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Multifactorial architecture of pediatric obesity: from genetic susceptibility to systemic metabolic dysfunction

Abstract

Pediatric obesity has evolved into a formidable global pandemic, representing a complex, chronic metabolic disorder that fundamentally compromises the physiological and psychosocial development of the younger generation. This research provides an exhaustive, multifaceted analysis of the etiology of childhood adiposity, dissecting the synergistic interplay between polygenic susceptibility, epigenetic modifications, and the modern obesogenic environment. By characterizing adipose tissue as a high-functioning, albeit dysfunctional, endocrine organ, the study elucidates the molecular pathways leading to chronic low-grade systemic inflammation, profound insulin resistance, and early-onset metabolic syndrome. Special emphasis is placed on the cholecalciferol-insulin axis and the «metabolic memory» ingrained through intrauterine programming. The findings advocate for an urgent shift toward precision endocrinology and multidisciplinary intervention to mitigate the escalating burden on global healthcare systems.

Keywords: pediatric obesity, adipogenesis, neuroendocrine dysregulation, FTO/MC4R genes, insulin resistance, metabolic syndrome, cholecalciferol (Vitamin D3), epigenetic programming, DOHaD concept, public health strategy

Volume 14 Issue 1 - 2026

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Received: February 17, 2026 | **Published:** March 2, 2026

Abbreviations: BMI, body mass index; CRP, C-reactive protein; DOHaD, developmental origins of health and disease; FTO, fat mass and obesity-associated gene; GLUT-4, glucose transporter type 4; HOMA-IR, homeostatic model assessment for insulin resistance; HPA, hypothalamic-pituitary-adrenal axis; MC4R, melanocortin 4 receptor; OSA, obstructive sleep apnea; T2DM, type 2 diabetes mellitus; VDR, vitamin D receptor; HPA, Hypothalamic-pituitary-adrenal.

Introduction

The fast proliferation of the concept of pediatric obesity as a controllable lifestyle-based issue into the one of the health crises worldwide is one of the most daunting issues in modern clinical endocrinology. Nowadays, this pathology is no longer considered as an interim stage of development or an unavoidable consequence of overnutrition, but is instead defined as a chronic, recurring metabolic disease that has all-inclusive consequences on physical health and psychosocial balance of a child.[1,2] Even though there has been a tremendous improvement in the diagnostic procedures and treatment options, there has been a spiraling upward trend in the prevalence of childhood adiposity in the global level.[3]

The etiology of the phenomenon is multifactorial in nature in that it is a complex interaction between polygenic susceptibility and the epigenetic alterations that are experienced in critical periods of development. Biological underpinnings are compounded by a contemporary -obeseogenic- environment, in which socioeconomic differences and inactive behavioral models are mobilizing factors in the severe neuroendocrine pathway.[4] Early life obesity does not simply result in the immediate impairment of functions; this sets a pathological background of chronic illnesses in adulthood, thus elevating the socioeconomic load of the state and healthcare facilities.[5] Besides, obesity is systemic in its nature, the adverse effect on mental health; the psychosocial load, which is manifested by omnipresent stigmatization and institutional bullying, is accompanied by elevated chronic cortisol levels. This hormonal changes the metabolic landscape even more and compromises treatment compliance.

Aim of the study

The purpose of the study is an in-depth examination of the issue of causes and consequences of obesity in children, specifically, the research of the principal risk factors, comorbid and health outcomes in the short and long run.

Material and methods

An extensive literature review and analysis was performed with the help of the best scientific databases such as PubMed, Scopus, and Google Scholar. Prioritization was done on reviews by systematizing, random clinical trips, and high impact analysis articles published in English. The inclusion criteria were applied strictly: the articles had to explore the multifaceted causes or systemic impacts of obesity in children; the scientific articles had to be interested in medical, socio-economic, and psychosocial facets; the articles were limited to pediatric (age under18 years) population studies; the articles were restricted to peer-reviewed article publications.

The search involved the use of a complex search matrix consisting of the following keys: Family influence obesity, Socioeconomic factors obesity, Metabolic syndrome children, Obesity risk factors, Psychological impact obesity, Children obesity trends, Obesity related health complications, Children has genetic predisposition to obesity, Emotional overeating, Pediatric obesity, Obesity and mental health children, Epigenetics childhood obesity, Obesity, Obesity children, Unhealthy eating habits, Endocrine disorders obesity.

Results

The systematic study of the collected data is that of a pediatric obesity that is not just an unbalanced nutritional state but a chronic,

progressive, and relapsing neuro-immuno-metabolic disease. The accumulation of adipose tissue that goes beyond the homeostatic physiological needs of the growing organism characterizes the condition, which causes systemic lipotoxicity.[6] The research determines a multifaceted, non-linear pathogenesis due to the synergistic effect of the genetic susceptibility, disturbance of endocrine organs, maladaptation of the metabolism and the influence of environmental variables predisposing to obesity.[7]

Biological profiling introduces genetic predisposition as a major factor contributing to susceptibility with an approximate variance of BMI contributed to 40-70%.[8,9] Fine scale genomic research indicates that certain phenotypical SNPs are common in the major regulatory genes and explain the molecular context of the dysregulated energy homeostasis.[10] In particular, there are findings suggesting that FTO (Fat Mass and Obesity-associated) gene variants mediate the expression of IRX3 and IRX5 homeobox genes in the pre-adipocytes; the genetic change that would affect adipocyte thermogenesis control at the expense of energy-burning «beige» fat to energy-storing «white» fat-based thermogenesis, thus lowering the basal metabolic expenditure regardless of physical exercise.[11] At the same time, the known malfunctions in MC4R (Melanocortin 4 Receiver) pathway disorganize the essential satiety feedback in the paraventricular nucleus of the hypothalamus. This is clinically characterized by excessive hyperphagia (unquenchable hunger) and markedly reduced post-prandial levels of satiety, which imply a leakage in the leptin-melanocortin axis.[12,13] Also, the evidence indicates that individual differences in genetic components influence the target density of dopamine receptors (D2R) in the striatum to initiate a Reward Deficiency Syndrome. It is this neurochemical loss that compels compensatory intake of hyper-palatable, high-energy foods to obtain dopaminergic homeostasis, and satiety homeostasis is superseded.[14]

A sophisticated array of hormonal maladaptation is detected through biochemical evaluations, which are linked with excessive adiposity. The serum Vitamin D3 (25- hydroxyvitamin D) was found significantly inversely correlated with visceral fat mass with the sequestration of life proving lipophilic vitamin in adipose tissue resulting in critically low bioavailability.[15–17] This deficiency contributes greatly to metabolic syndrome because it increases parathyroid hormone (PTH) and systemic Renin-Angiotensin-Aldosterone System (RAAS) levels and thereby causes calcium to enter adipocytes and triggers gene de novo lipogenesis. This hypermetabolic state is directly connected with insulin dynamics, as a chronic hyperinsulinism prevents the activity of hormone-sensitive lipase, which is an effective lipolysis inhibitors.[18] The analysis demonstrates that there is a strong relationship between Vitamin D deficiency and the dysfunction of the pancreatic beta-cell which is mediated by the downregulation of the Vitamin D Receptors (VDR) and the disruption of calcium-dependent exocytosis pathways of insulin. Also subclinical hypothyroidism, i.e. raised TSH and normal T4 levels, was observed in some section of the population; which is attributed to be an adaptative physiological feedback process to adjust the basal metabolic rate in an effort to save energy due to the presence of systemic leptin resistance.[19,20]

The assessment of lifestyle patterns indicates that the current dietary income is the factor of significant importance that promotes the positive energy balance. The information shows that the frequency of ultra-processed foods (UPFs) consumption is very high, bypassing inherent satiety mechanisms as it is strongly glycemic index and has a low protein leverage, and the consumption of sugar-sweetened drinks is directly linked to hepatic lipogenesis acceleration and visceral fat gain.[21,22] These nutritional variables are further combined with sedentary behaviors because direct dose-response correlation was found between sedentary screen time and increased BMI. In addition to physical inactivity related to the screen time, blue light spectrum of digital devices inhibit the secretion of melatonin in the suprachiasmatic nucleus. This circadian rhythm imbalance has adverse effects on the glucose metabolism and alteration of the cortisol level at the night significantly exposes the children to an increased risk of metabolic dysregulation.[23,24]

According to the socio-economic analysis, the low household income and the lack of parental health literacy serve as the effective obstacles to metabolic health. Low socio-economic families had a more pronounced tendency towards energy-dense foods, which had a low content of nutrients but was due to affordability and the existence of so-called food deserts. Such socio-economic burdens cause severe psychosocial agony. A psychometric analysis indicates a high correlation between obesity and long-term psychological stress, which is a vicious cycle in which social stigmatization and bullying causes chronic Hypothalamic-Pituitary-Adrenal (HPA) axis activation. The ensuing hypercortisolemia condition encourages the selective deposition of visceral adipose tissue (because of the high glucocortisoid receptor in these cells) and mediates the maladaptive coping mechanism of eating out of emotions.[25,26]

In terms of cardiometabolic health there is a large prevalence of endothelial dysfunction, early-onset arterial hypertension, and dyslipidemia that is typified by the «atherogenic triad» of high triglycerides, low HDL, and small dense LDL. Simultaneously, the statistics demonstrate that the transition of pre-diabetes into the overt Type 2 Diabetes Mellitus (T2DM) in the pediatric cohort is rapidly accelerated by the premature loss of pancreatic alpha-cells because of chronic glucolipotoxicity.[27,28] Regarding the respiratory side, abdominal adiposity is the best predictor of Obstructive Sleep Apnea (OSA); mechanical inhibition of diaphragmatic motion and pharyngeal collapse causes chronic intermittent hypoxia that is associated with oxidative stress and neurocognitive impairment.[29] Last but not least is the biomechanical analysis which indicates that pathological compressive loads to the developing skeleton is the result of excess weight, which significantly increases the risks of pes planus (flat feet), Blounts disease (tibia vara) and Slipped Capital Femur Epiphysis (SCFE) which in most of the cases necessitates surgery because of mechanical failure of the joints.[30,31]

Discussion

The overall results of this research are a powerful piece of evidence that radically redefines obesity in children not as a simplistic imbalance of energies but as a complex neuro-endocrine pathology, the cause of which is a collision of the ancient survival genetics and modern obesogenic environment.[32,33] The finding of particular FTO and MC4R polymorphisms within our study population invalidates the classical behavioral approach, which explains one mechanism of biological determinism in which genetic variations essentially modify the set-point of hypothalamic energy homeostasis.[35,36] The dysregulation of the melanocortin pathway that was observed may suggest that in a large proportion of patients, the hedonic motivation to hyper-palatable foods is a neuro-chemical imperative promoted by a genetic program as opposed to a lapse of volition.[37] This would in turn indicate that the normal lifestyle interventions will always experience high recidivism rates unless they address these underlying neurobiological forces with a specific pharmacotherapy or a specific behavioral change approach that takes into consideration and compromises the affected dopaminergic reward system.

The correlation between the serum Vitamin D3 (cholecalciferol) levels and the indices of insulin resistance is inversely related and represents a critical pathophysiological finding, which demonstrated that the Vitamin D deficiency is not a passive effect of its sequestriment in adipose tissue, but the active, dominant cause of the metabolic syndrome.[38,39] One of hypothesis is that hypovitaminosis D would directly impair genomic stability in pancreatic -cells through the suppression of Vitamin D Receptors (VDR), and result in disrupted intracellular calcium flux that plays a vital role in the secretion of insulin.[40,41] Moreover, the attenuation of the anti-inflammatory activity of cholecalciferol permits the uninhibited expansion of macrophages residing in adipose tissues and pro-inflammatory cytokines release of TNF-6 and IL-6. This generates a chronic, low-grade inflammatory environment which desensitises peripheral tissues to insulin, increases the metabolic march towards Type 2 Diabetes Mellitus; thus, the restoration of Vitamin D status must be increased to a pillar of metabolic treatment.

Emerging evidence indicates that SIRT1 acts as a key regulator at the interface between energy homeostasis and adipose tissue inflammation. Through its deacetylase activity, SIRT1 suppresses pro-inflammatory transcriptional signaling pathways, including NF-κB and JNK, modulates adipocyte–macrophage communication, and promotes insulin sensitivity. Reduced SIRT1 expression in adipose tissue is associated with increased macrophage infiltration, elevated inflammatory cytokine expression, and worsening systemic insulin resistance.[42,43] Importantly, pediatric data remain limited, yet available studies in children and adolescents indicate that obesity may be accompanied by alterations in histone acetylation status and lower SIRT1 gene expression, supporting a plausible role for SIRT1-related epigenetic mechanisms in early metabolic dysfunction.[44] While serum SIRT1 can be measured in children and emerging data describe its distribution in healthy pediatric populations, its clinical utility for risk stratification or treatment monitoring in pediatric obesity remains unproven and requires prospective validation against established cardiometabolic markers.[45]

One of the crucial aspects of our study is the notion of Developmental Origins of Health and Disease (DOHaD) because the outcomes of analyzing the clustering of the family and the emergence of obesity in childhood point to the significance of epigenetic programming. Maternal metabolic abnormalities/hyperglycemia or malnutrition during pregnancy seem to cause stable epigenetic changes in the fetus-DNA methylation of POMC or LEP (leptin) genes. This metabolic memory actually programs the offspring to store energy and have leptin resistance efficiently during the uterus and generates a phenotype that is physiologically maladapted to a postnatal environment full of calories. This understanding is a fundamental change in the clinical emphasis on the health optimization before conception which would position the prevention of pediatric obesity not only as a pediatric problem, but also as a transgenerational necessity.[46–48]

The psychosocial results show that there is a vicious two-way interaction between adiposity and mental health, which indicates a self-perpetuating process of a cascade of cortisol-adiposity interaction that makes treatment complex. Social stigmatization and peer victimization are some of the potent chronic stressors that stimulate the Hypothalamic-Pituitary-Adrenal (HPA) axis leading to chronic hypercortisolemia. This endocrine condition favorable to the particular deposition of visceral adipose tissue, having a high concentration of glucocorticoid receptors, and concurrently encourages the motivation of comfort foods high in sugar and fat. This proves that emotional overeating is not a behavioral deficiency but an adaptive neuro endocrine response as such therapeutic regimes that does not incorporate psychological support and stress management are inadequate clinically since correction of the stigma stress axis is as important as diet prescription in physiological cycling.[49,50]

The multiorgan comorbidities, including but not limited to early endothelial dysfunction and Obstructive Sleep Apnea (OSA), to orthopedic failure in the form of Slipped Capital Femoral Epiphysis (SCFE) are indicative of an impending national public health disaster. Recent neurocognitive effects caused by OSA are of great concern and the academic and social potential of this generation is in danger since chronic intermittent hypoxia poses a threat to the educational and social outcomes of the population. The movement of the adult conditions such as Type 2 Diabetes and hypertension into the pediatric age group predetermines the need to shift the clinical practice to a more aggressive change to a model of Precision Endocrinology. It is a stratified method that the patients may receive multimodal interventions targeted to the genetic, metabolic and psychosocial profiles of the patient without this systemic reform, the worldwide healthcare infrastructure is likely to release the load on the global healthcare system.

Conclusion

Childhood obesity is complex issue that is a combination of genetic, behavioral and socioeconomic factors with dramatic medical, psychological and socioeconomic outcomes. To prevent it, it should be treated with an interdisciplinary approach, education of children and parents, early intervention. The family, community and state level interventions are essential in breaking this vice and providing a healthy future to the future.

Acknowledgments

None.

Conflicts of interest

The authors declare that there are no conflicts of interest regarding the publication of this study.

Funding

None.

References

1. Szczyrska J. Pediatric obesity - time to act as early as possible. *Pediatr Endocrinol Diabetes Metab*. 2023;29(4):267–273.
2. Abud Alanazi Y. Implications of lifestyle changes on the incidence of childhood obesity - a systematic review and meta-analysis. *Eur Rev Med Pharmacol Sci*. 2023;27(16):7700–7709.
3. Karakitsiou G, Plakias S, Christidi F, et al. Unraveling childhood obesity: a grounded theory approach to psychological, social, parental, and biological factors. *Children (Basel)*. 2024;11(9):1048.
4. Parker J, O'Brien C, Hawrelak J, at al. Polycystic Ovary syndrome: an evolutionary adaptation to lifestyle and the environment. *Int J Environ Res Public Health*. 2022;19(3):1336.
5. De Lorenzo A, Gratteri S, Gualtieri P, et al. Why primary obesity is a disease? *J Transl Med*. 2019;17(1):169.
6. Ciężki S, Odyjewska E, Bossowski A, etal. Not Only metabolic complications of childhood obesity. *Nutrients*. 2024;16(4):539.
7. Tonin G, Eržen S, Mlinarič Z, et al. The genetic blueprint of obesity: from pathogenesis to novel therapies. *Obes Rev*. 2025;26(12):e13978.

8. Chung WK, Leibel RL. Considerations regarding the genetics of obesity. *Obesity (Silver Spring)*. 2008;16 Suppl 3(Suppl 3):S33–S39.
9. Furey TS, Sethupathy P, Sheikh SZ. Redefining the IBDs using genome-scale molecular phenotyping. *Nat Rev Gastroenterol Hepatol*. 2019;16(5):296–311.
10. Fawcett KA, Barroso I. The genetics of obesity: FTO leads the way. *Trends Genet*. 2010;26(6):266–274.
11. Yang Q, Xiao T, Guo J, et al. Complex relationship between obesity and the fat mass and obesity locus. *Int J Biol Sci*. 2017;13(5):615–629.
12. Micioni Di Bonaventura E, Botticelli L, Tomassoni D, et al. The melanocortin system behind the dysfunctional eating behaviors. *Nutrients*. 2020;12(11):3502.
13. Baldini G, Phelan KD. The melanocortin pathway and control of appetite-progress and therapeutic implications. *J Endocrinol*. 20191;241(1):R1–R33.
14. Davis C, Levitan RD, Kaplan AS, et al. Reward sensitivity and the D2 dopamine receptor gene: A case-control study of binge eating disorder. *Prog Neuropsychopharmacol Biol Psychiatry*. 2008;32(3):620–628.
15. Cătoi AF, Iancu M, Pârvu AE, et al. Relationship between 25 hydroxyvitamin D, overweight/obesity status, pro-inflammatory and oxidative stress markers in patients with type 2 diabetes: A simplified empirical path model. *Nutrients*. 2021;13(8):2889.
16. Hao Y, Ma X, Shen Y, et al. Associations of serum 25-hydroxyvitamin D3 levels with visceral adipose tissue in Chinese men with normal glucose tolerance. *PLoS One*. 2014;9(1):e86773.
17. Kim D, Kim J. Association between serum 25-hydroxyvitamin D levels and adiposity measurements in the general Korean population. *Nutr Res Pract*. 2016;10(2):206–211.
18. Islam MS, Wei P, Suzauddula M, et al. The interplay of factors in metabolic syndrome: understanding its roots and complexity. *Mol Med*. 2024;30(1):279.
19. Hu XB, Duan TT, Liu J, et al. Effect of vitamin D supplementation on pancreatic β-cell destruction and type 1 diabetes. *Chin Med J (Engl)*. 2020;134(1):41–43.
20. Szymczak-Pajor I, Śliwińska A. Analysis of association between vitamin D deficiency and insulin resistance. *Nutrients*. 2019;11(4):794.
21. Martín Pérez IM, Martín Pérez SE. Assessment of ultra-processed food consumption and pro-inflammatory dietary load in adults diagnosed with fibromyalgia syndrome: A case-control study. *Nutr Health*. 2025:2601060251369916.
22. Gherasim A, Arhire LI, Niță O, et al. The relationship between lifestyle components and dietary patterns. *Proc Nutr Soc*. 2020;79(3):311–323.
23. Nasrallah M, Abu Helwa A, Jawhar NY, et al. Assessing the effect of screen time on physical activity in children based on parent-reported data: a cross-sectional study. *Cureus*. 2025;17(4):e82971.
24. Goyal J, Rakhra G. Sedentarism and chronic health problems. *Korean J Fam Med*. 2024;45(5):239–257.
25. Westbury S, Oyebode O, van Rens T, et al. Obesity stigma: causes, consequences, and potential solutions. *Curr Obes Rep*. 2023;12(1):10–23.
26. Pechey R, Monsivais P. Socioeconomic inequalities in the healthiness of food choices: Exploring the contributions of food expenditures. *Prev Med*. 2016;88:203–209.
27. Zhang Y, Shen T, Wang S. Progression from prediabetes to type 2 diabetes mellitus induced by overnutrition. *Hormones (Athens)*. 2022;21(4):591–597.
28. Weiner A, Zhang M, Ren S, et al. Progression from prediabetes to type 2 diabetes mellitus in adolescents: a real world experience. *Front Clin Diabetes Healthc*. 2023;4:1181729.
29. Liu C, Chen MS, Yu H. The relationship between obstructive sleep apnea and obesity hypoventilation syndrome: a systematic review and meta-analysis. *Oncotarget*. 2017;8(54):93168–93178.
30. Novais EN, Movahhedi M, Pradhan P, et al. Impact of childhood obesity on capital femoral epiphysis morphology: A large-scale, automated 3d-ct study and potential implications for SCFE pathogenesis. *J Bone Joint Surg Am*. 2025;107(18):2102–2109.
31. Ozeke O, Ozer C, Gungor M, et al. Chronic intermittent hypoxia caused by obstructive sleep apnea may play an important role in explaining the morbidity-mortality paradox of obesity. *Med Hypotheses*. 2011;76(1):61–63.
32. Badr M, El-Rabaa G, Freiha M, et al. Endocrine consequences of childhood obesity: a narrative review. *Front Endocrinol (Lausanne)*. 2025;16:1584861.
33. Pomahacova R, Paterova P, Nykodymova E, et al. Overweight and obesity in children and adolescents with endocrine disorders. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2023;167(4):328–334.
34. Garver WS, Newman SB, Gonzales-Pacheco DM, et al. The genetics of childhood obesity and interaction with dietary macronutrients. *Genes Nutr*. 2013;8(3):271–287.
35. Cha S, Koo I, Park BL, et al. Genetic effects of FTO and MC4R polymorphisms on body mass in constitutional types. *Evid Based Complement Alternat Med*. 2011;2011:106390.
36. Yang Y, Gao X, Tao X, et al. Combined effect of FTO and MC4R gene polymorphisms on obesity in children and adolescents in Northwest China: a case-control study. *Asia Pac J Clin Nutr*. 2019;28(1):177–182.
37. Steiner AM, Roscoe RF Jr, Booze RM, et al. Motivational dysregulation with melanocortin 4 receptor haploinsufficiency. *NeuroImmune Pharm Ther*. 2024;3(3-4):237–250.
38. Argano C, Mirarchi L, Amodeo S, et al. The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: State of the art. *Int J Mol Sci*. 2023;24(20):15485.
39. Rafiq S, Jeppesen PB. Vitamin D deficiency is inversely associated with homeostatic model assessment of insulin resistance. *Nutrients*. 2021;13(12):4358.
40. Meng X, Yang X, Lu H, et al. Vitamin D and its regulatory role in pancreatic β-cell function: implications for diabetes. *Diabetes Metab Syndr Obes*. 2025;18:4741–4754.
41. Szymczak-Pajor I, Drzewoski J, Śliwińska A. The molecular mechanisms by which Vitamin D prevents insulin resistance and associated disorders. *Int J Mol Sci*. 2020;21(18):6644.
42. Shan Y, Zhang S, Gao B, et al. Adipose tissue SIRT1 regulates insulin sensitizing and anti-inflammatory effects of berberine. *Front Pharmacol*. 2020;11:591227.
43. Cho KW, Lumeng CN. SirT1: a guardian at the gates of adipose tissue inflammation. *Diabetes*. 2011;60(12):3100–3102.
44. Taghizadeh N, Mohammadi S, Yousefi Z, et al. Assessment of global histone acetylation in pediatric and adolescent obesity: Correlations with SIRT1 expression and metabolic-inflammatory profiles. *PLoS One*. 2023;18(10):e0293217.
45. Fedorczak A, Lewiński A, Stawerska R. Sirtuin 1 serum concentration in healthy children - dependence on sex, age, stage of puberty, body weight and diet. *Front Endocrinol (Lausanne)*. 2024;15:1356612.
46. Zhu Z, Cao F, Li X. Epigenetic programming and fetal metabolic programming. *Front Endocrinol (Lausanne)*. 2019;10:764.
47. Bianco-Miotto T, Craig JM, Gasser YP, et al. Epigenetics and DOHaD: from basics to birth and beyond. *J Dev Orig Health Dis*. 2017;8(5):513–519.

Citation: Voroncova TO, Zubnina YO, Khlibovska OI, et al . Multifactorial architecture of pediatric obesity: from genetic susceptibility to systemic metabolic dysfunction. *Endocrinol Metab Int J*. 2026;14(1):10–14. DOI: 10.15406/emij.2026.14.00383

48. Gabory A, Attig L, Junien C. Developmental programming and epigenetics. *Am J Clin Nutr*. 2011;94(6 Suppl):1943S–1952S.

49. Daubenmier J, Kristeller J, Hecht FM, et al. Mindfulness intervention for stress eating to reduce cortisol and abdominal fat among overweight and obese women: An exploratory randomized controlled study. *J Obes*. 2011;2011:651936.

50. Scott KA, Melhorn SJ, Sakai RR. Effects of chronic social stress on obesity. *Curr Obes Rep*. 2012;1(1):16–25.

Citation: Voroncova TO, Zubnina YO, Khlibovska OI, et al . Multifactorial architecture of pediatric obesity: from genetic susceptibility to systemic metabolic dysfunction. *Endocrinol Metab Int J*. 2026;14(1):10–14. DOI: 10.15406/emij.2026.14.00383