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MATERNAL CARDIORENAL-METABOLIC AXIS IN OBESITY: DIGITAL BIOMARKERS AND FETAL PROGRAMMING OF FUTURE CARDIOVASCULAR AND KIDNEY DISEASE

Kinga Bojdo *(Corresponding Author, Email: kinga.bojdo@stud.umed.lodz.pl)*
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Zofia Dąbrowska
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Hanna Frankenstein
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Magdalena Gąsior
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Katarzyna Kazimierczuk
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Agata Mikołajczyk
Institute of Medical Sciences, Jan Kochanowski University of Kielce, IX Wieków Kielc 19a, 25-317 Kielce, Poland

Julia Piotrowska
Medical University of Lodz, Tadeusza Kościuszki 4, 90-419 Łódź, Poland

Amelia Wardak
Institute of Medical Sciences, Jan Kochanowski University of Kielce, IX Wieków Kielc 19a, 25-317 Kielce, Poland

ABSTRACT

Maternal obesity is an increasingly prevalent global health challenge and a major determinant of adverse cardiometabolic outcomes across generations. This review introduces the concept of the maternal cardiorenal–metabolic axis as an integrated framework linking hemodynamic stress, renal vulnerability, endothelial dysfunction, metabolic imbalance, and chronic inflammation in obese pregnancy. These interrelated disturbances create a maladaptive intrauterine environment that contributes to fetal programming of future cardiovascular disease (CVD) and chronic kidney disease (CKD). Evidence from clinical, epidemiological, genetic, and experimental studies demonstrates that maternal adiposity before and during pregnancy is associated with subclinical alterations in maternal cardiovascular and renal function, impaired placentation, and long-term structural and functional changes in the offspring heart and kidneys.

The review further explores the emerging role of digital biomarkers—including remote blood pressure monitoring, continuous glucose monitoring, heart rate variability, wearable-derived vascular signals, and AI-based cuff-less blood pressure estimation—in early detection and intergenerational risk stratification. Integration of multimodal physiological data with artificial intelligence–driven analytics may enable personalized monitoring and earlier identification of maladaptive trajectories within the maternal cardiorenal–metabolic axis. While promising, clinical translation requires longitudinal validation, standardized methodologies, and equitable implementation. Linking maternal digital phenotypes with long-term offspring outcomes represents a critical next step toward precision prevention of intergenerational cardiovascular and kidney disease.

KEYWORDS

Maternal Obesity, Cardiorenal–Metabolic Axis, Fetal Programming, Cardiovascular Disease, Digital Biomarkers, Wearable Technologies, Artificial Intelligence, Heart Rate Variability

CITATION

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1. Introduction

Obesity is a complex, multifactorial condition defined by excessive adiposity and is recognized by the World Health Organization (WHO) as a global epidemic. Since 1975, the prevalence of overweight and obesity has nearly tripled worldwide, with hundreds of millions of adults affected. Projections indicate a continued rise, with over one billion adults expected to live with obesity by 2030 [1, 2]. During pregnancy, maternal obesity is associated with increased risks of gestational diabetes, hypertensive disorders, preeclampsia, preterm birth, cesarean delivery, and pregnancy loss. It also elevates the likelihood of fetal macrosomia and congenital anomalies. The prevalence of maternal obesity is rising globally, with particularly high rates reported in certain regions and among older mothers, ethnic minorities, and socioeconomically disadvantaged populations. Importantly, in-utero exposure to maternal obesity is linked to adverse cardiometabolic outcomes in offspring that may persist into adulthood, underscoring its intergenerational impact [2].

In line with global trends, Poland has also experienced a marked increase in excess body weight among women. In 2022, 42.6% of women aged ≥20 years were classified as overweight and 11.7% as obese, indicating a high prevalence of abnormal body weight among those of reproductive age. National reports show a consistent upward trend in obesity over the past decade, with projections for 2020–2035 estimating an average annual increase of 1.7% in adult obesity rates. If this trajectory continues, nearly one-third of Polish women may be living with obesity by 2035. These data highlight the urgent need to improve awareness and promote early preventive strategies among women of reproductive age [3].

Cardiovascular disease (CVD) remains the leading cause of global mortality, accounting for 17.9 million deaths in 2020 according to the WHO. It encompasses a spectrum of chronic disorders affecting the heart and blood vessels, including coronary artery disease, heart failure, arrhythmias, and cerebrovascular disease. Long-term data from the Framingham Heart Study have identified hypertension, coronary artery disease, cardiac hypertrophy, diabetes, and particularly obesity as major predictors of future cardiovascular events. In the context of rising obesity rates, increasing numbers of women enter pregnancy with pre-existing cardiometabolic risk. Even in the absence of overt diabetes, obesity is often accompanied by impaired fasting glucose, hyperinsulinemia, and dyslipidemia prior to conception, indicating underlying metabolic dysfunction. These disturbances may predispose to gestational complications and reflect broader cardiometabolic vulnerability that can influence both maternal and offspring cardiovascular health. Consistent with the Developmental Origins of Health and Disease framework, evidence from population studies, clinical cohorts, and animal models demonstrates that the early-life environment—particularly exposure to maternal obesity—can program long-term cardiovascular risk in the offspring. Experimental models suggest mechanisms such as cardiac mitochondrial dysfunction and persistent molecular alterations, although translation to humans remains limited. Differences between animal and human pregnancy, including placental structure and developmental timing, must also be considered [4].

Overall, current evidence suggests that maternal obesity contributes to the intergenerational transmission of cardiovascular risk through complex metabolic and molecular mechanisms initiated as early as fetal life. Understanding the maternal cardiorenal–metabolic axis and identifying early biomarkers of dysfunction may be crucial for preventing future cardiovascular and kidney disease. These concepts and underlying mechanisms will be further explored in this review.

2. Maternal Cardiorenal-Metabolic Dysfunction in Obesity

2.1. Hemodynamic Adaptations in Obese Pregnancy

Obesity is characterized by increased circulating blood volume, elevated stroke volume, and higher systemic and pulmonary pressures, promoting biventricular hypertrophy and left atrial enlargement. Both systolic and diastolic left ventricular (LV) function may be impaired, and in severe cases right ventricular dysfunction can occur.

Because pregnancy itself represents a state of chronic volume overload, maternal obesity may further amplify cardiovascular stress. Even among healthy pregnant women, a subset demonstrates signs of cardiac maladaptation at term. Evidence from echocardiographic studies indicates that obese pregnant women exhibit higher blood pressure (BP), heart rate, cardiac output, stroke volume, and LV mass compared with normal-weight counterparts. Advanced imaging, including speckle-tracking echocardiography, has revealed reduced LV global longitudinal strain and a higher prevalence of diastolic dysfunction in obese women, suggesting subclinical myocardial impairment despite apparently uncomplicated pregnancies. These findings point to a maladaptive cardiovascular response to pregnancy in the context of obesity, which may contribute to the increased risk of hypertensive and uteroplacental complications. However, it remains unclear to what extent these alterations precede pregnancy or are exacerbated by gestational hemodynamic demands [5].

Further evidence from prospective longitudinal data indicates that maternal hemodynamic adaptation differs according to the underlying phenotype. Pregnancies complicated by hypertensive disorders and fetal growth restriction were characterized by lower cardiac output and concentric LV remodeling, suggesting impaired cardiovascular adaptation. In contrast, women with hypertensive disorders and appropriately grown fetuses showed increased cardiac output combined with elevated vascular resistance, concentric hypertrophy, and diastolic dysfunction, with a higher likelihood of persistent abnormalities postpartum. Importantly, obesity and other metabolic risk factors were more prevalent in this latter group, supporting the concept that a pre-existing cardiometabolic profile may predispose to sustained cardiovascular dysfunction. Together, these findings reinforce the view that maternal obesity shapes distinct hemodynamic trajectories within the cardiorenal–metabolic axis, with potential implications for long-term cardiovascular risk [6].

Complementary longitudinal echocardiographic studies in otherwise uncomplicated obese pregnancies confirm that these women exhibit persistent alterations in cardiac geometry and function across gestation. Despite preserved ejection fraction, obese pregnant women demonstrate greater relative wall thickness and LV mass, together with impaired diastolic indices and subtle reductions in longitudinal function. These structural and functional changes occur in the absence of overt hypertension and resemble early patterns of cardiac remodeling described in cardiometabolic disease. Such findings suggest that obesity itself may establish a subclinical myocardial phenotype during pregnancy, potentially lowering the threshold for subsequent hypertensive or placental complications. Within the maternal cardiorenal–metabolic axis, this supports the concept that pregnancy unmasks pre-existing cardiovascular vulnerability rather than acting as an isolated stressor [7].

2.2. Subclinical Cardiorenal Dysfunction

Emerging data also indicate that maternal obesity is linked to early renal alterations during pregnancy. In a cohort study evaluating body mass index (BMI), gestational weight gain (GWG), and urinary albumin excretion, higher first-trimester BMI was associated with increased albuminuria later in pregnancy, whereas GWG itself was not independently related. These findings suggest that pre-existing adiposity, rather than weight gain during gestation, may be the primary driver of renal stress. Obesity and albuminuria are both associated with adverse pregnancy outcomes and increased long-term cardiovascular risk. Proposed mechanisms include glomerular hyperfiltration, hyperinsulinemia, chronic low-grade inflammation, and endothelial dysfunction. Physiological increases in glomerular filtration during pregnancy may further exacerbate obesity-related renal vulnerability, leading to progressive rises in urinary albumin excretion across gestation. Given that even mild elevations in albuminuria are linked to future renal and cardiovascular disease, these observations support the concept of subclinical cardiorenal dysfunction in obese pregnancy. They also underscore the importance of preconception weight optimization and adherence to gestational weight recommendations as potential strategies to mitigate long-term maternal cardiometabolic risk within the maternal cardiorenal–metabolic axis [8].

Beyond hemodynamic and renal alterations, endothelial dysfunction represents another central component of the maternal cardiorenal–metabolic axis in obesity. The vascular endothelium functions as an active endocrine organ that maintains metabolic and vascular homeostasis; however, in states of metabolic

stress it shifts toward a dysfunctional phenotype characterized by impaired vasodilation, oxidative stress, and inflammation [9].

During normal pregnancy, profound changes occur in lipid metabolism to meet increasing maternal and fetal energy demands. Total cholesterol, low-density lipoprotein (LDL), and particularly triglyceride concentrations rise progressively, especially in the second and third trimesters. High-density lipoprotein (HDL) levels initially increase and may decline toward term. This physiological hyperlipidemia supports steroid hormone synthesis and placental development, but it also modifies lipoprotein composition and function. Qualitative changes in lipoproteins appear to be as important as quantitative increases. Pregnancy is associated with a higher proportion of triglyceride-rich lipoproteins and small, dense LDL particles, which are more susceptible to oxidation. Oxidized LDL can impair endothelial nitric oxide bioavailability, promote vasoconstriction, and enhance expression of adhesion molecules, thereby contributing to endothelial activation. Alterations in HDL functionality have also been described, including reduced antioxidative and anti-inflammatory capacity, which may further diminish endothelial protection. Endothelial dysfunction in pregnancy is characterized by impaired flow-mediated vasodilation, increased arterial stiffness, and altered angiogenic balance (e.g., shifts in pro- and anti-angiogenic factors). In uncomplicated pregnancies, these changes are usually subtle and reversible postpartum. However, in complicated pregnancies—such as those with hypertensive disorders—atherogenic lipid profiles, increased lipid peroxidation, and disturbed lipoprotein function are more pronounced and correlate with markers of vascular dysfunction. Thus, pregnancy represents a state in which physiological hyperlipidemia intersects with endothelial adaptation. Variations in lipid composition and lipoprotein quality may influence the degree of endothelial stress, potentially shaping both short-term obstetric outcomes and long-term maternal cardiovascular risk [9].

Inflammation is a tightly regulated and necessary component of normal pregnancy, coordinating implantation, placental development, and parturition through precisely timed cytokine signaling. The biological impact of inflammation depends on its intensity, duration, and cellular composition. In obesity, however, chronic low-grade inflammation originates primarily from hypertrophic adipose tissue. Lipid accumulation, adipocyte stress, and increased release of free fatty acids promote immune cell infiltration—particularly monocytes that differentiate into pro-inflammatory macrophages—leading to sustained production of cytokines such as TNF-α, IL-1β, IL-6, and MCP-1. These mediators contribute to systemic insulin resistance and endothelial dysfunction, processes also implicated in hypertensive and metabolic pregnancy complications [10].

Within the maternal–fetal interface, immune cells such as uterine natural killer (uNK) cells, macrophages, dendritic cells, and regulatory T cells orchestrate vascular remodeling and maternal–fetal tolerance. Maternal obesity may alter this balance. Evidence suggests increased activation of uNK cells, including enhanced cytotoxic potential and TNF-α production, as well as shifts in decidual macrophage polarization and changes in T cell subsets. Such alterations may disturb trophoblast invasion, spiral artery remodeling, and placental vascular development. Indeed, higher pre-pregnancy BMI has been associated with greater placental weight, reduced placental efficiency, inflammatory villous lesions, and impaired angiogenic signaling. Experimental models further support that obesity is linked to defective decidualization, altered uterine vascular remodeling, and changes in placental gene expression related to lipid metabolism, cytokine activity, and angiogenesis. Although compensatory immune mechanisms may partially restore vascular remodeling later in gestation, persistent immune activation could lower the threshold for maladaptation, particularly in the presence of additional inflammatory stimuli. Together with previously described hemodynamic, lipid, and endothelial alterations, these immune and placental changes reinforce the concept that maternal obesity establishes a pro-inflammatory and metabolically stressed intrauterine environment. Within the maternal cardiorenal–metabolic axis, this milieu may amplify vascular dysfunction, impair placentation, and contribute to both short-term obstetric complications and long-term cardiovascular risk [10].

Together, these renal, endothelial, metabolic, and immunological alterations define a state of subclinical cardiorenal–metabolic dysfunction that extends beyond the mother and shapes the intrauterine environment. This maladaptive milieu provides a biological framework linking maternal obesity with fetal programming mechanisms that may predispose the offspring to future cardiovascular and kidney disease. To better illustrate and structurally summarize these interconnected mechanisms, Table 1 presents a schematic overview of the key components of maternal cardiorenal–metabolic dysfunction described above.

Table 1. Maternal Cardiorenal–Metabolic Dysfunction in Obese Pregnancy

Section / Domain	Main Mechanisms	Key Clinical or Subclinical Findings	Implications within the Maternal Cardiorenal–Metabolic Axis	References
Hemodynamic adaptations in obese pregnancy	Increased circulating blood volume, elevated stroke volume and cardiac output, higher systemic and pulmonary pressures, concentric LV remodeling, increased LV mass and relative wall thickness	Higher BP and heart rate; reduced LV global longitudinal strain; impaired diastolic indices despite preserved ejection fraction; persistence of abnormalities postpartum in some phenotypes	Obesity amplifies physiological gestational volume overload, unmasks pre-existing cardiovascular vulnerability, and promotes subclinical myocardial maladaptation	[5], [6], [7]
Hypertensive phenotype variability	Distinct hemodynamic patterns in pregnancies complicated by hypertensive disorders and fetal growth restriction versus appropriately grown fetuses	Lower cardiac output with concentric remodeling vs. higher cardiac output with increased vascular resistance and diastolic dysfunction	Pre-existing metabolic risk profile shapes hemodynamic trajectory and may predispose to sustained cardiovascular dysfunction	[6]
Renal alterations (subclinical cardiorenal dysfunction)	Obesity-associated glomerular hyperfiltration, hyperinsulinemia, chronic low-grade inflammation, endothelial dysfunction; physiological pregnancy-related rise in GFR	Higher first-trimester BMI associated with increased albuminuria later in pregnancy (independent of gestational weight gain)	Suggests early renal stress and subclinical cardiorenal dysfunction; mild albuminuria linked to future CVD and CKD risk	[8]
Endothelial dysfunction and lipid alterations	Oxidized LDL, triglyceride-rich lipoproteins, altered HDL functionality, impaired nitric oxide bioavailability, increased arterial stiffness, altered angiogenic balance	Subtle endothelial impairment in uncomplicated pregnancy; more pronounced dysfunction in hypertensive disorders	Intersection of physiological hyperlipidemia and metabolic stress promotes vascular maladaptation	[9]
Inflammatory and immune dysregulation	Chronic adipose tissue–derived inflammation; altered uNK cell activation; macrophage polarization; impaired trophoblast invasion and spiral artery remodeling	Increased placental weight, inflammatory villous lesions, impaired angiogenic signaling	Establishes a pro-inflammatory intrauterine environment contributing to vascular dysfunction and placental maladaptation	[10]
Integrated subclinical cardiorenal–metabolic dysfunction	Interaction of hemodynamic overload, renal stress, endothelial activation, metabolic dysregulation, and immune imbalance	Increased susceptibility to hypertensive and uteroplacental complications	Defines a maladaptive maternal phenotype shaping the intrauterine environment prior to fetal programming	[5–10]

3. Fetal and Offspring Consequences: Programming of Future Cardiorenal Disease

3.1. Renal and Vascular Programming

The number of nephrons formed before birth is crucial for lifelong kidney health. In humans, nephrogenesis is completed by 32–36 weeks of gestation, and no new nephrons are formed afterward. Lower nephron endowment, which is associated with low birth weight, prematurity, or intrauterine growth restriction, increases the risk of hyperfiltration, glomerular hypertrophy, hypertension, and eventually chronic kidney disease (CKD). Maternal obesity is an important factor contributing to CKD risk in offspring. Key mechanisms include inflammation, oxidative stress, and lipid disturbances. Offspring of obese mothers are more prone to increased fat accumulation, adipocyte hypertrophy, and long-term weight gain due to upregulation of genes involved in adipogenesis and lipogenesis. This leads to greater visceral fat deposition and metabolic disturbances such as insulin resistance. Lipid accumulation in the kidneys, along with increased inflammation and fibrosis, contributes to impaired renal function. Additionally, altered levels of adipokines play a role: adiponectin levels are reduced, promoting insulin resistance, while elevated leptin levels are associated with leptin resistance [11].

The placenta plays a central role in developmental programming by regulating the transfer of oxygen and nutrients between mother and fetus. Maternal obesity alters placental structure and function, affecting nutrient delivery. Glucose crosses the placenta mainly via GLUT1 transporters, and increased maternal or fetal insulin levels can enhance fetal glucose exposure through a mechanism known as “glucose steal.” The placenta is also highly permeable to free fatty acids. In maternal obesity, elevated maternal lipids and increased expression of placental fatty acid transporters result in greater lipid transfer to the fetus, contributing to metabolic alterations that may increase the risk of CKD later in life [11].

Recent human data addressing the relationship between maternal adiposity and offspring kidney development remain limited, particularly in Indigenous populations at high risk of CKD. One observational study conducted in an Indigenous Australian cohort examined fetal kidney growth after 26 weeks of gestation and early postnatal kidney function. Although greater maternal adiposity and higher pre-pregnancy BMI were associated with increased estimated fetal weight, absolute fetal kidney size in late gestation was not significantly related to maternal adiposity after adjustment for relevant confounders. Importantly, fetal kidney volume relative to estimated fetal weight was inversely associated with maternal adiposity, suggesting a disproportion between overall fetal growth and renal development. Given that neonatal kidney volume is considered a surrogate marker of nephron number, these findings raise the possibility that offspring of mothers with higher adiposity may have relatively reduced nephron endowment in proportion to body size. Such a mismatch may predispose to compensatory glomerular hyperfiltration and subsequent structural changes, providing a potential mechanistic link to increased CKD risk later in life. However, no association was observed between maternal adiposity and kidney function in early childhood, indicating that adverse renal consequences may not become clinically apparent until later stages of life. Methodologically, the study was strengthened by detailed fetal ultrasound assessment and the use of bioelectrical impedance analysis to characterize maternal body composition beyond BMI alone. Nevertheless, the relatively small sample size and limited follow-up highlight the need for larger, longitudinal studies to clarify the long-term renal implications of exposure to an obesogenic intrauterine environment [12].

Beyond functional and metabolic programming, structural congenital abnormalities of the kidney and urinary tract (CAKUT) represent another pathway linking maternal health to long-term cardiorenal risk in offspring. Although genetic factors account for a minority of cases, increasing evidence suggests that maternal metabolic and endocrine disorders contribute substantially to disrupted nephrogenesis. Epidemiological data indicate that gestational diabetes and thyroid dysfunction are particularly associated with antenatally detected renal anomalies, including urinary tract dilatation and structural kidney abnormalities. Other maternal conditions, such as gestational hypertension, kidney disease, and gynecological disorders, have also been linked to specific CAKUT phenotypes. Maternal hyperglycemia may interfere with early kidney development, including ureteric bud branching and nephron formation, thereby predisposing to reduced nephron endowment. Similarly, thyroid dysfunction during pregnancy may alter fetal growth patterns and renal maturation. These observations align with the concept of the maternal cardiorenal-metabolic axis, in which disturbances in glucose homeostasis, endocrine balance, and vascular function during pregnancy shape fetal renal structure and long-term susceptibility to hypertension and CKD. Importantly, antenatal detection of renal anomalies through ultrasound provides an opportunity for early risk stratification [13].

Experimental evidence further strengthens the concept that maternal obesity exerts persistent structural and molecular effects on the offspring kidney. Animal models demonstrate that exposure to a maternal high-

fat diet induces early lipid accumulation in renal tissue, suppression of key metabolic regulators such as the farnesoid X receptor, and activation of inflammatory and fibrotic pathways, even in the absence of differences in birth weight. These alterations persist into adulthood and are exacerbated by additional postnatal metabolic stressors, supporting a “second-hit” hypothesis in which intrauterine exposure increases vulnerability to later renal injury. Mechanistically, impaired autophagy and reduced expression of sirtuin-1, a critical regulator of mitochondrial function, oxidative balance, and lipid metabolism, appear to contribute to obesity-related renal programming. In parallel, epigenetic modifications established during fetal development may mediate long-term changes in gene expression, linking early nutrient excess, placental dysfunction, and oxidative stress to sustained cardiorenal risk. Notably, some effects show sex-specific patterns, suggesting differential susceptibility. Importantly, these experimental findings also point toward potential therapeutic strategies. Glucagon-like peptide-1 receptor agonists, which improve metabolic control and have demonstrated cardiovascular and renal benefits in adults with type 2 diabetes, attenuated inflammation, oxidative stress, and fibrosis in offspring exposed to maternal obesity in preclinical models [14].

Taken together, the available data support the concept that maternal obesity initiates a complex cascade of metabolic, molecular, and epigenetic events that program lifelong susceptibility to cardiovascular and kidney disease. Understanding this maternal cardiorenal-metabolic axis may facilitate the development of targeted preventive strategies, including digital risk stratification tools and early-life interventions aimed at breaking the intergenerational cycle of cardiometabolic and renal disease. Integrating maternal metabolic and endocrine profiles into predictive models may enhance identification of high-risk pregnancies and enable targeted monitoring. Within the framework of digital biomarkers and precision medicine, combining maternal clinical data with imaging findings could improve early detection of offspring at increased risk of future cardiovascular and kidney disease. Nonetheless, prospective longitudinal studies are required to determine how prenatal structural abnormalities interact with later metabolic programming to influence lifelong cardiorenal outcomes.

3.2. Metabolic and Long-Term Cardiovascular Outcomes

Children born to women with obesity during pregnancy are at increased risk of obesity, type 2 diabetes, cardiovascular disease, congenital heart defects, and premature cardiovascular mortality. Although part of this risk may be explained by higher rates of hypertension and excess body weight in the offspring, experimental studies indicate that maternal obesity may directly impair fetal cardiovascular development. Animal models demonstrate persistent systolic and diastolic dysfunction, cardiac hypertrophy, and fibrosis in exposed offspring, independent of postnatal body weight. Human evidence, although limited and heterogeneous, supports the presence of early structural and functional cardiac differences. Meta-analyses show no difference in fetal interventricular septal thickness, but increased septal thickness in infancy among offspring of mothers with obesity. LVr mass, ventricular volumes, and relative wall thickness were generally similar between groups, although single studies reported higher LV mass and thicker ventricular walls in later childhood. Importantly, maternal obesity was consistently associated with reduced LVr strain in fetal life and infancy, indicating subclinical systolic dysfunction, while ejection fraction was largely preserved. Diastolic parameters did not differ in pooled analyses, although some studies described lower early and late diastolic myocardial velocities after birth. Together, these findings suggest that maternal obesity is associated with subtle but measurable alterations in offspring cardiac structure and function from early life, which may contribute to increased cardiovascular risk later in life [15].

Large-scale genome-wide association meta-analyses of own birth weight and offspring birth weight identified numerous independent genetic signals, substantially expanding the number of known loci [16]. By applying structural equation modelling, the investigators partitioned these associations into direct fetal genetic effects and indirect maternal genetic effects, accounting for the correlation between maternal and fetal genotypes. This approach demonstrated that birth weight is influenced by a combination of fetal-only effects, maternal-only effects, and loci with concordant or opposing maternal and fetal contributions. Genome-wide analyses further showed that maternal genetic effects on birth weight are strongly correlated with maternal genetic determinants of gestational duration, whereas fetal genetic effects are not, indicating partly distinct biological pathways. Enrichment analyses suggested tissue- and mechanism-specific contributions for maternal and fetal variants, although functional validation is still required. Using Mendelian randomization based on maternal- and fetal-specific genetic estimates, the study disentangled intrauterine effects from inherited genetic influences. Both maternal and fetal height-associated variants were causally linked to higher birth weight. In contrast, maternal genetic predisposition to higher fasting glucose increased offspring birth

weight, whereas fetal variants related to insulin secretion were associated with greater fetal growth, highlighting the central role of fetal insulin pathways. For blood pressure, maternal genetic predisposition to higher systolic BP was causally associated with lower offspring birth weight, while fetal BP variants primarily influenced adult BP rather than birth weight itself. Importantly, the inverse association between lower birth weight and higher adult BP appeared to be driven by shared genetic factors and maternal BP effects during pregnancy, rather than by long-term intrauterine programming alone. Overall, separating maternal and fetal genetic contributions provided new insight into the biological mechanisms regulating birth weight and clarified how early growth relates to later cardiometabolic risk [16].

Growing evidence indicates that maternal adiposity before conception represents a critical determinant of long-term cardiovascular risk in the offspring. Across observational cohorts, higher maternal pre-pregnancy BMI (MppBMI) has been consistently associated with adverse cardiometabolic phenotypes from birth through adulthood, supporting the concept of early-life programming within the maternal cardiorenal–metabolic axis. The most robust associations concern offspring adiposity and BP, with a graded increase in risk across maternal BMI categories. In neonates, higher MppBMI correlates with altered metabolic markers, including elevated insulin concentrations. During childhood, these associations extend to greater fat mass and higher systolic and diastolic BP, while findings related to lipid profiles and cardiac structure remain heterogeneous. In adulthood, maternal obesity is linked to higher offspring BMI and, in some cohorts, increased risk of hypertension, type 2 diabetes, and cardiovascular events, although part of this effect appears mediated by adult adiposity. Although residual confounding cannot be excluded, the overall pattern aligns with experimental and mechanistic data suggesting that maternal metabolic status before and in early pregnancy may influence offspring cardiovascular trajectories independently of shared postnatal environment [17].

Given the consistent associations between maternal adiposity and adverse cardiometabolic trajectories in the offspring, an important question is whether these risks are modifiable through maternal lifestyle interventions before or during pregnancy. Follow-up analyses from randomized controlled trials evaluating dietary and physical activity interventions in women with overweight or obesity provide preliminary insight into this issue. Although limited by modest sample sizes and substantial attrition, available follow-up studies suggest that maternal lifestyle intervention may attenuate early cardiac remodeling in the offspring. Reduced interventricular septal thickness and lower LV mass indices have been observed in some intervention groups, accompanied by subtle differences in ventricular geometry on advanced imaging. Functional findings indicate modest improvements in systolic performance, including higher ejection fraction and myocardial systolic velocities, while diastolic parameters were largely comparable between groups. No consistent effects were demonstrated for BP or arterial stiffness in early childhood. Overall, interventional data remain limited and heterogeneous; however, they support the concept that elements of offspring cardiovascular programming associated with maternal obesity may be at least partially modifiable. Whether these early structural and functional differences translate into meaningful long-term reductions in cardiovascular risk requires confirmation in larger cohorts with extended follow-up [18].

In a large population-based cohort including 28,540 women and 37,709 offspring, 21% of mothers were overweight and 4% were obese. Maternal obesity was associated with older age, higher parity, and lower socioeconomic status. Offspring of obese women had higher birth weight. Over long-term follow-up, 6,551 deaths occurred among the offspring, with cardiovascular disease representing the leading cause in men and a major cause in women. Maternal overweight and obesity were significantly associated with increased all-cause and premature mortality in adult offspring. These associations persisted after adjustment for gestational age at weight measurement, maternal age, social class, offspring age and sex, and birth weight. No independent association was observed between birth weight and all-cause mortality, and no interaction between birth weight and maternal obesity was detected. In addition, 7.6% of offspring had at least one hospital admission for a cardiovascular event. Maternal overweight and obesity were associated with a higher risk of composite cardiovascular hospitalizations in the offspring, with similar patterns observed in analyses restricted to women whose body mass index was recorded in early pregnancy. Spline modelling suggested a non-linear association between maternal body mass index and offspring mortality, with the lowest risk observed at maternal body mass index values between 24 and 28. These findings support the concept that maternal adiposity is linked to long-term cardiovascular risk in the offspring, independent of measured socioeconomic confounders. Proposed mechanisms include intrauterine overnutrition, fetal hyperinsulinaemia, chronic low-grade inflammation, alterations in neuroendocrine regulation, structural and functional vascular changes, and epigenetic modifications. Although shared genetic and postnatal lifestyle factors cannot be excluded, the persistence of

associations after multivariable adjustment and the consistency with experimental data reinforce the hypothesis that maternal obesity contributes to developmental programming of later cardiovascular disease [19].

Human cohort studies similarly demonstrate that higher maternal pre-pregnancy BMI is associated with higher systolic BP, greater adiposity, and adverse metabolic profiles in children and young adults. Large population-based analyses from Scandinavia and Israel further show dose–response relationships between maternal BMI and offspring cardiovascular morbidity, including cerebrovascular disease, ischemic heart disease, heart failure, and increased cardiovascular hospitalizations. Maternal overweight and obesity have also been linked to higher all-cause and cardiovascular mortality in adult offspring, independent of major confounders. Evidence from fetal and neonatal imaging studies indicates that maternal obesity is associated with early structural and functional cardiac alterations, including impaired myocardial strain, increased septal thickness, altered ventricular geometry, and increased LV mass in childhood. In addition, several large registry-based studies report increased risks of selected congenital heart defects with rising maternal BMI. Proposed mechanisms include intrauterine overnutrition, fetal hyperinsulinemia, chronic low-grade inflammation, oxidative stress, placental dysfunction, altered adipokine signaling, sympathetic overactivation, and epigenetic modifications. Collectively, these data support the concept that maternal obesity contributes to long-term cardiovascular risk in the offspring through early metabolic and cardiac programming, although the relative contributions of intrauterine, genetic, and shared environmental factors require further clarification [20].

In summary, converging evidence from experimental models, imaging studies, genetic analyses, interventional trials, and large population-based cohorts indicates that maternal adiposity before and during pregnancy is associated with subtle alterations in fetal cardiac development, higher offspring adiposity and BP, and increased long-term cardiovascular morbidity and mortality. These findings support a developmental programming framework in which intrauterine metabolic and hemodynamic exposures interact with maternal and fetal genetic factors to shape lifelong cardiometabolic trajectories. Given the early onset and often subclinical nature of these alterations, there is a clear need for innovative approaches to identify at-risk individuals across generations. This provides the rationale for exploring digital biomarkers and data-driven strategies for intergenerational cardiovascular risk prediction.

4. Digital Biomarkers and Intergenerational Risk Prediction

4.1 Continuous Maternal-Fetal Monitoring via Wearables

Maternal health, encompassing pregnancy, childbirth, and the postpartum period, remains a global priority, as preventable complications continue to contribute substantially to maternal morbidity and mortality. Continuous monitoring of maternal and fetal status throughout gestation is therefore critical. In recent years, wearable and mobile sensing technologies have emerged as potential digital biomarkers capable of capturing physiological signals related to both maternal and fetal health in real time.

Current systems primarily focus on fetal electrocardiography (ECG) fetal heart rate, and fetal movement, as well as maternal parameters such as heart rate, stress level, temperature, oxygen saturation, and physical activity. Data acquisition typically relies on abdominal and chest electrodes, inertial measurement units, accelerometers, Doppler ultrasound sensors, and photoplethysmography, with signals transmitted to smartphones or computers for storage and analysis. Signal preprocessing commonly includes band-pass and notch filtering, artifact removal, and feature extraction, followed by machine learning–based classification or prediction models. Although reported diagnostic performance in several studies reached acceptable accuracy levels, most devices were evaluated in controlled or short-term settings, with limited sample sizes and without continuous monitoring across the entire pregnancy. These limitations highlight the need for validation in free-living conditions and longitudinal designs. Nevertheless, the integration of wearable-derived physiological metrics with advanced data analytics represents a promising step toward scalable digital biomarkers that may improve early risk detection and contribute to intergenerational cardiometabolic risk prediction [21].

A large U.S.-based survey of women of childbearing age further illustrates the readiness for integrating wearable ECG monitoring into routine prenatal care [22]. Participants were recruited across most states, achieving a high response rate. Approximately half reported plans for pregnancy within the next five years, and among these women the vast majority expressed interest in using a wearable device for continuous maternal–fetal monitoring at home, during sleep, and in daily life. Most respondents indicated willingness to wear the device continuously or during defined periods and to purchase it within a moderate consumer price range. ECG-based monitoring offers potential advantages over conventional ultrasound, as it enables continuous, real-time assessment rather than episodic, operator-dependent measurements. Such systems may provide more detailed characterization of maternal–fetal physiology. However, technical challenges remain,

particularly related to signal attenuation and low signal-to-noise ratios caused by maternal tissues and amniotic fluid, as well as the higher intrinsic fetal heart rate, which complicates signal extraction. The implementation of continuous monitoring may also carry psychosocial implications. Increased awareness of fetal status could promote healthier maternal behaviors, yet may also heighten anxiety or increase demand for clinical reassurance. Despite these considerations, survey findings suggest broad acceptance of telemedicine-based antepartum monitoring, supported by declining hardware costs and the availability of mature software solutions. Given that the pregnant woman and fetus function as a single interconnected physiological system, continuous dual monitoring through wearable ECG technology represents a plausible next step in advancing digital biomarkers for maternal–fetal risk stratification [22].

Within this context, digital lifestyle interventions have been explored as tools to support weight management among pregnant women with overweight and obesity. The SLIM (Supporting Lifestyle Change in Pregnant Mothers with Obesity through the Wearable Internet-of-Things) intervention was developed in collaboration with public health nurses and pregnant women, and integrated wearable technology into routine maternity care with the aim of enhancing self-efficacy and optimizing gestational weight gain.

In a cohort of 55 enrolled women (54 included at baseline; median BMI 30), most entered the study in the first trimester [23]. Despite the structured intervention and wearable-based monitoring, self-efficacy in eating and physical activity remained stable over time, with no statistically significant improvement. Baseline self-efficacy levels were already relatively high, which may have limited detectable change. Depressive symptoms were associated with lower eating-related self-efficacy, while higher education and lower smart ring non-wear time were associated with higher physical activity self-efficacy. However, fidelity indicators, including device wear time and food diary completion, were not associated with GWG. The intervention did not reduce excessive GWG. A substantial proportion of women exceeded recommended weight gain ranges, although those who gained within or below recommendations were markedly more likely to reduce weight postpartum. Women over 30 years of age were more likely to achieve optimal gestational weight gain. These findings reinforce the importance of recommended weight gain targets for long-term weight trajectories, even when behavioral self-efficacy remains unchanged. From a digital biomarker perspective, the study highlights both the promise and the limitations of wearable-supported perinatal interventions. Implementation in real-world maternity clinics was feasible, yet constrained by limited consultation time, organizational factors, and the absence of randomization or control groups. Importantly, psychosocial determinants—including depressive symptoms and educational level—emerged as modifiers of behavioral outcomes, underscoring the need for individualized, data-informed approaches. Future randomized trials are required to determine whether refinement of wearable-integrated strategies can more effectively influence gestational weight gain and, consequently, intergenerational cardiometabolic risk [23].

Remote BP monitoring represents one of the most clinically actionable digital tools in cardio-obstetrics. Home BP devices integrated with telehealth platforms enable longitudinal assessment of maternal hemodynamics beyond episodic office measurements, improving early detection and management of hypertensive disorders. In post-partum women, particularly after gestational hypertension or pre-eclampsia, remote BP monitoring combined with structured follow-up and medication adjustment has achieved better BP control and higher follow-up completion rates than standard care. Home measurements also correlate more closely with cardiovascular risk than clinic readings, strengthening their role in risk stratification. Emerging cuff-less and wearable technologies may further enhance continuous BP assessment, although validation in pregnant populations remains necessary. While implementation barriers and disparities in access persist, remote BP monitoring constitutes a scalable digital biomarker approach with potential to modify long-term maternal cardiovascular trajectories and, indirectly, intergenerational risk [24].

Evidence from studies in women with gestational diabetes mellitus indicates that continuous glucose monitoring (CGM) enhances metabolic insight by capturing 24-hour glucose profiles and identifying otherwise undetected hyperglycaemic excursions, particularly fasting elevations. Visualization of glucose trends supports individualized dietary adjustments and may reduce glycaemic variability over time, reinforcing behavioral change. However, discrepancies between interstitial and capillary measurements, alarm burden, sensor discomfort, and fragmented data platforms can reduce usability and trust. Thus, while CGM provides a clinically meaningful digital biomarker in pregnancy, its effectiveness depends on user-centered design, seamless data integration, and translation of glucose variability into actionable, risk-relevant insights for long-term cardiometabolic prediction [25].

Recent progress in bioengineered wearables and portable diagnostics underscores the expanding role of digital biomarkers in women’s health, particularly in the context of pregnancy-related metabolic and

hypertensive disorders. Beyond cardiovascular and glycaemic parameters, emerging platforms increasingly incorporate renal biomarkers that are highly relevant to cardio-obstetric risk. Markers such as cystatin C, uric acid, creatinine surrogates, and electrochemically detected metabolites associated with renal stress enable continuous or repeated assessment of kidney function, which is closely linked to hypertensive disorders of pregnancy and long-term cardiovascular risk. Miniaturized electrochemical and microfluidic sensors, integrated into skin-adherent devices or textile-based systems, allow multiplex detection of renal and metabolic biomarkers from interstitial fluid, sweat, or other accessible biofluids. When combined with physiological data streams—such as heart rate, activity, sleep, and BP—these platforms provide a multidimensional representation of maternal cardiorenal adaptation. However, their clinical utility depends not only on analytical sensitivity and stability, but also on robust data interpretation, user adherence, interoperability with electronic health records, and equitable access across diverse populations. When combined with cardiovascular and metabolic streams, renal digital phenotyping strengthens intergenerational risk prediction frameworks. The next step involves integrating these multimodal data into machine learning–based predictive models for personalized obstetric and long-term cardiovascular risk stratification [26].

4.2 AI-Powered Digital Biomarkers in Pregnancy

Heart rate variability (HRV) represents a promising autonomic digital biomarker within the maternal cardiorenal–metabolic axis. As a non-invasive measure of autonomic regulation, HRV has been widely applied in cardiovascular risk assessment and early disease detection. In pregnancy, growing interest has focused on maternal HRV (mHRV) as a potential tool for earlier identification of complications associated with autonomic dysfunction, including hypertensive disorders. Because many clinical manifestations—such as overt BP elevation—occur relatively late in gestation, mHRV may offer a window for earlier risk stratification. Regression-based analyses in healthy pregnant populations demonstrate that mHRV is significantly influenced by maternal and cardiorespiratory characteristics. These findings have direct implications for AI-driven risk modeling. Given the strong effects of gestational age, heart rate, and respiratory dynamics—as well as the large inter-subject variability—mHRV-based prediction models should prioritize longitudinal, personalized trajectories rather than absolute thresholds derived from population norms. Repeated measurements and mixed-effects modeling frameworks better capture dynamic autonomic adaptation across gestation and provide a more suitable foundation for machine learning–based classifiers. Within the broader framework of digital biomarkers, mHRV exemplifies the transition from episodic assessment to continuous, individualized phenotyping. When integrated with multimodal physiological streams and analyzed using advanced computational approaches, autonomic metrics may contribute to earlier identification of maladaptive maternal cardiovascular patterns, thereby refining intergenerational cardiometabolic and renal risk prediction [27].

Beyond autonomic indices, artificial intelligence has also been applied to arterial pulse–derived signals acquired from wearable piezoelectric sensors. After wireless transmission, pulse wave features are processed using machine learning pipelines that include unsupervised clustering, regression models, and deep neural networks. Among conventional approaches, random forest regression demonstrated the highest predictive accuracy for cardiovascular parameters, while deep learning–based pattern recognition achieved high classification performance for age, BMI, and vascular indices such as augmentation and reflection metrics. Within the maternal cardiorenal–metabolic axis, such AI-driven analysis enables automated detection of subtle changes in vascular stiffness and hemodynamic adaptation that may precede overt clinical manifestations. Importantly, model performance was supported by receiver operating characteristic analyses, underscoring the robustness of classification. These findings illustrate how continuous signal acquisition combined with machine learning transforms raw physiological data into clinically interpretable digital biomarkers, supporting scalable, remote cardiovascular risk stratification in pregnancy [28].

Invasive catheter-based BP monitoring remains the reference standard but is limited to critical care, while cuff-based methods provide only intermittent measurements. These constraints have driven the development of cuff-less, AI-supported approaches based primarily on photoplethysmography, alone or combined with ECG. Traditional surrogate markers such as pulse transit time and pulse arrival time rely on biophysical assumptions and repeated calibration, limiting long-term robustness. Machine learning reframes BP estimation as a data-driven prediction task, using temporal and morphological pulse wave features. Conventional models (e.g., regression, support vector machines, random forests) show acceptable accuracy in controlled or homogeneous datasets but often lack generalizability. Recurrent and deep learning architectures better capture temporal cardiovascular dynamics and demonstrate improved stability in longitudinal data, though validation in diverse, real-world pregnant populations remains limited. Overall, AI-enabled cuff-less

BP monitoring represents a promising digital biomarker within the maternal cardiorenal–metabolic axis. However, clinical translation requires larger cohorts, standardized validation, and integration with multimodal obstetric and metabolic data to support reliable intergenerational risk prediction [29].

To better illustrate the interconnections between the functionality of digital biomarkers within the cardiorenal–metabolic axis, we developed a simplified schematic representation (Figure 1). This conceptual framework is intended to clarify the complex and multidirectional relationships among cardiovascular, renal, and metabolic pathways, highlighting how digital biomarkers can capture dynamic physiological changes across these interconnected systems. By integrating diverse data streams into a unified model, the schematic facilitates a clearer understanding of shared mechanisms, overlapping risk factors, and potential points of clinical intervention.

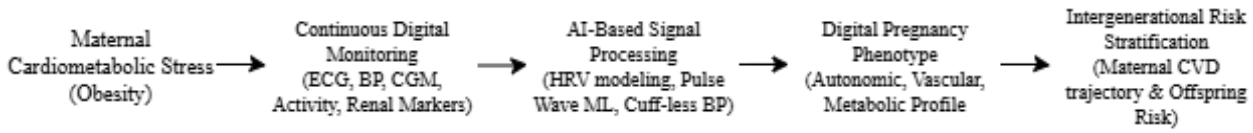

Fig. 1. *Digital Biomarkers in the Maternal Cardiorenal–Metabolic Axis*

Conclusions

Maternal obesity disrupts the maternal cardiorenal–metabolic axis through hemodynamic, renal, endothelial, metabolic, and inflammatory pathways, creating an intrauterine environment that contributes to fetal programming of future cardiovascular and kidney disease. Evidence from clinical, experimental, and population-based studies supports the concept that these alterations may initiate intergenerational cardiometabolic risk long before overt disease becomes clinically apparent. Digital biomarkers—derived from wearable cardiovascular, glycaemic, autonomic, and emerging renal monitoring—offer a shift from episodic assessment to continuous, individualized phenotyping during pregnancy. When integrated with artificial intelligence–based analytics, these technologies have the potential to enhance early risk stratification and personalize preventive strategies.

Nevertheless, large longitudinal studies, standardized validation, and equitable implementation are essential before routine clinical adoption. Linking maternal digital phenotypes with long-term offspring outcomes remains the critical next step in translating the maternal cardiorenal–metabolic framework into effective intergenerational prevention.

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Abbreviations

AI - Artificial Intelligence
BMI - Body Mass Index
BP - Blood Pressure
CAKUT - congenital abnormalities of the kidney and urinary tract
CMG - continuous glucose monitoring
CKD - chronic kidney disease
CVD - Cardiovascular disease
ECG - electrocardiography
GWG - gestational weight gain
HDL - high-density lipoprotein
HRV - heart rate variability
LDL - low-density lipoprotein
LV - left ventricular
mHRV - maternal heart rate variability
MppBMI - maternal pre-pregnancy BMI
WHO -World Health Organization

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