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MATERNAL OBESITY AS A DETERMINANT OF LONG-TERM OFFSPRING HEALTH: INSIGHTS FROM METABOLIC, CARDIOVASCULAR, AND NEURODEVELOPMENTAL PERSPECTIVES

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ABSTRACT

Introduction and Objective: The prevalence of maternal overweight and obesity has increased dramatically worldwide, posing significant risks for offspring health. Maternal obesity has been associated with multisystemic consequences, including metabolic, cardiovascular, neuroendocrine, renal, and immune disturbances. This review aims to synthesize current evidence on the impact of maternal obesity on offspring health and to identify potential mechanisms underlying these effects.
Methods: A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science for studies published up to 2026. Both human epidemiological studies and experimental animal models were included to evaluate the effects of maternal obesity on offspring outcomes. Keywords included “maternal obesity”, “offspring health”, “developmental programming”, “metabolic disorders”, “neurodevelopment”, and “immunology”. Relevant studies were critically assessed, with particular focus on mechanisms, sex-specific effects, and long-term consequences.
Results / Methodology: Evidence indicates that maternal obesity programs offspring health through multiple pathways, including altered metabolism, epigenetic modifications, inflammatory processes, and microbiome-mediated effects. Offspring of obese mothers exhibit higher risk of hypertension, insulin resistance, type 2 diabetes, dyslipidemia, impaired renal development, neurodevelopmental and behavioral disorders, and immune dysregulation. These effects are often sex-specific and may persist throughout childhood and adulthood. Both preconception and gestational interventions, including weight management and lifestyle optimization, can partially mitigate these risks.
Conclusion: Maternal obesity is a major determinant of long-term multisystem health outcomes in offspring. Understanding the mechanisms of developmental programming is essential for developing preventive strategies, emphasizing the importance of maternal health optimization before and during pregnancy.

KEYWORDS

Maternal Obesity, Offspring Health, Developmental Programming, Metabolic Disorders, Neurodevelopment, Immunology

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Introduction

A vast increase in the occurrence of overweight or obesity among reproductive-age women poses a challenge in developed and even developing societies (1). In the United States, it is estimated that 25-29% of women of reproductive age have obesity, and together with being overweight, it affects up to 2/3 of women in the USA (1, 2, 3). A multitude of studies, both in humans and using animal models, have confirmed the negative impact of excessive maternal weight on her offspring. Animal models using rodents, such as mice and rats, constitute an extremely valuable and irrefutable source of knowledge about the pathogenesis of these processes (2). Studies have established that complications in children are related not only to the mother's initial overweight or obesity in the preconception period, but also to excessive gestational weight gain (4). Unfortunately, the exact mechanisms responsible for the association between maternal body weight and complications in her offspring remain unclear (5). Some studies indicate intrauterine reprogramming of the autonomic nervous system and metabolism, which leads to the development of cardiovascular diseases, hypertension, and lipid disorders later in life (3, 6). One of the concepts attempting to explain the link between maternal obesity and metabolic disorders in her offspring is the developmental overnutrition hypothesis. It suggests that increased concentrations of glucose, fatty acids, and amino acids in the mother's blood contribute to impaired development of the endocrine system, autonomic nervous system, and energy metabolism (7, 8).

Hypertension

The increase in childhood blood pressure, especially systolic blood pressure, at the level of 1-2 mmHg contributes to the much higher risk of cardiovascular incidents (9). There is evidence indicating the presence of prenatal factors of maternal origin leading to hypertension later in the offspring's life. One of the confirmed factors is maternal obesity, which affects both systolic and diastolic pressure (4,9), however the link between maternal overweight and offspring's increased blood pressure etiology remains vague (5). It is suggested that the metabolism of obese mother may change the central regulatory pathways controlling the blood pressure of the child. Supposedly the excessive production of leptin plays the great role - it takes part in the central control of appetite, activates the efferent sympathetic nervous system and is involved in the hypothalamus development. The change in leptin secretion leads to the impaired structure and dysfunction of the hypothalamus (4). Other studies using a rat model suggest that the structural abnormalities in vessels of obese mothers' offsprings comprise endothelial cells dysfunction and impaired dilatation of the vessels (6, 7, 10). The great vessels, such as the aorta, show the reduced possibility of endothelium-dependent relaxation (11). The fact that gestational weight gain contributes to offsprings' excessive body fat and birth weight is of great significance in the pathophysiology of increased blood pressure (12). The study using a mouse model showed not only the increased risk of hypertension but impaired glucose tolerance as well. In this study researchers assessed metabolic functions, adipose cell functions and adipose gene expression of 3 and 6-month-old mice. It turned out that the mean night- time systolic blood pressure was much higher in those mice of obese mothers compared to the control group. It is worth highlighting that the mean night diastolic blood pressure was increased only in males (2). Many studies have shown so far the positive association with maternal body mass index and increased blood pressure in humans (13). In 2010, researchers from the United States confirmed the link between prepregnancy maternal obesity and higher blood pressure in their children by examining 30461 pairs mather-child. In this study both low and excessive weight gain during pregnancy presumably does not affect childhood SBP The research also pointed that both genetic and environmental factors as well must be taken into consideration and it needs additional explorations (5). In 2010, the study conducted in Southern

Brazil indicated positive correlations between maternal anthropometric variables, such as prepregnancy weight or BMI and BP of their 11-year-old children. The research clearly showed the increase of SBP/DBP both in male and female offsprings at the level of 3/2,4 mmHG and 4,5/3,5, respectively (9). It is supported by another study showing the positive correlation between prepregnancy obesity in females and increased blood pressure in their offsprings at age of 5-6 (3). The researchers take notice that distortion in the form of familial weight gain propensity and the higher body mass index in children must be taken into consideration (2, 3).

Diabetes

The rise of diabetes prevalence among citizens of developed countries is highly associated with maternal glucose intolerance during pregnancy which is significantly correlated with increased weight and limited physical activity of females (14). Diabetes type 2 is a long-term, confirmed complication of maternal obesity. The influence of mother obesity on diabetes type 1 remains vague and needs more research (15). The hormonal profile during pregnancy changes significantly. The insulin signaling is highly impacted by hormones, such as progesterone and cortisol, especially in the second trimester. The altered metabolism of the mother is crucial for the foetus development however it leads to boosting insulin resistance. Finally, insulin sensitivity decreases by 50-70% (16). In obese women the process is exacerbated by oxidative stress and inflammatory cytokines, such as Il-1alfa, Il-1beta, Il-6, Il-8 and TNF, among others (17). There may be a 50% surge in the plasma concentration of Il-6 in comparison to women of normal weight (18). The study is supported by other research using a rodent model. The exposure to Il-6 of male offspring contributed to reduced insulin sensitivity. It also resulted in the higher level of leptin and the increased abdominal fat depot, whereas the changes in female offspring metabolism were not of statistical significance (19).

Obese mothers are also at risk of perturbed metabolism, including gestational diabetes mellitus (GDM) (18). The risk is directly proportional to body mass index. In general, the probability of GDM occurrence is about 3-fold higher in females of BMI >30 compared to those of <20 (20, 21). The potential mechanism of decreased insulin sensitivity may comprise of a higher concentration of triglycerides and non-estrified fatty acids in plasma and a decreased level of adiponectin (20) The exact mechanism is not yet discovered but it is known that insulin does not cross to the foetus blood through the placenta. Insulin is highly involved in the trophoblast development in early gestation – it has potential implications for the later placental structure and its functions (22). Hyperinsulinaemia in pregnant women is regarded as the most important factor contributing to hyperinsulinaemia in foetus and thus to insulin resistance (23). In the histopathology examination hypertrophy and hyperplasia of pancreatic islets with the surge in beta-cells was observed (24). The study conducted in 2020 found increased levels of insulin and HOMA-IR in new born children of obese mothers (25).

The huge research conducted on 23316 blinded participants reported that high body mass index of the mothers contributes to hyperglycaemia in their child, independent of maternal glucose level. The study also showed that excessive maternal body weight can lead to other potential complications, such as birthweight >90th percentile, body fat percentage >90th percentile and pre-eclampsia (26). Another large study, conducted in Scotland, included 118 201 participants whose mothers' BMI was obtained during pregnancy. The data showed that maternal obesity was associated with a more frequent occurrence of diabetes type 2 in the offspring, especially in males (15). The study is in accord with the findings of the Helsinki Birth Cohort Study. In the research, including 13 345 participants, both men and women, death due to cancer, cancer incidence, coronary heart disease, cardiovascular disease, stroke, diabetes and all-cause mortality were assessed according to mothers' body mass index. Each of seven studied conditions was positively associated with the mother's BMI. Obesity had the highest impact on cardiovascular outcomes in both men and women. Women seemed to be more vulnerable to develop diabetes than men (27).

Dyslipidemia

Changes in the plasma lipid profile of pregnant women is a physiological process consisting of a prominent increase in plasma triglyceride and plasma cholesterol – both high-density lipoprotein (HDL) and low-density lipoprotein (LDL). Maternal LDL and HDL are transported to the fetus circulation through the trophoblast and the endothelial cells (28). During the first and the second trimester, which is an anabolic period for pregnant females, lipogenesis accelerates, whereas in the third trimester it significantly decreases (catabolic phase) (16, 27, 29). The adipose tissue excessive depot produces a great amount of proinflammatory factors leading to chronic inflammation. It contributes to high concentration of reactive oxygen species (ROS), inflammatory cytokines and profibrotic growth factors. It affects both embryo and foetus development and contributes to long-term complications of the offspring as well (30, 31). Leptin, free fatty acids and cytokines affect endometrium structure and function and ultimately lead to infertility. Additionally, both leptin and insulin as well may affect regulatory pathways of adipocyte proliferation and differentiation (4). The proinflammatory cytokines, glucose and lipid levels of maternal origin may contribute to the so-called 'developmental overnutrition hypothesis’. The constant excessive supply from overnutrited or obese mothers may lead to long-term changes in child metabolism, appetite regulatory pathways and behaviour (16). The study using female rodent models feeding with excessive amounts of sugars and fat showed the changes in plasma concentration of lipids in their offsprings. In 3-month-old mice there was an elevated level of plasma triglyceride and leptin. In the 3 month period of time, the level of leptin remained constant, whereas triglyceride did not differ from the control group. Cholesterol was also increased. The hypertrophy of abdominal cells was present in offspring of obese dams. What is interesting, the activity of both male and female mice was lower in comparison to the control group, which enhanced lipid metabolism disorders (2). Another study conducted in USA showed that obesity during pregnancy affects lipid profiles in both maternal and fetal plasma. The study found that the number of LDL receptors (LDLR) are highly decreased in the placenta of the male fetus resulting in long-term disturbances, such as hyperglycemia, atherosclerosis and renal failure. There were no sex-dependent differences in plasma lipids or insulin sensitivity (25, 32).

The impact of maternal obesity to offspring’s urinary tract

The rapidly growing obesity in developed societies is reflected in the increase in complications in newborns of obese or overweight mothers, which also applies to renal health (33). The process is intensified by diseases accompanying maternal obesity (BMI ≥ 30 kg/m2), such as type II diabetes or hypertension (33), prebirth and, later in life, both bad eating habits of the family and foods rich in saturated animal fats (34, 35). The research clearly suggests that even maternal overweight (BMI 25-30 kg/m2), contributes to impaired renal development and chronic kidney disease (CKD) later in an offspring's life. In the study, Hsu et al. observed the risk of CKD increases by 24% in overweight mothers and 26% in obese mothers (36). This observation can be confirmed using an animal model using female rodents fed a high-fat diet. The renal abnormalities demonstrated included increased levels of reactive oxidative species (ROS) leading to oxidative stress, and the presence of proinflammatory cytokines such as monocyte chemoattractant protein-1 (MCP-1), IL-6, and tumor growth factor β (TGF-β) (35, 37). The inflammatory process was also mediated by cells such as monocytes/macrophages, which, through their influx into the renal interstitial tissue under the influence of cytokines such as MCP-1, caused inflammation, tubulointerstitial fibrogenesis, and tubular atrophy (37, 38). The histological picture shows thickening of the glomerular basement membrane, cell proliferation in the mesangium, matrix expansion and glomerulosclerosis, both focal and segmental (39). A significant increase in the production of type IV collagen and fibronectin in the interstitial tissue was demonstrated in a mouse model study. This resulted in renal dysfunction, which in turn manifested itself through increased serum creatinine and albuminuria (40). A study by Macumber et al. showed that children with congenital abnormalities of the kidney and urinary tract (CAKUT) were 1.24 times more likely to have an obese mother than those without CAKUT. Furthermore, the increased likelihood of having a child with CAKUT was found to depend on the severity of maternal obesity. The study found that excessive preconceptional weight gain in mothers of normal weight was not associated with the development of CAKUT, suggesting that factors contributing to impaired renal development occur early in pregnancy or even prepregnancy (41). Interestingly, this only applies to the upper urinary tract (ren, ureter), the lower urinary tract (bladder, urethra) is not affected (41).

Hypothalamic–pituitary–adrenal (HPA) axis

The increasing prevalence of obesity in populations is associated with an increase in long-term health consequences in offspring, including not only metabolic disorders but also neuroendocrine dysregulation. A growing body of evidence suggests that maternal obesity may influence the programming of the fetal hypothalamic-pituitary-adrenal (HPA) axis, thereby altering stress reactivity and increasing susceptibility to various disorders later in life (42). The HPA axis plays a crucial role in regulating the body's response to stress. Its activation occurs in response to various stressors, including psychosocial stress, hunger, temperature fluctuations, infections, and physical injuries (43). Subsequently, this leads to the synthesis and secretion of ACTH (Adrenocorticotropic hormone) from the pituitary gland. ACTH then stimulates the adrenal cortex to produce cortisol, a hormone that plays a central role in the physiological stress response and in the regulation of metabolic processes. (44). In healthy individuals, elevated cortisol levels inhibit the further secretion of CRH (corticotropin-releasing hormone) and ACTH through a negative feedback mechanism. This mechanism is crucial for regulating the body's response to stress (45). Under normal physiological conditions, the placenta acts as a protective barrier that limits excessive fetal exposure to cortisol. This protection is mediated by the enzyme 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2), which is highly expressed in the placenta and converts biologically active cortisol into its inactive form, cortisone. By inactivating maternal glucocorticoids, this enzyme acts as a functional barrier that limits fetal exposure to excessive cortisol levels and thereby protects the developing fetus from potential adverse effects of glucocorticoid overexposure. However, a higher body mass index (BMI) in the mother is associated with reduced activity of this enzyme in the placenta, resulting in increased cortisol transfer to the fetal circulation. Chronic exposure of the fetus to elevated levels of glucocorticoids may adversely affect the processes of neurogenesis, neuron differentiation and myelination in the developing brain (42). A study conducted by Long et al. demonstrated that the offspring of ewes that were obese before and during pregnancy had higher basal concentrations of ACTH and cortisol compared to the offspring of ewes of normal body condition ($p < 0.05$). These results suggest that maternal obesity may affect the basal activity of the hypothalamic-pituitary-adrenal axis in offspring (46). In addition, female offspring appear to be more sensitive to prenatal stressors such as maternal obesity, particularly in the context of HPA axis reactivity, which may lead to emotional and metabolic disorders later in life. In males, on the other hand, changes in diurnal cortisol secretion are observed (47). Such differences in stress responses suggest that HPA axis programming may differ depending on sex, which may ultimately lead to different health consequences for offspring at different stages of their lives.

Neurodevelopment and Behavior

Neurodevelopmental disorders comprise a heterogeneous group of conditions affecting the development of the central nervous system and are typically characterised by impairments in motor function, language development, cognition, and behaviour (48). Increasing evidence suggests that maternal obesity may represent an important prenatal risk factor for these disorders, potentially influencing fetal neurodevelopment through multiple biological mechanisms. Maternal metabolic disturbances associated with obesity, including hyperinsulinaemia and hyperglycaemia, may disrupt leptin and insulin signalling pathways in the developing fetal brain, thereby impairing neuronal maturation and synaptic development. In addition, obesity-related systemic inflammation, characterised by elevated concentrations of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α), together with increased oxidative stress, may compromise placental function. These alterations may facilitate the transfer of inflammatory mediators and metabolic signals to the fetal circulation, potentially exerting adverse effects on brain development (49). Epidemiological evidence supports an association between maternal pre-pregnancy adiposity and an increased risk of neurodevelopmental disorders in offspring. A meta-analysis including 41 studies from eight developed countries reported that children born to mothers who were overweight before pregnancy had a 17% higher risk of neurodevelopmental disorders, whereas maternal obesity was associated with a 51% increased risk. However, the magnitude of these associations was somewhat weaker in studies of higher methodological quality, suggesting that residual confounding factors such as genetic background, shared environment, or maternal lifestyle may partially explain these findings (50,51). Maternal obesity has also been linked to subtle alterations in cognitive outcomes. Some studies indicate that maternal pre-pregnancy obesity may be more strongly associated with lower offspring IQ than paternal obesity, suggesting a potential intrauterine effect. Although the magnitude of this association appears relatively modest, maternal obesity prior to conception as well as excessive gestational weight gain may contribute to small but measurable reductions in cognitive performance during childhood (52). In addition to cognitive outcomes, maternal obesity has been associated with behavioural difficulties in offspring, including increased impulsivity and impairments in social functioning. Notably, both low and high maternal BMI values have been linked to less favourable behavioural outcomes in children, suggesting that deviations from optimal maternal nutritional status may adversely affect neurodevelopment (53). Large population-based cohort studies conducted in Denmark and Sweden, encompassing more than two million children, have further demonstrated a significant association between maternal pre-pregnancy BMI and the risk of autism spectrum disorder (ASD) in offspring. After adjustment for demographic characteristics, parental education, income, and parental psychiatric history, both low and high maternal BMI were associated with an increased risk of ASD. Specifically, elevated risk was observed in mothers with below-normal BMI (HR =1.16; 95% CI 1.06-1.27 for BMI 15) as well as in those with obesity (HR = 1.50; 95% CI 1.46-1.53 for BMI 30) (54). However, the relationship between parental adiposity and ASD risk may be complex. An analysis by Pål Surén and colleagues involving 92,909 children from the Norwegian MoBa cohort (Norwegian Mother, Father and Child Cohort Study) indicated that paternal obesity, rather than maternal obesity, was independently associated with an increased risk of ASD, including autistic disorder (OR = 1.73; 95% CI 1.07-2.82) and Asperger syndrome in older children (OR = 2.01; 95% CI 1.13-3.57). These findings suggest that genetic, epigenetic, or shared environmental mechanisms related to paternal obesity may also contribute to ASD risk (55). Maternal obesity prior to pregnancy has additionally been associated with an increased risk of emotional and behavioural problems in offspring, including internalising symptoms such as anxiety and depression, as well as externalising behaviours such as aggression and oppositional behaviour. These difficulties may emerge during childhood or adolescence and can negatively affect long-term psychosocial functioning (56). Furthermore, retrospective studies suggest that maternal obesity may be associated with an elevated risk of cerebral palsy in offspring, with higher maternal BMI linked to increased incidence of this disorder (57). Overall, although a consistent association between maternal obesity and adverse neurodevelopmental outcomes in offspring has been reported, the interpretation of these findings requires caution. The observed relationships likely reflect a complex interplay between intrauterine biological mechanisms, genetic susceptibility, and postnatal environmental factors. Further longitudinal and mechanistic studies are therefore required to clarify the causal pathways linking maternal obesity with neurodevelopmental disorders.

Future Risk of Obesity in Offspring

Maternal obesity is not limited to perinatal risks but also shapes the metabolic trajectory of the child. Disturbances in the intrauterine environment can lead to both macrosomia and abnormal fetal growth, activating metabolic programming mechanisms. As a result, offspring become more susceptible to obesity, disorders of carbohydrate metabolism, and cardiovascular disease later in life, particularly with rapid weight gain in early childhood (58). Genetic predisposition plays a significant role in obesity, particularly in individuals with overweight, arising from both monogenic mutations and multiple polygenic variants of small effect. The complexity of these factors makes it challenging to prevent or treat obesity solely based on genetic information (59). Maternal obesity affects the risk of obesity in children not only through the inheritance of genetic variants regulating appetite and energy metabolism-which can increase preferences for fats and simple carbohydrates, reduce energy expenditure, and promote fat storage-but also through the family environment, which may further reinforce these predispositions (60). During pregnancy, maternal obesity is associated with higher risks of complications, such as pre-eclampsia and gestational diabetes, as well as increased birth weight in children. The risk of cesarean section due to cephalopelvic disproportion or lack of labor progress is six times higher in obese women compared with women of normal weight. Thus, maternal obesity influences child health both through direct physiological mechanisms and indirectly through increased fetal weight (61). Evidence indicates that overweight and obese mothers are associated with a higher risk of obesity in daughters compared with sons and with higher body composition values in girls. These differences may result from sex-specific mechanisms predisposing to obesity, including differences in pubertal timing and energy requirements (62,63). Maternal obesity during pregnancy increases the risk of overweight, obesity, and metabolic disorders in children, and these effects persist throughout childhood, adolescence, and adulthood. Breastfeeding during the first six months of life confers significant benefits: it partially reduces the risk of overweight and supports beneficial metabolic programming, although it cannot fully offset the effects of high maternal BMI during pregnancy. Early fetal exposure to maternal overweight may induce permanent metabolic changes, whereas breastfeeding may mitigate some of these effects by modifying the bioactive components of milk and supporting child development (64). Modifiable lifestyle factors can significantly reduce the risk of obesity, even in individuals with high genetic predisposition. People who maintain a healthy lifestyle-balanced diet, regular physical activity, adequate sleep, and limited sedentary behavior and alcohol consumption-exhibit lower risk of obesity and obesity-related diseases, regardless of polygenic risk score. These findings suggest that promoting a healthy lifestyle is an effective preventive strategy, even for genetically predisposed individuals (65). In conclusion, maternal obesity during pregnancy may have long-lasting consequences for the health of offspring, increasing their susceptibility to overweight and obesity later in life.

Immune and Inflammatory Consequences

Maternal obesity increases the risk of long-term disorders in offspring, extending beyond perinatal complications. Metabolic and pro-inflammatory changes in the placenta may lead to epigenetic reprogramming of fetal immune cells, including monocytes and CD4+ T lymphocytes. As a result, offspring become more susceptible to chronic inflammation, metabolic disturbances, and immune dysfunction later in life (66). Additionally, excessive exposure to maternal lipids and elevated leptin levels affects cellular metabolism, mitochondrial function, and the fate of hematopoietic precursor cells. Epigenetic mechanisms, such as histone modifications, DNA methylation, and microRNAs, further modulate the expression of genes crucial for immune function (67). Consequently, offspring are rendered more vulnerable to chronic inflammatory states, metabolic disorders, and immune dysregulation in later life. Increasing evidence indicates the clinical consequences of these mechanisms: maternal obesity is associated with a higher risk of asthma in children. In a study of 95,723 mother–child pairs from the Kaiser Permanente system in Northern California, the risk of asthma at ages 4, 6, and 8 increased with higher maternal pre-pregnancy BMI (HR = 1.07 per 5 kg/m^2) (68). These findings suggest that immune and pro-inflammatory disturbances programmed during fetal development may manifest as atopic and autoimmune diseases during childhood and later in life. Furthermore, the maternal microbiome plays a key role in shaping the fetal and neonatal immune system (69). Maternal microorganisms constitute the primary source of neonatal gut colonization, and their interactions with the host influence immune development during the first days and months of life (70). The impact of the maternal microbiota and its metabolites on maternal and fetal immune function, the feedback between the microbiome and immune responses, and the role of milk components in modulating the microbiota and neonatal immunity remain poorly understood. Understanding these mechanisms is critical for elucidating how maternal obesity may contribute to the programming of chronic autoimmune and pro-inflammatory disorders in offspring, highlighting the need for further research in this area within the context of maternal and child health (71).

Discussion

Our review underscores that maternal obesity exerts a multisystemic and long-lasting impact on offspring health, encompassing metabolic, cardiovascular, renal, neuroendocrine, neurodevelopmental, and immunological domains. These findings provide strong support for the developmental overnutrition hypothesis, whereby excessive nutrient availability during critical windows of gestation programs offspring metabolism and organ function, predisposing them to chronic diseases later in life.

Importantly, our synthesis highlights that the effects of maternal obesity are highly context-dependent, varying according to the sex of the offspring, the tissue or organ system affected, and the timing of exposure during fetal development. For example, HPA axis dysregulation and neurobehavioral susceptibility appear particularly sensitive to early- to mid-gestational exposure, whereas metabolic disturbances such as insulin resistance and obesity often manifest during childhood or adolescence. These temporal differences underscore the need for longitudinal studies that track offspring outcomes across multiple developmental stages, rather than relying solely on cross-sectional or single-timepoint analyses. Our review also emphasizes the critical role of epigenetic and microbiome-mediated mechanisms. Alterations in DNA methylation, histone modifications, and microRNA expression, along with maternal microbiome-driven immune programming, likely contribute to long-lasting changes in metabolic and immunological function. These findings point to the possibility that early-life interventions -including maternal nutrition optimization, microbiome modulation, and targeted lifestyle support - may mitigate some of the intergenerational risks of obesity. Despite robust evidence linking maternal obesity to metabolic and cardiovascular outcomes, there remains a substantial gap in integrative understanding of its effects on neuroendocrine, neurodevelopmental, and immune systems within a single conceptual framework. Existing studies are often limited by cohort specificity, small sample sizes, or reliance on animal models, which constrains the generalizability of findings to diverse human populations. Furthermore, disentangling genetic, epigenetic, and environmental contributions remains a challenge, highlighting the need for multidisciplinary, mechanistic, and population-based research. From a public health perspective, our review reinforces that preconception and prenatal interventions offer enormous preventive potential. Promoting a healthy BMI before conception, monitoring and managing gestational weight gain, and supporting maternal diet quality and physical activity may partially counteract adverse metabolic and neuroendocrine programming. Additionally, strategies such as breastfeeding promotion and early-life lifestyle interventions could provide further protection, even in the context of high maternal BMI. Taken together, these observations highlight the intergenerational consequences of maternal obesity and the critical importance of holistic maternal health strategies for optimizing offspring outcomes. Finally, the synthesis of current evidence points toward a paradigm shift in maternal-fetal medicine: rather than focusing solely on short-term pregnancy outcomes, clinicians and researchers should consider long-term multisystemic trajectories in offspring. By integrating insights from epidemiology, animal models, epigenetics, and microbiome research, future studies can better inform precision interventions aimed at breaking the cycle of obesity and metabolic disease across generations.

Conclusions

Maternal obesity is a key factor in determining the health of offspring, influencing metabolic, cardiovascular, neuroendocrine and immunological development. These effects vary by sex, highlighting the need for research that takes into account the biological differences between boys and girls. Epigenetic mechanisms and microbiological modulations represent potential targets for intervention, and understanding them may help reduce the risk of chronic diseases in children. The literature also highlights the importance of pre-pregnancy preventive measures, including weight control, a healthy diet and physical activity. Further integrative research is needed to assess the long-term impact of maternal obesity on various systems in offspring, taking into account genetic and environmental factors.

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