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Review

Biological, environmental, and social influences on childhood obesity

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The prevalence of childhood obesity has increased globally over the past three decades, with evidence of recent leveling off in developed countries. Reduction in the, currently high, prevalence of obesity will require a full understanding of the biological and social pathways to obesity in order to develop appropriately targeted prevention strategies in early life. Determinants of childhood obesity include individual level factors, including biological, social, and behavioral risks, acting within the influence of the child's family environment, which is, in turn, imbedded in the context of the community environment. These influences act across childhood, with suggestions of early critical periods of biological and behavioral plasticity. There is evidence of sex and gender differences in the responses of boys and girls to their environments. The evidence that determinants of childhood obesity act at many levels and at different stages of childhood is of policy relevance to those planning early health promotion and primary prevention programs as it suggests the need to address the individual, the family, the physical environment, the social environment, and social policy. The purpose of this narrative review is to summarize current, and emerging, literature in a multilevel, life course framework.

INTRODUCTION

The prevalence of childhood obesity has increased globally over the past three decades, with more rapid increases recently occurring in low-income countries (1). In the United States, more than 30% of children are now overweight or obese (1), with evidence that the prevalence has leveled off (2). Children and adolescents are exhibiting obesity-related conditions such as type 2 diabetes, elevated blood pressure, low-density lipoprotein cholesterol, and higher fasting insulin levels (3–6). In addition, childhood obesity predicts adulthood obesity and its known health consequences (7,8). Treatment of obesity is notoriously difficult, with weight loss rarely sustained in adults (9). Therapeutic interventions in childhood are somewhat more successful, particularly if the intervention occurs prior to onset of puberty (10). However, real and sustained progress in combating the obesity epidemic will require a full understanding of the biological and social pathways to obesity in order to develop appropriately targeted prevention strategies in early life.

Pathways to childhood obesity are complex. It is therefore helpful to discuss determinants of obesity within a conceptual framework. A multilevel conceptual model, Bronfenbrenner's Bioecological Systems Theory (11), has previously been applied to the conceptualization of childhood obesity by Davison and Birch (12). This framework depicts individual-level factors, including biological, social, and behavioral risks, as acting within the influence of the child's family environment, which is, in turn imbedded in the context of the community environment. It is also helpful to consider critical periods for obesity risk and, as will be further illustrated in a later section, there are likely critical periods of biological and behavioral plasticity beginning as early as fetal life (13) with risk factors accumulating, and interacting with each other, across the life course. This is consistent with a life course model of chronic disease epidemiology (14). Specific determinants of obesity will be discussed below within this multilevel framework and life course perspective.

This narrative review will discuss both biological and social determinants of childhood obesity at three levels (individual, family, and community) and across early childhood. The relationship between childhood stress and obesity will be explored in greater detail as this is an important pathway of active interest in current literature. In addition, the review will address recent attention to sex- and gender-based differences in obesity risk. A key purpose in undertaking this review was to summarize evidence regarding pathways to obesity in boys and girls by integrating established plus emerging perspectives in the literature. These include an overview of important factors at each level. Given the breadth of the literature, it was not the intention to cover all literature on each determinant but rather to provide these as key examples of the many dimensions of obesity risk.

INDIVIDUAL-, FAMILY-, AND COMMUNITY-LEVEL DETERMINANTS OF OBESITY

At the individual level, the most direct determinant of children's obesity is the energy balance between nutritional

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Received 17 April 2015; accepted 30 September 2015; advance online publication 11 November 2015. doi:10.1038/pr.2015.208

intake and activity, the latter being influenced by both physical activity and sedentary behaviors (15–17). These behavioral factors are therefore frequent targets for both preventive and therapeutic interventions. However, nutrition and activity are "downstream" factors that can be influenced by many "upstream" causes. The energy balance required to maintain an appropriate fat mass varies among individuals due to differences in metabolism and in lipostatic set point, which will influence appetite and activity preferences (18). Metabolism and lipostatic set point, while to some degree influenced by genetic predisposition (18), can be altered by gene–environment interactions (19–21).

The family, physical, and social environment influence children's obesity risk in two ways: through a direct influence on children's nutrition and activity behaviors and through indirect influences via stress as will be discussed later in this paper. Higher parental education, parental nurturing, and higher self-esteem reduce obesity risk in girls (22). There is an abundance of evidence that the home food environment (23–25), shared family meals (26,27), and electronic media use influence children's obesity (28) largely through behavioral pathways. Mothers primarily establish the home food environment and are role models for eating behaviors (29) with evidence of strong correlation between the eating patterns of mothers and children (25,29). Appetite control and food preferences are established early in life (30), and there is a high correlation between parental obesity and their children's obesity (20,22).

The community environment is increasingly obesogenic, with increased use of convenience foods, automobiles, and electronic and televised forms of entertainment (31–33) leading to higher consumption of calorie-dense foods and more sedentary lifestyles. Food choices have been shown to be influenced by proximity to fast food outlets, supermarkets, and farmers markets (34–38). Physical activity levels are influenced by public recreation opportunities, transit availability, and neighborhood walkability (35,37,39–42). In addition, lower obesity levels are observed in areas where the natural environment has high recreational value (43). While evidence suggests that the above environmental factors affect risk behaviors and obesity, there is still a gap in understanding how children interface with the obesogenic environment (44).

PRENATAL AND POSTNATAL INFLUENCES

There is emerging interest in prenatal factors, postnatal factors, and their interactions. These are critical time periods of metabolic and endocrine plasticity and may condition later physiologic responses to environmental influences (13). This field of research has been labeled as the developmental origins of health and disease and is the subject of much attention in the biomedical and epidemiologic sciences.

For the past two decades, there has been intense interest in the possible effect of fetal undernutrition on later obesity. The interest in this proposed association was precipitated by seminal work by Barker (45). In humans, fetal undernutrition may be a consequence of maternal undernutrition, maternal smoking, or placental dysfunction from preeclampsia. Markers of fetal undernutrition, which include fetal growth restriction and its proxy indicator small birth weight for gestational age, have been shown to be associated with a modestly elevated risk of obesity. It has been suggested that this effect is due to an *in utero* adaptation that becomes a mismatch to a postnatal environment in which nutrition is abundant (46,47). Animal studies, often based on maternal dietary restriction, confirm evidence for such fetal metabolic adaptations to undernutrition (48). In both animal and human studies, there is evidence of the permanence of these adaptations. The greatest elevation in obesity risk is for those who were born small, but experienced rapid "catch up growth" postnatally (48–52).

Emerging literature is challenging the relationship of fetal undernutrition as a determinant of obesity. First, if the association does exist, is a genetic component partially responsible? Specific adult obesity gene loci have been implicated as associated both with fetal growth (53) and with growth velocity in infancy (54). In this genomic era, this will be an aspect of the literature to watch, although to date the predictive value of individual gene loci for obesity risk has been modest. There is emerging speculation as to whether this association indeed exists at all, despite the abundance of literature on the topic. Part of this speculation is based on a statistical argument that, in the zealous effort to control for the myriad of potential confounders, most studies looking at the relationship between fetal growth restriction or small birth weight for gestational age and later chronic conditions have controlled for variables along the causal pathway and thus introduced bias (55,56). Moreover, recent carefully analyzed studies have suggested the inverse; that small birth weight for gestational age is associated with a lower risk of obesity (57). This question remains an active topic of interest in the literature, despite the recognition that the association, if real, is a small magnitude association with no clear implications for prevention (58).

Fetal overnutrition, evidenced by large infant birth weight for gestational age, is a strong predictor of obesity in childhood and later life (59–61). A caveat is that, while large infant birth weight for gestational age is generally an indicator of excess fat mass, it may also reflect other growth parameters such that a subset of large infant birth weight for gestational age infants may have increased lean mass (62,63). Risk factors for large infant birth weight for gestational age include maternal obesity and maternal gestational diabetes (64,65) with African-American women exhibiting risk at lower maternal BMI thresholds (66). It is suggested that fetal hyperglycemia triggers fetal insulin production which in turn triggers fetal growth and adiposity (67). Animal studies demonstrate that fetal hyperinsulinemia may invoke permanent changes in the CNS mechanisms for regulating metabolism and body weight (67). Thus, fetal overnutrition may be a mechanism of intergenerational transmission of obesity and diabetes (67,68).

Early postnatal experiences are also important contributors to obesity risk. Breastfed infants are at lower risk for later obesity (69–73) for hypothesized reasons including that formula-fed infants develop greater reliance on external hunger cues (74) and have higher intake of protein (75), which may

contribute to obesity risk through behavioral and physiologic mechanisms, respectively. The benefits of breastfeeding appear to be confined to exclusive breastfeeding; mixed infant feeding of breastmilk and formula do not reduce obesity risks associated with formula feeding (76). In addition, the timing and choice of complementary foods introduced into an infant's diet may influence their food preferences in the long term (77). In general, obesity risk is elevated for those who experienced rapid early weight gain in infancy (78–80). Based on this knowledge, strategies for primary prevention in high-income countries may include support for long-term breastfeeding (81).

PSYCHOSOCIAL VULNERABILITIES

There is evidence that psychosocial stress is associated with obesity in children. Measures of stress vary from study to study (82), but the findings are consistent. Whether this association is causal is not known, but there are theoretical frameworks that suggest causality. For example, the life course–stress process perspective introduced by Pearlin *et al*. (83) has been discussed by Wickrama *et al*. (84) in the context of body mass. A pathway from stress to obesity could include inflammatory mechanisms (85) including arousal of the hypothalamic–pituitary–adrenal axis leading to increased cortisol levels and subsequent metabolic disruption and increased hunger (84,86–88). If so, nutrition may mediate the relationship between stress and obesity, or lifestyle factors may be coexisting with environmental stressors (89,90). Some of the reported associations of environmental stressors with childhood overweight and obesity include negative life events (82), maltreatment (91), how well the family communicates (90), and parental stress (92).

Depression and obesity are often comorbid in both children and adults. This comorbidity may be due to common genetic and environmental etiologies (93–96) or common pathways via dysregulation of the hypothalamic–pituitary–adrenal system (93,95,96). Increased food intake and reduced physical activity are characteristic of both conditions (94). Bidirectional causation is also plausible, with suggestions that obesity may be a determinant of later depression in children (97–99) and conversely hypothesized mechanisms for depression causing obesity (93,95,98–100). Indeed, it has more recently been suggested that these two comorbid conditions may mutually reinforce a progressive downward spiral in each other (101) and that additional insight into their longitudinal interaction may be important for intervention strategies (102).

Mothers' mental and emotional well-being has been shown to be associated with childhood obesity. Children of mothers with depressive symptoms are more likely to be obese or overweight in infancy (103,104), childhood (105,106), and adolescence (107). Prenatal exposure to maternal stress and distress has been shown to be associated with both children's obesity and rapid postnatal growth (108,109). Proposed mechanisms for the association include infant feeding practices (110), mother–infant interaction (111), mother–infant feeding interactions (112), parenting style (113), and a direct effect of stressors leading to central adiposity via arousal of the child's hypothalamic–pituitary–adrenal axis (86). It has also been suggested that, due to the comorbidity between maternal overweight and emotion regulation, these pathways may also play into the intergenerational transfer of overweight and obesity (112), as well as the roles of shared genes and environment (86). A recent systematic review noted the need for more prospective studies to confirm and explain these associations (114).

Consistently, in high-income countries, socioeconomic disadvantage has been shown to be associated with obesity risk in childhood and persistently throughout life (115–117). Socioeconomic disadvantage may exert its influence as early as the prenatal and postnatal period, through its association with maternal depression (106,118) and its consequences. Moreover, poverty may be associated with poorer individual diet (119), poorer retail food and recreational environment (34,120,121), suboptimal family food routines (118,122), and environmental stressors such as living in a higher crime neighborhood (121). The risks associated with socioeconomic disadvantage may accumulate and compound throughout childhood (123). Miller and Chen (124) present a theoretical model, with corresponding research evidence, linking poverty to the development of a proinflammatory phenotype and subsequent elevated risk for chronic conditions in childhood and beyond. Overall, it appears that poverty is associated with later obesity through its association with other obesity risk factors and through the stress process.

There is an increasing attention in the literature to the differences in vulnerabilities in boys and girls, suggesting different pathways to obesity. Much of the literature, to date, has looked at determinants of childhood obesity while statistically controlling for children's sex. However, to truly understand the developmental processes leading to obesity, researchers may need to look at boys and girls separately in order to recognize both sex-specific (biological) and gender-specific (social and cultural) differences in the ways in which boys and girls interact with their physical and social environments. Some biological differences include body composition and growth patterns, with clear sex differences in the distribution of adiposity beginning as early as the neonatal period and continuing through adulthood (125). Energy requirements and the aptitude for specific physical activities exhibit sex differences, while specific gender differences include how boys and girls interact with their family and their food environment as well as their overall physical activity levels (126). There are also gender differences in metabolic responses to stress (87) and family disruption or conflict (127). Responses to the physical and social environment will influence, and be influenced by, pubertal development (47,48,125). Pubertal timing itself has significant influence on insulin resistance and metabolic syndrome, particularly in girls (128,129). The pubertal transition is also well established as a time when depression rates rise dramatically, particularly for females; indeed, this developmental stage is when the gender difference in depression emerges (130,131). Finally, pubertal timing and growth influence later adult cardiovascular risk in both males and females (128). Additional research focusing on the gendered dimensions of childhood obesity is needed.

SUMMARY AND IMPLICATIONS

In undertaking a review of this broad area of significant health promotion interest, I have used the narrative review method. It has been argued that narrative reviews have advantages when the scope and literature coverage is broad and covers a range of issues within a given topic (132). This broader coverage comes at the expense of the more explicit methods, reporting and reproducibility, that are associated with systematic reviews, which tend to focus on narrower topics using prescribed search methods (132). Given the methodological limitations of the narrative method, and the acknowledged potential for selection biases in study selection when a nonsystematic review is undertaken, the reader should turn to determinant-specific systematic reviews for exhaustive discussion of the specific determinants covered in this review. The main objective of this review was to summarize key early determinants of childhood obesity within the important framework of individual-, family-, and community-level biological and social influences acting across early life.

Consideration of determinants of obesity within this broader multilevel framework may imply that strategies for health promotion and primary prevention should include attention to determinants at all levels. The upstream influences on childhood obesity occur at many levels, including the family and the community, and begin very early in the life course. Health promotion activities typically target individual lifestyle factors, despite emerging evidence of the importance of broader environmental prevention targets (133). Family-based interventions to improve the home food environment (90) and parenting style (134) and policies to reduce the costs of healthy food choices (135) are needed. Prevention efforts should also include programs to reduce financial stress in families and programs aimed at teaching children on how to cope with stressors in their environment (86). It has been suggested that overweight and obesity reductions may accrue if the prevention focus is shifted, more broadly, to promoting healthy lifestyles and healthy environments and beyond the focus on individual children's body weight as the outcome (136). The opportunities for early health promotion require attention simultaneously to many levels (30), suggesting the need to address the individual, family, and physical environment, the social environment, and social policy.

STATEMENT OF FINANCIAL SUPPORT
None.

Disclosure: none; there are no conflicts of interest.

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