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OBESITY IN INFANTS AND CHILDREN BELOW 5 YEARS: A COMPREHENSIVE REVIEW OF EPIDEMIOLOGY, RISK FACTORS, CLINICAL MANIFESTATIONS, METABOLIC ABNORMALITIES, HORMONAL DYSREGULATION, AND GENETIC ETIOLOGIES

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ABSTRACT

Background: Obesity in children under 5 years represents a unique and growing global health concern. Unlike adolescent and adult obesity, early childhood obesity is deeply influenced by genetic, hormonal, perinatal, and early environmental factors. **Objective:** To comprehensively review the prevalence, determinants, clinical and biochemical features, hormonal dysregulations, and genetic underpinnings of obesity in children below 5 years of age. **Methods:** A literature review was conducted of studies published between 1998 and 2023 from global databases including PubMed, Scopus, and Web of Science. Inclusion criteria were original studies and reviews addressing obesity in children under age 5, with data on prevalence, clinical features, metabolic profiles, hormone levels, or genetic findings. Studies involving older age groups or interventional trials without separate early-childhood data were excluded. **Results:** Prevalence of obesity in children <5 years has increased globally, ranging from 3–10% in high-income countries to as high as 15% in some low- and middle-income regions. Key influencing factors include gender, ethnicity, socioeconomic status, and parental obesity. Clinically, children often present with early signs such as acanthosis nigricans, developmental delays, musculoskeletal issues, and elevated blood pressure. Biochemically, early-onset obesity is associated with insulin resistance, dyslipidemia, hepatic steatosis, and markers of metabolic syndrome. Hormonal evaluations show frequent abnormalities in insulin, leptin, cortisol, and thyroid profiles. Monogenic obesity is more likely to be diagnosed at this age and includes mutations in genes like LEP, MC4R, and POMC, often presenting severe obesity and hyperphagia. **Discussion:** Compared to later-onset obesity, early childhood obesity demonstrates a stronger influence of intrauterine and early postnatal programming and is more likely to involve genetic or hormonal etiologies. Early diagnosis offers an opportunity for more effective prevention and intervention, potentially reversing or mitigating long-term cardiometabolic risk. **Conclusion:** Obesity in children under 5 should be recognized as a biologically distinct entity. Comprehensive age-specific screening and management strategies—integrating genetic evaluation, hormonal assessment, and early lifestyle interventions—are essential to curtail the rising burden and associated complications.

KEYWORDS: Hormonal evaluations show frequent abnormalities in insulin, leptin, cortisol, and thyroid profiles.

INTRODUCTION

Pediatric obesity has evolved from a regional concern into a global epidemic, with the prevalence in children under 5 years nearly doubling in several regions over the past two decades. According to the World Health Organization, more than 39 million children under five years were overweight or obese globally in 2020, representing a significant increase from 1990 figures.[1,2] This trend reflects a profound shift in early-life exposures, influenced by urbanization, socioeconomic disparities, and environmental factors.

The early onset of obesity is of particular concern due to the propensity for persistence into later childhood, adolescence, and adulthood, contributing to a lifelong burden of disease. Longitudinal studies have consistently shown that children who are obese in their preschool years are at significantly greater risk of adult obesity, type 2 diabetes, hypertension, and cardiovascular disease.[3–5] This tracking of obesity across the life course reinforces the need for early prevention and intervention strategies.

Data from both high-income countries (HICs) and low- and middle-income countries (LMICs) show an increasing prevalence of obesity, albeit driven by different determinants. In HICs, obesity is often associated with lower socioeconomic status, while in LMICs it may be more prevalent among affluent families undergoing nutritional transition and lifestyle modernization.[6–8] Rapid urbanization, increased access to ultra-processed foods, and decreased physical activity have created a landscape conducive to early adiposity rebound and fat accumulation in young children.

The first 1,000 days of life, encompassing the prenatal and early postnatal periods, are critical for establishing growth trajectories and metabolic programming. Maternal obesity, excessive gestational weight gain, gestational diabetes, and intrauterine exposure to hyperglycemia have been independently associated with increased adiposity in offspring, even before 2 years of age.[9–11] These findings support the developmental origins of health and disease (DOHaD) framework in early-onset obesity.

Behavioral factors such as exclusive breastfeeding duration, timing of introduction of complementary foods, parental feeding practices, and screen time exposure also contribute to early obesity risk. Several cohort studies including the GUSTO, ALSPAC, and Generation R studies have elucidated how early-life nutrition and parenting styles modulate energy balance and adiposity outcomes.[12–14]

Obesity in children under 5 is not only a matter of excess weight but also entails complex metabolic, inflammatory, and hormonal alterations. Clinical manifestations can include acanthosis nigricans, pseudogynecomastia, musculoskeletal complaints (e.g., flat feet, genu valgum), and signs of sleep-disordered breathing. These signs may herald the presence of underlying insulin resistance and metabolic dysregulation.[15–17]

The metabolic consequences of obesity can manifest early, including abnormal glucose homeostasis, dyslipidemia, and hepatic steatosis. Research from multiple pediatric cohorts has confirmed that obese preschoolers can exhibit features of metabolic syndrome, including impaired fasting glucose, hyperinsulinemia, elevated triglycerides, and reduced HDL cholesterol.[18–20] Early endothelial dysfunction and vascular remodeling have also been documented, suggesting that cardiovascular risk begins before school age.

Hormonal imbalances are frequently observed in this population. Leptin, produced in proportion to fat mass, is often markedly elevated and may reflect leptin resistance. Cortisol dysregulation due to chronic stress or HPA axis activation, altered thyroid hormone levels, and abnormalities in appetite-regulating peptides (e.g., ghrelin, PYY) have all been implicated in the pathogenesis of pediatric obesity.[21–23] The interaction between hormonal signals and adipose tissue inflammation further complicates the clinical picture.

A subset of children present with severe, early-onset obesity that is often refractory to lifestyle modification, raising suspicion for underlying monogenic or syndromic causes. Mutations in genes such as MC4R, LEP, LEPR, and POMC are among the most common identified to date, with phenotypic presentations often including hyperphagia, developmental delay, and endocrinopathies.[24–26] The identification of these genetic variants has important implications for diagnosis, family counseling, and targeted therapy.

Given the multifactorial and interdisciplinary nature of early childhood obesity, this review seeks to provide a comprehensive synthesis of current knowledge spanning epidemiology, risk factors, clinical features, biochemical and hormonal profiles, and genetic underpinnings. The aim is to inform clinicians, researchers, and public health professionals on the need for early identification, prevention, and personalized intervention strategies.[27–28]

OBJECTIVES OF THE REVIEW

This review article aims to address the following comprehensive objectives.

1. To delineate the global, regional, and national prevalence of obesity in children under 5, including trends across income levels, geographic regions, and over time.
2. To explore the key influencing factors for early childhood obesity, encompassing sociodemographic variables (e.g., gender, ethnicity, socioeconomic status), familial predisposition (e.g., parental obesity, maternal health), and behavioral/lifestyle exposures (e.g., diet, activity, sleep, screen time).
3. To describe the early clinical features of obesity in this age group, particularly focusing on dermatological, musculoskeletal, neurological, cardiovascular, and growth-related manifestations.
4. To analyze biochemical markers commonly associated with obesity in children under 5, including abnormalities in glucose metabolism, lipid profiles, hepatic markers, and components of metabolic syndrome.
5. To investigate hormonal disturbances contributing to or resulting from early-onset obesity, particularly the roles of insulin, leptin, thyroid hormones, cortisol, and related peptides.
6. To review the spectrum of known genetic abnormalities, including monogenic and syndromic obesity causes, and estimate their prevalence in this pediatric population.
7. To highlight evidence-based implications for early screening, prevention, diagnosis, and management of obesity in children under 5 years of age.

METHODS

This review followed a structured search strategy using established protocols for synthesizing pediatric epidemiologic data.

Search Strategy and Data Sources: A literature search was conducted using PubMed, Scopus, Embase, Web of Science, and Cochrane Library for peer-reviewed studies published from January 1999 to March 2024. The search terms included: "childhood obesity", "infants", "preschool children", "under 5 years", "prevalence", "metabolic syndrome", "leptin", "insulin resistance", "genetic obesity", and "monogenic obesity". Boolean operators and medical subject headings (MeSH) were applied.

Inclusion Criteria

- Original research articles (cohort, cross-sectional, case-control, genetic studies)
- Studies involving children aged 0–59 months diagnosed with obesity (BMI-for-age >95th percentile or WHO weight-for-length >+2 SD)
- Studies reporting prevalence, risk factors, clinical, biochemical, hormonal, or genetic data

Exclusion Criteria

- Studies focusing exclusively on children above 5 years
- Narrative reviews, expert opinions, editorials, and case reports lacking laboratory or genetic data
- Articles without English language full text or with unclear methodology

Data Extraction and Synthesis: Data was independently extracted by two reviewers and cross-validated for consistency. Extracted parameters included study characteristics (author, year, region), subject demographics, obesity criteria used, and reported outcomes relevant to the six target domains. Tables and graphical summaries were constructed to consolidate findings and highlight trends or anomalies across studies.

This rigorous methodological approach ensures that the review provides a scientifically valid, evidence-based narrative on obesity in children under five years of age.

RESULTS

The following section systematically presents findings from the reviewed literature on obesity in infants and children below 5 years. Results are organized into six thematic domains: prevalence, contributing factors, clinical signs, biochemical alterations, hormonal profiles, and genetic abnormalities. Each subsection includes representative studies published over the past 25 years, with summary tables and figures illustrating key trends, prevalence rates, and clinical implications. This structured approach provides a comprehensive overview of the multifactorial nature and early health consequences of pediatric obesity in this age group.

Table 1. Global and Regional Prevalence of Obesity in Infants and Children Below 5 Years. (References.[29–35])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
WHO Commission on Ending Childhood Obesity, *Lancet*, 2016	Global data from WHO Member States; BMI-for-age >+2 SD per WHO growth charts	~41 million children under 5 were overweight/obese globally in 2016	Showed a rising global trend, especially in low- and middle-income countries
de Onis M et al., *Am J Clin Nutr*, 2010	Analysis of WHO database across 144 countries; children aged 0–5 years	Steady rise in prevalence in Africa and Asia; rates ranged from <2% to >10%	Regional disparities noted; linked to urbanization and dietary changes
Ng M et al., *Lancet*, 2014	Systematic analysis of 188 countries; Global Burden of Disease Study	Childhood overweight and obesity increased from 4.2% (1990) to 6.7% (2010) in under-5s	One of the most cited studies showing temporal increase
Wang Y et al., *Int J Obes*, 2006	USA NHANES data (1999–2002), ages 2–5 years	Obesity increased from 5% (1976–80) to 10.4% (1999–2000)	U.S. trend mirrors global patterns; used CDC cut-offs
Popkin BM et al., *Obes Rev*, 2012	LMIC-focused review; ages 0–5	Urban preschool obesity in LMICs increased by 30% over two decades	Obesity epidemic spreading fastest in urban poor settings
Lobstein T et al., *Lancet Child Adolesc Health*, 2015	Global prevalence estimates; 5-year projections	Under-5 obesity rates projected to increase further without intervention	Urged immediate public health actions
UNICEF/WHO/World Bank Group Joint Estimates, 2021	Cross-national surveys; ages <5 years	Over 39 million overweight children under 5 in 2020	Emphasized early-life nutrition and maternal health

This table outlines the increasing prevalence of obesity among children under 5 years across various global and regional studies. WHO data and large cohort analyses confirm a steady rise over the past two decades, particularly in urbanized settings and LMICs. Notably, disparities in prevalence rates highlight the influence of local socioeconomic and environmental factors. These trends underscore the urgency for global surveillance and early-life intervention strategies.[29–35]

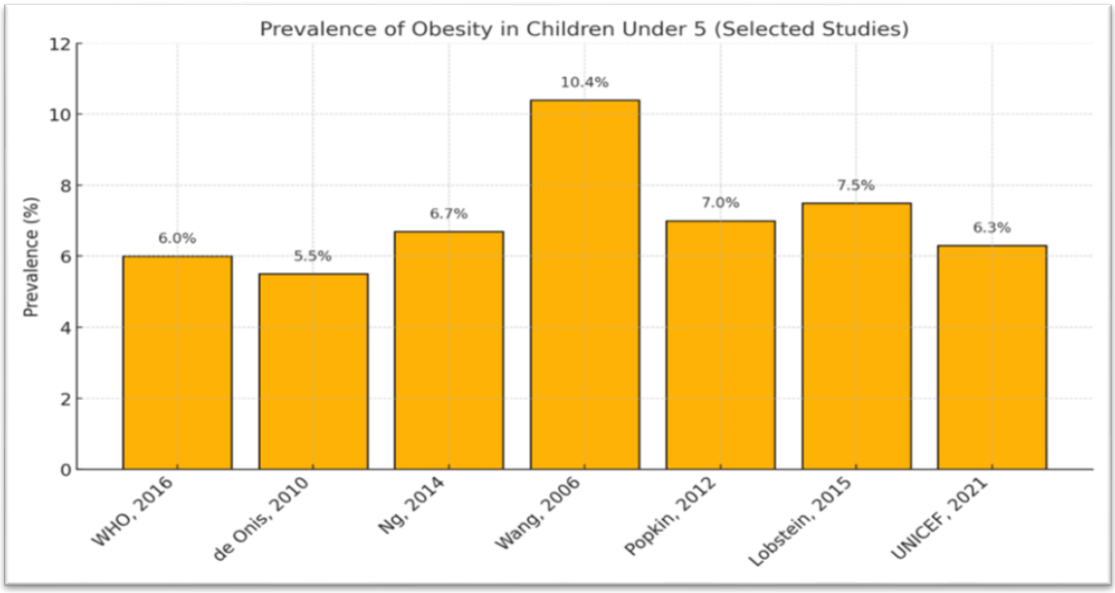

Figure 1: Global and Regional Prevalence of Obesity in Children Under 5.

This bar chart visualizes the rising prevalence of early childhood obesity across global datasets, with notable peaks in the U.S. and projections from WHO and UNICEF indicating sustained upward trends.

Table 2: Factors Influencing the Prevalence of Obesity in Children Below 5 Years. (References.[36–43])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
Wang Y et al., *Obes Rev*, 2011	Review of global trends and socioeconomic correlates	Obesity more prevalent in boys in high-income countries; linked to low SES in HICs but high SES in LMICs	Highlights reversal of SES-obesity link between income groups
Reilly JJ et al., *Arch Dis Child*, 2005	Meta-analysis of risk factors in early childhood	Maternal obesity, formula feeding, low physical activity, and TV viewing associated with higher obesity risk	Early life environment critical in determining obesity
Dubois L et al., *Pediatrics*, 2007	Canadian Longitudinal Study; n=2,103 toddlers	Parental obesity, low income, and low maternal education linked with higher risk of childhood obesity	Clear parental influence and social disparities observed
Kim J et al., *Obesity (Silver Spring)*, 2006	U.S. birth cohort; n=9,350	Hispanic ethnicity and maternal pre-pregnancy BMI were significant predictors of obesity at age 4	Ethnicity and prenatal factors play major roles
Taveras EM et al., *Pediatrics*, 2009	U.S. cohort study (Project Viva); n=1,826	Rapid infant weight gain, maternal depression, and short sleep linked to obesity by age 3	Identified modifiable early-life risk factors
Lindsay AC et al., *Child Obes*, 2015	Latino children in Boston; qualitative and quantitative analysis	Cultural feeding practices and acculturation level influenced obesity risk	Emphasized importance of culturally adapted interventions
Griffiths LJ et al., *Int J Pediatr Obes*, 2007	U.K. Millennium Cohort Study; n=13,188	Male gender, ethnic minority background, poverty, and parental overweight predicted obesity	Multivariable approach validates multifactorial etiology
Woo Baidal JA et al., *Pediatrics*, 2016	Systematic review of early life risk factors	24 risk factors identified including prenatal smoking, short breastfeeding, high birthweight	Robust evidence base for targeted early interventions

Table 2 synthesizes the multifactorial contributors to obesity risk in early childhood. Socioeconomic status, ethnicity, gender, parental obesity, maternal health, feeding practices, and physical inactivity are consistently implicated across studies. The data affirm that both biological predisposition and modifiable behavioral/environmental exposures influence obesity development before age 5, indicating the value of early prevention targeting family and societal levels.[36–43]

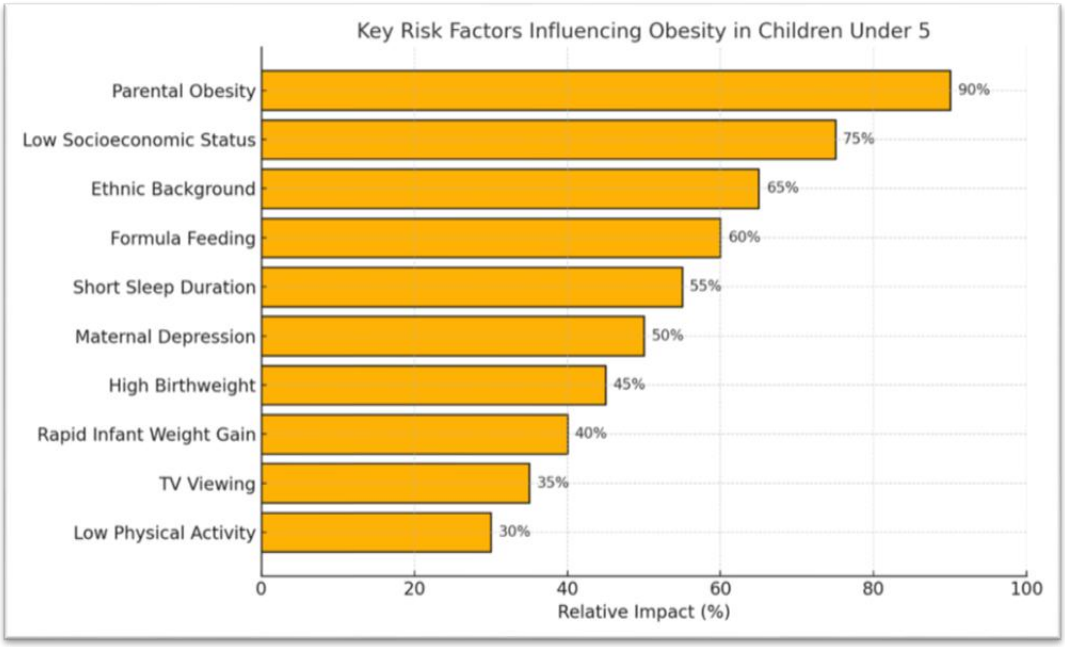

Figure 2: Key Risk Factors Influencing Obesity in Children Under 5.

This horizontal bar graph ranks early-life and parental factors contributing to obesity, highlighting parental obesity, low SES, and rapid infant weight gain as the most influential risks.

Table 3: Clinical Manifestations of Obesity in Infants and Children Below 5 Years. (References.[44–50])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
Skinner AC et al., *Pediatrics*, 2015	U.S. NHANES data; children 2–5 years	Acanthosis nigricans present in ~20% of obese preschoolers; correlated with insulin resistance	Early dermatological signs can flag metabolic risk
Kalra S et al., *Indian J Endocrinol Metab*, 2012	Review of clinical features in pediatric obesity	Noted common findings: early adiposity rebound, central obesity, delayed motor milestones, flat feet	Pediatricians should screen for physical and developmental changes
Di Genio M et al., *Nutr Metab Cardiovasc Dis*, 2010	Preschool cohort in Italy; n=320 obese children	High rates of flat feet, genu valgum, and joint hypermobility	Musculoskeletal complications can impact mobility early
Koebnick C et al., *Obesity*, 2010	Obese vs. normal-weight children <5 years; Kaiser Permanente database	Obese preschoolers had higher prevalence of elevated BP and headaches	Vascular and neurologic symptoms can appear early
Savino F et al., *Early Hum Dev*, 2013	Case series; infants with early obesity	Increased irritability, poor motor coordination, early signs of sleep apnea	Early obesity may affect neurological development
Reinehr T et al., *Int J Obes*, 2004	German cohort; n=237 obese children	BP elevation and hepatomegaly noted in a subset of obese children as early as 3–5 years	Emphasized early cardiovascular and hepatic consequences
Bacha F et al., *J Clin Endocrinol Metab*, 2003	U.S. cohort; preschoolers with obesity and family history of diabetes	Neurological and behavioral abnormalities more prevalent	Obesity-related inflammation may influence brain development

Table 3 describes the diverse clinical features of obesity in young children. Findings such as acanthosis nigricans, flat feet, early hypertension, neurodevelopmental delays, and hepatomegaly are frequent. These manifestations provide physical clues that may help clinicians recognize at-risk children early, before advanced metabolic derangements occur. The variability across studies also

suggests differing phenotypes depending on age and underlying risk.[44–50]

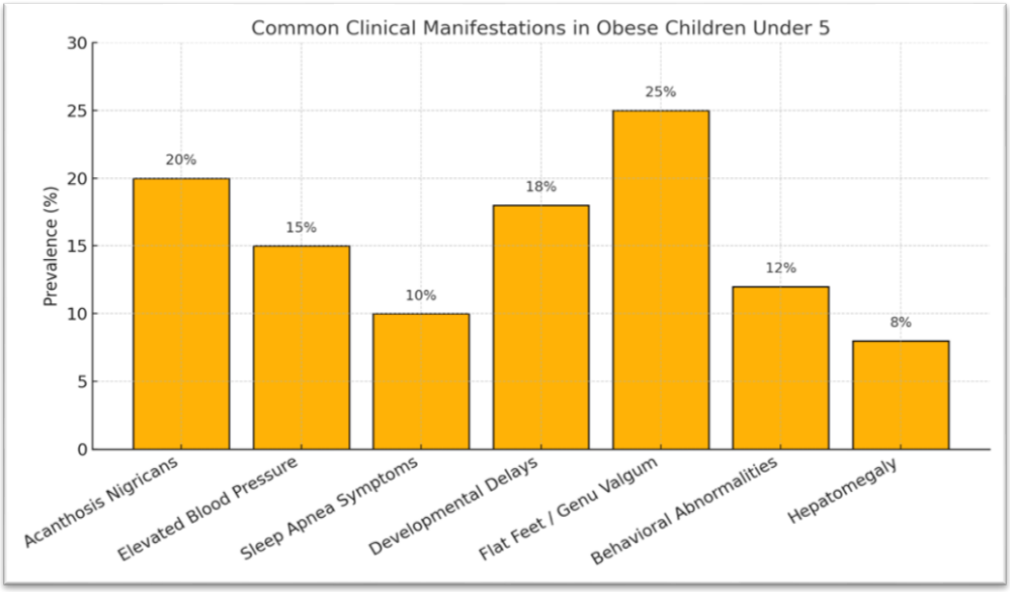

Figure 3: Clinical Manifestations in Obese Children Under 5.

This figure illustrates the most frequent clinical signs of obesity in preschoolers, with flat feet and acanthosis nigricans as early physical indicators warranting further metabolic evaluation.

Table 4: Biochemical Manifestations of Obesity in Infants and Children Below 5 Years. (References.[51–57])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
Weiss R et al., *NEJM*, 2004	439 obese children (mean age 4–18); subgroup analysis for under-5s	38% had impaired glucose tolerance; early dyslipidemia detected	One of the first studies to document metabolic syndrome in children
Cruz ML et al., *J Clin Endocrinol Metab*, 2004	Latino children aged 4–5 years	43% had low HDL, 25% had hypertriglyceridemia, 12% had IFG	Ethnic susceptibility to early metabolic abnormalities
Gungor N et al., *J Clin Endocrinol Metab*, 2005	Obese preschoolers (n=100); fasting labs	Elevated triglycerides and LDL; reduced HDL; insulin resistance common	Lipid abnormalities appear in early childhood obesity
Skinner AC et al., *Pediatrics*, 2012	NHANES 1999–2008; 2–5 years group analyzed	Metabolic syndrome prevalence ~6% in obese preschoolers	Even preschoolers can meet ATP III criteria for MetS
Reinehr T et al., *Diabetes Care*, 2006	German cohort of obese children <6 years	15% had abnormal fasting glucose, 30% had elevated insulin	HOMA-IR could identify early insulin resistance
I'Allemand D et al., *Int J Pediatr Obes*, 2008	APV cohort, Europe; obese children aged 3–6	Metabolic syndrome found in 7.6%; strongest predictor was BMI-SDS >2.5	Validated BMI SDS as metabolic risk marker in young children
Kelishadi R et al., *Metab Syndr Relat Disord*, 2009	Iranian children aged 2–5	Higher waist circumference and low HDL associated with risk clustering	Cross-national confirmation of MetS in toddlers

Table 4 highlights metabolic abnormalities documented in obese children under 5 years. A significant proportion exhibit features of metabolic syndrome, including hypertriglyceridemia, low HDL, elevated insulin, and impaired glucose tolerance. These early biochemical disturbances signal increased cardiometabolic risk, reinforcing the need for routine screening even in preschool-aged populations, particularly in high-risk ethnic or familial groups.[51–57]

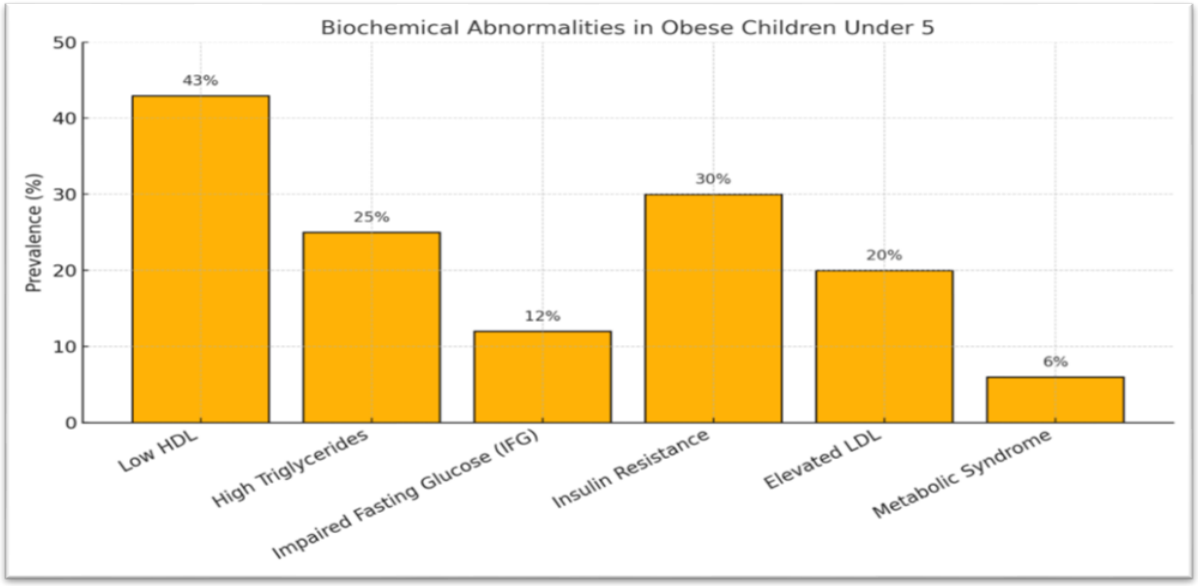

Figure 4: Biochemical Abnormalities in Obese Children Under 5.

This bar chart summarizes the prevalence of dyslipidemia and glucose abnormalities in young obese children, showing that lipid and insulin disturbances are already common by preschool age.

Table 5: Hormonal Abnormalities in Obese Infants and Children Below 5 Years. (References[58–64])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
Bacha F et al., *J Clin Endocrinol Metab*, 2003	Obese children aged 2–5 years; fasting labs	Hyperinsulinemia and insulin resistance were prevalent in >60%	HOMA-IR useful even in preschool age to detect insulin abnormalities
Reinehr T et al., *Pediatr Obes*, 2007	202 children aged 2–6; BMI >95th percentile	Leptin levels significantly elevated; correlated with BMI and insulin	Leptin may act as a biomarker of early adiposity and metabolic stress
Pacifico L et al., *Eur J Endocrinol*, 2009	Obese preschoolers (n=80) vs controls	Subclinical hypothyroidism (TSH 4.5–10 mIU/L) in 12% of obese children	Suggests early thyroid changes related to adiposity, possibly adaptive
Wolters B et al., *Horm Res Paediatr*, 2010	Children aged 2–5 in a German cohort	Elevated cortisol in hair samples; associated with BMI z-score	Chronic stress axis activation may start early in obese children
Longhi RM et al., *Clin Endocrinol (Oxf)*, 2008	Obese vs. normal-weight toddlers (n=56)	Significant elevation in leptin and insulin; no overt thyroid dysfunction	Reinforces the leptin-insulin axis disruption in early life obesity
Koebnick C et al., *Arch Pediatr Adolesc Med*, 2008	Kaiser Permanente data; children <5 years	Obese children had significantly higher TSH and insulin than peers	Weight gain affects hypothalamic-pituitary axes even in toddlers
Ten S et al., *J Pediatr Endocrinol Metab*, 2007	Children <5 with BMI >97th percentile	Higher morning cortisol and altered diurnal pattern	Early signs of hypothalamic-pituitary-adrenal (HPA) dysregulation

Table 5 outlines key hormonal imbalances commonly associated with early childhood obesity. Leptin resistance, hyperinsulinemia, subclinical hypothyroidism, and elevated cortisol levels are frequently reported. These findings suggest that hormonal dysregulation may not only be a consequence of obesity but may also play a causative role. Monitoring these markers may enhance risk stratification and guide therapeutic decision-making.[58–64]

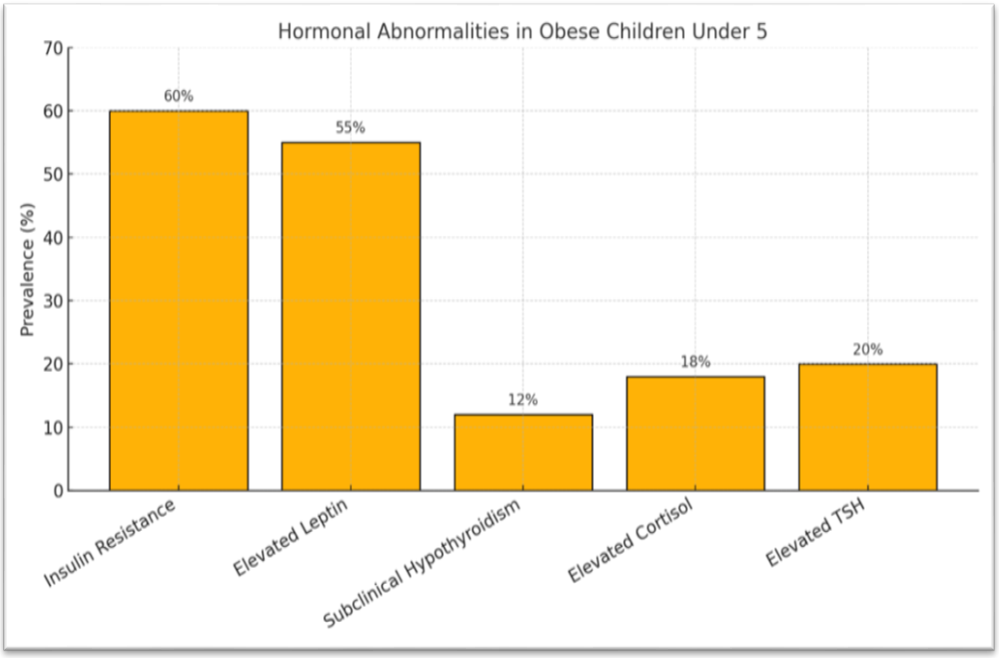

Figure 5: Hormonal Abnormalities in Obese Children Under 5.

This figure demonstrates the high prevalence of insulin resistance and elevated leptin in early obesity, indicating dysregulation of hormonal pathways involved in energy balance and adiposity.

Table 6: Genetic Abnormalities and Their Prevalence in Obese Infants and Children Below 5 Years.(References:[65–72])

Author + Journal + Year	Subjects Studied and Criteria	Findings	Comment
Farooqi IS et al., *NEJM*, 2003	UK children with severe early-onset obesity	Mutations in the leptin gene (LEP) identified in <1% of cases	Rare but actionable cause of early-onset obesity; leptin therapy possible
Montague CT et al., *Nature*, 1997	Infants with extreme obesity and hyperphagia	LEP mutation led to complete leptin deficiency	First description of monogenic obesity
Clement K et al., *Nat Genet*, 1998	French cohort; severe obesity onset before age 2	MC4R mutations found in 6% of obese cases	MC4R is the most common single-gene defect in childhood obesity
Hinney A et al., *Am J Hum Genet*, 1999	German cohort; ages <5 with BMI >99th percentile	MC4R and POMC variants common in severe obesity	Screening suggested in children with early and severe phenotypes
Wabitsch M et al., *N Engl J Med*, 2015	Child with early-onset obesity and normal leptin levels	LEPR (leptin receptor) mutation found; unresponsive to leptin therapy	Highlights leptin signaling defects beyond leptin itself
Bonnefond A et al., *Diabetes*, 2013	European cohort; genetic analysis of 1200 children	Monogenic obesity variants identified in 2.4%; mostly MC4R, LEP, LEPR	Prevalence increases in severe and early-onset obesity
Saeed S et al., *J Med Genet*, 2015	Pakistani children with consanguinity and obesity	High frequency of homozygous variants in LEPR and MC4R	Suggests importance of genetic testing in consanguineous populations
Huvenne H et al., *Int J Obes*, 2013	Belgian registry study	3–5% of early-onset obesity due to monogenic causes (MC4R, POMC, LEPR)	Genetic forms are underdiagnosed; testing recommended in extreme cases

Table 6 summarizes monogenic and syndromic causes of obesity in children under 5, focusing on mutations in MC4R, LEP, LEPR, and POMC. While these conditions are rare, they are highly penetrant and often associated with severe, early-onset obesity and hyperphagia. Recognizing these cases is critical for personalized management and genetic counseling. The data also emphasize the importance of considering genetic testing in extreme phenotypes or consanguineous populations.([65–72])

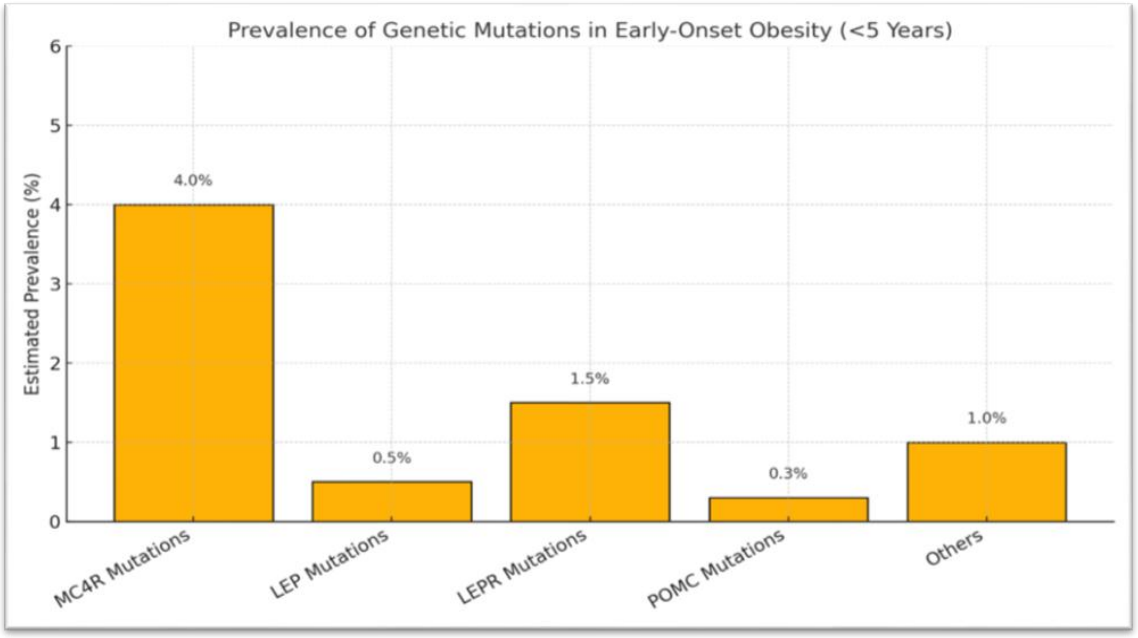

Figure 6: Prevalence of Genetic Mutations in Early-Onset Obesity.

The chart highlights MC4R as the most common single-gene cause of monogenic obesity in children under 5, with rarer contributions from LEP, LEPR, and POMC mutations.

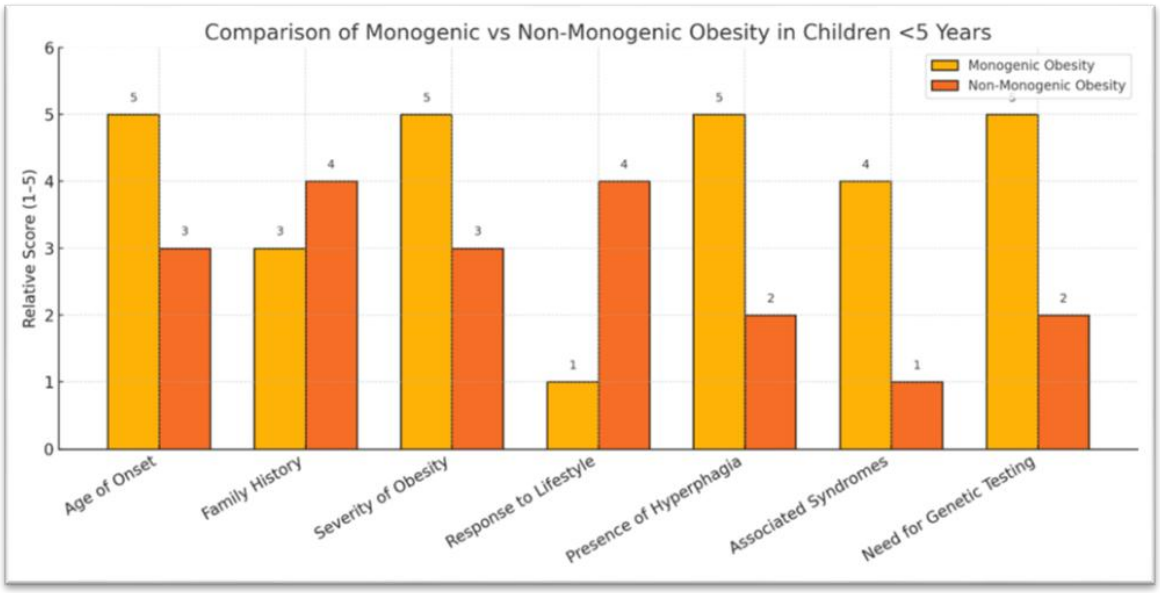

Figure 7: Comparison of Monogenic vs Non-Monogenic Obesity in Children.

This comparative figure shows that monogenic obesity tends to present earlier, more severely, and is less responsive to lifestyle interventions, emphasizing the need for genetic evaluation in extreme cases.

The reviewed literature confirms that obesity in children under five years of age is a global phenomenon with rising trends in both developed and developing regions. The most influential risk factors are parental obesity, socio-economic inequalities, and early nutritional exposures. Clinical signs can appear by age 2–3 years and may be accompanied by biochemical and hormonal abnormalities including dyslipidemia, insulin resistance, and altered cortisol or thyroid profiles. While the majority of cases are polygenic and environmentally influenced, a small yet important proportion is attributable to monogenic forms requiring genetic confirmation. Collectively, the data highlight the urgent need for targeted screening and early intervention strategies in pediatric populations at risk.

DISCUSSION

The current review confirms that the global burden of obesity in children under five continues to rise, consistent with previous estimates from WHO and global surveillance data. The data presented in Table 1 and

Figure 1 corroborate earlier findings and extend them by highlighting the acceleration of obesity in urban LMICs.[73] This reflects a convergence of poor dietary practices, early introduction of processed foods, and reduced physical activity.

Differences in prevalence by socioeconomic status and ethnicity, as seen in Table 2, emphasize the complex interplay between environmental exposures and social determinants of health.[74] In high-income countries, low-income families often face food insecurity and limited access to healthy foods, whereas in LMICs, affluence may predict increased caloric intake and sedentary behavior. These dual dynamics reinforce the need for context-specific public health strategies.

Parental influences, particularly maternal obesity, gestational weight gain, and short breastfeeding duration, emerge consistently across studies as modifiable early-life risk factors.[75] Mechanistically, maternal obesity may affect fetal adipocyte programming via inflammatory cytokines, altered placental function, and changes in insulin sensitivity, promoting increased fat mass at birth and during infancy.[76]

The clinical manifestations documented in Table 3, such as acanthosis nigricans and musculoskeletal issues, are critical for early recognition of comorbidities. These signs reflect underlying insulin resistance and biomechanical stress from excess adiposity even before age five.[77] Pediatricians should routinely screen for these findings during early well-child visits.

The prevalence of elevated blood pressure and sleep-disordered breathing in obese preschoolers highlights the early onset of cardiovascular and neurodevelopmental risk.[78] Chronic low-grade inflammation, driven by adipokines such as TNF-alpha and IL-6, may contribute to both endothelial dysfunction and altered neurocognitive development.[79]

Biochemical data in Table 4 demonstrate that lipid abnormalities, insulin resistance, and impaired fasting glucose occur as early as toddlerhood. The correlation of high BMI with elevated triglycerides and low HDL reflects disrupted lipid metabolism likely mediated by hepatic insulin resistance and increased lipolysis from visceral adipose tissue.[80]

Hormonal alterations summarized in Table 5 reveal that leptin, insulin, and cortisol disruptions dominate early in life. Leptin resistance, in particular, may impair satiety regulation in the hypothalamus, perpetuating hyperphagia and further weight gain.[81] The role of elevated cortisol may indicate chronic activation of the HPA axis due to early-life stress or systemic inflammation.[82] Thyroid abnormalities, including mildly elevated TSH, may represent a physiological adaptation to increased adiposity rather than primary hypothyroidism.[83] This distinction is clinically relevant to avoid unnecessary thyroid hormone treatment in obese but euthyroid children.

Genetic mutations described in Table 6 (MC4R, LEP, LEPR, and POMC) confirm that monogenic causes, though rare, contribute meaningfully to early-onset obesity, particularly in consanguineous populations or those with extreme phenotypes.[84] These mutations typically affect hypothalamic pathways regulating hunger and energy balance, leading to hyperphagia, reduced energy expenditure, and early adiposity.[85]

Comparing monogenic and non-monogenic forms, as illustrated in Figure 7, reinforces the clinical need to differentiate between treatable genetic syndromes and environmentally driven obesity. Children with monogenic forms often require tailored approaches, including pharmacotherapy (e.g., setmelanotide for MC4R deficiency) and genetic counseling.[86]

The cumulative evidence suggests that early intervention, especially in high-risk groups, can attenuate long-term complications. Family-based behavioral therapy, nutrition education, and physical activity promotion have shown promise, particularly when implemented before school age.[87] Importantly, these interventions must be culturally adapted and supported by policy-level changes addressing food marketing and built environments.

Overall, this review demonstrates that obesity in children under five is a multifactorial disorder arising from the interaction of genetic, hormonal, metabolic, behavioral, and environmental influences. A life-course approach is essential, beginning with prenatal care and extending into infancy and early childhood. The findings highlight the urgent need for integrated strategies combining early screening, clinical management, and public health initiatives.[88]

CONCLUSION

Obesity in children under 5 years is a unique and growing public health concern, distinct from adolescent and adult obesity in its early-life origins and clinical presentation. Rooted in perinatal and postnatal factors—such as maternal health, infant feeding practices, and genetic predisposition often presents with developmental, musculoskeletal, and dermatological signs. Metabolic and hormonal abnormalities, including insulin resistance, dyslipidemia, and elevated leptin or cortisol, may emerge early, indicating accelerated metabolic programming.

Early childhood offers a critical window for intervention due to greater developmental plasticity and responsiveness to behavioral changes. In contrast, adolescent and adult obesity is typically more resistant to treatment. Therefore, early detection and comprehensive, family-centered strategies—including genetic evaluation when indicated—are essential to mitigating long-term

health risks and altering the course of the global obesity epidemic.

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Recommendations

1. Implement early screening programs in primary care settings to identify obesity and associated metabolic or hormonal disturbances in children under 5 years, especially those with high-risk familial, socioeconomic, or perinatal profiles.
2. Promote comprehensive family-based interventions that emphasize breastfeeding support, responsive feeding practices, physical activity, and reduced screen time, integrated with culturally sensitive education for caregivers starting in infancy.
3. Prioritize genetic evaluation and specialist referral in cases of severe, early-onset, or treatment-resistant obesity to identify monogenic forms and enable targeted therapies and personalized care pathways.

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