

## From Maternal Diet to Childhood Obesity: How Pregnancy Shapes Metabolic Health in the First 1,000 Days



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Childhood obesity has become one of the major non-communicable disease burdens worldwide. In 2022, more than 1 billion people were living with obesity, and prevalence among children and adolescents has increased more than fourfold since 1990 [1]. Because excess adiposity in childhood often tracks into adult life and is associated with type 2 diabetes, cardiovascular disease and impaired psychosocial functioning, prevention needs to start before excess weight is established [2]. The generally modest effects of school-based obesity-prevention interventions in primary-school-aged children have therefore shifted attention towards earlier determinants of risk [3].

The “first 1,000 days” (from conception to a child’s second birthday) is increasingly viewed as a critical window for obesity prevention. The Developmental Origins of Health and Disease (DOHaD) framework shows that exposures during sensitive periods can shape organ development, endocrine function and health behaviours with long-term consequences [4,5]. Maternal nutrition and metabolic health during pregnancy are particularly influential because they determine the intrauterine milieu and may modify offspring susceptibility to obesity through metabolic and epigenetic pathways [4,5].

### **Programming pathways**

Metabolic programming refers to long-lasting adaptations in tissues that regulate appetite, adipogenesis and glucose–insulin and lipid homeostasis in response to intrauterine conditions [4,5]. Both undernutrition and overnutrition can induce changes that support short-term survival but become maladaptive in energy-dense postnatal environments [4,5]. Epigenetic mechanisms - DNA methylation, histone modifications and non-coding RNAs - provide a plausible biological substrate because many marks are established during early development and can be influenced by nutrient status, glycaemia, inflammation, microbiota-derived metabolites and exposure to environmental chemicals [5]. Most human evidence is observational; associations should be interpreted with attention to residual confounding and the difficulty of separating prenatal from shared familial and postnatal influences.

### **What matters most in pregnancy**

Evidence is most consistent for maternal adiposity and glycaemic dysregulation. Higher pre-pregnancy body mass index (BMI), excessive gestational weight gain and maternal obesity are associated with greater child adiposity and adverse cardiometabolic profiles across childhood and adolescence [6]. Gestational diabetes mellitus (GDM) and milder degrees of hyperglycaemia in pregnancy are also linked to higher offspring BMI and central adiposity, particularly when hyperglycaemia occurs early or coexists with maternal obesity [7].

Beyond total energy intake, overall diet quality and dietary patterns appear important. In pooled European cohort analyses, poorer maternal diet quality and more pro-inflammatory dietary profiles during pregnancy have been associated with higher childhood adiposity [8]. Western-style patterns often rich in refined carbohydrates, sugar-sweetened beverages and ultra-processed foods may promote excessive gestational weight gain and low-grade inflammation. Large prospective cohorts have reported that higher maternal ultra-processed food intake during the child-rearing period is associated with an increased risk of offspring overweight/obesity [9].

Conversely, Mediterranean-type patterns (vegetables, fruit, legumes, whole grains, nuts, olive oil and fish; limited red/processed meat and sweets) are consistently associated with more favourable offspring adiposity and cardiometabolic traits [10]. Mechanistic hypotheses include improved maternal glycaemic control and inflammatory status, but causal pathways remain to be clarified [5].

The maternal gut microbiota has been proposed as an additional link between maternal diet and offspring metabolic risk. Maternal obesity and highly processed diets are associated with gut dysbiosis (lower diversity and fewer short-chain fatty acid-producing taxa), which may influence early infant colonisation and has been related to later adiposity [5]. However, evidence for a consistent, viable placental microbiome in uncomplicated pregnancies remains debated, and contamination is a major concern in low-biomass microbiome studies [11]. Delivery by caesarean section and early antibiotic exposure also modify early colonisation patterns; links with later obesity are generally modest and may be confounded by indication [12,13].

Environmental endocrine-disrupting chemicals (“obesogens”) add a further layer of risk. Compounds such as bisphenols, phthalates and certain persistent organic pollutants can cross the placenta and may influence adipocyte differentiation and metabolic signalling [14]. Meta-analyses suggest prenatal exposure to persistent organic pollutants is associated with higher childhood BMI and adiposity, although exposure assessment, co-exposures and socioeconomic confounding remain important methodological challenges [15].

Implications for practice and policy

Implications for practice and policy follow directly from this evidence base: a) address weight and metabolic risk before conception and monitor gestational weight gain; b) provide early, structured dietary counselling (ideally dietitian-led) that emphasises a Mediterranean-type pattern and practical strategies to reduce ultra-processed foods and free sugars; c) prioritise prevention, early detection and optimal management of GDM, with postpartum follow-up for women and support for infant feeding and growth monitoring; d) support perinatal environments (food systems, maternity care pathways and social policies) that enable healthy choices and reduce inequities; and e) apply the precautionary principle to minimise exposure to endocrine-disrupting chemicals in pregnancy, alongside broader regulatory action [3,6–10,14,15].

## Conclusion

Pregnancy should be framed as a key intervention window for lifelong metabolic health, not only a period of fetal growth monitoring. Maternal diet quality, nutritional status, glycaemic control, microbiota-related exposures and environmental chemicals interact to shape developmental programming during the first 1,000 days [4,5,7,14,15]. Strengthening evidence-based nutritional care and metabolic risk management in and around pregnancy is a realistic step towards reducing the future burden of childhood obesity.

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