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Review Article

Maternal Obesity, Metabolic Syndrome, and Long-Term Effects on Offspring: A Developmental Origins of Health and Disease (DOHaD) Perspective

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ABSTRACT

Introduction: *The global prevalence of obesity among women of reproductive age has increased markedly over the past two decades, reaching 24% in high-income and 16% in low- and middle-income countries. This trend contributes to adverse maternal and neonatal outcomes, including a 2–4-fold higher risk of gestational diabetes, a threefold increase in preeclampsia, and up to a 50% higher caesarean delivery rate. Maternal metabolic disorders also have long-term consequences for offspring metabolic and cardiovascular health.* ***Objective:*** *To synthesize current biological and epidemiological evidence linking maternal metabolic status to offspring health within the Developmental Origins of Health and Disease (DOHaD) framework.* ***Methods:*** *A comprehensive search was conducted in PubMed, Scopus, and Web of Science from January 2015 to September 2025 using keywords related to maternal obesity, metabolic syndrome, DOHaD, offspring health, and epigenetics. Included studies comprised human and animal research with follow-up beyond two years, while case reports, editorials, and studies without long-term outcomes were excluded.* ***Findings:*** *From 22 eligible studies, consistent evidence indicates that maternal*

obesity and metabolic syndrome affect offspring through placental dysfunction, systemic inflammation, and epigenetic reprogramming. Offspring of obese mothers show a 1.5–2.8-fold increased risk of obesity and insulin resistance during adolescence and adulthood. Epigenome-wide analyses reveal altered methylation of genes regulating lipid metabolism (LEP, IGF2) and inflammatory pathways, supporting the DOHaD hypothesis. ***Conclusion:*** *Maternal metabolic health critically shapes offspring developmental and metabolic outcomes. Preconception and antenatal interventions targeting metabolic optimization provide key opportunities to prevent intergenerational transmission of metabolic risk and promote healthier future generations.*

1. INTRODUCTION

The prevalence of obesity among women of reproductive age has escalated globally, contributing to an increased risk for maternal and neonatal complications. The World Health Organization (WHO) estimates that more than 24% of pregnant women worldwide are obese, with the highest proportions observed in high-income and rapidly urbanizing countries, where prevalence rates have increased by nearly 40% over the past two decades[1]. This upward trend poses significant public health challenges, as obesity during pregnancy is not only associated with gestational diabetes mellitus (GDM), preeclampsia, and caesarean delivery, but also exerts long-lasting effects on the child's metabolic, cardiovascular, and neurodevelopmental health [2].

The Developmental Origins of Health and Disease (DOHaD) hypothesis posits that environmental and nutritional exposures during critical windows of development—particularly in utero—can permanently influence gene expression, organ structure, and physiological function [3]. Within this framework, maternal metabolic disorders, including obesity and metabolic syndrome (MetS), act as potent inducers of foetal programming, leading to increased susceptibility to obesity, type 2 diabetes, and hypertension in later life [4].

However, despite growing evidence, critical gaps remain in understanding how specific maternal metabolic profiles—such as insulin resistance, lipid dysregulation, and inflammatory markers—differentially affect offspring outcomes across populations and developmental stages. In addition, inconsistencies persist regarding the relative contribution of epigenetic mechanisms versus placental dysfunction in mediating these long-term effects. Furthermore, most available studies are concentrated in high-income countries, limiting the generalizability of findings to low- and middle-income settings, where the burden of maternal obesity is rapidly increasing.

The mechanisms underlying these intergenerational effects are multifactorial, encompassing placental dysfunction, epigenetic modifications, inflammatory signaling, oxidative stress, and altered nutrient transfer.[5] Understanding these pathways is critical for designing preventive interventions targeting maternal health and breaking the intergenerational cycle of metabolic disease.

Therefore, this review synthesizes recent biological and epidemiological evidence (2015–2025) linking maternal obesity and metabolic syndrome with long-term offspring outcomes through the DOHaD framework. By integrating mechanistic insights and population-level data, the review aims to clarify existing inconsistencies and identify priority areas for intervention and future research.

2. METHODS

2.1 Search Strategy and Selection Criteria

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science databases for studies published between January 2015 and September 2025. The following Boolean string was used to ensure comprehensive retrieval:
("maternal obesity" OR "maternal overweight" OR "metabolic syndrome" OR "MetS")
AND ("offspring health" OR "child health" OR "intergenerational effects")
AND ("developmental origins" OR "DOHaD" OR "foetal programming" OR "epigenetics")

Reference lists of eligible papers and relevant reviews were also hand-searched to identify additional studies. Inclusion criteria:

1) Human and animal studies examine the relationship between maternal obesity or metabolic syndrome and offspring health outcomes.
2) Studies addressing long-term outcomes (>2 years) related to metabolic, cardiovascular, or neurodevelopmental disorders.
3) Articles published in English and in peer-reviewed journals.

Exclusion criteria included case reports, editorials, conference abstracts, and studies lacking long-term outcome assessment were excluded.

In total, 1,247 records were retrieved across the three databases. After removing duplicates (n = 312), 935 abstracts were screened. Of these, 104 full-text articles were assessed for eligibility, and 22 studies meeting all inclusion criteria were included in the final synthesis. A PRISMA-style flow diagram was constructed to illustrate the screening and selection process and was displayed as Figure 1 to enhance transparency.

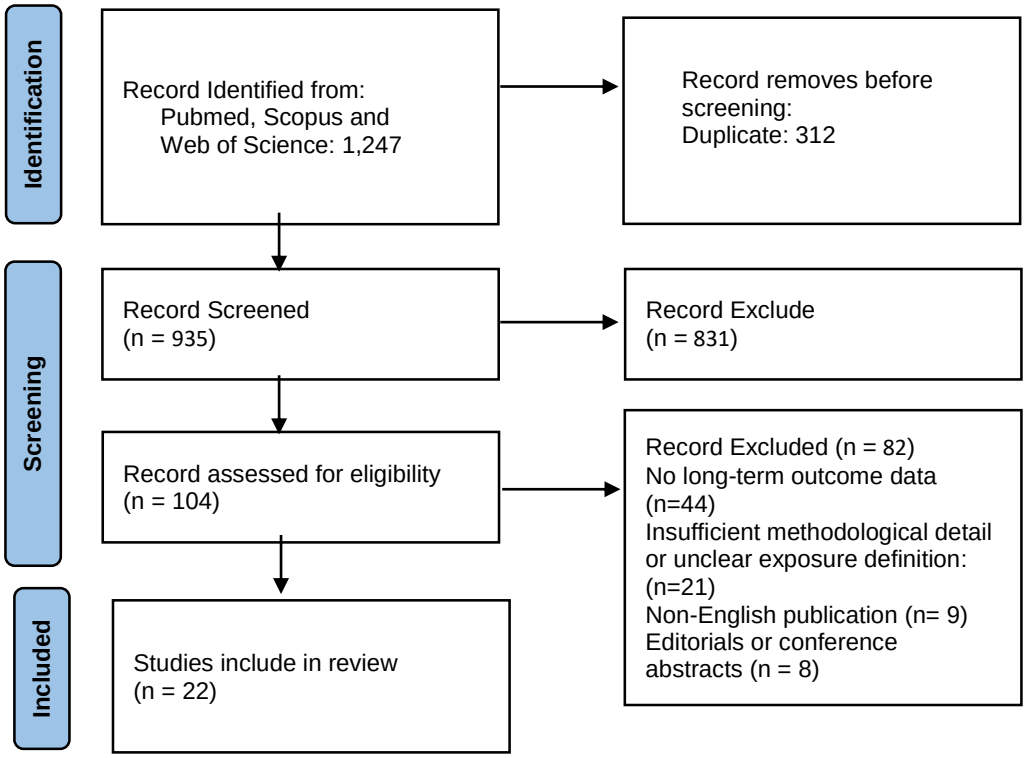

Figure 1. Flow diagram of study selection

2.2 Data Extraction and Synthesis

Two reviewers independently screened titles and abstracts, extracted data, and resolved discrepancies through discussion or consultation with a third reviewer. Extracted data included study design, sample size, exposure and outcome measures, follow-up duration, and key findings related to offspring metabolic, cardiovascular, and neurodevelopmental outcomes.

Given the heterogeneity in study designs and outcome measures, findings were synthesized narratively rather than quantitatively. Mechanistic studies were integrated with epidemiological data to construct a conceptual model linking maternal metabolic status, placental and epigenetic mechanisms, and offspring health trajectories.

3. RESULTS

3.1. Epidemiological Evidence Linking Maternal Obesity and Offspring Outcomes

Recent evidence consistently demonstrates that maternal obesity and metabolic syndrome (MetS) are strong predictors of adverse offspring outcomes, including increased adiposity, insulin resistance, and cardiometabolic dysfunction [2,6,7]. Large birth cohorts such as the *Avon Longitudinal Study of Parents and Children (ALSPAC)* and *Generation R* have shown a linear association between maternal pre-pregnancy body mass index (BMI) and child BMI, waist circumference, and systolic blood pressure extending into adolescence [8].

Maternal MetS—defined by central obesity, dyslipidemia, hypertension, and insulin resistance—exacerbates these intergenerational risks. In a pooled analysis of European and U.S. cohorts, offspring of mothers with MetS exhibited higher risks for

childhood obesity (adjusted odds ratio [aOR] 1.8–2.5) and metabolic syndrome (aOR 1.6–2.0) after adjustment for confounders such as maternal age, parity, socioeconomic status, and gestational diabetes [9]. Additionally, several studies reported associations with neurodevelopmental delays, suggesting broader developmental consequences beyond metabolic health [10–12].

Table 1. Summary of Key Studies on Maternal Obesity/Metabolic Syndrome and Long-Term Offspring Health

Study	Population	Exposure	Offspring Outcomes	Key Findings
Godfrey et al., 2017 [13]	UK Cohort (n = 6,500)	Maternal BMI (pre-pregnancy)	Adolescent adiposity	Higher maternal BMI associated with ↑ fat mass at age 15
Gaillard et al., 2015 [4]	Generation R (Netherlands)	Pre-pregnancy BMI	Cardiometabolic risk	Offspring showed ↑ blood pressure and insulin resistance at age 9
Catalano et al., 2018 [2]	U.S. multi-center study	Gestational diabetes mellitus (GDM) + maternal obesity	Childhood metabolic phenotype	Combined GDM and obesity amplified childhood insulin resistance
Perrine et al., 2021 [14]	France (n = 1,200)	Maternal BMI in third trimester	Placental gene expression and DNA methylation	↑ Leptin/adiponectin methylation → placental dysfunction → offspring metabolic dysregulation

Although most studies converge on the positive relationship between maternal obesity and offspring metabolic risk, the magnitude of association varies across populations and exposure definitions. For example, the Generation R cohort identified a moderate increase in child systolic BP with higher maternal BMI [4], whereas Catalano et al. [2] reported a more pronounced effect when obesity co-occurred with gestational diabetes. Differences in timing of exposure (pre-pregnancy vs. late gestation), ethnic composition, and postnatal environment partly explain these discrepancies. Nonetheless, the overall trend supports the Developmental Origins of Health and Disease (DOHaD) hypothesis, indicating that maternal metabolic status shapes offspring physiology through intrauterine and early-life mechanisms.

3.2 Mechanistic Insights: Biological Pathways in DOHaD Context

Maternal obesity disrupts intrauterine homeostasis, altering foetal developmental trajectories through multiple, interrelated biological mechanisms [15].

3.2.1 Placental Dysfunction

Obesity and MetS impair placental angiogenesis and nutrient transport, leading to foetal hyperinsulinaemia and increased adipose deposition [16]. Elevated TNF-α and IL-

6 concentrations in the placenta interfere with nutrient sensing via the mTOR and AMPK pathways, further amplifying metabolic stress [17].

3.2.2 Epigenetic Regulation

Epigenetic modifications, particularly DNA methylation and histone acetylation, mediate the transmission of metabolic risk. Hypermethylation at IGF2, LEP, and PPARγ loci has been associated with higher maternal BMI and subsequent offspring adiposity [10,18]. These alterations persist into childhood, suggesting stable metabolic reprogramming.

3.2.3 Oxidative Stress and Inflammation

Maternal obesity induces systemic oxidative stress, increasing reactive oxygen species (ROS) and lipid peroxidation, which damage placental tissue and disturb foetal redox balance [19]. Inflammatory cytokines crossing the placenta further reprogram foetal immune and metabolic pathways [20].

3.2.4 Gut Microbiota and Metabolomic Alterations

Emerging evidence highlights the role of the maternal gut microbiome in transmitting metabolic risk. Dysbiosis in obese mothers alters short-chain fatty acid (SCFA) profiles and increases lipopolysaccharide (LPS) exposure, affecting foetal immune maturation and metabolic homeostasis [21–23].

Table 2. Proposed Mechanistic Pathways Linking Maternal Obesity/MetS and Offspring Health (DOHaD Framework)

Mechanism	Biological Pathway	Representative Evidence	Consequences for Offspring
Placental dysfunction	↑ TNF-α, IL-6; mTOR/AMPK dysregulation	Myatt, 2006 [16]	Foetal hyperinsulinaemia, altered nutrient transfer
Epigenetic reprogramming	DNA methylation at IGF2, LEP, PPARγ loci	Godfrey et al., 2017 [13]	Persistent alterations in energy metabolism genes
Oxidative stress	ROS accumulation, lipid peroxidation	Burton & Fowden, 2020 [19]	Endothelial dysfunction, metabolic reprogramming
Maternal microbiota	Altered SCFA, ↑ LPS exposure	Calatayud et al., 2019 [23]	Foetal immune modulation, obesity risk

Collectively, these mechanisms illustrate how maternal metabolic dysregulation influences foetal growth and long-term health outcomes. While placental dysfunction and epigenetic reprogramming are consistently observed, emerging microbiome-based mechanisms offer new insights into intergenerational metabolic programming. However, heterogeneity in exposure measurement and postnatal confounders necessitates cautious interpretation and further mechanistic validation.

4. DISCUSSIONS

This narrative review integrates recent evidence demonstrating that maternal obesity and metabolic syndrome (MetS) exert profound and enduring effects on offspring health. These influences operate through intertwined biological, molecular, and environmental mechanisms, aligning with the Developmental Origins of Health and Disease (DOHaD) framework. The DOHaD paradigm conceptualizes the intrauterine environment as a “biological blueprint” that programs long-term metabolic trajectories and disease susceptibility. Thus, maternal metabolic health before and during pregnancy constitutes a pivotal determinant of intergenerational wellbeing, shaping population-level patterns of noncommunicable disease risk.

4.1 Intergenerational Transmission of Metabolic Risk

The reviewed studies highlight robust intergenerational associations between maternal metabolic status and offspring cardiometabolic profiles [24]. This persistence underscores the primacy of intrauterine metabolic programming rather than purely environmental transmission. Mechanistically, maternal hyperglycemia and dyslipidemia induce a nutrient-excess intrauterine milieu that modulates foetal insulin secretion, lipid metabolism, and vascular development. These processes foster adipocyte hypertrophy, β-cell dysfunction, and endothelial impairment, establishing a pro-inflammatory and insulin-resistant phenotype early in life.

Recent meta-analyses, including one published in The Lancet Diabetes & Endocrinology (2023), reinforce these associations, demonstrating that maternal pre-pregnancy obesity increases offspring risk of obesity by 2.3-fold and type 2 diabetes by 1.8-fold, independent of postnatal weight gain [25,26]. Notably, these studies also reveal sex-specific vulnerability: male offspring tend to exhibit greater insulin resistance and hepatic steatosis, while females show higher adiposity and altered leptin regulation [27,28]. Such dimorphic outcomes suggest differential epigenetic sensitivity to intrauterine metabolic stress, prompting a refinement of the classical DOHaD model to incorporate sex-dependent programming effects [26,28].

4.2 Epigenetic and Molecular Evidence

Epigenetic studies increasingly reveal that maternal metabolic disturbances leave persistent molecular imprints on offspring gene regulation. Altered methylation of LEP, PPARγ, and IGF2 genes has been identified in placental and cord blood samples of infants born to mothers with obesity or gestational diabetes, influencing pathways of adipogenesis and glucose metabolism. These findings extend the DOHaD framework by demonstrating that metabolic programming is not merely functional but also molecularly encoded [26,24].

Importantly, these modifications may be sex dependent. Evidence from recent cohorts (2022–2025) indicates that methylation changes in metabolic and inflammatory loci—such as NR3C1 and H19—are more pronounced in male fetuses, potentially explaining their higher cardiometabolic vulnerability in later life [27,28]. Conversely, female fetuses appear more resilient due to estrogen-mediated modulation of oxidative stress pathways and mitochondrial biogenesis [28]. Such findings advance the DOHaD model by suggesting that maternal obesity-induced epigenetic reprogramming interacts with foetal sex hormones and placental signaling, creating divergent adaptive responses [26,28].

Complementary animal studies further demonstrate that maternal high-fat diets alter hypothalamic appetite regulation, gut microbiota composition, and hepatic lipid metabolism in offspring [24,27]. Together, these insights highlight that the maternal metabolic milieu induces multi-organ reprogramming that transcends generational process better conceptualized as intergenerational metabolic imprinting rather than simple developmental adaptation.

4.3 Comparative Evidence and Synthesis

Synthesizing across meta-analyses and longitudinal cohorts, the consistency of evidence supports a dose–response relationship between maternal metabolic dysregulation and offspring cardiometabolic risk. The Generation R and ALSPAC cohorts show linear associations between maternal BMI and offspring BMI z-scores extending into adolescence, while systematic reviews (2023–2024) reveal that these associations persist after adjusting for paternal BMI and household environment [24,25]. This convergence of evidence validates the biological plausibility of intrauterine programming and reinforces the urgency of integrating maternal metabolic assessment into life-course public health frameworks.

4.4 Public Health and Policy Implications

The intergenerational nature of metabolic programming underscores the need for proactive, life-course–oriented interventions targeting women of reproductive age. Beyond general recommendations for optimizing preconception BMI and micronutrient intake, emerging frameworks emphasize preconception health optimization programs, integrating nutrition, metabolic screening, and behavioral counselling into routine primary care [25,26].

Integrating DOHaD-informed counselling—covering gestational weight management, metabolic risk screening, and tailored dietary guidance—within existing antenatal systems could substantially mitigate intergenerational disease transmission. Furthermore, addressing structural determinants such as urbanization, food insecurity, and healthcare inequities remain essential to sustainable progress. Interdisciplinary collaboration among obstetricians, pediatricians, nutritionists, and public health policymakers is imperative to translate molecular insights into actionable community-level interventions.

4.4 Limitations of Current Evidence

While current findings robustly link maternal metabolic health to offspring outcomes, several limitations constrain causal inference. Most available studies are observational in nature, subject to recall and selection bias, and often rely on self-reported maternal weight or non-standardized definitions of metabolic syndrome (MetS). Sample sizes are frequently modest, and follow-up durations are insufficient to capture long-term or multi-generational effects. Moreover, few studies adequately account for confounding by shared genetic, socioeconomic, or environmental factors, further limiting causal interpretation. Although this review did not conduct a formal risk of bias assessment, the synthesis highlights consistent methodological weaknesses across the literature.

Future research should therefore prioritize longitudinal and multi-generational cohort designs with large, diverse populations and standardized phenotyping. Integration of multi-omics approaches—including genomics, epigenomics, transcriptomics, metabolomics, and microbiome profiling—will be essential to elucidate biological pathways underlying intergenerational transmission. Additionally, well-controlled intervention trials are needed to determine whether optimizing maternal metabolic health before and during pregnancy can alter offspring risk trajectories. Identifying critical windows of developmental plasticity—when interventions exert the greatest benefit—will be pivotal for translating the Developmental Origins of Health and Disease (DOHaD) framework into effective public health strategies.

5. CONCLUSION

Maternal obesity and MetS are pivotal drivers of intergenerational metabolic vulnerability. Through intertwined physiological, molecular, and epigenetic pathways, they shape the developmental programming of offspring health and contribute to the global rise in metabolic diseases. Targeted preconception interventions and integrated maternal health programs could represent pivotal strategies to disrupt intergenerational transmission of metabolic risk, translating current evidence into actionable approaches that promote healthier future generations.

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Conflict of Interest Statement:

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.