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Original Research Article

The association between obesity and hormonal imbalances in children: a prospective study

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ABSTRACT

Background: Hormonal alterations, may complicate the metabolic consequences linked to childhood obesity. To study the relation between pediatric obesity and hormonal abnormalities, along with the correlation between body mass index (BMI) and other metabolic and endocrine markers.
Methods: A prospective observational study including 325 children aged 6 to 14 years was conducted at a tertiary care teaching hospital in north India over 30 months (January 2024 to July 2026). Individuals were classified into three categories based on their BMI percentiles for age and gender as normal weight, overweight, and obese. In addition to blood pressure and anthropometric data, fasting biochemical markers such as cortisol, free thyroxine (free T4), glucose, insulin, and thyroid-stimulating hormone (TSH) were measured. The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated to determine insulin resistance. One-way analysis of variance and multivariate linear regression with age and gender adjustments were used for statistical analysis.
Results: Obese children demonstrated markedly elevated levels of cortisol, TSH, HOMA-IR, and fasting insulin compared to their normal-weight counterparts, along with reduced levels of free T4 ($p<0.05$). A multivariate regression analysis, after controlling for age and gender, indicated that BMI was significantly associated with indicators of insulin resistance and thyroid hormone levels.
Conclusions: Childhood obesity is associated with significant hormonal and metabolic alterations, suggesting early endocrine adaptations that may have metabolic implications. While these changes (particularly thyroid-related shifts) represent reversible, adaptive responses rather than primary pathology, findings highlight need for early identification and intervention to prevent potential long-term cardiometabolic risks associated with pediatric-onset obesity.

Keywords: Childhood obesity, Hormonal dysregulation, Insulin resistance, Thyroid function, Hyperthyrotropinemia, Prospective study

INTRODUCTION

Childhood obesity represents a consequential epidemiological transition with profound global health implications. Global childhood obesity prevalence has increased dramatically since 1990.[1] This phenomenon is particularly pronounced in low-and middle-income countries undergoing rapid socioeconomic transformation, where nutrition transition has accelerated dramatically. South Asia, harboring nearly one-quarter of the global pediatric population, has experienced a dramatic surge, with recent meta-analyses in India indicating pooled prevalence estimates of 8.4% for obesity and 12.4% for overweight.[2] This epidemiological shift is compounded by persistent undernutrition in marginalized communities coexisting with burgeoning overnutrition in urban populations, creating a syndemic that challenges public health frameworks.[3,4]

The conceptual evolution of obesity from an energy balance model to an endocrine paradigm has transformed our understanding of its pathophysiology. Adipose tissue is now recognized as a dynamic endocrine organ secreting adipokines-leptin, adiponectin, and resistin-that regulate appetite, insulin sensitivity, and metabolism.[5] In obesity, this network undergoes dysregulation characterized by leptin resistance, suppressed adiponectin, and pro-inflammatory adipokines establishing low-grade systemic inflammation.[6] This imbalance initiates endothelial activation, macrophage infiltration, and cytokine elaboration-including tumor necrosis factor-α and interleukin-6-promoting insulin resistance. The recognition that these perturbations originate in childhood has catalysed a paradigm shift toward early-life prevention.

Insulin resistance constitutes the mechanistic fulcrum linking childhood obesity to type 2 diabetes mellitus, dyslipidemia, and hypertension.[7] Accumulation of intramyocellular and intrahepatic lipids generates bioactive metabolites-diacylglycerols and ceramides-that activate serine/threonine kinase cascades, notably protein kinase C isoforms, impairing insulin receptor signaling.[8] Hypertrophied adipocytes recruit pro-inflammatory macrophages elaborating tumor necrosis factor-α and interleukin-6, further impairing insulin action. This convergent lipotoxic and inflammatory insult creates a self-amplifying cycle. Pediatric studies consistently document elevated fasting insulin and HOMA-IR among overweight and obese children. In South Asian populations, vulnerability is amplified by greater adiposity at lower BMI thresholds, potentially reflecting thrifty genotype adaptations.[9] High vitamin D deficiency prevalence, impairing pancreatic β-cell function, may synergize with adiposity to accelerate metabolic deterioration.[10]

Thyroid function exhibits characteristic alterations in childhood obesity, though clinical interpretation demands distinction between adaptive responses and true pathology. Elevated TSH with normal or modestly reduced free T4 in obese children superficially resembles subclinical hypothyroidism yet diverges fundamentally. Leptin stimulates thyrotropin-releasing hormone synthesis, augmenting TSH secretion.[11] Crucially, these alterations are reversible with weight reduction, normalizing as adiposity declines.[12] This reversibility has led to consensus characterization as "hyper thyrotropinemia of obesity"-a benign compensatory response rather than primary dysfunction.[13] Unnecessary thyroid hormone replacement exposes children to adverse effects and diverts attention from weight management. Guidelines recommend reserving hormone therapy exclusively for cases with clear primary pathology-persistently elevated TSH after weight loss, low free T4 below reference range, or positive autoantibodies.[14]

The hypothalamic-pituitary-adrenal axis represents a third pathway through which obesity may influence metabolic health. Chronic HPA axis activation—driven by stress, sleep disruption, or metabolic burden-results in sustained cortisol excess promoting visceral adiposity through local glucocorticoid amplification by 11β-hydroxysteroid dehydrogenase type 1, converting inactive cortisone to active cortisol in adipose tissue, creating a feed-forward loop enhancing lipolysis, promoting adipocyte differentiation, and inducing insulin resistance through impaired glucose uptake and augmented gluconeogenesis.[15] Hair cortisol measurements reveal subtle alterations in obese children linked to socioeconomic disadvantage and chronic stress, suggesting bidirectional association. HPA axis modulation through behavioural interventions-stress reduction, sleep optimization, and physical activity-offers supplementary strategies for comprehensive management.

Interpretation of endocrine parameters in pediatric populations is constrained by pubertal development, which can confound obesity-related alterations. Puberty induces physiological insulin resistance via growth hormone-insulin-like growth factor-1 axis, peaking during Tanner stage III, confounding assessment in peripubertal children aged 10-14 years.[16] Rising gonadal steroids influence thyroid hormone-binding globulin, complicating total thyroid hormone interpretation.[17] Vitamin D deficiency, prevalent in South Asian children due to pigmentation, limited sun exposure, and pollution, has been independently linked to insulin resistance, potentially acting as confounder.[18] Failure to account for these factors may lead to erroneous conclusions regarding independent adiposity contribution. The literature is characterized by limited integration of multiple endocrine axes, scarcity of South Asian data, inconsistent confounder assessment, and predominance of cross-sectional designs precluding causal inference. This prospective investigation addresses these gaps by examining associations between childhood obesity and multiple endocrine parameters-fasting insulin, HOMA-IR, TSH, free T4, and cortisol-within a cohort of South Asian children aged 6-14 years. By providing integrated data across three endocrine axes, this research generates testable hypotheses, informs population-specific screening, and provides methodological foundation for future longitudinal investigations. This study reinforces that childhood obesity represents a complex endocrine condition requiring comprehensive multidisciplinary approaches addressing metabolic, hormonal, and psychosocial dimensions.

METHODS

A prospective observational study was performed to examine the association between hormonal alterations and childhood obesity. The study was conducted at a tertiary care teaching hospital in north India over 30 months (January 2024 to July 2026). The study complied with the declaration of Helsinki (6th revision, 2008) and received institutional ethics committee approval (IEC

Code 23/25). Written informed consent was obtained from parents or legal guardians, and assent was obtained from children where appropriate.

Children aged 6-14 years attending pediatric outpatient services or recruited through school health screening programs were considered eligible for inclusion. Participants were enrolled after obtaining written informed consent from parents or legal guardians and assent from children where appropriate. The study included children aged between 6 and 14 years, clinically healthy children without any known chronic systemic illness, and willing to participate with informed parental consent. Children with diagnosed endocrine disorders (e.g., type 1 diabetes mellitus, hypothyroidism, Cushing's syndrome), history of chronic illness, genetic syndromes, or long-term medication affecting metabolism or hormonal status, acute illness at the time of assessment, or receiving steroid therapy or thyroid hormone supplementation were excluded.

The study enrolled 325 children and adolescents. Participants were categorized into three groups according to BMI percentiles associated with age and gender, in accordance with the World Health Organization's growth reference standard; i.e., normal weight (BMI-for-age between the 5th and 84th percentile), overweight (BMI-for-age between the 85th and 94th percentile), and obese (BMI-for-age ≥95th percentile).[19]

Anthropometric measurements were performed by trained personnel utilizing established procedures. Children donned lightweight clothing and were barefoot, with their body weight assessed to the nearest 0.1 kg utilizing a calibrated digital scale. A wall-mounted stadiometer was used to measure height with an accuracy of 0.1 cm. BMI was calculated using the standard formula [weight (kg) / height (meters)^2]. A non-elastic measuring tape was used to measure the child's waist circumference in an upright position and breathing calmly, positioned midway between the inferior rib margin and the iliac crest. Blood pressure was measured using an appropriately sized cuff and a calibrated digital sphygmomanometer. Measurements were conducted after a minimum of fifteen minutes of relaxation in a seated position. Three readings were obtained at five-minute intervals, with the mean value utilized for analysis.

After an overnight fast of 10-12 hours, venous blood samples were collected between 08:00 and 09:00 hours in the morning to minimize diurnal variation. Serum and plasma were separated and analysed on the same day or stored at -20°C until analysis as per standard protocol. The parameters were estimated using standardized laboratory methods: fasting plasma glucose by enzymatic glucose oxidase-peroxidase method, fasting serum insulin by chemiluminescent immunoassay, TSH and free thyroxine (free T4) by electrochemiluminescence immunoassay, and serum cortisol by chemiluminescent immunoassay. Insulin resistance was calculated using the HOMA-IR formula: [fasting insulin (μIU/ml) × fasting glucose (mg/dl)]/405.[20] Each batch had internal quality control samples run on it, and every biochemical experiment was performed in duplicate. Instruments were calibrated on a regular basis according to manufacturer's instructions to ensure accuracy and precision.

Statistical software was utilized to evaluate data. Descriptive statistics for categorical variables were reported as frequencies and percentages, while continuous variables were described using mean±standard deviation. The Shapiro-Wilk test was used to ascertain whether the data exhibited a normal distribution. One-way ANOVA was used to compare the BMI groups based on continuous factors. Post-hoc analyses were conducted wherever required. The chi-square test was used to analyse categorical variables. Following adjustment for age and gender, multivariate linear regression was used to evaluate the independent association between BMI and hormonal markers. Regression coefficients (β), p values, 95% confidence intervals, and standard errors were shown. P values less than 0.05 were considered statistically significant.

RESULTS

The mean age and sex distribution of children were comparable across normal-weight, overweight, and obese groups, with no statistically significant differences observed ($p>0.05$), indicating adequate group matching (Table 1). In contrast, anthropometric parameters such as BMI and waist circumference showed a progressive and statistically significant increase from normal weight to obese children ($p<0.001$) (Table 1). Consistently, overweight and obese children exhibited markedly elevated systolic and diastolic blood pressure compared to their normal-weight counterparts ($p<0.001$) (Table 1). These findings suggest a clear association between increasing adiposity and elevated cardiometabolic risk indicators even in childhood.

A statistically significant increase in fasting insulin levels and HOMA-IR values was observed with increasing BMI category, indicating progressively worsening insulin resistance among overweight and obese children ($p<0.001$) (Table 2). Thyroid function analysis revealed significantly higher TSH levels and lower free T4 concentrations in obese children compared to normal-weight children, suggesting altered thyroid homeostasis consistent with obesity-related hyper thyrotropinemia (Table 2). Additionally, obese children exhibited significantly elevated mean cortisol levels ($p=0.01$) (Table 2). Overall, these results suggest marked hormonal alterations associated with increasing adiposity in pediatric populations.

BMI was significantly associated with numerous hormonal indicators after adjusting for age and gender (Table 3). Multivariate linear regression analysis demonstrated a significant positive correlation between

BMI and fasting insulin, HOMA-IR, and TSH levels ($p<0.001$) (Table 3). BMI showed a substantial negative correlation with free T4 levels ($p=0.001$) (Table 3). These data suggest that higher BMI is independently associated with adverse hormonal alterations in children, although the absence of adjustment for pubertal status and vitamin D levels-critical confounders in this population-should be considered when interpreting these associations.[21]

Table 1: Baseline characteristics of the study population according to BMI category.

Variables	Normal weight	Overweight	Obese	P value
Age (in years)	10.8±2.1	11.1±2.0	11.3±1.9	0.28
Male: Female	62:58	47:48	56:54	0.94
BMI (kg/m²)	17.6±1.9	21.8±1.6	27.9±3.2	<0.001
Waist circumference (cm)	61.4±5.8	72.2±6.1	86.5±7.9	<0.001
Systolic BP (mmHg)	102.3±7.6	108.9±8.2	116.7±9.4	<0.001
Diastolic BP (mmHg)	64.8±6.3	69.5±6.7	74.6±7.1	<0.001

Table 2: Comparison of hormonal parameters across BMI categories.

Hormonal parameter	Normal weight	Overweight	Obese	P value
Fasting insulin (µIU/ml)	7.4±2.1	11.6±3.4	18.9±5.7	<0.001
HOMA-IR	1.6±0.5	2.8±0.9	4.7±1.6	<0.001
TSH (mIU/l)	2.1±0.7	2.8±0.9	3.6±1.1	<0.001
Free T4 (ng/dl)	1.22±0.18	1.16±0.17	1.09±0.15	0.004
Cortisol (µg/dl)	11.6±3.2	12.4±3.6	14.1±4.1	0.01

Table 3: Association between BMI and hormonal parameters (Multivariate linear regression).

Hormonal parameter	β coefficient	Standard error	95% CI	P value
Fasting insulin	0.48	0.06	0.36-0.60	<0.001
HOMA-IR	0.51	0.07	0.37-0.65	<0.001
TSH	0.29	0.07	0.15-0.43	<0.001
Free T4	-0.21	0.06	-0.33--0.09	0.001

DISCUSSION

The present prospective study demonstrates a clear association between childhood obesity and multiple hormonal alterations, including insulin resistance, altered thyroid function, and elevated cortisol levels. These findings highlight the complex endocrine changes accompanying excess adiposity in children and support the concept that obesity-related metabolic alterations begin early in life.

A progressive increase in fasting insulin and HOMA-IR values with rising BMI observed in our study is consistent with several national and international studies reporting insulin resistance as a hallmark of pediatric obesity. Research from Indian populations has documented significantly higher levels of insulin resistance among overweight and obese children, emphasizing the growing burden of early metabolic risk in South Asian populations.[22] Similar findings have been reported in European and North American cohorts, reinforcing the universality of this association irrespective of ethnicity.[23] Insulin resistance during childhood is clinically important, as it predisposes individuals to future type 2 diabetes mellitus and cardiovascular disease. The mechanistic basis for this association involves the accumulation of intracellular lipids in muscle and liver tissue, leading to impaired insulin signaling through the activation of serine kinases and the release of pro-inflammatory cytokines from adipose tissue.[24]

The association between obesity and insulin resistance in South Asian children may be further amplified by the high prevalence of vitamin D deficiency in this population.[25] Recent multicenter studies, including have highlighted the direct link between vitamin D deficiency and insulin resistance and metabolic syndrome in Indian children.[26,27] Vitamin D plays a crucial role in insulin secretion and sensitivity through its effects on pancreatic β-cell function and the regulation of calcium flux. Its deficiency is associated with increased risk of metabolic abnormalities. In our study, vitamin D status was not assessed, and this represents a significant limitation, as the observed associations between BMI and metabolic markers may be partially confounded by underlying vitamin D deficiency. Future studies should incorporate vitamin D assessment to better characterize the independent contribution of adiposity to insulin resistance in this population.

Our study demonstrated higher TSH levels and slightly lower free T4 concentrations in obese children, findings that agree with multiple pediatric studies suggesting

obesity-related hyperthyrotropinemia.[28] It is important to contextualize these findings within current understanding of obesity-related thyroid changes. Recent literature distinguishes between true subclinical hypothyroidism and benign, reversible hyperthyrotropinemia observed in obesity.[14] Studies indicate that these shifts in obese children often represent a functional adaptation-likely resulting from leptin-mediated stimulation of hypothalamic thyrotropin-releasing hormone-rather than primary thyroid pathology.[29] The clinical significance of these changes has been re-evaluated, with emerging consensus suggesting that the majority of such cases are benign and normalize with weight loss.[28]

In our cohort, elevated TSH levels with only modest reductions in fT4 are consistent with this adaptive pattern rather than overt hypothyroidism.[30] While we observed statistically significant association between BMI and thyroid parameters, characterizing this as "altered thyroid homeostasis" rather than "dysfunction" may more accurately reflect the current understanding in field.[14] It is noteworthy that many studies report no significant thyroid hormone differences after adjusting for confounders such as pubertal status and body composition, further supporting need for cautious interpretation.[31]

The reversibility of these thyroid changes following weight reduction has been well-documented in longitudinal studies.[28] This has important clinical implications: differentiating obesity-related hyperthyrotropinemia from true subclinical hypothyroidism is crucial to avoid unnecessary thyroid hormone supplementation and to focus management on weight reduction strategies. Our study reinforces the importance of this distinction, and we recommend that clinicians interpret elevated TSH in obese children cautiously, reserving thyroid hormone therapy for cases with clear evidence of primary thyroid dysfunction.

Elevated cortisol levels observed among obese children in our study further support the role of hypothalamic-pituitary-adrenal (HPA) axis alterations in pediatric obesity. Cortisol excess promotes central adiposity, insulin resistance, and metabolic syndrome through multiple mechanisms, including the activation of 11β-hydroxysteroid dehydrogenase type 1 in adipose tissue, which amplifies local glucocorticoid action.[32] Recent studies utilizing serum, salivary, and hair cortisol measurements have reported altered cortisol dynamics in obese children, particularly in relation to chronic stress and socioeconomic factors.[33] Although our findings align with this body of evidence, cortisol interpretation is limited by diurnal variability and single-time-point sampling. The HPA axis exhibits a characteristic circadian rhythm, and single measurements may not accurately reflect overall cortisol exposure.

In multivariate regression models, BMI emerged as a significant predictor of insulin resistance indices, TSH, and free T4 after controlling for age and sex. This supports the hypothesis that adiposity per se exerts direct endocrine effects independent of demographic factors. Similar associations between BMI and hormonal parameters have been reported in longitudinal and cross-sectional pediatric studies, reinforcing the role of excess adipose tissue in endocrine alterations.[34] However, the claim that BMI is an "independent" predictor should be interpreted with caution given the absence of adjustment for vitamin D deficiency and pubertal staging-both critical, recognized confounders for metabolic markers in this population.[26,27,35] The observed endocrine changes-ranging from hyperinsulinemia to HPA axis activation-underscore the premorbid status of these patients, who often remain asymptomatic despite underlying metabolic shifts.[36,37] Nonetheless, the complexity of these interactions necessitates further investigation with more comprehensive covariate adjustment.

Limitations

Despite its contributions, this study has several important limitations. First, pubertal staging (Tanner staging) was not assessed, which represents a significant methodological gap. Children aged 6-14 years span a wide range of pubertal development, and puberty itself induces physiological insulin resistance and significant hormonal shifts through the activation of the growth hormone-insulin-like growth factor-1 axis.[35] Without Tanner staging, it is difficult to fully isolate the effect of BMI from pubertal hormonal changes, particularly in the 10-to-14-year age group where pubertal changes are most pronounced. This omission may confound the interpretation of the association between BMI and the observed hormonal markers, as both insulin sensitivity and thyroid hormone levels are significantly influenced by pubertal status. Second, hormonal measurements were based on single fasting samples, particularly limiting cortisol interpretation due to well-documented circadian variation. The HPA axis follows a distinct diurnal rhythm, and a single morning sample may not capture the full spectrum of cortisol dynamics or the blunted diurnal variation often observed in obesity.[38] Third, vitamin D status was not evaluated, which is a notable limitation given the high prevalence of vitamin D deficiency in South Asian populations and its established role as a confounder for insulin resistance and metabolic syndrome.[26,27] Vitamin D deficiency has been independently associated with insulin resistance, and failure to account for this may overestimate the true contribution of adiposity to metabolic alterations. Fourth, inflammatory markers and detailed body composition analysis were not evaluated, which could have provided further mechanistic insights into the observed associations. Markers such as high-sensitivity C-reactive protein, interleukin-6, and tumor necrosis factor-α, as well as measures of fat distribution (e.g., visceral adiposity index), would have enhanced the understanding of the inflammatory pathways linking obesity to hormonal changes. Fifth, the single-center design limits

generalizability to other populations with different demographic and ethnic characteristics. Sixth, the cross-sectional design precludes causal inference and assessment of temporal relationships between obesity and endocrine alterations. Seventh, the adipokine profile, including leptin and adiponectin levels, was not assessed, which would have provided additional insight into the endocrine function of adipose tissue. These limitations should be considered when interpreting the findings of this study and highlight areas for improvement in future research.

CONCLUSION

This prospective study demonstrates that childhood obesity is associated with significant hormonal and metabolic alterations, including elevated insulin resistance, higher TSH levels with reduced free T4 concentrations, and increased cortisol levels, providing integrated evidence across multiple endocrine axes within a South Asian pediatric cohort-a population historically underrepresented in this research domain. The thyroid changes observed, characterized by elevated TSH with only modest reductions in free T4, likely represent a benign, adaptive hyper thyrotropinemia rather than primary thyroid pathology, reinforcing the emerging consensus that these alterations are reversible with weight management and should not prompt unnecessary thyroid hormone replacement. These findings advance current understanding by establishing that obesity-related endocrine perturbations manifest early in the disease continuum, often in asymptomatic children, underscoring the premorbid status of this population and the critical importance of early identification and intervention to prevent long-term cardiometabolic consequences. However, the absence of adjustment for pubertal status and vitamin D levels-critical confounders in this population-limits the strength of the independent association between BMI and hormonal markers, highlighting the need for future research incorporating comprehensive confounder assessment, pubertal staging, and longitudinal designs to provide more robust evidence for clinical practice. Establishing population-specific reference ranges for insulin resistance and hormonal parameters in Indian children may further improve early screening and intervention strategies, ultimately contributing to the development of targeted preventive approaches for this high-risk population.

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