

Childhood Obesity and Associated Risk Factors: A Review and Cross-Sectional Analysis

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Abstract-

Background: Childhood obesity has become one of the most pressing public health challenges of the twenty-first century, with prevalence continuing to rise across both high-income and low- and middle-income settings. Identifying the modifiable and non-modifiable factors that drive excess weight gain in children is essential for designing effective prevention strategies. **Objective:** This study aimed to review the global epidemiology of childhood obesity and to examine the principal risk factors associated with its development, integrating evidence from recent epidemiological literature with an illustrative cross-sectional analysis. **Methods:** A narrative synthesis of peer-reviewed literature published largely between 2015 and 2026 was combined with secondary analysis of risk-factor associations reported in large cohort and cross-sectional studies. Variables examined included parental obesity, birth weight, dietary patterns, screen time, sedentary behaviour, sleep duration, physical activity, socioeconomic status, and early-life feeding practices. **Results:** Global obesity prevalence among children and adolescents aged 5-19 years was estimated at 9.3% in boys and 6.9% in girls, equivalent to roughly 159 million affected children. Parental obesity, particularly maternal obesity, showed the strongest and most consistent association with childhood obesity (odds ratios ranging from 1.7 to 2.6). Excessive screen time, short sleep duration, low household income, low parental education, high birth weight, and frequent consumption of fast food and sugar-sweetened beverages were also significantly associated with elevated obesity risk, whereas regular family meals, outdoor physical activity, and exclusive breastfeeding were protective. **Conclusion:** Childhood obesity arises from a complex interaction of genetic, familial, behavioural, and environmental determinants. Multi-level interventions that simultaneously target parental health behaviours, household environments, screen-time regulation, sleep hygiene, and dietary quality are likely to be more effective than single-component programmes.

Keywords: *childhood obesity; overweight; risk factors; sedentary behaviour; screen time; parental obesity; pediatric.*

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INTRODUCTION

Childhood obesity is now recognised as a chronic, relapsing disease rather than simply a transient phase of excess weight, with measurable consequences for physical and psychological health well before adulthood¹. Globally, the prevalence of overweight and obesity in children and adolescents has risen sharply over the past four decades, and the World Health Organization's 2025 target of preventing any increase in adolescent obesity between 2010 and 2025 has already been missed in most countries¹. According to the most recent NCD Risk Factor Collaboration estimates, 9.3% of boys and 6.9% of girls aged 5 to 19 years were obese in 2022, corresponding to approximately 159 million school-aged children and adolescents worldwide¹. Projections from the World Obesity Federation suggest that the number of affected children and adolescents will continue to climb, from roughly 158 million in 2020 to an estimated 254 million by 2030³.

In the United States, national surveillance data indicate that approximately one in five children and adolescents aged 2 to 19 years has obesity, with disproportionately higher rates among adolescents, Hispanic and non-Hispanic Black children, and children living in lower-income households². Disparities of this kind are not unique to high-income countries; obesity prevalence is now rising rapidly in low- and middle-income settings as well, where urbanisation, changing food environments, and reduced opportunities for active play are reshaping childhood lifestyles¹. The clinical significance of this trend extends well beyond cosmetic or social concerns. Childhood obesity is associated with early-onset type 2 diabetes, hypertension, dyslipidaemia, metabolic-associated fatty liver disease, and orthopaedic and psychosocial complications, and it substantially increases the risk of remaining obese into adulthood⁴.

The aetiology of childhood obesity is multifactorial, reflecting an interaction of genetic predisposition, epigenetic programming, family environment, and broader social and commercial determinants of health⁵. Among the most consistently reported determinants is parental weight status. A landmark longitudinal analysis demonstrated that parental obesity roughly doubles a child's risk of becoming an obese adult, and this effect is particularly strong before the age of three years, when parental obesity is a stronger predictor of future weight than the child's own weight status at that age⁷. A birth-cohort study from Japan similarly reported odds ratios of 1.70 for paternal obesity and 2.56 for maternal obesity in predicting obesity among three-year-old children⁸, a pattern that has been replicated across diverse populations and is thought to reflect a combination of shared genetic susceptibility and shared household dietary and activity patterns⁵.

Beyond the family environment, early-life exposures occurring during the so-called first 1000 days, from conception to two years of age, have attracted growing research attention. A recent systematic review identified maternal pre-pregnancy weight, excessive gestational weight gain, maternal smoking during pregnancy, higher birth weight, and large-for-gestational-age status as the most strongly and consistently associated early-life risk factors for later childhood obesity⁶. Postnatal behavioural factors are equally important. Excessive recreational screen time, now exceeding the recommended two-hour daily limit in 45% to 80% of children in several high-income countries, has been linked to obesity both directly, through the displacement of physical activity, and indirectly, through its association with increased snacking, poorer diet quality, and shortened sleep duration¹¹. Short sleep duration itself appears to independently raise obesity risk, possibly through hormonal pathways affecting appetite regulation as well as behavioural pathways related to late-night screen exposure¹⁰.

Socioeconomic and dietary factors add a further layer of complexity. Lower household income and lower parental educational attainment have repeatedly been associated with higher childhood obesity risk, even after adjustment for ethnicity²⁶, while frequent fast-food consumption and purchasing lunch at school have been linked to increased odds of overweight, in contrast to the protective association observed with regular family meals¹³. Taken together, this body of evidence suggests that effective prevention of childhood obesity will require coordinated action across the family, school, and community environments rather than reliance on any single intervention target⁵. The present article reviews this evidence base and reports an illustrative analysis of risk-factor associations drawn from recent cohort and cross-sectional studies, with the aim of providing a consolidated, up-to-date picture of the determinants of childhood obesity to inform clinical practice and public health policy.

MATERIALS AND METHODS

Study design: This article combines a structured narrative review of the literature with a secondary, illustrative cross-sectional analysis of risk-factor associations reported in previously published cohort and cross-sectional studies of children and adolescents. The objective was to synthesise consistent, quantifiable associations between candidate risk factors and childhood obesity rather than to generate new primary data.

Search strategy: A literature search was conducted across PubMed/MEDLINE, Scopus, and Google Scholar for articles published predominantly between January 2015 and June 2026, supplemented by foundational studies of historical importance where relevant. Search terms combined "childhood obesity", "pediatric obesity", "overweight", "risk factors", "screen time", "sedentary behaviour", "parental obesity", "sleep duration", "socioeconomic status", and "dietary habits", using Boolean operators. Reference lists of identified articles and relevant systematic reviews were hand-searched for additional eligible studies.

Eligibility criteria: Studies were eligible for inclusion if they (a) enrolled children or adolescents aged 0-18 years, (b) reported obesity or overweight as an outcome defined using validated anthropometric criteria such as body mass index (BMI) percentile-for-age-and-sex, BMI z-score, or national/international growth reference standards, and (c) reported quantitative associations (odds ratios, relative risks, or prevalence estimates) between obesity/overweight and at least one candidate risk factor. Editorials, case reports, and studies without extractable quantitative risk estimates were excluded.

Population and definitions: For the purposes of this review, overweight and obesity in children and adolescents were defined according to World Health Organization and Centers for Disease Control and Prevention criteria, namely a BMI-for-age at or above the 85th percentile for overweight and at or above the 95th percentile for obesity, using sex-specific growth charts. Where individual studies used alternative national reference standards, the original study definitions were retained and are noted in the results tables.

Risk factor categories: Candidate risk factors were grouped into five domains for analysis: (1) perinatal and early-life factors (parental pre-pregnancy weight, gestational weight gain, birth weight, infant feeding practice); (2) family and genetic factors (parental obesity, family history); (3) behavioural and lifestyle factors (dietary patterns, physical activity, sedentary behaviour, screen time, sleep duration); (4) socioeconomic and environmental factors (household income,

parental education, neighbourhood characteristics, school food environment); and (5) demographic factors (age, sex, ethnicity).

Data extraction and synthesis: For each eligible study, the following data were extracted into a structured spreadsheet: first author, publication year, country, study design, sample size, age range, obesity definition used, and the adjusted effect estimate (odds ratio or relative risk) with 95% confidence interval for each reported risk factor. Where multiple adjusted models were reported, the most fully adjusted estimate was extracted preferentially. Because included studies varied substantially in design, population, and adjustment strategy, a formal meta-analytic pooling of effect sizes was not performed; instead, findings are synthesised narratively and summarised descriptively in tabular form, consistent with a scoping/narrative review methodology.

Statistical presentation: Descriptive prevalence estimates are reported as percentages with 95% confidence intervals where available. Associations between candidate risk factors and obesity are reported as adjusted odds ratios (aOR) with corresponding 95% confidence intervals, as published in the source studies. An odds ratio with a 95% confidence interval excluding 1.0 was considered statistically significant. All prevalence and association data presented in the Results section are drawn from the published literature identified through the search strategy described above; no new human-subjects data were collected for this article, and no ethical approval was therefore required.

Quality considerations: Because the review draws on observational studies of varying design (cross-sectional, prospective cohort, and birth-cohort studies), causal inference is limited, and associations should be interpreted as indicative of statistical relationship rather than proven causation. This limitation is addressed further in the Discussion.

RESULTS

The literature search and synthesis identified consistent evidence across multiple geographic settings regarding the prevalence of childhood obesity and its principal risk factors. Findings are summarised in Tables 1 to 4 below, followed by a narrative explanation of each table.

Table 1. Global and regional prevalence of childhood and adolescent overweight/obesity

Population / Region	Age group	Overweight/Obesity prevalence	Source
Global, boys	5-19 years	9.3% obese	NCD-RisC, 2022 data
Global, girls	5-19 years	6.9% obese	NCD-RisC, 2022 data
United States	2-19 years	19.7% obese (~14.7 million)	NHANES 2017-Mar 2020
United States, age 12-19	12-19 years	Highest among age bands	NHANES 2017-Mar 2020
Canada (provincial)	Grade 5 (~10-11 yrs)	32.9% overweight; 9.9% obese	Veugelers & Fitzgerald, 2005
China, preschool (Shaanxi)	0-3 years	Rising trend, 2012-2024	NCNHSS registry
Global forecast	5-19 years	~254 million projected by 2030	World Obesity Federation

Note: Definitions of overweight/obesity vary slightly by source (WHO/CDC BMI-for-age percentiles vs. national reference standards); see Materials and Methods.

As shown in Table 1, global obesity prevalence among school-aged children and adolescents is estimated at 9.3% in boys and 6.9% in girls, with substantial cross-national variation. United States national surveillance data show a considerably higher national prevalence of approximately 19.7%, reflecting both differing population characteristics and differing

measurement periods. Notably, prevalence tends to rise with age through adolescence, and projections indicate a continued upward trajectory globally through 2030 in the absence of effective intervention.

Table 2. Perinatal and family-level risk factors associated with childhood obesity

Risk factor	Comparison group	Adjusted OR (95% CI)	Source population
Maternal obesity	Non-obese mother	2.56 (2.07-3.17)	Toyama Birth Cohort, Japan
Paternal obesity	Non-obese father	1.70 (1.43-2.02)	Toyama Birth Cohort, Japan
Both parents obese (child age 3-9 yrs)	Neither parent obese	Substantially increased odds*	NEJM longitudinal cohort
High birth weight	Normal birth weight	Significant positive association*	Preschool logistic model, China
Ethnicity (Black, White/European) vs.	White/European	2.7 (1.9-3.9)	UK Millennium Cohort Study
Ethnicity (Asian, White/European) vs.	White/European	1.7 (1.2-2.3)	UK Millennium Cohort Study

*Reported as statistically significant in adjusted models; exact OR not uniformly reported across studies. CI = confidence interval.

Table 2 highlights the strength of family-level determinants. Maternal obesity showed the single strongest association with childhood obesity among the factors reviewed, with odds more than double those of children with non-obese mothers, while paternal obesity carried a smaller but still significant association. Children with both parents obese carried substantially higher risk than children with only one obese parent, particularly when parental obesity was present before the child's third birthday. Ethnic disparities persisted even after adjustment for household income and parental education, suggesting that ethnicity captures additional unmeasured genetic, cultural, or structural factors.

Table 3. Behavioural and lifestyle risk factors associated with childhood obesity

Risk factor	Comparison group	Adjusted OR (95% CI)	Source population
Television viewing >3 hrs/day	<3 hrs/day	1.38 (1.09-1.76)	US YRBS, high school students
Not participating in school sports team	Participating	1.61 (1.31-1.98)	US YRBS, high school students
Purchasing lunch at school	Bringing lunch from home	1.39 (1.16-1.67)	Canadian Grade 5 cohort
Family supper together \geq 3x/week	Less frequent family meals	0.68 (0.52-0.88) [protective]	Canadian Grade 5 cohort
Short sleep duration	Recommended sleep duration	Dose-response increase in risk*	Toyama Birth Cohort; multiple reviews
Excessive recreational screen time (>2 hrs/day)	Within guideline	Consistently positive association*	Multiple cross-sectional studies
Frequent junk food / fast-food intake	Infrequent intake	Significant positive association*	Preschool logistic model, China; US cohort

*Pooled effect size not calculable across heterogeneous studies; direction and significance consistently reported. OR = odds ratio.

As summarised in Table 3, modifiable behavioural factors showed consistent, statistically significant associations with obesity risk, although individual effect sizes were generally more modest than those observed for parental obesity. Reduced physical activity, indexed either by non-participation in organised sport or by high television viewing time, was associated with 38% to 61% higher odds of obesity. School food environment also mattered: children who purchased lunch at school had higher odds of overweight than those who brought lunch from home, while frequent shared family meals were associated with substantially lower odds, an effect of similar magnitude in the opposite direction. Short sleep duration and excessive screen time showed consistent positive associations with obesity across multiple independent cohorts, though heterogeneity in measurement methods precluded a single pooled estimate.

Table 4. Socioeconomic and protective factors associated with childhood obesity

Factor	Direction of association	Strength of evidence	Representative source
Low household income	Increased risk	Consistent across high-income countries	UK Millennium Cohort Study
Low parental educational attainment	Increased risk	Consistent	UK Millennium Cohort Study
Higher parental education level	Protective	Consistent	Preschool logistic model, China
Longer daily outdoor activity time	Protective	Consistent	Preschool logistic model, China
Exclusive/longer breastfeeding duration	Protective	Moderate, some heterogeneity	First-1000-days systematic review
Regular family meal routines	Protective	Consistent	US preschool household routines study

Table 4 summarises socioeconomic and household-level factors. Lower household income and lower parental education were consistently linked to greater obesity risk, independent of ethnicity, whereas longer parental education, regular outdoor activity, structured household routines, and longer breastfeeding duration were associated with reduced risk. These protective associations point toward feasible, low-cost targets for community-level prevention programmes.

DISCUSSION

The findings synthesised in this review confirm that childhood obesity is driven by a layered set of determinants spanning the perinatal period, the family environment, individual behaviour, and the broader socioeconomic context, consistent with current conceptual models of pediatric obesity aetiology⁵. The magnitude of the association observed for parental, and particularly maternal, obesity is striking and aligns with decades of longitudinal evidence showing that parental weight status is among the strongest single predictors of a child's long-term weight trajectory⁷. This pattern likely reflects the combined influence of heritable genetic susceptibility and a shared "obesogenic" household environment, in which food choices, portion sizes, and activity habits are transmitted from parent to child well before the child can exercise independent dietary or behavioural autonomy⁸. The stronger effect of maternal compared with paternal obesity observed across multiple cohorts may additionally reflect intrauterine programming effects related to maternal metabolic status during pregnancy, a hypothesis supported by the first-1000-days literature identifying maternal pre-pregnancy weight and gestational weight gain as leading early-life risk factors⁶.

The behavioural risk factors identified, particularly excessive screen time, physical inactivity, and short sleep duration, are notable because they are, at least in principle, highly modifiable. However, the relatively modest individual effect sizes observed for these factors, compared with parental obesity, are consistent with findings from a 2024 Cochrane review showing that population-based interventions targeting diet or physical activity in isolation produced only limited or marginal benefit, particularly among adolescents⁵. One plausible explanation is that these behaviours rarely operate in isolation; screen time, sedentary behaviour, and sleep curtailment appear to cluster together and to reinforce one another, such that children who fail to meet recommendations in all three domains simultaneously face markedly higher cumulative risk than would be predicted from any single behaviour alone¹⁰. This clustering effect has important implications for intervention design: programmes that address screen time, physical activity, and sleep together may be more effective than those targeting a single behaviour, although robust trial evidence for this combined approach remains limited.

The socioeconomic gradient observed in this review, whereby lower household income and lower parental education were consistently associated with higher obesity risk even after adjustment for ethnicity, mirrors a broader pattern documented in national surveillance data showing disproportionate obesity burden among children from lower-income families and certain racial and ethnic minority groups². This gradient is unlikely to be explained by individual choice alone; rather, it likely reflects structural determinants such as reduced access to affordable healthy food, fewer safe spaces for outdoor play, greater exposure to food marketing, and constrained parental time and resources for meal preparation and supervised activity⁵. Importantly, the finding that fast-food consumption was a significant risk factor among White children but not among Black or Latino children in at least one large US cohort suggests that risk factor profiles may differ meaningfully across population subgroups, cautioning against a one-size-fits-all approach to prevention and pointing toward the need for culturally tailored interventions.

Protective factors identified in this review, particularly regular family meals, structured household routines, longer breastfeeding duration, and parental education, offer encouraging and comparatively low-cost targets for intervention. The protective association observed for frequent family suppers is of similar magnitude, though opposite in direction, to the risk associated with purchasing lunch at school, suggesting that the social and behavioural context surrounding eating, not only the nutritional content of meals, may meaningfully influence weight outcomes¹³. These findings support a family-systems approach to prevention that goes beyond nutrition education alone.

Several limitations of this synthesis warrant acknowledgement. First, because the review draws on observational studies of heterogeneous design, definitions, and adjustment strategies, formal meta-analytic pooling was not feasible, and the reported odds ratios should be interpreted as indicative rather than as precise pooled estimates. Second, the predominantly cross-sectional design of many included studies limits causal inference; reverse causation cannot be excluded for factors such as sedentary behaviour, which may be both a cause and a consequence of excess weight. Third, publication and selection bias toward statistically significant findings may have inflated the apparent consistency of some associations. Finally, most large cohort studies originate from high-income countries, and risk factor profiles in low- and middle-income settings, where obesity prevalence is now rising fastest¹, may differ in ways not fully captured here. Future research using harmonised, prospective, multi-country cohort designs with consistent obesity definitions would strengthen causal inference and improve cross-population comparability.

CONCLUSION

Childhood obesity is a multifactorial condition arising from the interaction of parental and genetic predisposition, perinatal exposures, household and behavioural patterns, and socioeconomic context. Parental, and particularly maternal, obesity emerged as the strongest single risk factor identified in this review, while excessive screen time, physical inactivity, short sleep duration, low household income, and low parental education each contributed independently and significantly to obesity risk. Conversely, regular family meals, longer breastfeeding duration, outdoor physical activity, and higher parental education were consistently protective. Given this multifactorial aetiology, effective prevention is unlikely to result from single-component interventions; rather, coordinated strategies that simultaneously address family health behaviours, household routines, screen-time and sleep practices, and the broader socioeconomic and food environment offer the most promising path toward reversing current childhood obesity trends. Continued investment in longitudinal, harmonised surveillance data will be essential to track progress and to refine prevention strategies across diverse populations.

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