

Etiology and Pathophysiology

Factors associated with development of excessive fatness in children and adolescents: a review of prospective studies

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Summary

The purpose of this review was to examine the factors that predict the development of excessive fatness in children and adolescents. Medline, Web of Science and PubMed were searched to identify prospective cohort studies that evaluated the association between several variables (e.g. physical activity, sedentary behaviour, dietary intake and genetic, physiological, social cognitive, family and peer, school and community factors) and the development of excessive fatness in children and adolescents (5–18 years). Sixty-one studies met the eligibility criteria and were included. There is evidence to support the association between genetic factors and low physical activity with excessive fatness in children and adolescents. Current studies yielded mixed evidence for the contribution of sedentary behaviour, dietary intake, physiological biomarkers, family factors and the community physical activity environment. No conclusions could be drawn about social cognitive factors, peer factors, school nutrition and physical activity environments, and the community nutrition environment. There is a dearth of longitudinal evidence that examines specific factors contributing to the development of excessive fatness in childhood and adolescence. Given that childhood obesity is a worldwide public health concern, the field can benefit from large-scale, long-term prospective studies that use state-of-the-art measures in a diverse sample of children and adolescents.

Keywords: Childhood obesity, dietary intake, fat mass, physical activity.

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Introduction

Childhood obesity has reached epidemic proportions and has become a critical public health threat. Over the past 30 years, the prevalence of obesity has more than doubled for

both preschool children and adolescents, and has more than tripled for elementary school children (1). Currently, 33.2% of children and adolescents (ages 6–19 years) in the United States are overweight or obese (1). The burden of obesity is disproportionately high among minorities;

41.8% of Non-Hispanic black and 41.2% Hispanic children and adolescents are overweight or obese (1). Because obesity is a multifaceted problem, public health interventions aimed at preventing it should be informed by a comprehensive understanding of the physiological, behavioural, environmental and cultural factors that associate with development of excessive fatness in childhood and adolescence. Social ecological models that take into account a wide range of factors and contextual influences on obesity are well suited to understanding and ultimately preventing obesity.

Obesity is a condition characterized by an excessive fat mass relative to total body mass. The vast majority of children begin life with normal body composition, and in normally developing children, fat mass increases at a rate of 1.9 kg year⁻¹ (2). However, in some children fat mass increases more rapidly, ultimately producing overweight or obesity. Typically fat mass increases very gradually, and even when the increase is excessive, a period of several years is required for the obese condition to be attained. The factors that relate importantly to this gradual, excessive increase in fat mass are not well understood. Designing research to identify these factors is challenging because they exert their influence in children, who are initially normal in body composition, over an extended period of time. Studies focusing on fat loss in children who are already overweight do not necessarily provide information that is relevant to prevention of obesity in children who are initially normal. In contrast, prospective observational studies have the potential to identify the factors that associate with excessive fat gain.

The purpose of this systematic review was to examine current scientific literature on the factors that predict the development of excessive fatness in children and adoles-

cents. The review focused exclusively on prospective cohort studies that evaluated the association between multiple individual and contextual variables (e.g. physical activity, sedentary behaviour, dietary intake and genetic, physiological, social cognitive, family and peer, school and community factors) and the development of excessive fatness. We limited the review to prospective studies because through temporal sequencing, prospective studies identify the factors that associate with development of excessive fatness in children and adolescents.

Methods

Conceptual framework

This systematic review was guided by the social ecological model of health behaviour, which posits that an individual's behaviour is influenced by factors operating at multiple levels, including intrapersonal, interpersonal, institutional, community and public policy (3,4). A social ecological framework has been used in previous studies and is increasingly acknowledged as a promising approach for studying the aetiology and prevention of obesity (5–8). In the context of this review, the social ecological model provides a framework for examining a wide range of factors that associate with energy balance, including physical activity, sedentary behaviour and dietary intake, and other intrapersonal (e.g. genetic, physiological and social cognitive), interpersonal (e.g. family and peers), institutional (e.g. school) and community (e.g. physical activity and nutrition environment) factors. A conceptual model of our interpretation of the factors that relate to a child's body composition is presented in Fig. 1.

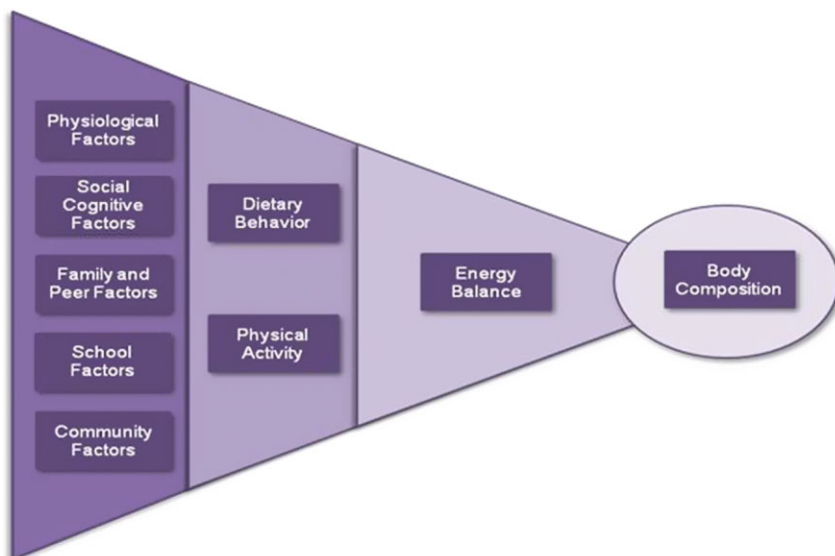


Figure 1 Conceptual model.

Table 1 Search terms

Variables	Search terms
Dietary intake	total energy intake, energy intake, fat, dietary fats, protein, dietary protein, carbohydrate, dietary carbohydrate, dairy intake, dairy products, milk, soft drinks, fruit juice, sugar-sweetened beverage, beverage consumption, soda, fruit and vegetable intakes, vegetables, snack food intake, dietary sucrose, energy-dense, low-fiber, dietary fiber, high fat, meal frequency, breakfast eating, restrained eating, feeding behavior
Physical activity	physical activity, accelerometry, motor activity, exercise
Sedentary behaviour	sedentary, sedentary behavior, sedentary lifestyle, sitting, accelerometry
Genetic	genetic(s), twin(s), heritability, parental overweight, quantitative trait, heritable
Physiologic	blood pressure, triglyceride, low density lipoprotein, LDL, LDL cholesterol, high density lipoprotein, HDL, HDL cholesterol, leptin, adiponectin, insulin, ghrelin
Social cognitive	self-esteem, depression, depressive symptoms, self-regulation, restrained eating, self-concept, protein restricted diet, fat restricted diet, carbohydrate restricted diet
Family and peers	socio-economic status, social class, income, family income, family structure, family characteristics, maternal work status, employment status, time spent in childcare, feeding practices, feeding behavior, meals eaten together, frequency of family meals, parental monitoring on weight, parental monitoring on physical activity, peer weight status, peer group
School	school food, school lunch, school breakfast, physical education, recess, school physical activity
Community physical activity	green space, residential density, parks, public open spaces, sprawl index, physical activity facilities, playground, sidewalks, trail, connectivity, built environment, neighborhood
Community nutrition	proximity of food retailers, food environment, neighborhood food environment, food outlet, fast food restaurant, super market

Review methods

We conducted an electronic database search of Medline, Web of Science and PubMed to identify relevant articles published between January 1990 and October 2011. We chose this time frame to focus on the more recent studies of the past 22 years. A keyword search in Medline and Web of Science and a MeSH search in PubMed focused on children and adolescents as the target population, and longitudinal, cohort or prospective studies as the study design. The outcome variables of interest were overweight, obesity, adiposity and body mass index (BMI). The exposure variables of interest were objectively measured physical activity, objectively measured sedentary behaviour, dietary intake, genetic factors, physiological factors, social cognitive factors, family and peers, school, the community physical activity and nutrition environments. A detailed list of the search terms and their associated exposures are presented in Table 1.

Inclusion criteria for the articles were as follows: (i) published in peer-reviewed journals between January 1990 and October 2011; (ii) written in English; (iii) used a prospective cohort study design and included longitudinal analyses to evaluate the independent association between the exposure and outcome variables of interest and (iv) included only children and adolescents between the ages of 5 and 18 years. Because sedentary behaviour has received increased attention in recent years, we extended the publication search for this construct through 30 June 2012.

The selection of articles was performed independently by two investigators. A total of 7,735 articles were identified

in the search and 61 articles met the inclusion criteria and were included in the review (Fig. 2). Data from the selected articles were extracted into a summary table by one investigator and confirmed by another investigator. The summary table included the following information for each article: study location, participant characteristics (i.e. sample size, age, sex and race/ethnicity), study design and follow-up duration, exposure variables and measures, outcome variables and measures, statistical analysis method and main findings. A summary of the studies is presented in Table 2.

Results

Intrapersonal factors related to energy balance

Behaviours related to energy balance

Physical activity. Low levels of physical activity relate to energy imbalance and may associate with the development of excessive fatness in children and adolescents. Many studies that have measured physical activity in children and adolescents have used subjective (self-report) measures, which involve considerable recall error, especially in children <11 years of age (9–11). Objective methods (e.g. heart rate monitor, pedometer and accelerometer) provide more accurate estimates of physical activity by removing recall error. Accelerometry is currently considered the 'gold standard' for measuring physical activity in children (12).

This review identified seven prospective cohort studies that used accelerometry to examine the relationship between physical activity and excessive fatness (13–19).

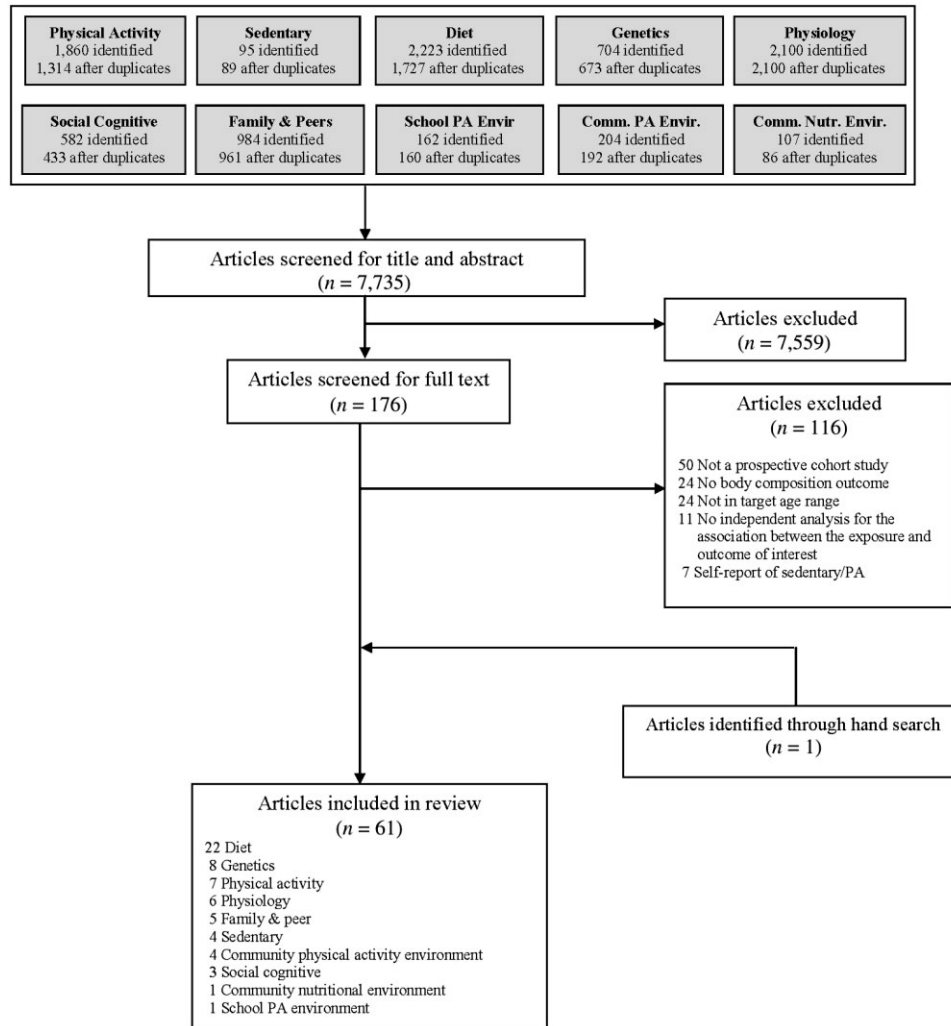


Figure 2 Flow diagram of study selection process.

The follow-up period for these studies ranged from 1 to 7 years. For the measure of body composition, three of the studies used a criterion measure, dual-energy X-ray absorptiometry (DXA) (13–15), and four used other measures (i.e. BMI, skin-fold thickness or bioelectrical impedance analysis [BIA]) (16–19). In the Iowa Bone Development Study (13,14), one of the two that used DXA, Janz *et al.* (13) followed 379 five-year-old children for 3 years. At follow-up, children in the lowest quartile of percent body fat had higher total physical activity, vigorous physical activity and 5-min bouts of vigorous physical activity than children in the upper three quartiles of percent body fat (13). An additional follow-up with the same cohort of children 3 years later revealed that those in the highest moderate-to-vigorous physical activity (MVPA) quartile at age 5 had significantly lower fat mass at ages 8 and 11 than those in the lowest MVPA quartile (14). The second study, the Avon Longitudinal Study of Parents and Children, followed

4,150 twelve-year-old children for 2 years. This study found that an increase of 15 min day⁻¹ in MVPA was associated with 2.4 and 2.3% lower fat mass in boys and girls respectively (15).

The other four cohort studies that used accelerometry to measure physical activity used BMI, skin-fold thickness or BIA to measure body composition. Fisher *et al.* (16) examined the association between MVPA and a 1-year change in BMI and fat mass index (FMI: fat mass divided by height in m²) in 280 children ages 8 to 9 years. They found that higher MVPA levels at baseline were associated with lower BMI and FMI 1 year later, independent of both total physical activity and sedentary behaviour (16). Stevens *et al.* (17) followed 454 American-Indian children ages 7 to 10 years and found that physical activity was associated with lower percent body fat in normal weight children at the 3-year follow-up. This effect was not seen in overweight children, whose physical activity levels were associated with higher

Table 2 Summary of included studies*

Variable	Number of studies	Summary	Description	Refs
Physical activity	7	----- 0	6 studies (13–18): PA reduces fat mass 1 study (19): no association	(13–19)
Sedentary behaviour	4	00 0 +	3 studies (13,16,22): no association 1 study (23): ↑ sedentary behaviour associated with ↑ in BMI	(13,16,22,23)
Energy intake	2	(+ F, 0 M) 0	1 study (24): ↑ energy intake associated with ↑ BMI in girls, not boys 1 study (25): no association	(24,25)
Energy-dense foods	5	+0 0 0 0	1 study (29): ↑ energy-dense foods associated with ↑ FMI in children 4 studies (24,26–28): no association in adolescents	(24,26–29)
Macronutrient intake	2	-0	1 study (26): macronutrient intake inversely associated with body fat 1 study (27): no association	(26,27)
Dairy intake	4	-- 0 0	2 studies (30,31): ↓ dairy intake associated with ↑ fat mass and BMI 2 studies (32,33): no association	(30–33)
Fruit and vegetable intake	1	0	1 study (34): no association	(34)
Snack food intake	3	00 +	2 studies (35,36): no association 1 study (37): ↑ snack food intake associated with ↑ BMI z-score	(35–37)
Sugar-sweetened beverage intake	8	+++++ (+ F, 0 M) 0 0	5 studies (35,38–41): ↑ SSB associated with ↑ BMI 1 study (43): for girls, ↑ SSB associated with ↑ BMI, for boys, no evidence of association 2 studies (42,44): no association	(35,38–44)
Energy-dense, low-fibre, high-fat pattern	1	Mixed	1 study (45): pattern associated with ↑ fat mass from 7- to 9-year-olds, no association from 5- to 9-year-olds	(45)
Heritability of BMI	8	+++++++	7 studies (47–53): children with overweight or obese parents more likely to be overweight 1 study (54): heritability only associated with BMI of parent of same sex	(47–53)
Plasma insulin	2	+0	1 study (55): ↑ insulin associated with ↑ fat mass 1 study (56): no association	(55,56)
Insulin sensitivity	3	---	3 studies (55,57,58): ↓ insulin sensitivity associated with ↑ fatness	(55,57,58)
Leptin	2	++	2 studies (60,61): ↑ leptin associated with ↑ fat mass	(60,61)
Plasma adiponectin	1	0	1 study (61): no association	(61)
Depressive symptoms	1	+	1 study (63): depressive symptoms associated with ↑ fat mass	(63)
Self-esteem	1	0	1 study (64): no association	(64)
Self-control	1	-	1 study (65): higher self-control associated with ↓ BMI	(65)
Parental concern for child's weight	1	Mixed	1 study (66): ↑ parental concern associated with ↓ fat mass for white children, no association among African-American children	(66)
Parental feeding restriction	2	Mixed	1 study (67): higher parental feeding restriction scores associated with ↓ BMI z-score for 5–6-year-olds, but no association in 10–12-year-olds 1 study (68): for high-risk children, ↑ parental feeding restriction and ↓ pressure to eat were associated with ↓ BMI z-score	(67,68)
Parental monitoring	1	Mixed	1 study (68): for low-risk children, ↑ parental monitoring of high-fat food associated with ↓ BMI z-scores; for high-risk children, no association	(68)
Meal frequency	1	0	1 study (69): no association	(69)
Maternal work status	1	+	1 study (64): mother's full-time employment associated with ↑ BMI	(71)
School PA environment	1	Recess: -(PE: - M, 0 F)	1 study (73): meeting recess recommendation associated with ↓ in BMI, meeting PE recommendation associated with ↓ in BMI for boys only	(73)
Community PA environment	4	-- 0 - 0 0	1 study (76): park space and recreation programmes associated with ↓ BMI z-score 1 study (74): greenness associated with ↓ BMI z-score; residential density - no association 1 study (75): ↑ road connectivity associated with ↓ BMI z-score; access to destinations and traffic exposures - no association 1 study (77): no association	(74–77)
Community nutrition environment	1	+	1 study (80): higher availability of convenience stores to child's home (0.25 mile) associated with ↑ BMI	(80)

*There were no articles identified for peer factors, time spent in child care, nor school food environment.
+, indicates positive association between the variable and obesity/overweight; -, indicates negative association; 0, indicates no association.
BMI, body mass index; F, female; FMI, fat mass index; M, male; PA, physical activity; PE, physical education; SSB, sugar-sweetened beverage.

BMI and fat mass 3 years later (17). Moore *et al.* (18) followed 103 four-year-old children for 7 years and found that children in the highest tertile of daily physical activity (accumulated over the 7 years) had significantly smaller gains in BMI and skin-fold thickness at age 11. However, one study that followed 212 five-year-old children for 3 years did not find a significant association between MVPA and BMI, body fatness or waist circumference (19).

Overall, the prospective evidence to date indicates that MVPA and total physical activity are associated with lower fat mass in children and adolescents over time. However, the maximum amount of follow-up time was only 7 years. Longer follow-up periods across early childhood into adolescence would be valuable because the association between physical activity and relative fatness gain may exist during critical periods in childhood and adolescence. This is in line with our current understanding of critical periods for the development of fat cells in individuals (20). For example, the adiposity rebound period (ages 4 to 7 years) may be a critical period for the development of subsequent adiposity (20). Once body fatness status is established during adiposity rebound, the fatness level may then be sustained throughout childhood and adolescence (18). Future longitudinal studies using criterion measures and including participants from early childhood through adolescence could explore these relationships more fully.

Sedentary behaviour. Sedentary behaviour, defined as activities with an energy expenditure ranging from 1.0 to 1.5 metabolic equivalent units (21), likely plays a role in energy balance. This review identified four prospective cohort studies that used accelerometry to examine the relationship between sedentary behaviour and excessive fatness (13,16,22,23).

The Iowa Bone Development Study examined the association between objectively measured sedentary time and the 3-year change in adiposity among 378 five-year-old children (13). There was no significant difference between children in the lowest quartile for percent body fat compared with those in the upper three quartiles in sedentary time (283 ± 39 min day⁻¹ and 285 ± 41 min day⁻¹ respectively). Similarly, there was no difference between children in the upper quartile for percent body fat compared with those in the lower three quartiles in sedentary time (286 ± 44 min day⁻¹ and 284 ± 40 min day⁻¹ respectively).

Another cohort study investigated the relationship between objectively measured sedentary time and 1-year change in BMI, FMI and waist circumference among 280 eight-year-old children (16). After adjusting for baseline MVPA and total physical activity, sedentary time was not significantly associated with the 1-year change in BMI, FMI and waist circumference.

Basterfield *et al.* (22) followed 403 six- and seven-year-old children for 2 years and measured sedentary time, FMI

and BMI z-score. After adjusting for baseline MVPA, changes in sedentary time were not associated with changes in FMI in boys but were marginally associated in girls. However, changes in sedentary time were not associated with changes in BMI z-score for either sex.

Mitchell *et al.* (23) examined the relationship between objectively measured sedentary time and 6-year change in BMI among 789 nine-year-old children. Longitudinal quantile regression was used to determine if sedentary time was associated with changes at the 10th, 25th, 50th, 75th and 90th BMI percentiles over time. The researchers found that time spent in sedentary behaviour was positively associated with change in BMI at the 50th, 75th and 90th percentiles between ages 9 and 15 years, independent of MVPA.

In sum, the limited prospective evidence to date yields mixed findings on whether sedentary behaviour is associated with excessive fatness in children and adolescents. There is some indication that among children (ages 5 to 9 years), sedentary time is not associated with fat mass; whereas there is a positive association between sedentary time and BMI in older children and adolescents (ages 9 to 15 years). This limited evidence indicates the need for more prospective studies to evaluate the predictors of objectively measured sedentary behaviour on the development of excessive fatness in children and adolescents.

Dietary intake. There is consensus that dietary intake plays a key and complementary role to energy expenditure (i.e. physical activity) in the regulation of energy balance. This review identified 22 studies that examined the relationship between dietary intake and the development of excessive fatness in children and adolescents.

In a large cohort of U.S. adolescents, Berkey *et al.* (24) reported that higher energy intake in 9- to 14-year-old girls, but not boys, was associated with a 1-year increase in BMI. In contrast, Koutedakis *et al.* (25) did not find an association between higher energy intake and 2-year changes in percent body fat estimated from skin-fold measurements in 12-year-old Greek children.

Studies on the relationship between macronutrient intake and changes in BMI in children and youth are mixed (24,26–29). For example, one study reported an inverse association with body fat (26), whereas another has not (27). The intake of energy-dense foods was positively associated with a 1-year increase in FMI among 6- to 8-year-old children (29); however, studies with adolescents have not supported these findings (24,26–28).

Intake of specific types of foods including dairy, fruit and vegetables, snack foods and sugar-sweetened beverages have been evaluated with respect to their impact on the development of excessive fatness. There is some indication that reduced intake of dairy during childhood may be related to greater increases in body fat and BMI (30,31),

although findings have not been consistent (32). In fact, among adolescents, those who reported higher milk intake experienced greater increases in BMI; however, this finding was no longer significant after adjustment for total energy intake (33). One study looked specifically at children's fruit and vegetable intake and found that, when adjusted for caloric intake, fruit and vegetable intake was not associated with 3-year change in BMI z-score in 9- to 14-year-old children (34).

We identified three studies that examined the relationship between snack food consumption (e.g. cookies, pastries, crackers, chips, sweets, confectionaries) and change in BMI z-score in children and adolescents. Two studies found no relationship between snack food consumption and change in BMI z-score after controlling for dieting behaviours and caloric intake (35,36); however, one study found a positive association between snack food consumption and 4-year change in BMI z-score in 5-year-old girls from overweight families (37). There is mixed evidence regarding sugar-sweetened beverage intake and the development of excessive fatness in children and adolescents. Some studies demonstrated that sugar-sweetened beverage intake is associated with longitudinal increases in BMI or BMI z-scores in children and adolescents (35,38–41). One study found no association between sugar-sweetened beverage intake at ages 5 or 7 years and fat mass at 9 years (42); whereas another found a positive association among girls, but no association among boys (43). Further, one study found a positive association which disappeared after adjustment for total energy intake (44). Another study examined food intake patterns and identified an energy-dense, low-fibre and high-fat pattern. Among 7-year-old children, this dietary pattern was associated with higher fat mass at 9 years of age; however, this association effect was not present from ages 5 to 9 years (45).

In sum, the prospective evidence regarding the association of dietary intake on excessive fatness in children and adolescents is mixed. Major methodological challenges include inaccuracies in estimation of energy intake by dietary assessment methods, errors in self-report from both parents and children and systematic underreporting. None of the studies included in this review used specific methods to adjust for measurement error. It has been well documented that dietary intake and patterns change markedly in childhood (46); however, the question remains of whether there is a critical period during childhood and adolescence where dietary intake is truly relevant to changes in BMI over time.

Additional intrapersonal factors related to energy balance

Genetic factors. This review identified eight studies that examined the relationship between genetic factors and the

development of excessive fatness in children and adolescents. Studies of the genetic predictors on the development of excessive fatness in children and adolescents have primarily estimated the heritability of BMI over time through prospective studies of twins and children in adoptive families (47–49). Haworth *et al.* (47) followed more than 3,000 pairs of monozygotic and dizygotic twins from ages 7 to 11 and found that the heritability of children's BMI became progressively stronger (i.e. 0.48 at age 7 to 0.78 at age 11) with increasing age. This study also found a significant association between the FTO gene and change in child BMI over time; children with the AA genotype consistently had a higher BMI than children with the AT or TT genotype at ages 7, 10 and 11 (47). Three other twin studies with adolescent samples found high heritability of BMI with age, and the variance was largely explained by a similar set of genetic factors (50–52). Additional findings revealed that genetic factors had a stronger association on BMI heritability for females than males from ages 10 to 12 (51), and at age 12 (52).

This review identified four prospective studies that estimated the heritability of BMI between parents and their children (48,49,53,54). Among 5-year-old children with overweight or obese parents, the risk of becoming overweight or obese at age 14 was 6 to 10 times higher than for children with normal weight parents (53). Likewise, among 9-year-old girls, the risk of becoming overweight at age 18 was 3.8 times and 7.9 times higher for those with an obese father or overweight mother respectively (48). Further, 9-year-old boys with obese fathers had a 4.4 times higher risk of being overweight at age 18 than boys with a normal weight father (48).

However, the association of parental BMI on their child's overweight risk may not be entirely attributable to genetic factors. One study that followed 5-year-old children annually until age 8 demonstrated that child BMI was only associated with the BMI of the parent of the same sex, possibly indicating the role of parenting practices associating with child BMI (54). Another study found a positive association between parental BMI and child BMI in both biologically related parent-child pairs ($r = 0.11$ – 0.22) and adoptive parent-child pairs ($r = 0.10$) at ages 11 to 13, possibly indicating the role of the familial environment on BMI change in children (49).

In sum, the genetic association with the development of excessive fatness during childhood and adolescence, while significant, remains small relative to other factors. For example, the FTO polymorphism (rs9939609) only explained about 1% of the variance of the BMI heritability (47). Overall, evidence suggests high heritability of child and adolescent BMI with increasing age. More research is needed into the association between specific genes and change in child BMI over time. There is also evidence of heritability of BMI between parents and their children,

although the contribution of the gene-environment interaction to the development of excessive fatness in childhood and adolescence remains unknown.

Physiological factors. This review identified six studies that have examined the relationship between various physiological factors and the development of excessive body fatness in children and adolescents. Prospective research to date has focused mainly on the association between body fat with plasma insulin, leptin and plasma adiponectin rather than other biomarkers, such as cholesterol or ghrelin.

Prospective studies on the relationship of fasting plasma insulin concentration to excessive fatness have yielded mixed results. One study with 8-year-old children who were followed between 3 and 6 years found that those children with higher fasting plasma insulin concentration at baseline had a greater increase in fat mass over time (55); however, another study with 5- to 9-year-old Pima Indian children found no association between fasting plasma insulin concentration and fat gain 9 years later after adjusting for maternal BMI (56). Studies have also examined the relationship between insulin sensitivity and longitudinal change in fat mass in children and adolescents (55,57,58). These studies reported that reduced insulin sensitivity was independently associated with a 3- to 10-year increase in fat mass among children (55,57) and adolescents (58) after controlling for age, sex, pubertal stage and race/ethnicity.

Leptin is a hormone that plays a key role in energy balance, including regulation of appetite and metabolism (59). This review identified only two prospective studies that have examined the association of baseline leptin level on change in fat mass in children. These studies examined 3-year change in body fat among 8-year-old (60) and 9-year-old children (61) and found that higher baseline leptin levels were associated with a greater increase in fat mass after adjusting for baseline fat mass and sex. Only one study was identified that examined the relationship between change in BMI and plasma adiponectin. This study examined 3-year change in BMI ratio (follow-up BMI/baseline BMI) among 9- to 10-year-old Japanese children, and found no relationship between plasma adiponectin and change in BMI ratio (61).

In sum, the limited prospective evidence to date indicates mixed findings surrounding the association of physiological biomarkers on the development of excessive fatness in children and adolescents. Of the studies identified, there were mixed results regarding the association with plasma insulin, limited evidence of a positive association with leptin, and limited evidence of a negative association with insulin sensitivity. We are unable to make a conclusion about the relationship between adiponectin and the development of excessive fatness in children and adolescents.

Further, the collective effect of these biomarkers on excessive fatness is not well understood. One challenge to examining the impact of these biomarkers on the development of excessive fatness in youth is that many are highly interrelated. For example, reduced insulin sensitivity was associated with excessive fatness, but it also was associated with decreased leptin (59). Further, genetic and environmental factors interact and add to the challenge of examining the impact of these physiological factors on excessive fatness in children and adolescents (62). It is worthwhile to note that there is the potential for reverse causality (i.e. overweight causing insulin resistance, an increase in adipose tissue increases the secretion of adiponectin); however, this review was designed to identify predictors of overweight (i.e. overweight as the dependent variable, not the independent variable).

Social cognitive factors. This review identified three studies that have examined the relationship between various social cognitive factors (e.g. depressive symptoms, self-esteem, self-control) and the development of excessive fatness in children and adolescents. Only one prospective study examining depressive symptoms and change in body fat was identified (63). This study found that adolescents who had depressive symptoms at baseline were at increased risk of overweight 1 year later, after controlling for socio-demographics, self-esteem, physical activity and smoking. Another prospective study examined the association of self-esteem on the development of overweight in a cohort of 5- to 10-year-old children found that a low self-esteem score at baseline did not predict which non-overweight children would move into the overweight category 3 years later (64).

Duckworth *et al.* (65) examined the effects of self-control (i.e. impulsivity and delay of gratification) and prospective weight gain in 105 fifth grade children. This study found that children with higher self-control in fifth grade had a lower BMI in eighth grade. However, we are not aware of any longitudinal studies that specifically investigated the association between children's self-regulation of eating behaviour on excessive fatness.

Due to the limited prospective evidence, we are unable to draw any clear conclusions surrounding the relationship between social cognitive factors and the development of excessive fatness in children and adolescents. Drawing from social ecological theory, we would hypothesize these variables to be greatly correlated with excessive weight gain in children; however, social cognitive research on obesity has yet to take advantage of prospective designs. Additional prospective studies could clarify how social cognitive factors associate with the development of excessive fatness, the factors that are the most predictive over time, the developmental phases that are the most relevant for nurturing social cognitive attributes and the relationship between these different social cognitive factors.

Interpersonal factors related to energy balance

Family and peer factors. Family and peers are two interpersonal factors that are hypothesized to associate with the development of excessive fatness in children and adolescents. We identified three prospective studies that examined the relationship between parental concern for child's weight, parental feeding practices and longitudinal change in child fat mass and BMI z-score (66–68). One study found that among 11-year-old children, parental concern for child's weight was inversely associated with change in total fat mass over time among white, but not African-American children (66). Two other studies examined the relationship between parental feeding practices and change in child BMI z-score (67,68). One study found that higher parental feeding restriction scores of energy-dense foods and drinks were associated with lower BMI z-scores in 5- to 6-year-old children 3 years later, but no association was found in 10- to 12-year-old children (67). Another study found that the association of parental feeding practices on change in children's BMI z-score varied among children by obesity risk status (low risk as maternal pregnancy weight <33rd percentile, and high risk as >66th percentile) (68). Among low-risk children, higher parental monitoring of child consumption of high-fat food was associated with lower BMI z-scores 2 years later. Among high-risk children, increased parental feeding restriction and reduced pressure to eat, but not parental monitoring, were associated with decreased BMI z-score. The literature search identified only one prospective study that examined the relationship between meal frequency and longitudinal change in child overweight status determined from BMI. Fulkerson *et al.* (69) found no association between family meal frequency and adolescent risk for excessive fatness at 5-year follow-up.

Family structure, particularly maternal work status and time spent in child care, may predict the development of excessive fatness. Children who are enrolled in child care are more likely to be obese (70). However, prospective evidence on these relationships is limited, as only one study was identified that met our inclusion criteria. Researchers reported that 7-year-old children with a full-time employed mother had an increased probability of being overweight at age 16 (71). No prospective study of the association of time spent in child care was identified.

Overall, there is little data on the role of family processes in the development of excessive fatness in children and adolescents, and results are mixed. Limited evidence suggests that parental concern for child's weight may be inversely associated with fat mass change, although these results may differ by race. Further, the relationship between child feeding practices (e.g. monitoring, restriction) and excessive fatness is unclear and appears to differ by age. Less is known about the longitudinal association of family

structure (i.e. maternal hours worked, child care) on child and adolescent weight status. The tenets of the social ecological model suggest that peers influence the development of excessive fatness in children and adolescents; however, no longitudinal studies explored the impact of peers' influence on individual child weight trajectories.

Institutional factors related to energy balance

School factors. Because children and adolescents spend the majority of their weekday time at school, the school environment carries significant authority on their dietary and physical activity patterns (72). The school food environment is critically important for child and adolescent weight trajectories given that children consume more than 35% of their daily energy intake at school (72). However, this review did not identify any prospective studies that examined the association between the school food environment and excessive fatness.

With regard to the school physical activity environment, only one prospective study investigated the association between existing school physical activity programmes (i.e. recess and physical education) and BMI trajectories in children from first to fifth grade. Fernandes *et al.* (73) found that children who met the National Association for Sport and Physical Education (NASPE) guideline of 100 min of recess per week exhibited a 0.74 unit decrease in BMI percentile over time, while meeting the NASPE guideline of 150 min of physical education per week was associated with a 1.56 unit decrease in BMI percentile for boys only.

Given the lack of prospective data examining the relationship between the school nutrition and physical activity environment and the development of excessive fatness in children and adolescents, no conclusions can be drawn. It is currently unknown how the school environment, including school dietary and physical activity policies and practices, predict longitudinal changes in the weight status of children and adolescents.

Community factors related to energy balance

Physical activity environment factors. Longitudinal studies of the relationship between the physical activity environment (e.g. parks, roadways, landscaping) and excessive fatness are limited, especially in children and adolescents. The present review identified only four prospective studies (74–77).

One study that sampled 3,173 children between the ages of 9 and 10 investigated the association between park space, recreation programmes and an 8-year change in BMI z-score (76). Park space was defined as acres of parkland within a 500 m distance of a child's home. Availability of recreation programmes was defined as the number of municipal recreation programmes within a

10 km buffer of a child's home that targeted children under 18 and required physical exertion. Both park space and recreation programmes were inversely associated with BMI z-score at age 18 after controlling for demographic and environmental confounders. Further, recreation programmes had a larger effect on BMI z-score compared with park space, and boys had larger effect sizes relative to girls for both outcomes.

Another study examined greenness (i.e. the presence of open green spaces within a 1 km buffer of a child's home) and residential density (within the child's census block) on change in BMI z-score in a sample of 3,831 youth ages 3–16 years (74). Greenness was inversely associated with BMI z-score at the 2-year follow-up with and without controlling for residential density. Residential density was not significantly associated with BMI z-score at follow-up when greenness was not included in the model, and only marginally associated when greenness was controlled for in the model.

Finally, two studies involving a small cohort of Australian children examined the association of physical activity environmental factors on change in BMI z-score in 140 children ages 5 to 6 and 269 children ages 10 to 12 over 3 years (75), and on average BMI z-score in 301 children ages 10 to 12 over 5 years (77). Both studies examined multiple physical activity environmental features, including destinations (i.e. public open spaces, sports options, walking/cycling tracks, distance to school), road connectivity (i.e. number of cul-de-sacs, density of intersections, proportion of four-way intersections, length of access paths) and traffic exposure (i.e. length of busy and local roads). Two different buffer sizes were used, 800 m and 2 km. Analyses controlled for various socio-demographic and home characteristics. In the first study, road connectivity was inversely associated with a 3-year change in child BMI z-score, whereas access to destinations and traffic exposures were not (75). In the second study, none of the neighbourhood (i.e. physical activity environment) variables were associated with a 5-year change in BMI z-score after controlling for age, maternal education, role modelling, parental social support and rules and restrictions for sedentary behaviour (77).

In sum, there are mixed results regarding the association between the community physical activity environment and excessive fatness. Methodological issues, particularly the inconsistency in operationalization of physical activity environmental variables, heterogeneity in measurement approaches (78) and salience of the geographic buffer (e.g. distance of 800 m, 2 km, around a child's home) are evident and likely contribute to the mixed results. In order to effectively examine the role of the community physical activity environment and the development of excessive fatness throughout childhood and into adolescence, these current limitations need to be considered.

Nutrition environment factors. The community nutrition environment may associate with dietary behaviour and ultimately weight status in children and adolescents through various factors, including type and location of food outlets and accessibility of food outlets (e.g. hours of operation, availability of drive-through windows) (79). This review identified only one study that examined the association of the nutrition environment on change in BMI in children. Leung *et al.* (80) examined the association between the density of different types of food outlets (e.g. convenience stores, supermarkets, fast food restaurants) within 0.25 mile and 1.0 mile buffer of the child's home and change in BMI z-score over 3 years in a sample of 353 girls ages 6 to 7. Of all of the food outlets, only a higher availability of convenience stores within 0.25 mile of the child's home was significantly associated with a 3-year increase in BMI z-score and overweight/obesity status; however, this association was not present when the buffer around the home was extended to 1.0 mile.

Given the lack of prospective evidence of the association between the community nutrition environment and the development of excessive fatness in children and adolescents, no conclusions can be drawn.

Discussion

This review summarized the current scientific evidence on the predictors of the development of excessive fatness in children and adolescents. Adopting a social ecological framework, this review examined key variables across multiple levels (intrapersonal, interpersonal, institutional and community) that may relate to excessive gain in BMI and fat mass over time. Overall, the available prospective evidence indicates that genetic predisposition and low levels of physical activity are associated with the development of excessive fatness in children and adolescents. Current studies yielded mixed evidence for the contribution of sedentary behaviour, dietary intake, some physiological biomarkers, family factors and the community physical activity environment. Due to the limited number of prospective studies identified, no conclusions could be made about the association of the social cognitive factors, peer factors, school nutrition and school physical activity factors and the community nutrition environment. Therefore, it appears that the closer to the right side of our conceptual model, the stronger the evidence, whereas further to the left, the evidence is less studied, possibly due to the presence of the additional factors.

For the majority of studies identified, there was limited and inconclusive evidence of a relationship with the development of excessive fatness in children and adolescents. This lack of association could be explained by several issues. It is possible that the samples used in the studies are not representative of the wider population, or that the

model does not apply to certain ages, genders or race/ethnicities. Further, the lack of relationship may be due to poor measurement of the constructs contributing to low reliability and validity. Targeted behaviours are complex but some have been measured simply. Studies adopting a social ecological framework are limited by the number of factors they can operationalize and evaluate (6).

Heterogeneity between the studies including differences in the measures used, populations sampled and limited adjustment for pubertal status are likely important factors contributing to the mixed results. For example, the inconsistencies could be explained in part by measurement issues related to the use of non-criterion measures of fat mass (e.g. BMI, skin-folds and waist circumference). There is evidence that non-criterion measures produce relatively larger measurement errors when assessing body fatness in children than criterion measures (81). These measurement errors may mask the true relationship between the various factors examined and the development of childhood obesity. Specifically, it is recognized that BMI is not equivalent to body fatness, and studies that used BMI as the dependent variable should be interpreted with that limitation in mind. Heterogeneity in participant characteristics, environment, culture and geographic location (all important contextual variables in the social ecological theory) could also explain the mixed findings. Additionally, some researchers examined very specific populations (17,56,61) which do not allow for adjustment for race/ethnicity. Lastly, pubertal status is an important consideration as children develop and mature at different rates (82).

While most of the studies adjusted for potential confounders (e.g. sex, age, race/ethnicity), very few studies examined the interaction effect among factors on the association of interest. For instance, only one study (63) examined the interaction between depressed mood and several factors (e.g. physical activity, smoking) on the development of excessive fatness. Therefore, the literature is characterized by a narrow focus, typically evaluating the relationship between longitudinal change in fat mass and one individual or environmental factor at a time. This approach differs markedly from the social ecological model of health behaviour, which recognizes that multiple factors interact and simultaneously affect individual health behaviour and health outcomes. Future prospective studies need to assess multiple factors and examine their interactions with the development of excessive fatness in children and adolescents. Because human behaviour is highly complex, advanced statistical models are needed to examine the association of multiple variables as well as any interactions between variables (5).

We limited the review to prospective studies because through temporal sequencing, prospective studies identify the factors that associate with the development of excessive fatness in children and adolescents. This is particularly

important with children and adolescents, as they are growing and developing at a rapid rate. The critical factors that predict energy balance for young children may differ from the critical factors for older adolescents. We recognize that prospective studies demonstrate association, and not necessarily causation, and that prospective studies have the potential for biases and confounding, similar to cross-sectional studies. While we chose to limit this review to prospective studies published from 1990 to 2011, we note that researchers could extend this work by examining the findings of other study designs, and by examining earlier publications.

The limited number of identified prospective studies underscores the need to conduct more of this type of research in order to fully understand the aetiology of childhood overweight and obesity. While cross-sectional studies are valuable, a prospective design can add substantially to our understanding of the development of excessive fatness in youth by focusing on the following issues: how factors associate with the development of excessive fatness, the most salient factors contributing to the development of excessive fatness in youth, the developmental phases or critical periods that are instrumental in contributing to weight gain over time and the interrelationship between these factors. Longitudinal studies that include longer follow-up periods beginning in early childhood and into adolescence is an important next step to identifying the prospective (and potentially causal) association between key variables and excessive weight gain over time. Researchers should plan and seek funding for longitudinal studies with extended follow-up periods. If feasible, resources should be used to outreach children from previous studies in order to provide more complete longitudinal data. Further, the use of criterion measures and state-of-the-art measures integrated uniformly between studies will allow researchers to draw more accurate conclusions across such studies.

In conclusion, the current review highlights the degree to which research on development of overweight and obesity in youth has yet to take advantage of prospective designs. Given that childhood obesity is a critical public health problem of the 21st century, the field can benefit from additional large-scale, long-term prospective studies that use criterion and state-of-the-art measures. Longitudinal studies are needed to identify the relationships between factors and the underlying mechanisms. Future prospective studies should examine multiple factors across the social ecological model in order to develop an understanding of the complex nature of factors contributing to the aetiology of obesity in children and adolescents. Although there is evidence to suggest some variables predict the development of excessive fatness in children and adolescents, no prospective study has comprehensively assessed the association of multiple variables and the interactions between variables

on the development of excessive fatness using state-of-the-art measures in a large, diverse sample of children and adolescents.

Conflict of interest statement

Authors declare no conflicts of interest.

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References

- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999–2010. *JAMA* 2012; 307: 483–490.
- Huang TT, Johnson MS, Figueroa-Colon R, Dwyer JH, Goran MI. Growth of visceral fat, subcutaneous abdominal fat, and total body fat in children. *Obes Res* 2001; 9: 283–289.
- McLeroy K, Bibeau D, Steckler A, Glanz K. An ecological perspective on health promotion programs. *Health Educ Q* 1988; 15: 351–377.
- Bronfenbrenner U. *The Ecology of Human Development: Experiments by Nature and Design*. Harvard University Press: Cambridge, MA, 1979.
- Baranowski T, Cullen KW, Nicklas T, Thompson D, Baranowski T. Are current health behavioral change models helpful in guiding prevention of weight gain efforts? *Obes Res* 2003; 11: 23S–43S.
- Lytle LA. Examining the etiology of childhood obesity: the IDEA study. *Am J Community Psychol* 2009; 44: 338–349.
- Egger G, Swinburn B. An ‘ecological’ approach to the obesity pandemic. *BMJ* 1997; 315: 477–480.
- Huang TT, Glass TA. Transforming research strategies for understanding and preventing obesity. *JAMA* 2008; 300: 1811–1813.
- Sirard J, Pate RR. Physical activity assessment in children and adolescents. *Sports Med* 2001; 31: 439–454.
- Welk GJ, Corbin CB, Dale D. Measurement issues in the assessment of physical activity in children. *Res Q Exerc Sport* 2000; 71: S59–S73.
- Calfas KJ, Sallis JF, Nader PR. The development of scales to measure knowledge and prevalence for diet and physical activity behavior in 4- to 8-year-old children. *J Dev Behav Pediatr* 1991; 12: 185–190.
- Rowlands AV, Eston RG. The measurement and interpretation of children’s physical activity. *J Sports Sci Med* 2007; 6: 270–276.
- Janz KF, Burns TL, Levy SM. Tracking of activity and sedentary behaviors in childhood: the Iowa Bone Development Study. *Am J Prev Med* 2005; 29: 171–178.
- Janz KF, Kwon S, Letuchy EM *et al.* Sustained effect of early physical activity on body fat mass in older children. *Am J Prev Med* 2009; 37: 35–40.
- Riddoch CJ, Leary SD, Ness AR *et al.* Prospective associations between objective measures of physical activity and fat mass in 12–14 year old children: the Avon Longitudinal Study of Parents and Children (ALSPAC). *BMJ* 2009; 339: b4544.
- Fisher A, Hill C, Webber L, Purslow L, Wardle J. MVPA is associated with lower weight gain in 8–10 year old children: a prospective study with 1 year follow-up. *PLoS ONE* 2011; 6: e18576.
- Stevens J, Suchindran C, Ring K *et al.* Physical activity as a predictor of body composition in American Indian children. *Obes Res* 2004; 12: 1974–1980.
- Moore LL, Gao D, Bradlee ML *et al.* Does early physical activity predict body fat change throughout childhood? *Prev Med* 2003; 37: 10–17.
- Metcalf BS, Voss LD, Hosking J, Jeffery TN, Wilkin TJ. Physical activity at the government-recommended level and obesity-related health outcomes: a longitudinal study (EarlyBird 37). *Arch Dis Child* 2008; 93: 772–777.
- Taylor RW, Williams SM, Carter PJ, Goulding A, Gerrard DF, Taylor BJ. Changes in fat mass and fat-free mass during the adiposity rebound: FLAME study. *Int J Pediatr Obes* 2011; 6: e243–e251.
- Pate RR, O’Neill JR, Lobelo F. The evolving definition of ‘sedentary’. *Exerc Sport Sci Rev* 2008; 36: 173–178.
- Basterfield L, Pearce MS, Adamson AJ *et al.* Physical activity, sedentary behavior, and adiposity in English children. *Am J Prev Med* 2012; 42: 445–451.
- Mitchell JA, Pate RR, Beets MW, Nader PR. Time spent in sedentary behavior and changes in childhood BMI: a longitudinal study from ages 9 to 15 years. *Int J Obes (Lond)* 2013; 37: 54–60. doi: 10.1038/ijo.2012.41.
- Berkey CS, Rockett HR, Field AE *et al.* Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics* 2000; 105: E56.
- Koutedakis Y, Bouziotas C, Flouris AD, Nelson PN. Longitudinal modeling of adiposity in periadolescent Greek schoolchildren. *Med Sci Sports Exerc* 2005; 37: 2070–2074.
- Magarey AM, Daniels LA, Boulton TJ, Cockington RA. Does fat intake predict adiposity in healthy children and adolescents aged 2–15 y? A longitudinal analysis. *Eur J Clin Nutr* 2001; 55: 471–481.
- Hoppe C, Molgaard C, Thomsen BL, Juul A, Michaelsen KF. Protein intake at 9 mo of age is associated with body size but not with body fat in 10-y-old Danish children. *Am J Clin Nutr* 2004; 79: 494–501.
- Brixval CS, Andersen LB, Heitmann BL. Fat intake and weight development from 9 to 16 years of age: the European Youth Heart Study – a longitudinal study. *Obes Facts* 2009; 2: 166–170.
- McCaffrey TA, Rennie KL, Kerr MA *et al.* Energy density of the diet and change in body fatness from childhood to adolescence; is there a relation? *Am J Clin Nutr* 2008; 87: 1230–1237.
- Moore LL, Bradlee ML, Gao D, Singer MR. Low dairy intake in early childhood predicts excess body fat gain. *Obesity (Silver Spring)* 2006; 14: 1010–1018.

31. Skinner JD, Bounds W, Carruth BR, Ziegler P. Longitudinal calcium intake is negatively related to children's body fat indexes. *J Am Diet Assoc* 2003; 103: 1626–1631.
32. Phillips SM, Bandini LG, Cyr H, Colclough-Douglas S, Naumova E, Must A. Dairy food consumption and body weight and fatness studied longitudinally over the adolescent period. *Int J Obes Relat Metab Disord* 2003; 27: 1106–1113.
33. Berkey CS, Rockett HR, Willett WC, Colditz GA. Milk, dairy fat, dietary calcium, and weight gain: a longitudinal study of adolescents. *Arch Pediatr Adolesc Med* 2005; 159: 543–550.
34. Field AE, Gillman MW, Rosner B, Rockett HR, Colditz GA. Association between fruit and vegetable intake and change in body mass index among a large sample of children and adolescents in the United States. *Int J Obes Relat Metab Disord* 2003; 27: 821–826.
35. Phillips SM, Bandini LG, Naumova EN *et al.* Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obes Res* 2004; 12: 461–472.
36. Field AE, Austin SB, Gillman MW, Rosner B, Rockett HR, Colditz GA. Snack food intake does not predict weight change among children and adolescents. *Int J Obes Relat Metab Disord* 2004; 28: 1210–1216.
37. Francis LA, Lee Y, Birch LL. Parental weight status and girls' television viewing, snacking, and body mass indexes. *Obes Res* 2003; 11: 143–151.
38. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001; 357: 505–508.
39. Striegel-Moore RH, Thompson D, Affenito SG *et al.* Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr* 2006; 148: 183–187.
40. Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA. Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study. *Int J Obes (Lond)* 2006; 30: 1091–1093.
41. Fiorito LM, Marini M, Francis LA, Smiciklas-Wright H, Birch LL. Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. *Am J Clin Nutr* 2009; 90: 935–942.
42. 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.
43. Libuda L, Alexy U, Sichert-Hellert W *et al.* Pattern of beverage consumption and long-term association with body-weight status in German adolescents—results from the DONALD study. *Br J Nutr* 2008; 99: 1370–1379.
44. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. *Obes Res* 2004; 12: 778–788.
45. 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.
46. Northstone K, Emmett PM. Are dietary patterns stable throughout early and mid-childhood? A birth cohort study. *Br J Nutr* 2008; 100: 1069–1076.
47. Haworth CM, Carnell S, Meaburn EL, Davis OS, Plomin R, Wardle J. Increasing heritability of BMI and stronger associations with the FTO gene over childhood. *Obesity (Silver Spring)* 2008; 16: 2663–2668.
48. Burke V, Beilin LJ, Dunbar D. Family lifestyle and parental body mass index as predictors of body mass index in Australian children: a longitudinal study. *Int J Obes Relat Metab Disord* 2001; 25: 147–157.
49. Sorensen TI, Holst C, Stunkard AJ. Childhood body mass index – genetic and familial environmental influences assessed in a longitudinal adoption study. *Int J Obes Relat Metab Disord* 1992; 16: 705–714.
50. Lajunen HR, Kaprio J, Keski-Rahkonen A *et al.* Genetic and environmental effects on body mass index during adolescence: a prospective study among Finnish twins. *Int J Obes (Lond)* 2009; 33: 559–567.
51. Beunen G, Maes HH, Vlietinck R *et al.* Univariate and multivariate genetic analysis of subcutaneous fatness and fat distribution in early adolescence. *Behav Genet* 1998; 28: 279–288.
52. Cornes BK, Zhu G, Martin NG. Sex differences in genetic variation in weight: a longitudinal study of body mass index in adolescent twins. *Behav Genet* 2007; 37: 648–660.
53. Mamun AA, Lawlor DA, O'Callaghan MJ, Williams GM, Najman JM. Family and early life factors associated with changes in overweight status between ages 5 and 14 years: findings from the Mater University Study of Pregnancy and its outcomes. *Int J Obes (Lond)* 2005; 29: 475–482.
54. Perez-Pastor EM, Metcalf BS, Hosking J, Jeffery AN, Voss LD, Wilkin TJ. Assortative weight gain in mother-daughter and father-son pairs: an emerging source of childhood obesity. Longitudinal study of trios (EarlyBird 43). *Int J Obes (Lond)* 2009; 33: 727–735.
55. Johnson MS, Figueroa-Colon R, Huang TT, Dwyer JH, Goran MI. Longitudinal changes in body fat in African American and Caucasian children: influence of fasting insulin and insulin sensitivity. *J Clin Endocrinol Metab* 2001; 86: 3182–3187.
56. Odeleye OE, de Courten M, Pettitt DJ, Ravussin E. Fasting hyperinsulinemia is a predictor of increased body weight gain and obesity in Pima Indian children. *Diabetes* 1997; 46: 1341–1345.
57. Morrison JA, Glueck CJ, Horn PS, Schreiber GB, Wang P. Pre-teen insulin resistance predicts weight gain, impaired fasting glucose, and type 2 diabetes at age 18–19 y: a 10-y prospective study of black and white girls. *Am J Clin Nutr* 2008; 88: 778–788.
58. Travers SH, Jeffers BW, Eckel RH. Insulin resistance during puberty and future fat accumulation. *J Clin Endocrinol Metab* 2002; 87: 3814–3818.
59. Huang KC, Lin RC, Kormas N *et al.* Plasma leptin is associated with insulin resistance independent of age, body mass index, fat mass, lipids, and pubertal development in nondiabetic adolescents. *Int J Obes Relat Metab Disord* 2004; 28: 470–475.
60. Johnson MS, Huang TT, Figueroa-Colon R, Dwyer JH, Goran MI. Influence of leptin on changes in body fat during growth in African American and white children. *Obes Res* 2001; 9: 593–598.
61. Nishimura R, Sano H, Matsudaira T *et al.* Changes in body mass index, leptin and adiponectin in Japanese children during a three-year follow-up period: a population-based cohort study. *Cardiovasc Diabetol* 2009; 8: 30.
62. Butte NF, Cai G, Cole SA *et al.* Metabolic and behavioral predictors of weight gain in Hispanic children: the Viva la Familia Study. *Am J Clin Nutr* 2007; 85: 1478–1485.
63. Goodman E, Whitaker RC. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 2002; 110: 497–504.
64. Hesketh K, Wake M, Waters E. Body mass index and parent-reported self-esteem in elementary school children: evidence for a causal relationship. *Int J Obes Relat Metab Disord* 2004; 28: 1233–1237.
65. Duckworth AL, Tsukayama E, Geier AB. Self-controlled children stay leaner in the transition to adolescence. *Appetite* 2010; 54: 304–308.

66. Spruijt-Metz D, Li C, Cohen E, Birch L, Goran M. Longitudinal influence of mother's child-feeding practices on adiposity in children. *J Pediatr* 2006; **148**: 314–320.
67. Campbell K, Andrianopoulos N, Hesketh K *et al*. Parental use of restrictive feeding practices and child BMI z-score. A 3-year prospective cohort study. *Appetite* 2010; **55**: 84–88.
68. Faith MS, Berkowitz RI, Stallings VA, Kerns J, Storey M, Stunkard AJ. Parental feeding attitudes and styles and child body mass index: prospective analysis of a gene-environment interaction. *Pediatrics* 2004; **114**: e429–e436.
69. Fulkerson JA, Neumark-Sztainer D, Hannan PJ, Story M. Family meal frequency and weight status among adolescents: cross-sectional and 5-year longitudinal associations. *Obesity (Silver Spring)* 2008; **16**: 2529–2534.
70. Lumeng JC, Rahnama S, Appugliese D, Kacirotin N, Bradley RH. Television exposure and overweight risk in preschoolers. *Arch Pediatr Adolesc Med* 2006; **160**: 417–422.
71. von Hinke Kessler SS. Maternal employment and overweight children: does timing matter? *Health Econ* 2008; **17**: 889–906.
72. Briefel RR, Wilson A, Gleason PM. Consumption of low-nutrient, energy-dense foods and beverages at school, home, and other locations among school lunch participants and nonparticipants. *J Am Diet Assoc* 2009; **109**: S79–S90.
73. Fernandes MM, Sturm R. The role of school physical activity programs in child body mass trajectory. *J Phys Act Health* 2011; **8**: 174–181.
74. Bell JF, Wilson JS, Liu GC. Neighborhood greenness and 2-year changes in body mass index of children and youth. *Am J Prev Med* 2008; **35**: 547–553.
75. Timperio A, Jeffery RW, Crawford D, Roberts R, Giles-Corti B, Ball K. Neighbourhood physical activity environments and adiposity in children and mothers: a three-year longitudinal study. *Int J Behav Nutr Phys Act* 2010; **7**: 18.
76. Wolch J, Jerrett M, Reynolds K *et al*. Childhood obesity and proximity to urban parks and recreational resources: a longitudinal cohort study. *Health Place* 2011; **17**: 207–214.
77. Crawford D, Cleland V, Timperio A *et al*. The longitudinal influence of home and neighbourhood environments on children's body mass index and physical activity over 5 years: the CLAN study. *Int J Obes (Lond)* 2010; **34**: 1177–1187.
78. Brownson RC, Hoehner CM, Day K, Forsyth A, Sallis JF. Measuring the built environment for physical activity: state of the science. *Am J Prev Med* 2009; **36**: S99–S123.
79. Glanz K, Sallis JF, Saelens BE, Frank LD. Healthy nutrition environments: concepts and measures. *Am J Health Promot* 2005; **19**: 330–333.
80. Leung CW, Laraia BA, Kelly M *et al*. The influence of neighborhood food stores on change in young girls' body mass index. *Am J Prev Med* 2011; **41**: 43–51.
81. Himes JH. Challenges of accurately measuring and using BMI and other indicators of obesity in children. *Pediatrics* 2009; **124**: S3–C22.
82. Baxter-Jones AD, Eisenmann JC, Sherar LB. Controlling for maturation in pediatric exercise science. *Pediatr Exerc Sci* 2005; **17**: 18–30.