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Cross-Sectional Association of Parental Obesity History with BMI-for-Age and Biochemical Risk Markers in School-Going Children

Homeshwar Mandawliya* **Meenu Sharma****

*Food and Nutrition, Mansarovar Global University, Sehore (M.P.) INDIA
** Food and Nutrition, Mansarovar Global University, Sehore (M.P.) INDIA

Abstract: Family history is widely regarded as a determinant of childhood obesity, but its association with routinely available biochemical markers in a general school population is rarely tested. A cross-sectional study was conducted on 950 school-going children aged 10–18 years; 915 (838 girls, 77 boys) were retained after excluding 35 records with biologically implausible weight-for-height combinations. Height, weight, BMI, random blood sugar (RBS) and haemoglobin (Hb) were recorded, and BMI-for-age z-scores computed against the WHO 2007 reference. Independent t-tests, chi-square tests, Pearson correlation and multiple regression were used (α=0.05). Overall, 30.4% of children had a family history of overweight/obesity. WHO BMI-for-age categories showed a double-burden pattern (Normal 68.5%, Thinness 14.5%, severe thinness 9.1%, Overweight 5.4%, Obese 2.5%). Family history was not significantly associated with overweight/obesity prevalence, BMI-for-age z-score, or Hb (all p>0.39), but showed a consistent, borderline association with higher RBS (99.6 vs 97.4 mg/dL; adjusted β=2.17, p=0.094). BMI-for-age z-score correlated significantly with RBS (r=0.109, p=0.001) but not Hb; sex was the strongest independent predictor of both. Family history does not significantly predict anthropometric or haemoglobin status but is associated with a near-significant glycaemic signal meriting confirmation in a larger sample.
Keywords: Family history of obesity; BMI-for-age z-score; random blood sugar; haemoglobin; anthropometric status; school-going children.

Introduction - Childhood obesity is a growing public-health concern, with WHO estimating that overweight/obesity prevalence among 5-19-year-olds worldwide rose from about 4% in 1975 to over 18% in 2022 [1,2]. In urban India, childhood overweight/obesity prevalence is estimated at 8-12% and continues to rise with urbanisation [3]. Family history is considered an important, if under-investigated, risk pathway: a classic prospective study showed that parental obesity roughly doubles a child's risk of adult obesity [4], and children born into families with obesity may carry both genetic predisposition and a shared obesogenic environment [5] that can manifest as early metabolic change - including sub-clinical insulin resistance - before overweight itself becomes clinically apparent [6]. In this study, anthropometric measurements (height, weight, BMI) and two routinely obtainable biochemical parameters - random blood sugar and haemoglobin - were used to examine whether family history of obesity is associated with early anthropometric or biochemical risk in a general school-going population, complementing an earlier conference report from the same research programme on family history and anthropometric status.

Review of Literature:

In this section, a critical review of the existing studies has been done to identify their studies, findings, and research gap. Table 1 shows the summarized table of key studies on family history with relevant analysis.

Table 1 (see in last page)

Table 1 shows, no identified study jointly examines family history alongside anthropometric status (BMI-for-age z-score) and both available biochemical markers (RBS and Hb) within a single general school-going sample the specific gap this study addresses.

Research Gap: As the literature summarized above shows, family history has been repeatedly linked to childhood obesity risk [4-6], and both the WHO BMI-for-age reference [7] and blood glucose/haemoglobin [11-13] are well-validated, low-cost screening tools individually. However, no identified Indian study jointly examines family history alongside anthropometric status (BMI-for-age z-score) and both available biochemical markers (RBS and Hb) within a single, general (not purposively overweight/obese-selected) school-going sample. Existing evidence, including the classic Whitaker et al. cohort [4], is also skewed toward purposively sampled or already-overweight cohorts, leaving the true, population-level strength of the family history – anthropometric – biochemical relationship largely untested.

Methodology: In this section, the methodology has been done that evaluate the dataset step by step:

Subjects: Initial dataset had 950 school-aged children aged 10-18 years old. After data screening, 35 entries (3.7%) exhibited body mass index-for-age (z-score) beyond ±4 SD of the WHO 2007 median, and were likely due to errors during data entry, and were dropped from analyses. Final data set had 915 subjects (838 females, 77 males; mean age 15.4 ± 1.6 years), of which 278 (30.4%) had family history of obesity (Table 2).

Data Reliability: Body height, body weight and biochemical values (random blood sugar [RBS], hemoglobin [Hb]) were measured using standard procedures. Each data entry was internally consistent before analyses, where anthropometric outliers were dropped as noted above, and RBS/Hb were also verified to be within physiologically possible range.

Criterion Measures: BMI was calculated by the use of height (cm) and weight (kg) (kg/m²); BMI z-scores with respect to age were calculated against the WHO 2007 growth standards for ages 5 to 19 years old [7], and then categorized as: Severe thinness ($z \leq -3$), Thinness ($-3 < z \leq -2$), Normal ($-2 < z \leq 1$), Overweight ($1 < z \leq 2$) or Obese ($z > 2$). Blood sugar (random) (mg/dL) and Haemoglobin (g/dL) were the only biochemical tests conducted; anaemia was defined as Hb<12 g/dL using the Indian criteria [12]. Family history of obesity (Yes/No), parental health conditions, dietary habit (vegetarian/non-vegetarian), and physical activity (Low/Medium/High) were recorded as covariates.

Experimental Design: This study utilized cross-sectional and descriptive-correlational study design. Independent samples t-test (Cohen's d) was employed to compare the BMI-for-age z-score, RBS, and Hb among family history categories; chi-square test for categorical was performed; Pearson correlation analysis to correlate BMI-for-age z-score to RBS and Hb; Multiple Linear Regression analysis with BMI-for-age z-score, RBS, and Hb as dependent variables, and age, gender, dietary habits, physical activity, family history, and parental morbidity as independent variables. Results with p<0.05 were significant, while those with p<0.10 were considered as a trend.

Table 2: Sample Characteristics (N=915)

Characteristic	Value
Age, years, mean ± SD	15.4 ± 1.6 (range 10–18)
Sex	838 Female (91.6%), 77 Male (8.4%)
Height, cm, mean ± SD	155.9 ± 6.7
Weight, kg, mean ± SD	45.0 ± 9.6
BMI, kg/m², mean ± SD	18.5 ± 3.7
BMI-for-age z-score,mean±SD	-1.06 ± 1.38
Random blood sugar (RBS), mg/dL, mean ± SD	98.1 ± 17.6
Haemoglobin (Hb), g/dL, mean ± SD	11.9 ± 1.54
Family history of overweight/obesity, n (%)	278 (30.4%)

Table 3: WHO BMI-for-age Category Distribution (N=915)

Category	n	%
Severe thinness	83	9.1
Thinness	133	14.5
Normal	627	68.5
Overweight	49	5.4
Obese	23	2.5

Table 4 (see in last page)

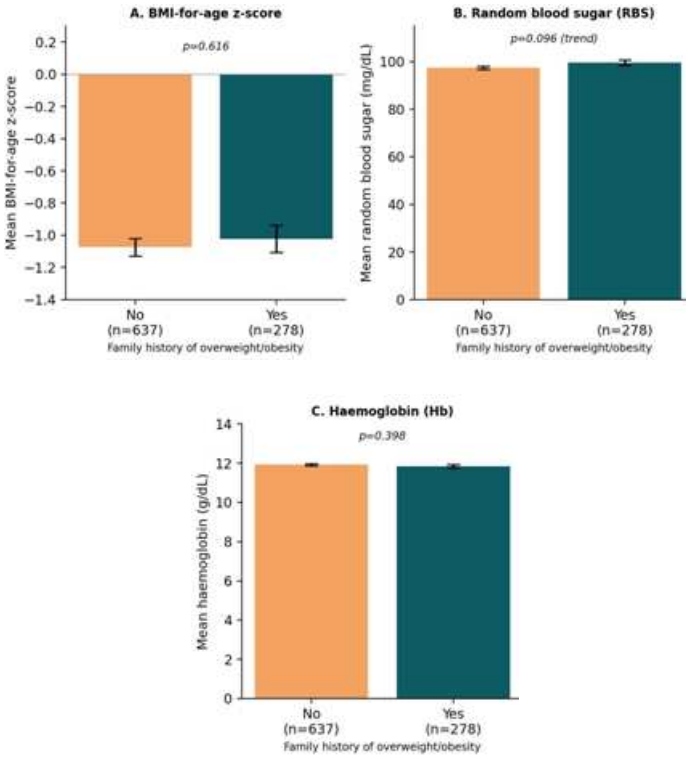

Figure 1: BMI-for-age z-score (A), random blood sugar (B) and haemoglobin (C) by family history of overweight/obesity. Error bars = SEM.

Table 5 (see in last page)

Results and Discussion: From 950 screened records, 35 records were rejected because of improbable weight-for-height ratios, which led to the inclusion of 915 children (278 with a family history, 30.4%). The categories based on WHO BMI-for-age showed the double burden phenomenon, that is, 68.5% Normal, 14.5% Thinness, 9.1% Severe thinness, 5.4% Overweight, and 2.5% Obese (Table 3).

Overweight/obesity family history was not found to have a significant relation with overweight/obesity prevalence, BMI-for-age z-score, and hemoglobin (all p>0.39; Table 4, Figure 1), contrary to the strong purposive sampling-based association observed by Whitaker et al. [4]. On the other hand, there were some positive, weak associations between overweight/obesity family history and increased RBS levels in three analyses: the bivariate comparison (99.6 vs. 97.4 mg/dL, p=0.096), the elevated-RBS prevalence (46.8% vs. 41.3%, p=0.143), and the regression analysis (β=2.17, p=0.094). This is similar to studies showing early subclinical

insulin resistance among Asian Indian children who had a family history of obesity [6].

BMI-for-age z-score was significantly correlated with RBS (r = 0.109, p = 0.001) but not with haemoglobin (r = 0.048, p = 0.150) – meaning that anaemia in this group is influenced by some factors other than adiposity, similar to national studies that demonstrated anaemia in Indian schoolchildren to be an independent problem from obesity [11,13].

Gender was the major independent and consistent predictive factor in all three regression models: boys had significantly higher BMI-for-age z-score, RBS (+11.2 mg/dL), and haemoglobin (+1.70 g/dL) compared with girls ($p<0.001$ for all; Table 5), while girls presented with a higher prevalence of anaemia that is observed nationally [11, 12]. Age and level of physical activity were also found to be independent predictors of BMI-for-age z-score (p=0.017 and p=0.019, respectively), which corresponds with WHO recommendations on physical activity being protective from adiposity [14]; diet and parents' morbidity were not predictive.

Overall, there is no evidence for family history of obesity as a significant predictor of anthropometric or anaemia status in this general population sample; however, the association between family history of obesity and significant increase in blood glucose concentration (near-significant) looks reasonable [4-6].

Conclusion and Future Scope:

1. Among this particular population of students attending schools (N=915), there was no statistically significant relationship between the presence of family history of obesity and the prevalence of obesity, BMI-for-age Z score, and haemoglobin levels.
2. Family history of the condition was statistically significantly related to the presence of high random blood sugar levels (p=0.09-0.14 for three different tests), indicating early signs of glycaemia risk.
3. The z-scores of BMI-for-age were significantly associated with RBS (r = 0.109; p = 0.001) but not with Hb levels, suggesting that the anthropometric state is a good predictor of glycaemic but not haematological risk.
4. Sex proved to be the most consistent predictor of BMI-for-age z-score, RBS and Hb levels, preceding family history, dietary habits, physical activity, and parent morbidity status; hence, the screening procedure has to be stratified based on sex.
5. Further research needs to broaden the range of biochemical parameters (fasting glucose level, lipid profile) to better define the metabolic risk pathway associated with family history [6].

References:-

1. World Health Organization. Obesity and overweight fact sheet. Geneva: WHO; 2024.
2. Zhang X, et al. Global prevalence of overweight and obesity in children and adolescents: a systematic review and meta-analysis. JAMA Pediatr. 2024;178(8): 800-813.
3. Singh S, et al. Childhood obesity in India: a two-decade meta-analysis of prevalence and socioeconomic correlates. ClinEpidemiol Glob Health. 2023;23: 101390.
4. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med. 1997;337(13):869-873.
5. Jebeile H, et al. Obesity in children and adolescents: epidemiology, causes, assessment, and management. Lancet Diabetes Endocrinol. 2022;10(5):351-365.
6. Bhardwaj S, Misra A, Khurana L, Gulati S, Shah P, Vikram NK. Childhood obesity in Asian Indians: a burgeoning cause of insulin resistance, diabetes and sub-clinical inflammation. Asia Pac J ClinNutr. 2008;17(Suppl 1):172-175.
7. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ. 2007;85(9):660-667.
8. Popkin BM. The nutrition transition and obesity in the developing world. J Nutr. 2001;131(3):871S-873S.
9. Black RE, Victora CG, Walker SP, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. Lancet. 2013;382(9890):427-451.
10. Lobstein T, Jackson-Leach R, Moodie ML, et al. Child and adolescent obesity: part of a bigger picture. Lancet. 2015;385(9986):2510-2520.
11. International Institute for Population Sciences (IIPS) and ICF. National Family Health Survey (NFHS-5), 2019-21: India Report. Mumbai: IIPS; 2021.
12. Sachdev HPS, Porwal A, Acharya R, et al. Haemoglobin thresholds to define anaemia in a national sample of healthy children and adolescents in India: a population-based study. Lancet Glob Health. 2021;9(6):e822-e831.
13. Kapil U, Kapil R, Gupta A. Nutritional anaemia in children and adolescents in India: a persistent public health problem. Indian J Pediatr. 2019;86(3):265-266.

Table 1: Summary of Key Studies on Family History, Anthropometric Status and Biochemical Risk in Children

Author(s) & Year	Study Type	Focus of Study	Key Findings	Research Gap
Whitaker RC, et al., 1997 [4]	Prospective cohort; USA	Parental obesity as predictor of adult obesity	Parental obesity ~doubled adult obesity risk	Purposive cohort; untested in general population
Zhang X, et al., 2024 [2]	Meta-analysis; global	Global overweight/obesity prevalence	Prevalence rose sharply over four decades	Limited disaggregation by family-history status
Singh S, et al., 2023 [3]	Meta-analysis; Indian children	Prevalence & correlates in India	8-12% urban prevalence, rising with urbanisation	Few studies combine anthropometric & biochemical risk
Jebeile H, et al., 2022 [5]	Narrative review	Epidemiology & management of paediatric obesity	Family history & shared environment are key pathways	Biochemical correlates rarely assessed directly
Bhardwaj S, et al., 2008 [6]	Narrative review; Asian Indian	Obesity and insulin resistance	Early sub-clinical insulin resistance in Asian Indian children	Glycaemic risk rarely quantified with a simple marker
de Onis M, et al., 2007 [7]	Methodological; 5-19 years	WHO growth reference development	Established the BMI-for-age reference	Does not identify risk factors driving deviation
Popkin BM, 2001; Black RE, et al., 2013; Lobstein T, et al., 2015 [8,9,10]	Lancet reviews; global, LMICs	Double burden of malnutrition	Under- and over-nutrition coexist, especially in India	Rarely applied jointly to anthropometric & biochemical data
IIPS and ICF (NFHS-5), 2021 [11]	National survey; India	Nutrition &anaemia indicators	Anaemia highly prevalent in Indian children/girls	Limited link with family history or anthropometric status
Sachdev HPS, et al., 2021 [12]	Population study; India	Hb thresholds &anaemia definition	Confirmed Hb<12 g/dL; sex differences in anaemia	Anaemia studied independent of obesity/glycaemic markers
Kapil U, et al., 2019 [13]	Review; India	Nutritional anaemia burden	Anaemia& thinness persist despite rising obesity	Little integration of anaemia, glycaemic& anthropometric data

Table 4: Anthropometric and Biochemical Status by Family History

Outcome	Family history: No (n=637)	Family history: Yes (n=278)	Statistic	p-value
Overweight/obese, %	8.0%	7.6%	X^2=0.01	0.920
BMI-for-age z-score, mean	-1.07	-1.02	t=0.50, d=0.04	0.616
RBS, mg/dL, mean	97.4	99.6	t=1.67, d=0.12	0.096
Elevated RBS (≥100 mg/dL), %	41.3%	46.8%	X^2=2.15	0.143
Haemoglobin, g/dL, mean	11.93	11.83	t=-0.85, d=-0.06	0.398

Table 5: Multiple Regression — Predictors of BMI-for-age z-score, RBS and Hb

Predictor	BMI-for-age z (β, p)	RBS (β, p)	Hb (β, p)
Age	0.068, p=0.017	-0.34, p=0.341	0.002, p=0.954
Sex (Male)	0.569, p=0.001	11.17, p<0.001	1.70, p<0.001
Diet (Vegetarian)	-0.014, p=0.887	0.66, p=0.584	0.11, p=0.283
Physical activity	-0.221, p=0.019	0.03, p=0.980	—
Family history (Yes)	0.039, p=0.700	2.17, p=0.094	-0.12, p=0.264
Parental morbidity (Yes)	0.116, p=0.292	-1.16, p=0.405	0.02, p=0.837
Model fit	F(6,908)=4.21, p<0.001, R^2=0.027	F(6,908)=5.52, p<0.001, R^2=0.035	F(5,909)=19.28, p<0.001, R^2=0.096

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