormal fasting glucose and elevated hemoglobin A1c is prevalent among 17% of youth in the USA, and is highly associated with obesity (40). Type 2 DM, once diagnosed in middle-aged adults with obesity, is increasingly detected among adolescents by age 14 y. Type 2 DM increased to an incidence of 13.8/100,000 among US youth aged 10–19 y in 2014–2015, and is projected to quadruple between 2010 and 2050 (39, 41). By adulthood, these youth have experienced years of chronic exposure to metabolic and atherogenic abnormalities, who have higher odds of diabetic-related complications and premature cardiovascular mortality compared with those with type 1 DM (42). Moreover, those with type 2 DM who develop acute myocardial infarction have increased complication rates and mortality (43).

Effects of obesity on reproductive health.

For adolescent and young adult females, obesity is associated with polycystic ovarian syndrome, decreased fertility, and increased risks of complications of miscarriage, preterm delivery, or stillbirth with pregnancy (44, 45). Offspring born to obese women are at increased risk of birth defects, including congenital heart disease, as well as elevated blood pressure and lipid abnormalities (46, 47). The likelihood of obesity in young adulthood for offspring of obese parents has increased by 2- to almost 6-fold, depending on if 1 or both parents were obese (48).

TABLE 2 Lifelong health consequences of obesity (6, 49, 50, 51)

Domain	Example
Psychological	Sports and social isolation
	Bullying and cyber-texting
	Attention deficit disorder
	Work bias
	Low self-esteem
	Depression
Neurological	Cognitive dysfunction
	Reduced school performance
	Headaches
	Pseudotumor cerebri
	Stroke
Pulmonary	Asthma
	Obstructive sleep apnea
Cardiovascular	Elevated blood pressure, hypertension
	Left ventricular hypertrophy
	Decreased cardiac contractility
	Lipids: elevated cholesterol and triglycerides, low high-density lipoprotein
	Premature atherosclerosis, ischemic heart disease
	Atrial fibrillation
	Heart failure
Gastrointestinal	Nonalcoholic fatty liver disease, cirrhosis, and end-stage liver disease
	Gallstones
	Gastroesophageal reflux
Renal	Chronic kidney disease
	Urolithiasis
Endocrine	Early puberty
	Insulin resistance
	Diabetes mellitus, type 2
Reproductive	Polycystic ovarian syndrome
	Infertility
	Miscarriage
	Preterm birth
	Macrosomia
	Birth defects
Musculoskeletal	Pes planus, genu valgum
	Slipped capital-femoral epiphysis
	Blount's disease of knees
	Lower extremity fractures
	Degenerative joint disease/arthritis
	Gout
Skin	Striae
	Acanthosis nigricans
	Venous stasis disease
Cancer	Meningioma, thyroid, breast (postmenopausal), esophageal adenocarcinoma, liver, gall bladder, pancreas, kidney, uterus, ovarian, colorectal, multiple myeloma

Effects of obesity on adult-onset, chronic noncommunicable diseases.

The life-long consequences of obesity from childhood into adulthood include increased risk of cardiovascular disease, cancer, disability, and shortened life expectancy (6, 52–55). Obesity-related cardiovascular disease accounted for >4 million deaths and 120 million disability-adjusted life-years globally in 2015 (6). The economic burden of caring for obesity-related cardiovascular diseases is staggering: total costs are estimated to increase in the USA by $28 billion dollars annually between 2015 and 2035, from the current level of ≥$351 billion dollars (4, 56).

The origins of atherosclerotic vascular disease associated with childhood obesity have been documented for several decades, although the clinical consequences of hypertension, premature ischemic heart disease, and stroke become clinically apparent in adulthood (36, 52, 54, 57). The age at incident cardiovascular events is decreasing in some demographic groups and may be a principal driver of the plateau in national age-adjusted death rates due to cardiovascular diseases over the past decade (4).

The pathophysiology of adipose tissue promoting and accelerating cancer development shares a common pathway of proinflammatory changes with cardiometabolic disorders (58). By adulthood, excess weight is associated with a higher risk of ≥13 different types of cancer, accounting for 40% of all cancers diagnosed in 2014 (59). These cancers, particularly colon cancer, are being diagnosed with increasing frequency among young adults with obesity (60). Improved awareness of the role of obesity in the promotion of multiple forms of cancer should lend urgency to the need for prevention, treatment, and control of childhood obesity.

Strategies for the prevention of early childhood obesity

The US Healthy People 2020 goal aimed to lower the proportion of obesity among young children aged 2–5 y from 10.4% in 2008 to 9.4%; the 2030 goals are in progress (61). Comprehensive recommendations to address obesity in older children and adults focus on individual nutritional and behavioral changes, which demonstrate limited success (17, 49). Primordial prevention of overweight and obesity in early childhood incorporating environmental, societal, and policy changes may have the largest impact and opportunity for lasting improvement in cardiovascular health across future generations, with strategies listed in **Table 3** (10, 11, 17, 62, 63).

Broaden support for early breastfeeding.

Improving the rate of initiation and duration of exclusive breastfeeding for the first 6 mo of life could be expected to reduce childhood obesity by ≥13–30% (64, 65). Governmental and health care policy support and changes in infant formula purchasing agreements are necessary to increase participation in the effective Baby-Friendly Hospitals Initiative for successful breastfeeding (66). A major barrier to continued breastfeeding includes the need to return to work soon after birth, which disproportionately affects lower income households (67). Extending paid maternity leave has been associated with increased rates and longer duration of breastfeeding; in turn, this can be expected to reduce obesity in later childhood and adulthood by ≥12–15% (64, 68).

TABLE 3 New strategies for primordial prevention of early childhood obesity

Domain	Recommendation
Support for early breastfeeding	Increase proportion of deliveries at Baby-Friendly Hospitals Extend support for breastfeeding for first 6 mo of life
Focus attention on growth in first 2 y of life	Family screening for newborns at increased risk of development of obesity Monitor crossing of height-for-length percentiles in first 2 y of life Family-based feeding interventions
Reduce consumption of added sugars in children's diets	Target reduction of added sugars to <12 g daily for first 3 y of life Reduce fruit juice intake in first years of life
Improve dietary quality and availability	Subsidize fruit and vegetables
Invest in nutritional support for highest risk populations	Expand SNAP food supplementation benefits Increased support for food banks
Reduce obesity among adolescent females	Educational and activity programs Require physical education throughout high school Ensure healthy options for school lunch and afterschool programs Food delivery programs for highest risk adolescents
Increase efforts to reduce exposure to environmental obesogens	Restrict pesticide usage Reduce exposure to environmental obesogens Invest in research regarding obesogens effects during pregnancy and early childhood

SNAP, Supplemental Nutritional Assistance Program.

Focus attention on growth parameters in the first years of life.

Identifying families and newborns at increased risk of obesity as listed in Table 2 could prompt early home health visits and more frequent weight and feeding monitoring (17). Avoidance of fruit juices in the first year of life, with efforts to make water the normative beverage after the age-appropriate consumption of milk is advocated (69).

Reduce the consumption of added sugars in children's diets.

The implementation of taxation on sugar-sweetened beverages could reduce consumption by >25% and result in estimated reduced direct medical costs of $23 billion dollars annually by 2025, in addition to averting over 101,000 disease-attributable disability-adjusted life years (70). In January 2017, a 1.5 penny-per-ounce sugar-sweetened and artificially sweetened beverage tax was implemented in Philadelphia to support prekindergarten education, resulting in both a net 27% decline in sugar-sweetened and artificially sweetened beverage purchasing based on sales volume and the creation of new programming (71).

Improve dietary quality and availability.

Specific policy strategies to modify the poor diet that is the leading cause of cardiovascular disease globally have been proposed (72–74). A combined approach of subsidies for fruits, vegetables, nuts, and whole grains of 15–30% with taxation of sugar and sugar-sweetened beverages was estimated to produce the greatest reduction in cardiovascular disease and reduction of disparities in disease burden (73). Marketing of calorie-dense, nutrient-poor products to children, supported by enormous federal subsidies, are demonstrated as important contributors to childhood and adolescent obesity (72).

Invest in nutritional support for populations at highest risk of obesity in childhood.

Recommendations to modify the federal Supplemental Nutritional Assistance Program (SNAP) to benefit families at highest risk of suboptimal nutrition include restrictions to purchasing taxable products not consumed for nourishment and incentives to increase purchasing of healthy foods (75). An analysis of the impact of SNAP changes estimated that over 5 y, >11,900 cardiovascular deaths in adults would be averted, while achieving health care cost savings of >$5 billion dollars annually (75). By reducing food insecurity and improving dietary quality for children, the improvements in health quality and cost savings could be even greater (73).

Reduce obesity among adolescent females.

Healthy People 2020 aims to reduce the proportion of obese adolescents to a target of 16%; currently, over 21% of adolescent females are obese. Reducing obesity in adolescent and young adult females could be expected to reduce childhood obesity by 10–22%, with ongoing effects for subsequent generations. Food banks and medically tailored meals (low-fat or low-glycemic index meals with reduced calories) may be successful in reducing obesity among adolescents at highest risk (76).

Increase research, governmental, and industry efforts to guide reduced exposure to environmental obesogens.

Environmental chemical exposures contribute health care costs that may exceed 10% of the global domestic product (77). Obesogens are estimated to contribute ≥2% to 4% of obesity prevalence; however, this may be underestimated due to the irreversible, transgenerational effects of exposures in early childhood. To reduce environmental exposure to

endocrine-disrupting chemicals which contribute to obesity, legislation is necessary (77).

Improve the economics of direct health care costs of obesity. Direct health care costs related to childhood and adult obesity are estimated at >$275 billion dollars annually (56, 63). Medical costs related to children with obesity accounted for ≥$14 billion dollars annually in 2008 and childhood obesity has since increased by 10% (78). Small weight reductions of 1% in early childhood obesity among children aged 6 y, requiring expenditure of $103 million dollars, are projected to result in annual savings in adult medical expenditures of $845 million dollars annually (62). Paid maternity leave has been shown to increase breastfeeding duration, which would reduce childhood and adult obesity by ≥15%. Estimates of cost savings related to breastfeeding are impressive: if 90% of US families could comply with recommendations to breastfeed exclusively for the first 6 mo of life (a high goal compared with the 25% rate in 2018, and the Healthy People 2020 goal of 61%), then $13 billion dollars in annual health care costs would be saved (18, 79, 80).

Conclusions

Obesity is a severe, chronic disease associated with shortened life expectancy due to cardiovascular events, diabetes, chronic kidney disease, and several forms of cancer. Determinants of obesity in childhood are well established before the age of 5 y, with these children especially likely to become obese adults. New policies directed at reducing obesity at the earliest stages by targeting the nutritional environments and well-being of infants, toddlers, and preschool children could alter the trajectory of childhood and adult obesity and improve population health, longevity, and quality of life throughout the life course. Starting early isn't an option, it's essential.

Acknowledgments

The authors' responsibilities were as follows—all authors: wrote, read and approved the final manuscript.

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