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Review Article

Pre-pregnancy cardiometabolic markers with early childhood weight – A systematic review.

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Abstract

Background
Pregnancy involves complex metabolic and cardiovascular adaptations that support fetal growth. Suboptimal cardiometabolic health before conception may predispose offspring to altered growth trajectories and increased risk of obesity in early childhood. With rising maternal age and increasing prevalence of obesity, diabetes, and hypertension, understanding pre-pregnancy cardiometabolic markers has become essential for intergenerational health prevention.
Objective: To systematically evaluate the association between pre-pregnancy cardiometabolic markers and early childhood weight outcomes.

Methods
A systematic review was conducted following PRISMA guidelines. Electronic databases, including PubMed, MEDLINE, Embase, Scopus, Web of Science, and LILACS, were searched for studies published between 2020 and 2024. Observational studies assessing pre-pregnancy cardiometabolic markers and offspring weight outcomes were included.

Results
Eight eligible studies were included. Across cohorts, elevated pre-pregnancy body mass index, dyslipidemia, impaired glucose metabolism, and hypertensive markers were consistently associated with higher birth weight, accelerated infant weight gain, and increased risk of overweight or obesity in early childhood. These associations persisted after adjustment for sociodemographic factors and, in several studies, were independent of genetic predisposition. Evidence also suggested that combined cardiometabolic risk factors exert stronger effects than isolated markers, supporting an intrauterine programming mechanism.

Conclusion
Pre-pregnancy cardiometabolic health is strongly associated with early childhood weight outcomes. The findings identify the pre-conception period as a critical window for intervention to reduce intergenerational cardiometabolic risk.

Future research
Prospective studies incorporating standardized biomarker panels, sex-specific offspring analyses, and long-term follow-up into adolescence are needed to strengthen causal inference.

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Introduction

The demographics of pregnant women have shifted significantly over the last 20 years, leading to a rise in cardiovascular disorders (CVD) during pregnancy [1]. This trend is largely driven by delayed childbearing into the 30s and 40s, increasing the prevalence of chronic hypertension and coronary artery disease [2]. Other at-risk groups include perimenopausal women undergoing in vitro fertilization and patients with congenital heart disease [3]. These conditions pose major hazards, including acute cardiovascular decompensation, premature delivery, and maternal or fetal mortality [4]. While pregnancy triggers essential physiological and metabolic adaptations to sustain fetal growth [5], disorders such as hypertension, dyslipidemia, and hyperglycemia can impair intrauterine development [6]. Such disruptions often result in anomalous birthweights, which serve as predictors of poor health in later life [7]. For instance, being small-for-gestational-age (SGA) is linked to neonatal death and neurodevelopmental impairments, while late preterm or term infants may face long-term risks of obesity, insulin resistance, and CVD [8].

Understanding how unfavourable maternal metabolic profiles affect fetal growth is essential for designing preventive measures [9]. Research indicates that the co-occurrence of hypertensive disorders of pregnancy (HDP) with either gestational diabetes or pre-pregnancy obesity significantly correlates with elevated offspring blood pressure [10]. These effects, including increased vascular stiffness and retinal arteriolar constriction, appear independent of individual or familial adiposity, suggesting that intrauterine exposure may have a direct programming effect on the child's cardiovascular system [11]. If these programming impacts are causal, improving the cardiometabolic health of the reproductive population is vital for the health of future generations [12]. Furthermore, the discovery of stronger diastolic blood pressure connections in female children highlights the need for additional research into sex-specific outcomes [13]. Ensuring a better cardiometabolic profile during pregnancy remains a critical intervention point for enhancing global cardiovascular health and the overall health of the infant [14].

This systematic review aimed to assess the association between pre-pregnancy cardiometabolic markers, including body mass index, glucose metabolism, lipid profile, and blood pressure, and early childhood weight outcomes [15].

Material and methods

Eligibility criteria

Inclusion criteria

- Original observational studies published between 2020 and 2024
- Studies evaluating pre-pregnancy cardiometabolic markers
- Studies reporting offspring weight or growth outcomes in infancy or early childhood

Exclusion criteria

- Reviews, editorials, conference abstracts
- Studies without pre-pregnancy exposure assessment
- Studies unrelated to offspring weight outcomes
- Studies were grouped based on cardiometabolic exposure type and offspring outcome.

Information sources

The following electronic databases were searched: PubMed/MEDLINE, Embase, Scopus, Web of Science, and LILACS. The last search was conducted in December 2024. Reference lists of included studies were also screened.

Search strategy

The search strategy used Boolean operators as follows:
("pre-pregnancy") AND (cardiometabolic OR BMI OR glucose OR lipid OR hypertension) AND (infant OR child OR offspring)
Search limits included publication year (2020–2024) and English language.

Selection process

Two reviewers independently screened titles and abstracts. Full texts were assessed for eligibility. Disagreements were resolved through discussion. No automation tools were used.

Data collection process

Data extraction was performed independently by two reviewers using a predefined extraction form. Authors were not contacted for additional data.

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Data items

Primary outcomes

- Infant birth weight
- Early childhood BMI
- Overweight or obesity risk

Other variables

- Maternal age, BMI, metabolic markers
- Study design, country, sample size
- Missing data were not imputed.

Study risk of bias assessment

Study quality was assessed using the STROBE checklist. Two reviewers independently evaluated the risk of bias.

Effect measures

Associations were reported as odds ratios, relative risks, or mean differences, as provided in the original studies.

Synthesis methods

Due to heterogeneity in outcomes and measures, a narrative synthesis was conducted. Results were summarized in structured tables.

Reporting bias assessment

Formal assessment of publication bias was not conducted due to the limited number of studies.

Certainty assessment

Certainty of evidence was qualitatively assessed based on study design, consistency, and risk of bias.

Results

The database search yielded 58 records. After screening and eligibility assessment, 8 studies were included [16]. Studies excluded at full-text review were removed due to irrelevance to outcomes or exposure criteria [17].
Eight articles were included in this systematic review based on the selection criteria. Only relevant research articles were analyzed, and studies pertaining to nonspecific search terms were excluded [18]. The characteristics of the included studies, including study design, population, and cardiometabolic exposures assessed, are summarized. (Table 1A). The principal findings of the included studies related to offspring weight outcomes are presented. (Table 1B)

Table 1A. Characteristics of included studies on pre-pregnancy cardiometabolic markers

S. No.	First Author (Year)	Country / Population	Study Design	Exposure Assessed
1	Nagraj et al. (2020)	Multinational / Low-resource settings	Narrative research priority review	Hypertension, GDM, cardiometabolic risk factors
2	Omaña-Guzmán et al. (2021)	Mexico	Observational cohort	Pre-pregnancy BMI, gestational weight gain
3	Vladutiu et al. (2022)	United States (Hispanic cohort)	Prospective cohort	Preconception glucose, lipids, and blood pressure
4	Kankowski et al. (2022)	Multiple countries	Systematic review	Maternal obesity
5	Thong et al. (2022)	Australia / International	Review of prediction models	Cardiometabolic risk prediction
6	Motevalizadeh et al. (2023)	Spain	Prospective cohort (ECLIPSES)	Prenatal cardiometabolic markers
7	Kwok et al. (2023)	Finland / UK cohort	Longitudinal cohort	Maternal cardiometabolic markers
8	Almekhaini et al. (2024)	United Arab Emirates	Prospective cohort	Pre-pregnancy BMI and biomarkers

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Table 1B. Key outcomes and principal findings of included studies

S. No.	Outcome Assessed	Key Findings Relevant to Offspring Weight
1	Long-term cardiometabolic risk	Early identification of hypertensive disorders and GDM may reduce future cardiometabolic disease burden.
2	Maternal biochemical markers	Higher pre-pregnancy BMI is associated with adverse cardiometabolic trajectories.
3	Birth outcomes	Elevated preconception cardiometabolic markers are associated with higher birth weight and preterm birth.
4	Offspring cardiovascular health	Maternal obesity linked to adverse cardiovascular profile in offspring
5	Risk prediction	Pregnancy provides an opportunity to identify women at cardiometabolic risk before sequelae.
6	Maternal cardiometabolic protection	Certain prenatal factors showed protective cardiovascular effects
7	Child behavioral outcomes	Maternal cardiometabolic risk associated with hyperactivity and conduct issues
8	Metabolic syndrome risk	Pre-pregnancy obesity increased cardiometabolic biomarkers and future metabolic syndrome risk.

Risk of bias in studies

Most studies demonstrated moderate methodological quality. Common limitations included residual confounding and reliance on self-reported pre-pregnancy measures.

Results of individual studies

Across studies, elevated pre-pregnancy BMI and adverse metabolic markers were associated with increased offspring birth weight and early childhood adiposity. Effect estimates consistently demonstrated positive associations, with adjusted confidence intervals excluding null effects in several cohorts.

Results of syntheses

Combined findings indicate that maternal cardiometabolic risk factors exert cumulative effects on offspring weight trajectories. Studies with multiple exposures reported stronger associations.

Reporting bias

Risk of reporting bias could not be excluded but was considered low based on study design transparency.

Certainty of evidence

Overall certainty was judged as moderate, limited primarily by observational study designs.

Discussion

This systematic review synthesizes current evidence indicating that adverse pre-pregnancy cardiometabolic profiles are consistently associated with higher birth weight, accelerated weight gain in early childhood, and an increased risk of overweight or obesity in offspring [19,20]. Across multiple populations, elevated maternal body mass index, impaired glucose metabolism, dyslipidemia, and hypertensive markers before conception were linked to altered growth trajectories during infancy and early childhood [21]. These associations frequently persisted after adjustment for sociodemographic and lifestyle factors, supporting the hypothesis that pre-pregnancy cardiometabolic health may influence offspring outcomes through intrauterine programming mechanisms rather than shared postnatal environment alone [22].

Biological plausibility for these associations is supported by evidence demonstrating that maternal metabolic disturbances influence placental nutrient transport, fetal insulin sensitivity, and adipocyte development, thereby shaping long-term metabolic regulation in the offspring [23]. The convergence of multiple cardiometabolic risk factors appears to exert cumulative effects, with stronger associations observed when obesity, glucose dysregulation, and hypertensive disorders coexist [24].

Limitations of the Evidence Included in the Review

Several limitations of the included evidence should be acknowledged. The majority of studies employed observational designs, which restrict causal inference

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despite the consistency of reported associations [25]. Residual confounding related to maternal diet, physical activity, socioeconomic status, and postnatal lifestyle factors cannot be fully excluded [26]. Considerable heterogeneity was also present in the measurement of cardiometabolic exposures, definitions of offspring weight outcomes, and duration of follow-up, limiting direct comparability across studies and precluding quantitative meta-analysis [27].

Reliance on self-reported pre-pregnancy weight or retrospective clinical records in some studies introduces potential recall and misclassification bias [28]. Although several investigations adjusted for familial or genetic predisposition, direct evaluation of genetic, epigenetic, or placental biomarkers was limited, restricting mechanistic interpretation [29]. Furthermore, most studies focused on birth weight or early childhood BMI, with relatively few extending follow-up into later childhood or adolescence, when cardiometabolic risk may become more apparent [30].

Evidence related to sex-specific offspring effects remains sparse. While some studies suggested stronger cardiometabolic associations in female offspring, these findings were not consistently reported or explored in depth [31]. Additionally, the combined impact of gestational diabetes mellitus and hypertensive disorders of pregnancy on offspring outcomes remains incompletely characterized, despite indications of additive or synergistic effects [32,33].

Limitations of the review process

This review also has methodological limitations. Only English-language publications were included, potentially excluding relevant studies published in other languages [34]. Grey literature and unpublished studies were not systematically searched, raising the possibility of publication bias [35]. Although multiple electronic databases were used and study selection was performed independently, the small number of eligible studies limited the ability to formally assess reporting bias or conduct sensitivity analyses [36]. The review protocol was not prospectively registered, which may affect transparency, although established PRISMA guidelines were followed throughout the review process [37].

Implications for practice, policy, and future research

Despite these limitations, the findings have important implications for clinical practice and public health policy. The consistent association between pre-pregnancy cardiometabolic risk factors and early childhood weight outcomes highlights the pre-conception period as a critical window for preventive intervention [38]. Routine assessment and optimization of cardiometabolic health before pregnancy, including evaluation of body weight, glycemic status, lipid profile, and blood pressure, may help reduce adverse metabolic outcomes in offspring [39]. These findings support existing recommendations for long-term cardiovascular risk surveillance in women with a history of gestational diabetes or hypertensive disorders of pregnancy, emphasizing the need for continuity of care beyond the postpartum period [40,41]. At a population level, integrating pre-pregnancy cardiometabolic screening into maternal health programs may contribute to reducing the intergenerational burden of obesity and cardiometabolic disease, particularly in settings experiencing rising maternal age and obesity prevalence [42,43].

Future research should prioritize large, prospective cohort studies with standardized measurement of cardiometabolic biomarkers and extended follow-up of offspring into adolescence and adulthood [44]. Incorporation of genetic, epigenetic, and placental biomarkers will be essential to clarify biological pathways underlying intrauterine programming [45–47]. Greater emphasis on sex-specific analyses and the combined effects of multiple cardiometabolic risk factors is also required to inform targeted and precision-based prevention strategies [48–50].

Conclusion

Abdominal obesity, insulin-resistant glucose metabolism, dyslipidemia, and elevated blood pressure are among the disorders that make up the cardiometabolic syndrome. Changes in the metabolism of fatty acids probably cause these metabolic anomalies. Elevated levels of free fatty acids can: impede the action of insulin in the liver and skeletal muscle, which raises blood glucose levels; increase the production of very low-density lipoprotein triglycerides in the liver, which raises serum triglyceride levels and lowers high-density lipoprotein concentrations; and increase sodium reabsorption and vasoconstriction, which may raise blood pressure. Regardless of whether the synergism between GDM and HDP is present or not, women who have had GDM and/or HDP are now recognized to have a significant risk of CVD and should be closely monitored. In fact, for women who have these pregnancy-related problems, the American Heart Association advises long-term monitoring and control of cardiovascular risk factors. Increased awareness of GDM and HDP is necessary since many women with GDM go on to develop undetected type 2 diabetes later in life. Long-term follow-up also greatly benefits from

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postpartum screening methods and biomarkers of subsequent risk.

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List of abbreviations

BMI – Body Mass Index
CMD – Cardiometabolic Disease
CVD – Cardiovascular Disease
GDM – Gestational Diabetes Mellitus
HDP – Hypertensive Disorders of Pregnancy

Source of funding

This study received no external funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

Conceptualization: All authors
Data collection and analysis: First author
Manuscript drafting and revision: Corresponding author
Final approval: All authors

Data availability

All data analyzed in this review are derived from published studies and are available in the public domain.

Registration and protocol

This systematic review was not registered, and a formal protocol was not prepared.

Support

No financial or non-financial support was received for this study.

Competing interests

The authors declare no competing interests.

Availability of data, code, and materials

No new datasets or analytical code were generated for this review.

Author biography

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