
**International Journal of Innovative Technologies in Social Science**

e-ISSN: 2544-9435

**Operating Publisher**
**SciFormat Publishing Inc.**
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

| | |
|---|---|
| **ARTICLE TITLE** | THE INFLUENCE OF MATERNAL NUTRITION DURING PREGNANCY ON CHILDHOOD OBESITY RISK |
| **DOI** | https://doi.org/10.31435/ijitss.1(49).2026.4617 |
| **RECEIVED** | 09 January 2026 |
| **ACCEPTED** | 20 March 2026 |
| **PUBLISHED** | 25 March 2026 |
| **LICENSE** | The article is licensed under a **Creative Commons Attribution 4.0 International License.** |

# THE INFLUENCE OF MATERNAL NUTRITION DURING PREGNANCY ON CHILDHOOD OBESITY RISK

***Julia Kamińska*** *(Corresponding Author, Email: juliakam502@gmail.com)*
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0009-2162-0712*

***Karolina Strzelecka***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0007-9797-9815*

***Ewa Jagodzińska***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0008-4089-2708*

***Zuzanna Jabłońska***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0003-3592-879X*

***Maja Jabłońska***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0008-8498-0603*

***Antoni Plasota***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0006-1789-2094*

***Joanna Zygadło***
*Infant Jesus Clinical Hospital UCC MUW, Warsaw, Poland*
*ORCID ID: 0000-0001-5377-5254*

***Wiktor Milewczyk***
*Medical University of Lublin, Lublin, Poland*
*ORCID ID: 0009-0004-5539-5553*

***Wojciech Michalski***
*Medical University of Warsaw, Warsaw, Poland*
*ORCID ID: 0009-0000-2612-515X*

***Mikołaj Olczak***
*Samodzielny Publiczny Specjalistyczny Szpital Zachodni im. św. Jana Pawła II in Grodzisk Mazowiecki, Grodzisk Mazowiecki, Poland*
*ORCID ID: 0009-0006-2232-143X*

**ABSTRACT**

Childhood obesity is an urgent global concern. Growing evidence suggests its roots often extend back to prenatal development. This review examines how maternal nutrition influences the risk of childhood obesity through fetal metabolic programming. A literature search (October–December 2025) was conducted using PubMed, Scopus, Web of Science, and Google Scholar. It identified peer-reviewed studies on maternal diet, metabolic pathways, and long-term offspring health. Maternal macronutrient imbalances, such as excessive energy intake, high glycemic load, high saturated fat intake, or inadequate protein, were associated with altered fetal hormonal regulation. They also led to changes in placental nutrient transfer and epigenetic modifications. These changes affect insulin sensitivity and adipose tissue development. Micronutrient deficiencies, especially vitamin D, folate, and B12, were linked to disrupted metabolic signaling. They were also linked to adverse growth patterns. Maternal conditions, including obesity and gestational diabetes, further increased fetal susceptibility to obesity. This occurs by promoting hyperinsulinemia and adiposity before birth. Other factors, such as maternal genetics, gut microbiota, and early postnatal feeding, also interact with prenatal nutrition to shape long-term outcomes. The evidence highlights pregnancy as a key window for prevention. More longitudinal studies and biomarker-based dietary assessment are needed to support tailored nutritional guidance.

**KEYWORDS**

Obesity, Pregnancy, Nutrition

**CITATION**

Julia Kamińska, Karolina Strzelecka, Ewa Jagodzińska, Zuzanna Jabłońska, Maja Jabłońska, Antoni Plasota, Joanna Zygadło, Wiktor Milewczyk, Wojciech Michalski, Mikołaj Olczak. (2026) The Influence of Maternal Nutrition During Pregnancy on Childhood Obesity Risk. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.4617



## Introduction

Over the past two decades, childhood excess weight has emerged as a major global health challenge. A recent systematic review and meta-analysis by Zhang et al. (2024) synthesized data from 2033 studies across 154 countries. This included data from almost 46 million participants aged 18 or younger. The research revealed that 8.5% of children and adolescents were obese. Additionally, 22.2% were overweight or obese in the time period between 2012 and 2023. This means that almost one in five children globally has an excess body weight. These results mark a rise in obesity prevalence compared to the results from 2000 to 2011. Namely, obesity increased by about 1.5-fold. The same study showed that maternal weight status is strongly associated with obesity risk in offspring. Children born to mothers with obesity had a higher obesity prevalence (15.9%) compared with those born to mothers without obesity (8.1%). Additionally, birth weight showed a graded association. Obesity prevalence increased from low birth weight (6.2%) to normal (9.2%) and high birth weight (12.8%) [1]. These findings point to the importance of early developmental influences.

A growing body of research suggests that the intrauterine environment can shape long-term metabolic health. When conditions in the womb are suboptimal, they may alter fetal development and increase the likelihood of excess adiposity later in life. Such effects may arise through mechanisms including epigenetic modifications, changes in adipose tissue development, or altered regulation of hunger and satiety. Maternal metabolic disturbances, such as obesity or insulin resistance, may promote fetal overgrowth and increase susceptibility to postnatal weight gain. This risk is particularly relevant when combined with genetic susceptibility and postnatal environmental factors [2].

Beyond prevalence, children with obesity show a significantly higher risk of chronic conditions such as type 2 diabetes, hypertension, atherosclerosis, joint problems, and hypercholesterolemia [2]. They are also at higher risk for depression and asthma [1]. This emphasizes the need for early prevention. Among other factors, an appropriate maternal diet may be crucial for prevention, as it can induce metabolic and epigenetic changes in the fetus [3].

Considering this evidence and current global trends, this review aims to summarize the current research on how maternal nutrition during pregnancy influences the risk of childhood obesity through fetal metabolic programming. It examines the key mechanisms involved and the developmental periods most vulnerable. It also explores how different macronutrients and micronutrients shape metabolic outcomes. In addition, the review considers the effects of maternal conditions such as obesity and gestational diabetes, as well as overall dietary patterns, and discusses how factors such as genetics, maternal gut microbiota, and early postnatal influences interact with prenatal nutrition. Finally, it highlights the public health implications of these findings.

## Methods

*Study design and scope*

This narrative review summarizes current evidence on how maternal nutrition during pregnancy influences offspring obesity and metabolic outcomes. Human studies form the core of the review, complemented by mechanistic animal and experimental data when relevant to fetal programming pathways.

*Literature search strategy*

A structured search was conducted in PubMed, Scopus, Web of Science, and Google Scholar (October–December 2025). Search terms covered maternal diet, macronutrients, micronutrients, dietary patterns, gestational diabetes, maternal obesity, epigenetics, fetal programming, and childhood obesity. No publication-year limits were applied.

*Eligibility criteria*

We included peer-reviewed English-language studies examining associations between maternal nutritional exposures during pregnancy (energy/macronutrients, micronutrients, dietary patterns, obesity, metabolic syndrome, gestational diabetes) and offspring outcomes related to adiposity or metabolic health (birth size, fat mass, BMI, overweight/obesity, metabolic biomarkers). Cohort, case–control, interventional studies, and systematic reviews/meta-analyses were eligible. Mechanistic epigenetic and animal studies were used selectively to clarify biological pathways.

*Study selection and data extraction*

Titles and abstracts were screened, followed by full-text review. From each study, we extracted: design, population, exposure assessment, timing of exposure, outcome measures, effect estimates, and confounder adjustment. Mechanistic studies were reviewed for specific programming pathways (e.g. IGF2/H19 methylation, leptin signaling, placental nutrient transfer).

Due to heterogeneity, results were synthesized narratively across thematic domains rather than meta-analyzed.

## Maternal Nutrition and Fetal Metabolic Programming

### Mechanisms of metabolic programming.

Adverse intrauterine environments induced by maternal nutritional imbalances initiate epigenetic modifications, primarily DNA methylation and histone acetylation, that persistently alter gene expression without altering DNA sequences [4]. These modifications program fetal metabolic pathways toward obesity and insulin resistance by targeting key regulatory genes, with effects transmitted through cell divisions into adulthood [4]. For instance, hypomethylation at the IGF2 promoter and hypermethylation at the H19 promoter in placental and cord blood cells elevate IGF2 expression, driving macrosomia in fetuses exposed to gestational diabetes [4]. Similarly, placental leptin hypermethylation in maternal impaired glucose tolerance reduces leptin expression, disrupting appetite regulation and promoting hyperphagia [4]. Maternal high-fat diets further induce global DNA hypermethylation in fetal liver, muscle, and adipose tissues, alongside histone changes such as elevated H3K14 acetylation, which remodel chromatin to favor lipogenic gene activity [4,5].

Building on epigenetic changes, adverse nutrition disrupts the fetal hormonal milieu, elevating insulin and leptin while dysregulating cortisol, which amplifies insulin resistance [4]. Maternal overnutrition directly increases fetal insulin levels, epigenetically silencing pancreatic genes such as Pdx1 and impairing beta-cell proliferation and function [4]. Gestational diabetes sustains fetal hyperinsulinemia—even under glucose control—through inflammation, adipokine dysregulation, and endoplasmic reticulum stress, establishing early insulin resistance independent of hyperglycemia [4]. Concurrently, maternal obesity increases leptin transfer, inducing hypothalamic leptin resistance via altered neuropeptide Y (NPY) upregulation and pro-opiomelanocortin (POMC) downregulation [4]. Stress or undernutrition compounds this by downregulating placental 11β-HSD2, enhancing cortisol flux to the fetus, and prioritizing cerebral glucose allocation over peripheral tissues [6].

Placental adaptations integrate these epigenetic and hormonal signals to alter nutrient transfer, directly shaping fetal growth trajectories [6]. The enzyme OGT acts as a nutrient sensor via the hexosamine pathway, modifying transport proteins and epigenetic regulators in response to maternal glucose and lipids [6]. High-fat maternal diets boost placental efficiency for fatty acids and glucose, producing large-for-gestational-age (LGA) fetuses predisposed to non-alcoholic fatty liver disease (NAFLD) [6]. Conversely, undernutrition epigenetically represses energy homeostasis genes, such as SLC13A5, leading to small-for-gestational-age (SGA) neonates who are vulnerable to postnatal catch-up obesity [4]. Sex-specific OGT expression heightens male placental sensitivity, leading to mitochondrial dysfunction and greater growth restriction [6]. In gestational diabetes, reduced LPL methylation enhances lipid shuttling, correlating with elevated childhood adiposity [5].

**Key developmental periods of vulnerability.**

The first trimester marks peak vulnerability during organogenesis, when maternal nutrition shapes foundational epigenetic landscapes through sensitive germ cell and preimplantation windows [4]. Nutrient deficiencies, such as folate shortages, disrupt neural tube and cardiac development while initiating genome-wide demethylation cycles that program transgenerational metabolic risks [4]. Placental OGT expression diverges sexually early, with females upregulating it to stabilize trophoblast invasion amid fluctuating nutrient levels, while males remain susceptible [6]. High-fat exposure triggers hepatic histone acetylation shifts by mid-gestation, priming insulin signaling defects before organ maturity [4]. These early calibrations set hypothalamic nutrient sensors, establishing lifelong leptin resistance patterns [6].

Transitioning to the second and third trimesters, rapid adipose accretion amplifies vulnerability as placental nutrient transport peaks, channeling maternal excesses into fetal fat deposition [4]. Hyperglycemia from gestational diabetes upregulates transporters, fueling fetal hyperinsulinemia and LGA outcomes through sustained epigenetic repression of adiponectin and PPARGC1A [5]. High-fat diets enhance histone H3K14 acetylation in adipose precursors, upregulating Zfp423 to accelerate adipogenesis and causing hypothalamic NPY/POMC imbalance, leading to hyperphagia [4]. Maternal obesity intensifies leptin signaling, while sex differences exacerbate male OGT deficits, yielding mitochondrial impairments in fat progenitors [6]. Undernutrition, conversely, hypermethylates SLC13A5, restricting growth but priming postnatal obesity rebound [4].

**Macronutrient Intake and Childhood Obesity Risk**

Maternal nutrition during pregnancy is a key determinant of long-term metabolic outcomes in the offspring, shaping their susceptibility to obesity through metabolic, hormonal, and epigenetic pathways. Evidence synthesized in a systematic review demonstrates that both excessive and insufficient maternal intake of energy and macronutrients can significantly influence fetal development [7]. Excessive caloric consumption is consistently associated with increased fetal adiposity and higher birth weight, frequently resulting in macrosomia - a strong predictor of childhood overweight and obesity. High maternal energy intake enhances placental glucose transfer, promotes fetal hyperinsulinemia, and alters fetal insulin-like growth factor concentrations, thereby facilitating adipose tissue deposition. Energy-dense maternal diets induce epigenetic modifications, particularly changes in DNA methylation within genes regulating metabolism and appetite [8]. These epigenetic alterations persist after birth and contribute to accelerated weight gain and heightened obesity risk later in life.

Protein intake during pregnancy exerts similarly profound effects on fetal metabolic programming. Low maternal protein intake may impair intrauterine growth and promote compensatory metabolic adaptations, increasing the risk of insulin resistance in childhood and adulthood [8]. These adaptations arise from nutrient-sparing processes that prioritize essential organ development while predisposing offspring to adiposity when exposed to a postnatal environment with adequate or excessive energy intake. Some scientific evidence suggests that excessive protein intake, particularly from animal sources, may also alter fetal growth trajectories and metabolic signaling, although results across population studies remain inconsistent [7]. Furthermore, both insufficient and excessive protein intake can modify DNA methylation patterns in key metabolic tissues, affecting gene networks related to insulin sensitivity and amino acid metabolism [8].

The type and amount of dietary fat consumed by the mother are also important regulators of fetal metabolic development. A high intake of saturated fatty acids is associated with increased fetal fat mass and impaired insulin sensitivity, whereas monounsaturated and polyunsaturated fats are associated with more favorable metabolic profiles [7]. Moreover, omega-3 polyunsaturated fatty acids (particularly DHA and EPA)

may modulate gene expression related to inflammation, lipid metabolism, and neurodevelopment. Although randomized trials of omega-3 supplementation during pregnancy have yielded mixed metabolic outcomes in offspring, emerging evidence suggests that these fatty acids may exert protective effects by altering epigenetic signatures that regulate appetite and adiposity [8].

Carbohydrate quality, particularly glycemic index (GI) and glycemic load (GL), is another major determinant of fetal metabolic programming. High-GI and high-GL diets contribute to maternal postprandial hyperglycemia, increasing placental glucose transfer and inducing fetal hyperinsulinemia [7]. These processes enhance adipose tissue deposition and elevate the risk of macrosomia, mirroring pathophysiological mechanisms observed in gestational diabetes, even in otherwise healthy pregnancies. Chronic exposure of the fetus to elevated glucose levels can also induce epigenetic alterations in genes regulating carbohydrate metabolism, reinforcing a metabolic trajectory that predisposes the child to obesity. Interventions aimed at reducing dietary GI, such as increasing the intake of dietary fiber and complex carbohydrates, have shown potential to reduce the incidence of macrosomia and improve maternal glycemic control, although findings vary across populations and study designs [7].

Overall, the evidence supports the conclusion that the quantity and quality of maternal macronutrient intake are crucial determinants of childhood obesity risk [7, 8].

**Micronutrients and Bioactive Compounds**

Studies have demonstrated that prenatal vitamin D deficiency programs adipose tissue metabolism in offspring in a sex-specific manner. Measurements such as neonatal subcutaneous fat mass or bone densitometry may reveal a stronger association with accelerated DNAmGA aging. Elevated maternal plasma homocysteine concentrations are associated with accelerated gestational age in the fetus, and increased homocysteine levels often result from vitamin B12 deficiency. This indicates that disruptions in maternal one-carbon metabolism may influence the rate of epigenetic aging during the prenatal period. Furthermore, maternal plasma fatty acid profiles—particularly higher concentrations of n-3 polyunsaturated fatty acids—have been shown to correlate with accelerated epigenetic gestational aging. These findings suggest that an integrative maternal dietary pattern may cooperatively contribute to DNAmGA programming.[9]

Given the substantial presence of vitamin D receptors in adipocytes, the association between vitamin D and obesity has become a relevant subject of investigation. A 2018 study found that offspring with prenatal vitamin D deficiency exhibited progressively higher body weight compared with non-deficient offspring, with significant differences emerging by week 10 of life. At 14 weeks of age, 24-hour heat production, peak blood glucose levels, adipose tissue volume, and blood lipid indices were all significantly increased in vitamin D-deficient offspring relative to controls. Therefore, maternal vitamin D deficiency not only affects body weight, adiposity, glucose metabolism, and lipid metabolism in adult offspring, but also influences preadipocyte proliferation and differentiation. This suggests that inadequate nutrient supply during early developmental windows may directly contribute to the obesity epidemic later in life. [10]

Authors of a recent review reported an overall effect of excessive maternal carbohydrate intake on increased body weight and hepatic lipid accumulation in offspring, particularly in males. Intake of fructose-containing nutrients has risen over recent decades during pregnancy, despite growing evidence of their numerous adverse effects on offspring health—including the development of obesity, hepatic steatosis, dyslipidemia, insulin resistance, hypertension, and hypoadiponectinemia. Animal studies indicate that a high-fructose maternal diet during pregnancy is associated with reduced HDL cholesterol levels (potentially female-specific), hyperlipidemia, and insulin resistance through changes in the methylation patterns of genes such as LXRα and Pparα, which play essential roles in lipid homeostasis, fatty acid oxidation, and fatty acid transport.

Animal research also suggests that a low-protein maternal diet is associated with reduced birth weight, increased obesity, and lower blood glucose concentrations, mediated by alterations in the methylation patterns of key genes (LEP, G6PC) and global hepatic DNA methylation. These genes are critical for regulating food intake and glucose homeostasis. Evidence from animal studies further indicates that higher multivitamin intake during pregnancy increases body weight, food intake, and features of metabolic syndrome in offspring.

During critical stages of early embryonic development, insufficient maternal intake of one-carbon metabolism nutrients has been linked to DNA hypomethylation at the agouti locus, predisposing offspring to adult-onset obesity and diabetes. The findings cited above collectively highlight the clear need for further research to better elucidate the relationships between maternal nutrient availability during pregnancy, alterations in DNA methylation, and the development of obesity and associated metabolic disturbances in human offspring later in life [11].

**Specific Maternal Conditions and Dietary Patterns**

**Gestational diabetes mellitus (GDM)**

Gestational diabetes mellitus (GDM) is defined as hyperglycemia during pregnancy resulting from an inadequate insulin response, most often due to insulin resistance induced by human placental lactogen (hPL). According to the Developmental Origins of Health and Disease (DOHaD) theory, maternal hyperglycemia disrupts fetal programming through multiple mechanisms [12]. Exposure of the fetus to abnormal glucose concentrations results in both immediate and long-term complications. The pathogenesis involves increased transplacental glucose transfer, which leads to fetal hyperinsulinemia and subsequent metabolic dysfunction [13].

Short-term consequences of GDM include fetal macrosomia (birth weight ≥4000 g), which affects 15–45% of infants born to mothers with GDM compared to 12% in non-GDM pregnancies. Additional immediate outcomes are neonatal hypoglycemia, resulting from persistent fetal hyperinsulinemia, and increased early adiposity, with GDM-exposed infants demonstrating a 16% greater adipose tissue volume by 2–3 months of age. The long-term metabolic consequences are significant: GDM exposure elevates the risk of obesity during childhood and adolescence, with affected children exhibiting a 4.56 kg/m² increase in BMI between ages 10 and 13, compared to a 3.51 kg/m² increase in unexposed peers. Furthermore, offspring exposed to GDM have a two-fold higher risk of developing metabolic syndrome later in life [12], and intrauterine exposure to maternal diabetes accounts for 47% of type 2 diabetes cases diagnosed before age 22 [14]. Altered hunger and satiety signalling, potentially due to hypothalamic changes during hyperglycemic exposure, contributes to increased caloric intake and obesity. Cardiovascular complications include thickening of the posterior left ventricular wall and a higher susceptibility to valvular defects [12]. These intergenerational effects are mediated by epigenetic modifications and alterations in gut microbiota [14], perpetuating a cycle of metabolic disease across generations [13].

**Maternal obesity and metabolic syndrome**

Maternal obesity and metabolic syndrome during pregnancy are significant public health concerns. These conditions have lasting effects on the offspring's health. Metabolic syndrome (MetS) includes metabolic abnormalities such as abdominal obesity, hypertension, dyslipidemia, and impaired glucose tolerance [15]. Maternal obesity or GDM creates an intrauterine environment with excess nutrients. This environment alters fetal development, especially adipose tissue formation and metabolic programming [15, 16, 17].

The main link between maternal metabolic disease and adverse outcomes in offspring is abnormal fetal adipogenesis during a key developmental period. In late fetal life, adipocyte progenitors are specified and committed. Maternal hyperglycemia, hyperlipidemia, and increased adiposity accelerate this differentiation. These factors lead to excessive fat accumulation in utero [15, 16]. Maternal BMI directly predicts neonatal adiposity. Each unit increase in maternal BMI corresponds to about an 8 mL increase in neonatal white adipose tissue volume [16]. Elevated cord leptin levels in pregnancies affected by obesity are also associated with greater skinfold thickness. This finding indicates increased fetal fat deposition [16, 17].

The effects on offspring are significant and long-lasting. Newborns from pregnancies with metabolic disorders often have excessive adiposity at birth and face a higher risk of childhood obesity and cardiometabolic disease. Studies show that 50% of children born large-for-gestational-age from GDM pregnancies meet MetS criteria by ages 6 to 11, compared to 30% of those with normal birth weight [15]. Accelerated adipogenesis in utero may also reduce white adipose tissue plasticity later in life. Offspring of mothers with obesity often have limited subcutaneous adipose tissue expandability and show signs of adiposopathy, including impaired lipolysis and insulin resistance [15, 17]. Male offspring are especially vulnerable to diet-induced metabolic dysfunction, suggesting possible sex-specific programming effects [15].

**Dietary patterns**

Maternal diet during pregnancy plays a key role in determining the offspring's health. The nutritional environment influences fetal growth, metabolic programming, and the long-term risk of obesity and metabolic complications in children. Evidence shows that specific dietary patterns can have positive or negative effects on neonatal outcomes [18, 19, 20].

A systematic review and meta-analysis found that healthful dietary patterns, such as high-fiber diets, the Mediterranean diet, and the DASH diet, are linked to reduced adverse metabolic outcomes in pregnant women and their children. The Mediterranean diet was associated with a lower risk of childhood overweight (OR: 0.85; 95% CI: 0.74–0.97), as well as reduced risks of small for gestational age (SGA) and fetal growth restriction (FGR). The DASH diet was linked to a reduced risk of gestational diabetes mellitus (GDM) (OR:

0.36; 95% CI: 0.26–0.51) and excessive gestational weight gain [18]. A prospective cohort study supported these findings, showing that higher adherence to the alternate Mediterranean diet (aMed) and DASH scores in early pregnancy was associated with increased birthweight and neonatal length. For example, greater adherence to aMed was linked to increased length (quartile 4 vs. 1: 0.54 cm; 95% CI: 0.10, 0.99) and lower odds of low birthweight (ORadj = 0.42; 95% CI: 0.18, 1.00). However, the study also found that certain quartiles of the aMed and AHEI-2010 scores were associated with higher odds of large-for-gestational-age (LGA) and macrosomia, suggesting these patterns may increase neonatal size across the birthweight distribution [19]. This underscores that healthful patterns, such as the Mediterranean and DASH diets, appear to offer protective benefits against childhood overweight and metabolic dysfunction [18, 19].

In contrast, a systematic review of vegan diets during pregnancy found that children born to vegan mothers had lower birth weights and a higher risk for SGA compared to those born to mothers on an omnivorous diet. Several studies reported significantly lower birth weights among infants of vegan mothers. For example, one study found a mean birth weight of 3015.2 ± 420.4 g for infants of vegan mothers versus 3328 ± 495.8 g for those of omnivorous mothers ($p = 0.002$). This may be related to low protein intake, as nearly half of vegan mothers in one study had insufficient protein intake [20].

## Interaction with Other Factors

### Genetic predisposition and gene–diet interactions

Besides the mother's diet and its effects on offspring's obesity, there are intrinsic factors, such as genetic predispositions, that not only affect the mother's food intake but also dictate the probability of a child developing adiposity. For instance, fat mass and obesity-associated (FTO) gene polymorphisms show distinct patterns among mothers with greater total energy intake during pregnancy [21]. One study shows that the FTO polymorphism shifts towards increased carbohydrate consumption, leading to higher caloric intake to meet metabolic demands [21]. This finding suggests that maternal genetic predisposition influences gestational nutrition choices, which may, in turn, affect an offspring's obesity risk.

On top of the maternal genetic predisposition, the mother's BMI plays a crucial role in the development of obesity in children. A study shows that the mother's weight not only affects her child through shared genes but also through the nutritional environment provided during pregnancy [22]. As a result, the mother's indirect effect on the offspring contributes to their postnatal body weight and development of adiposity [22].

At the level of epigenetics, the mother's high BMI influences DNA methylation at birth much more significantly compared to that of normal-weight mothers. Even if the differences fade over time, the methylation patterns undoubtedly suggest that the risk of adiposity is higher in the child whose mother is obese [23]. These genetic patterns explain why maternal obesity is associated with a higher risk of adiposity in offspring.

### Maternal gut microbiota and fetal metabolic programming

Gut microbiota is another important factor that contributes to maternal metabolic status. It is so significant because it can influence the signals that the child receives during pregnancy. If the woman becomes obese or gains substantial weight during pregnancy, she may experience reduced microbial diversity and shifts in key bacterial groups, including Bifidobacterium, Blautia, Christensenellaceae, Lachnospira, and Parabacteroides [24]. The changes in the infant's gut microbiota are not big and long-lasting. However, some of the mother's bacteria may persist in the child even after birth. It is likely to influence how the infant's metabolism develops later in life, especially in relation to bacteria linked to energy regulation and inflammation [24].

An additional insight into how maternal gut microbiota shapes fetal metabolic development can be gained from animal research. Studies in mice showed that maternal microbiota produce short-chain fatty acids (SCFAs) that later enter the placenta and reach the embryo, where they support the development of neural, intestinal, and pancreatic cells. Then, microbial metabolites help regulate energy balance via fetal receptors such as GPR41 and GPR43. If the mother is fed a low-fiber diet, the SCFA signaling in the offspring is impaired, and they develop obesity and glucose intolerance on a much larger scale. Meanwhile, when the SCFAs are restored during pregnancy, the child has a much lower chance of developing metabolic problems [25]. Thus, a healthy maternal microbiota contributes to better metabolic programming and lowers the chances of obesity in the child.

**Postnatal influences**

There are many postnatal factors that contribute to a higher risk of developing obesity. They include infant feeding practices and quick weight gain. Studies show that formula-fed infants have higher BMI scores and are more likely to develop obesity by age six compared to children who were exclusively breastfed in the first months of their lives. Moreover, formula-fed newborns usually gain weight more rapidly during the first six months of life, which later often leads to adiposity. Therefore, breastfeeding may help reduce the risk of excessive weight gain, as early rapid growth is associated with feeding type and childhood BMI [26]. This aligns with the results of multiple other studies, which suggest that breastfeeding, early growth patterns, and maternal pre-pregnancy BMI work together to influence childhood obesity risk. Infants whose mothers decided to exclusively breastfeed them tend to have lower BMI and are at a lower risk of developing early childhood obesity [27]. This is because breastfeeding leads to slower, more regulated weight gain. However, rapid weight gain can lead to being overweight later in life, regardless of feeding type. The studies also showed that mothers with higher pre-pregnancy BMI were more likely to breastfeed for a shorter time, linking maternal health factors to postnatal feeding patterns [27]. This highlights the importance of early feeding practices for the child's later development.

**Importance of prenatal nutrition counseling**

A mother's diet and nutritional status, both before conception and throughout pregnancy, strongly influence the fetus's health and disease risk during intrauterine development and even into adulthood. Inadequate nutrient intake during the prenatal period can affect the programming of multiple organ systems and may heighten the offspring's overall susceptibility to disease [28]. Maternal excess weight has been linked to higher fetal heart rate and reduced heart rate variability and is further associated with the early onset of chronic conditions—including childhood obesity—that can have serious long-term consequences [29].

**Evidence-based dietary recommendations for pregnant women**

Greater adherence to a fast-food and sweets dietary pattern before pregnancy increases the likelihood of delivering a large-for-gestational-age infant, whereas consuming more vegetables and dairy products is associated with a reduced risk of preterm birth [30]. Higher rates of maternal nutrient deficiencies and lower dietary diversity scores have been significantly associated with a lower risk of small-for-gestational-age newborns [31].

Numerous nutrients have been examined for their influence on fetal development. For example, sufficient intake of folate, leafy green vegetables, and folic acid supplements lowers the risk of neural tube defects such as spina bifida and reduces the incidence of small-for-gestational-age infants [32]. Moderate fish consumption, high adherence to a Mediterranean diet, use of olive oil, and adequate intake of key vitamins (A, D, B1, B3, B6, B9, and B12) and iron supplements have been shown to reduce the risk of preterm birth [33]. A recent prospective study by Kesary et al. found that a maternal vegan diet may protect against excessive gestational weight gain, but it is also associated with an increased risk of small-for-gestational-age infants and lower birth-weight percentiles [34].

It is generally recommended that pregnant women consume four to five meals per day, with breakfast providing the highest caloric intake and including dairy products, fruits, and cereals. Lunch should be moderate in size, and dinner should be eaten several hours before bedtime, with an optional small serving of fruit or dairy before sleep. Water intake should reach at least 2–2.5 L per day, while foods high in fats, sugars, fried items, and excess salt should be limited. Physical activity for at least 30 minutes per day can also help women to achieve recommended gains [35].

**Conclusions**

Childhood excess weight remains a major global health concern, and growing evidence suggests its origins may lie in early developmental influences. Adverse intrauterine environments resulting from imbalanced maternal nutrition can shape long-term metabolic health by inducing persistent epigenetic modifications in fetal metabolic pathways. These include alterations in DNA methylation and histone modifications that influence genes involved in insulin sensitivity, appetite regulation, lipid metabolism, and adipose tissue development. Maternal nutritional imbalances also disrupt the fetal hormonal equilibrium, with overnutrition elevating fetal insulin levels, impairing pancreatic beta-cell proliferation, and increasing insulin-like growth factor concentrations. All these changes, taken together, promote adipose tissue deposition and increase the risk of obesity. Excess maternal intake of glucose and fatty acids increases fetal susceptibility to

NAFLD, especially among large-for-gestational-age (LGA) infants, whereas maternal undernutrition may epigenetically predispose small-for-gestational-age (SGA) neonates to accelerated postnatal catch-up growth and later-life obesity. Deficiencies in critical micronutrients such as folic acid, vitamin D, and vitamin B12 interfere with organogenesis, disrupt neural and cardiac development, and have been linked to accelerated epigenetic gestational aging, underscoring the importance of balanced micronutrient intake during pregnancy.

Despite substantial progress, current evidence has several limitations that restrict the strength of conclusions. First, significant heterogeneity in study designs and dietary assessment methods, including reliance on food-frequency questionnaires, self-reporting, and varied definitions of dietary patterns. This limits comparability across studies. Second, many findings derive from animal research or short-term human cohorts, highlighting substantial challenges in conducting long-term follow-up of offspring to track metabolic outcomes into adolescence and adulthood. Third, maternal nutrition is closely tied to socioeconomic status, genetic predispositions, cultural factors, and lifestyle behaviors, making it difficult to fully isolate the effects of prenatal diet; thus, confounding factors remain a major constraint in the interpretation of available evidence.

Overall, the evidence consistently demonstrates that maternal diet exerts a powerful influence on fetal metabolic programming. Both overnutrition and undernutrition alter epigenetic marks, disrupt hormonal regulation, reshape placental nutrient transfer, and affect adipose tissue development in sex-specific ways. Macronutrient quality, particularly excessive energy intake, high glycemic load, saturated fats, or low protein, plays a crucial role in shaping adiposity and insulin sensitivity. Similarly, micronutrients and bioactive compounds, including vitamin D, B12, folate, and n-3 fatty acids, influence fetal metabolic trajectories by modulating DNA methylation and accelerating gestational age. Collectively, these findings emphasize the critical role of maternal diet in shaping lifelong metabolic health, and the growing need for early-life interventions to prevent childhood obesity, beginning as early as pregnancy or even preconception.

Future research must address existing gaps by prioritizing longitudinal cohort studies that incorporate objective dietary biomarkers rather than relying solely on self-reported intake. Integrating omics technologies, such as epigenomics, metabolomics, and microbiome profiling, will be essential to uncover the mechanistic pathways linking maternal diet to offspring metabolic outcomes. Moreover, advancing personalized nutrition approaches during pregnancy, potentially informed by maternal metabolic status, genetic susceptibility, and microbiota composition, could enable more targeted interventions to reduce obesity risk in future generations.

## REFERENCES


1. Zhang, X., Liu, J., Ni, Y., Yi, C., Fang, Y., Ning, Q., Shen, B., Zhang, K., Liu, Y., Yang, L., Li, K., Liu, Y., Huang, R., & Li, Z. (2024). Global prevalence of overweight and obesity in children and adolescents: A systematic review and meta-analysis. *JAMA Pediatrics, 178*(8), 800–813. https://doi.org/10.1001/jamapediatrics.2024.1576
2. Cristian, A., Tarry-Adkins, J. L., & Aiken, C. E. (2023). The uterine environment and childhood obesity risk: Mechanisms and predictions. *Current Nutrition Reports, 12*(3), 416–425. https://doi.org/10.1007/s13668-023-00482-z
3. Rasmussen, L., Knorr, S., Antoniussen, C. S., Bruun, J. M., Ovesen, P. G., Fuglsang, J., & Kampmann, U. (2021). The impact of lifestyle, diet and physical activity on epigenetic changes in the offspring: A systematic review. *Nutrients, 13*(8), 2821. https://doi.org/10.3390/nu13082821
4. Zhu, Z., Cao, F., & Li, X. (2019). Epigenetic programming and fetal metabolic programming. *Frontiers in Endocrinology, 10*, 764. https://doi.org/10.3389/fendo.2019.00764
5. Lesseur, C., & Chen, J. (2018). Adverse maternal metabolic intrauterine environment and placental epigenetics: Implications for fetal metabolic programming. *Current Environmental Health Reports, 5*(4), 531–543. https://doi.org/10.1007/s40572-018-0217-9
6. Nugent, B. M., & Bale, T. L. (2015). The omniscient placenta: Metabolic and epigenetic regulation of fetal programming. *Frontiers in Neuroendocrinology, 39*, 28–37. https://doi.org/10.1016/j.yfrne.2015.09.001
7. Khammarnia, M., Ansari-Moghaddam, A., Kakhki, F. G., Clark, C. C. T., & Barahouei, F. B. (2024). Maternal macronutrient and energy intake during pregnancy: A systematic review and meta-analysis. *BMC Public Health, 24*(1), 478. https://doi.org/10.1186/s12889-024-17862-x
8. Bokor, S., Csölle, I., Felső, R., Vass, R. A., Funke, S., Ertl, T., & Molnár, D. (2024). Dietary nutrients during gestation cause obesity and related metabolic changes by altering DNA methylation in the offspring. *Frontiers in Endocrinology, 15*, 1287255. https://doi.org/10.3389/fendo.2024.1287255
9. Kawai, T., Jwa, S. C., Ogawa, K., Tanaka, H., Aoto, S., Kamura, H., Morisaki, N., Fujiwara, T., & Hata, K. (2025). Maternal vitamin D deficiency is a risk factor for infants' epigenetic gestational age acceleration at birth in Japan: A cohort study. *Nutrients, 17*(2), 368. https://doi.org/10.3390/nu17020368

10. Wen, J., Hong, Q., Wang, X., et al. (2018). The effect of maternal vitamin D deficiency during pregnancy on body fat and adipogenesis in rat offspring. *Scientific Reports, 8*, 365. https://doi.org/10.1038/s41598-017-18770-4
11. Bokor, S., Csölle, I., Felső, R., Vass, R. A., Funke, S., Ertl, T., & Molnár, D. (2024). Dietary nutrients during gestation cause obesity and related metabolic changes by altering DNA methylation in the offspring. *Frontiers in Endocrinology, 15*, 1287255. https://doi.org/10.3389/fendo.2024.1287255
12. Al Bekai, E., Beaini, C. E., Kalout, K., Safieddine, O., Semaan, S., Sahyoun, F., Ghadieh, H. E., Azar, S., Kanaan, A., & Harb, F. (2025). The hidden impact of gestational diabetes: Unveiling offspring complications and long-term effects. *Life, 15*(3), 440. https://doi.org/10.3390/life15030440
13. Burlina, S., Dalfrà, M. G., & Lapolla, A. (2019). Short- and long-term consequences for offspring exposed to maternal diabetes: A review. *The Journal of Maternal-Fetal & Neonatal Medicine, 32*(4), 687–694. https://doi.org/10.1080/14767058.2017.1387893
14. Franzago, M., Fraticelli, F., Stuppia, L., & Vitacolonna, E. (2019). Nutrigenetics, epigenetics and gestational diabetes: Consequences in mother and child. *Epigenetics, 14*(3), 215–235. https://doi.org/10.1080/15592294.2019.1582277
15. Scheidl, T. B., Brightwell, A. L., Easson, S. H., & Thompson, J. A. (2023). Maternal obesity and programming of metabolic syndrome in the offspring: Searching for mechanisms in the adipocyte progenitor pool. *BMC Medicine, 21*(1). https://doi.org/10.1186/s12916-023-02730-z
16. Xue, L., Chen, X., Sun, J., Fan, M., Qian, H., Li, Y., & Wang, L. (2024). Maternal dietary carbohydrate and pregnancy outcomes: Quality over quantity. *Nutrients, 16*(14), 2269. https://doi.org/10.3390/nu16142269
17. Jovandaric, M. Z., Babic, S., Raus, M., & Medjo, B. (2023). The importance of metabolic and environmental factors in the occurrence of oxidative stress during pregnancy. *International Journal of Molecular Sciences, 24*(15), 11964. https://doi.org/10.3390/ijms241511964
18. Fan, M., Chu, Y., Zheng, Y., Zhang, Z., & Hou, M. (2025). Association of pregnancy diet with metabolic adverse outcomes in pregnant women and their children: A systematic review and meta-analysis. *Annals of Nutrition and Metabolism, 81*(3), 123–140. https://doi.org/10.1159/000543423
19. Yisahak, S. F., Mumford, S. L., Grewal, J., Li, M., Zhang, C., Grantz, K. L., & Hinkle, S. N. (2021). Maternal diet patterns during early pregnancy in relation to neonatal outcomes. *The American Journal of Clinical Nutrition, 114*(1), 358–367. https://doi.org/10.1093/ajcn/nqab019
20. Meulenbroeks, D., Otten, E., Smeets, S., Groeneveld, L., Jonkers, D., Eussen, S., Scheepers, H., & Gubbels, J. (2024). The association of a vegan diet during pregnancy with maternal and child outcomes: A systematic review. *Nutrients, 16*(19), 3329. https://doi.org/10.3390/nu16193329
21. Martins, M. C., Trujillo, J., Freitas-Vilela, A. A., Farias, D. R., Rosado, E. L., Struchiner, C. J., & Kac, G. (2018). Associations between obesity candidate gene polymorphisms (FTO, MC4R, LEP and LEPR) and dietary intake in pregnant women. *The British Journal of Nutrition, 120*(4), 454–463. https://doi.org/10.1017/S0007114518001423
22. Wright, L., Shireby, G., Morris, T. T., Davies, N. M., & Bann, D. (2025). The association between parental BMI and offspring adiposity: A genetically informed analysis of trios. *PLoS Genetics, 21*(8), e1011775. https://doi.org/10.1371/journal.pgen.1011775
23. Sharp, G. C., Lawlor, D. A., Richmond, R. C., Fraser, A., Simpkin, A., Suderman, M., et al. (2015). Maternal pre-pregnancy BMI and gestational weight gain, offspring DNA methylation and later offspring adiposity. *International Journal of Epidemiology, 44*(4), 1288–1304. https://doi.org/10.1093/ije/dyv042
24. Stanislawski, M. A., Dabelea, D., Wagner, B. D., Sontag, M. K., Lozupone, C. A., & Eggesbø, M. (2017). Pre-pregnancy weight, gestational weight gain, and the gut microbiota of mothers and their infants. *Microbiome, 5*(1), 113. https://doi.org/10.1186/s40168-017-0332-0
25. Kimura, I., Miyamoto, J., Ohue-Kitano, R., Watanabe, K., Yamada, T., Onuki, M., et al. (2020). Maternal gut microbiota in pregnancy influences offspring metabolic phenotype in mice. *Science, 367*(6481), eaaw8429. https://doi.org/10.1126/science.aaw8429
26. Nagore González, C., Iglesia Altaba, I., Guillén Sebastián, C., Alvarez Sauras, M. L., García Enguita, S., Moreno, L. A., & Rodríguez, G. (2025). Mediating effect of infant rapid weight gain on the association between exclusive breastfeeding and the risk of obesity later in life. *Pediatric Obesity, 20*(10), e70039. https://doi.org/10.1111/ijpo.70039
27. Zheng, M., Hesketh, K. D., Vuillermin, P., Dodd, J., Wen, L. M., Baur, L. A., et al. (2023). Understanding the pathways between prenatal and postnatal factors and overweight outcomes in early childhood. *International Journal of Obesity, 47*(7), 574–582. https://doi.org/10.1038/s41366-023-01301-9
28. Krzeczkowski, J. E., Boylan, K., Arbuckle, T. E., Muckle, G., Poliakova, N., Séguin, J. R., et al. (2020). Maternal pregnancy diet quality is directly associated with autonomic nervous system function in 6-month-old offspring. *The Journal of Nutrition, 150*(2), 267–275. https://doi.org/10.1093/jn/nxz228
29. Catalano, P. M., & Shankar, K. (2017). Obesity and pregnancy: Mechanisms of short term and long term adverse consequences for mother and child. *BMJ, 356*. https://doi.org/10.1136/bmj.j1

30. Alves-Santos, N. H., Cocate, P. G., Benaim, C., Farias, D. R., Emmett, P. M., & Kac, G. (2019). Prepregnancy dietary patterns and their association with perinatal outcomes. *Journal of the Academy of Nutrition and Dietetics, 119*(9), 1439–1451. https://doi.org/10.1016/j.jand.2019.02.016
31. Cano-Ibáñez, N., Martínez-Galiano, J. M., Amezcua-Prieto, C., Olmedo-Requena, R., Bueno-Cavanillas, A., & Delgado-Rodríguez, M. (2020). Maternal dietary diversity and risk of small for gestational age newborn. *Clinical Nutrition, 39*(6), 1943–1950. https://doi.org/10.1016/j.clnu.2019.08.019
32. Martínez-Galiano, J. M., Amezcua-Prieto, C., Salcedo-Bellido, I., González-Mata, G., Bueno-Cavanillas, A., & Delgado-Rodríguez, M. (2018). Maternal dietary consumption of legumes, vegetables and fruit during pregnancy. *BMC Pregnancy and Childbirth, 18*(1), 486. https://doi.org/10.1186/s12884-018-2123-4
33. Amezcua-Prieto, C., Martínez-Galiano, J. M., Salcedo-Bellido, I., Olmedo-Requena, R., Bueno-Cavanillas, A., & Delgado-Rodríguez, M. (2018). Maternal seafood intake and the risk of small for gestational age newborns. *BMJ Open, 8*(8), e020424. https://doi.org/10.1136/bmjopen-2017-020424
34. Kesary, Y., Avital, K., & Hiersch, L. (2020). Maternal plant-based diet during gestation and pregnancy outcomes. *Archives of Gynecology and Obstetrics, 302*(4), 887–898. https://doi.org/10.1007/s00404-020-05689-x
35. Marshall, N. E., Abrams, B., Barbour, L. A., Catalano, P., Christian, P., Friedman, J. E., et al. (2022). The importance of nutrition in pregnancy and lactation: Lifelong consequences. *American Journal of Obstetrics and Gynecology, 226*(5), 607–632. https://doi.org/10.1016/j.ajog.2021.12.035