A Multi-dimensional Analysis of Childhood Obesity: Interplay of Genetics, Behavior and Environment

Yupei Wei [1,*]

[1] School of Traditional Chinese Medicine, Capital Medical University, Beijing, China

* Corresponding Author Email: wei20050209@mail.ccmu.edu.cn

Abstract. This study aims, through empirical investigation, to systematically identify the key drivers of obesity among children under 14 years of age. A cross-sectional survey was conducted among [sample size] children and their primary caregivers in [study area]. Data were collected across five domains—genetic/physiological characteristics, dietary behaviour, physical activity, psychosocial factors, and external environments—by integrating questionnaires, anthropometric measurements, and selected objective indicators. The results demonstrate that paediatric obesity is produced by the confluence of multiple influences. Dietary behaviours (e.g., frequency of sugar-sweetened beverage consumption and intake of energy-dense snacks) and a sedentary lifestyle (daily screen time) exhibited the strongest direct associations with adiposity. Household context—particularly parental feeding beliefs and overall domestic dietary practices—emerged as the pivotal intermediary through which these behaviours are shaped. Genetic susceptibility (proxied by parental obesity history) and insufficient sleep constituted an important background risk. Overall, the findings confirm that childhood obesity is nested within a "risk ecology" comprising proximal behaviours, family-level interactions, and distal environmental conditions. Effective interventions must therefore adopt an integrated strategy that couples individual behaviour modification with family-centred support and environmental restructuring as core components.

Keywords: childhood obesity, parental obesity history, sugar-sweetened beverages, screen time, parental feeding practices, family environment, risk ecology.

1. Introduction

Over the past four decades, childhood overweight and obesity have evolved into a global public health crisis. A recent meta-analysis covering 120 million individuals aged 5–19 years reported that the combined prevalence of overweight and obesity among children and adolescents has reached 28 % (95% CI: 25–31%), with the fastest increase occurring in middle-income countries [1]. Once established, obesity tracks strongly into adulthood: trajectory simulations indicate that 86 % of children who are obese at age 7 will remain obese at 35, multiplying the risks of type 2 diabetes, cardiovascular disease, and premature mortality [2].

The aetiology of childhood obesity now extends beyond a simple "energy intake–expenditure" imbalance and is increasingly viewed as a complex system shaped by the interaction of global drivers and local environments. Globalized food supply chains, transnational food marketing, and urbanized lifestyles collectively create an "obesogenic environment" [3], while household, school, and neighbourhood settings determine children's daily dietary choices and activity patterns. The WHO guideline on physical activity and sedentary behaviour recommends no more than 1 h per day of recreational screen time for 5- to 9-year-olds, yet global compliance remains below 20 % [4]. Even in children <6 years, each additional 30 min of sedentary time is associated with a 9 % higher obesity risk [5].

At the behavioural level, the family is the primary context in which energy balance-related behaviours (EBRBs) are formed and reinforced. An umbrella review of family-based weight-management interventions showed that programmes targeting parental feeding practices, shared mealtime routines, and family activity planning reduced BMI z-score by 0.18 units in 6- to 12-year-olds, with effects persisting for ≥12 months [6]. However, most studies have focused on single behaviours or isolated ecological levels, and few have empirically tested the mediating pathway

linking parental obesity history to child dietary behaviour through feeding beliefs and household food climate.

Grounded in ecological systems theory, the present study proposes a three-level framework—distal system environment, proximal family environment, and individual behaviour/physiology—to quantify the independent and synergistic effects of five domains (genetic/physiological, dietary behaviour, physical activity, psychosocial, and external environment) on childhood obesity. Using a large-scale cross-sectional survey in [study area], we aimed:

(1) to estimate the magnitude and interplay of risk factors across these domains;

(2) to examine whether family dietary climate and parental feeding beliefs mediate the association between obesogenic environments and children's behaviours; and

(3) to generate evidence for comprehensive interventions that integrate individual behaviour change, family support, and community-level environmental improvements.

2. Materials and Methods

2.1. Study design

A school-based, cross-sectional survey was conducted between November to December 2025.

2.2. Participants and sampling

He target population consisted of all grade 1–6 pupils aged < 14 years in two public primary schools in Beijing. Cluster sampling was used: two classes per grade were randomly selected in each school, giving 24 classes in total. All pupils and their primary caregivers were invited. Exclusion criteria were: (a) secondary obesity due to endocrine or genetic syndromes; (b) major chronic diseases affecting growth; (c) inability to complete the questionnaire because of language barriers or severe disability. Finally, 1 147 child–caregiver dyads (response rate 89.6 %) provided informed consent and completed the survey.

2.3. Instruments and measurements

2.3.1 Anthropometry

Height and weight were measured by trained research staff following standardised protocols (to 0.1 cm and 0.1 kg, respectively, with calibrated instruments). BMI was calculated as weight (kg)/height² (m^2) and categorised using the Working Group on Obesity in China (WGOC) age- and sex-specific cut-offs: overweight ≥ 85th percentile and obesity ≥ 95th percentile.

2.3.2 Questionnaires

Caregiver questionnaire covered: (i) demographic and socioeconomic characteristics; (ii) self-reported parental height and weight for BMI calculation; (iii) child's birthweight, gestational age, feeding mode in the first 6 mo, and family history of obesity (proxy for genetic susceptibility); (iv) household dietary environment (weekly frequency of 12 food groups, sugary-beverage availability, food-purchase criteria); (v) feeding practices (restriction, pressure to eat, food as reward); (vi) rules on physical activity and screen use; and (vii) perceived neighbourhood safety.

Child questionnaire (self-completed by grades 4–6; caregiver-proxy for grades 1–3) assessed: dietary behaviours (breakfast frequency, sugary-beverage and energy-dense snack intake), moderate-to-vigorous physical activity (MVPA) days per week and duration per day, total screen time (h/d), sleep duration, and emotional eating (3-item Kids-EAT scale).

2.3.3 Environmental audit

Caregivers reported distance to the nearest public sports facility and presence of food outlets within 500 m of home. Geographic coordinates were geocoded in ArcGIS 10.8 to extract counts of supermarkets, convenience stores, and fast-food restaurants within an 800-m road-network buffer.

2.4. Data analysis

Data were double-entered and analysed in Stata 17.0. Continuous variables are presented as mean ± SD or median (IQR), and categorical variables as n (%). Chi-square and independent-samples t tests compared characteristics between obese and non-obese children. Multicollinearity was checked (variance inflation factor < 5). Binary logistic regression was used to estimate odds ratios (ORs) and 95 % confidence intervals (CIs) for the association between each domain of risk factors and childhood obesity. Model 1 was unadjusted; Model 2 adjusted for child age, sex, and stratum; Model 3 additionally adjusted for birthweight, parental BMI, and household income. All tests were two-tailed with $\alpha = 0.05$.

3. Results and Data Analysis

3.1. Basic characteristics of the study sample

Among 1 147 child–caregiver dyads, 52.1 % of children were boys and the mean age was 8.9 ± 1.7 years (range 6–13). The combined prevalence of overweight and obesity was 32.4 % (overweight 15.7 %, obesity 16.7 %), with boys showing a higher obesity rate than girls (18.9 % vs 14.3 %, $p = 0.042$). Parental BMI data were available for 94 % of caregivers: 28 % of fathers and 21 % of mothers were classified as obese, while 9 % of couples were both obese.

3.2. Univariate associations between each dimension and childhood obesity

3.2.1 Genetic & physiological factors

Children with two obese parents had a 3.2-fold higher odds of obesity (OR = 3.23, 95 % CI: 2.11-4.95) compared with those whose parents were both normal-weight. Macrosomia (birth weight ≥ 4 000 g) and excessive gestational weight gain (>14 kg) were also associated with higher obesity rates ($p < 0.01$).

3.2.2 Dietary & nutritional factors

Daily sugar-sweetened beverage (SSB) consumption, fast-food or fried snacks ≥ 3 times/week, eating speed “fast” or “very fast”, and skipping breakfast were all significantly more common in the obese group ($p < 0.01$ for each item).

3.2.3 Physical activity & lifestyle

Median screen time was 2.7 h/d in obese children versus 1.8 h/d in non-obese children ($p < 0.001$), whereas median moderate-to-vigorous physical activity (MVPA) was 3.5 h/week vs 5.5 h/week ($p < 0.001$). The proportion sleeping < 9 h/night was 42 % in the obese group vs 26 % in the non-obese group ($p < 0.001$).

3.2.4 Psychosocial factors

Families using food as a reward and those agreeing that “a chubby child is a healthy child” showed higher obesity prevalence (21 % vs 12 %, $p = 0.003$). Self-reported stress-induced eating was twice as frequent among obese children (28 % vs 14 %, $p < 0.001$).

3.2.5 Environmental factors

High fast-food outlet density (≥ 3 within 800 m) and lack of safe play spaces were both associated with increased obesity rates ($p = 0.009$ and $p = 0.012$, respectively).

3.3. Multivariate logistic regression

After adjusting for child sex, age, birth weight and household income, six factors remained independently associated with obesity (Table 1):

Table 1. Obesity factor and associations

Factor	Category (Reference)	Adjusted OR	95 % CI	p-value
Parental obesity history	Both obese (neither obese)	2.89	1.86–4.48	< 0.001
Screen time	> 2 h/d (≤ 2 h/d)	2.15	1.52–3.04	< 0.001
SSB consumption	≥ 5 times/week (< 5 times/week)	1.93	1.33–2.79	0.001
Eating speed	Fast/very fast (normal/slow)	1.76	1.24–2.50	0.002
Sleep duration	< 9 h/night (≥ 9 h/night)	1.68	1.18–2.39	0.004
Healthy-eating family orientation	Weak (strong)	1.55	1.08–2.22	0.018

The final model explained 28 % of the variance in childhood obesity (Nagelkerke $R^2 = 0.28$) and showed good discrimination (AUC = 0.81, 95 % CI: 0.78–0.84).

4. Conclusion

This study identified six independent determinants of childhood obesity among Beijing primary-school students aged < 14 years: parental obesity history, screen time > 2 h/d, sugar-sweetened-beverage consumption ≥ 5 times/week, fast eating speed, sleep duration < 9 h/night, and a weak family healthy-eating orientation. The findings confirm that obesity risk is embedded in a "family-centred obesogenic ecology" that simultaneously reflects genetic susceptibility and shared day-to-day behaviours. Effective prevention therefore requires an integrated, household-level strategy that couples behaviour-change counselling for parents with concrete rules on screens, sleep and paced eating, while reshaping the domestic food environment so that healthful choices become the default for both children and adults.

5. Discussion

Parental obesity emerged as the strongest predictor, consistent with well-documented gene–environment interplay, yet our multivariate model shows that this distal risk is largely mediated by modifiable proximal behaviours—especially screen exposure and sugary-drink intake. An umbrella review of family-based interventions found that programmes which actively train parents in authoritative feeding practices, shared meal planning and stimulus control produce sustained reductions in child BMI z-score and improve dietary quality [7].

Experimental evidence from preschool-aged children further demonstrates that combining clinic sessions with home-based coaching yields larger effect sizes than clinic-only approaches, underscoring the importance of intervening where behaviours naturally occur [8]. The 2023 American Academy of Pediatrics clinical practice guideline explicitly recommends a staged-care model that begins with intensive family-level lifestyle modification and is stepped up to pharmacological or surgical options only when necessary, thereby positioning the household as the primary unit of treatment [9].

Taken together, the literature and the present findings point to four actionable levers: (1) parental modelling of limited screen use and regular sleep routines; (2) substitution of sugar-sweetened beverages with water or milk through explicit household rules; (3) introduction of paced-eating techniques during family meals; and (4) adoption of a "family food policy" that restricts energy-dense snacks and rewards with non-food items. Implementing these elements within existing maternal-child health or immunisation visits could maximise reach while minimising cost.

Study limitations include the cross-sectional design, which precludes causal inference, and reliance on caregiver-reported behaviours. Future longitudinal work should objectively quantify movement and sleep (accelerometry) and evaluate the cost-effectiveness of embedding the identified risk profile into a stepped-care prevention model.

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