Journal of Developmental Origins of Health and Disease

www.cambridge.org/doh

The role of paternal obesity in fetal development, pregnancy complications, and childhood outcomes: a systematic review

Tuba Güner Emül and Emine Kaplan Serin

Mersin University, Türkiye

Original Article

Cite this article: Güner Emül T and Kaplan Serin E. (2026) The role of paternal obesity in fetal development, pregnancy complications, and childhood outcomes: a systematic review. *Journal of Developmental Origins of Health and Disease* doi: 10.1017/S2040174426100737

Received: 7 April 2026 UTC
Revised: 24 June 2026 UTC
Accepted: 21 July 2026 UTC

Keywords:
Paternal obesity; fetal development; pregnancy complications

Corresponding author:
Tuba Güner Emül;
Email: tubaguner2007@gmail.com

Abstract

The Developmental Origins of Health and Disease theory proposes that environmental influences during early life have long-term effects on health outcomes. Although maternal factors have been extensively studied, the impact of paternal obesity on fetal development and pregnancy outcomes has received limited attention. This systematic review aimed to evaluate the effects of paternal obesity on fetal development and pregnancy complications within the DOHaD framework. English-language studies published between 2020 and 2025 were searched in PubMed, Scopus, Web of Science, and the Cochrane Library databases. Studies were selected according to the PICOS criteria, and methodological quality was assessed using the Joanna Briggs Institute (JBI) critical appraisal tools. The study selection process followed PRISMA 2020 guidelines, and the protocol was prospectively registered in PROSPERO. A total of sevenprimary studies met the inclusion criteria and were included in the final review. Paternal overweight and obesity were associated with increased risks of macrosomia, large-for-gestational-age infants, low birth weight, preterm birth, preeclampsia, and cesarean delivery. In addition, paternal obesity was found to be potentially associated with childhood obesity and neurodevelopmental disorders. However, the effect of paternal body mass index (BMI) on birth weight was more limited compared to maternal factors. Paternal obesity represents a significant risk factor for fetal development and pregnancy outcomes and should be considered in preconception care strategies.

Introduction

The Developmental Origins of Health and Disease (DOHaD) theory suggests that exposure to environmental and biological factors during early life and prenatal development can permanently influence an individual's physiological structure, function, and risk of disease later in life.[1,2] Developmental plasticity refers to the fetus's ability to flexibly regulate developmental and metabolic processes in response to environmental conditions. However, exposure to adverse stimuli such as maternal malnutrition may lead to permanent reprogramming of fetal metabolism, increasing the risk of obesity and other non-communicable diseases later in life.[3]

Previous research has primarily focused on maternal health and the intrauterine environment, extensively examining the effects of maternal nutritional status, metabolic disorders, and pregnancy complications on fetal development and long-term health outcomes.[4–10] In contrast, the role of paternal health in early developmental processes has long been overlooked, and research in this area remains limited.[7]

Obesity is recognized as a major global public health challenge of the 21st century, significantly increasing morbidity and mortality. According to the World Health Organization and national surveillance data, more than two-thirds of adults in Western nations are classified as overweight or obese, a trend mirrored globally and projected to rise continuously by 2030.[9] This global rise suggests that obesity not only affects individual health but may also influence reproductive health and the development of subsequent generations.

While the association between maternal obesity and adverse pregnancy outcomes such as gestational diabetes, hypertensive disorders of pregnancy (HDCP), abnormal fetal growth, and preterm birth is well established, the potential effects of paternal obesity on fetal development and pregnancy complications have been examined in relatively few studies. Existing evidence suggests that paternal obesity may affect sperm quality and epigenetic structure.[11,12] Pregnancy outcomes such as fetal growth restriction, macrosomia, preterm birth, congenital anomalies, and placental dysfunction are key determinants of early human development and have profound implications for lifelong health.[10,13]

Understanding the role of paternal factors in pregnancy and fetal development is therefore critical for adopting a more comprehensive DOHaD perspective. The methodological heterogeneity of existing studies investigating paternal obesity complicates the comparison and generalization of findings. This systematic review aims to evaluate studies examining the effects

of paternal obesity on fetal development and pregnancy outcomes within the DOHaD framework and to synthesize current evidence in a holistic manner. The findings are expected to increase awareness of paternal health factors in early human development and inform preconception care strategies and future research.

Methods

A literature search was conducted between October and December 2025 in PubMed, Scopus, Web of Science, and the Cochrane Library databases. The search covered studies published between 2020 and 2025 and included articles in English. The time restriction was applied to capture the most recent evidence reflecting contemporary preconception care practices, updated BMI classifications, and advances in epigenetic research related to paternal obesity. Earlier studies were screened during preliminary searches; however, many were either superseded by larger contemporary cohorts or did not separately analyze paternal BMI effects. Both MeSH terms and English keywords were used: The complete search strategy used in PubMed was: ('paternal' OR 'father' OR 'paternal health') AND ('obesity' OR 'overweight' OR 'body mass index' OR 'BMI') AND ('fetal development' OR 'pregnancy outcomes' OR 'pregnancy complications' OR 'birth weight' OR 'childhood outcomes'). Equivalent adaptations were applied for Scopus, Web of Science, and the Cochrane Library. Studies were selected based on predefined inclusion criteria using the PICOS framework[14] (Table 1). Titles, abstracts, and keywords were initially screened, and full texts of eligible studies were examined in detail. Studies unrelated to the review or duplicate articles were excluded.

A total of 5765 records were identified through the literature search. After a rigorous screening process, seven studies met the inclusion criteria and were included in the systematic review. The study selection process is presented using the PRISMA flow diagram[15] (Fig. 1). The initial review protocol was developed according to PRISMA-P and prospectively registered in PROSPERO (registration number: CRD420261295090), while the reporting of this completed review strictly adhered to the PRISMA 2020 guidelines. All steps of study selection were independently reviewed by the authors, and disagreements were resolved by consensus. Data were extracted using a standardized form that included: author(s) and year, country, study design, sample size and characteristics, intervention or exposure, main findings, and JBI quality score. The study selection process involved an initial screening of titles and abstracts, followed by a detailed full-text review of eligible articles. Data synthesis was performed descriptively by comparing the relationships between paternal BMI categories and reported fetal, pregnancy, and childhood outcomes across studies, factoring in methodological differences and sample sizes. In the review, the titles and abstracts of the studies were initially read and assessed, and then the full-text articles were examined in detail. A meta-analysis was not conducted because the included studies showed substantial heterogeneity in BMI classification systems (WHO vs. Chinese WGOC criteria), outcome definitions, study populations, and statistical adjustment models, which limited the comparability of effect estimates.

The methodological quality of the included studies was independently evaluated using the Joanna Briggs Institute (JBI) critical appraisal tools. Initial quality assessments were conducted independently by two reviewers (TGE and EKS), and any discrepancies were resolved via consensus involving all authors[16] Cohort studies were evaluated using the 11-item JBI checklist (score range: 0–11).

Table 1. PICOS-based eligibility criteria

	Inclusion criteria	Exclusion criteria
P	Pregnant women and their partners; studies including paternal body mass index (BMI) or obesity status assessed before conception or during pregnancy	Studies in which paternal BMI/obesity status was not reported or in which paternal data were excluded from the analysis. Animal studies were excluded.
I	Paternal overweight/obesity exposure	–
C	–	–
O	Fetal development outcomes (e.g., birth weight, fetal growth restriction, macrosomia), pregnancy complications (e.g., preterm birth, gestational diabetes, hypertensive disorders, placental dysfunction, congenital anomalies)	
S	Descriptive, cross-sectional, prospective, retrospective, comparative, and randomized controlled English studies with accessible full text.	Preprint studies, non-peer-reviewed manuscripts, conference abstracts, reviews, commentaries, qualitative studies, case reports, studies with inaccessible full text, and non-English publications were excluded.

Each item was scored as 1 if the criterion was met ("yes") and 0 if it was not met, unclear, or not applicable ("no," "not specified," or "not applicable"). Higher total scores indicated higher methodological quality. In this systematic review, the methodological quality scores of the included studies ranged from 8 to 9, indicating overall medium to high methodological quality. In addition to overall JBI scores, key domains of bias were considered qualitatively, including selection bias, exposure misclassification, outcome assessment, and residual confounding. Particular attention was paid to whether paternal BMI was self-reported or clinically measured and whether studies adjusted for major maternal and socioeconomic variables.

Results

The characteristics of the included studies are presented in Table 2. Seven studies met the inclusion criteria and were included in this review. Most studies were prospective cohort studies conducted in China, with sample sizes ranging from 412 to 4.8 million participants. Overall, the studies evaluated the associations between paternal preconception BMI and fetal development, pregnancy outcomes, and childhood health outcomes. Across studies, the principal risks of bias were related to self-reported paternal BMI, incomplete adjustment for maternal and socioeconomic confounders, and potential selection bias in cohort recruitment. Outcome assessment was generally based on clinical records, reducing the likelihood of differential misclassification for birth outcomes.

All studies evaluated the effects of paternal obesity on fetal development and pregnancy outcomes. Across studies, the principal risks of bias were related to self-reported paternal BMI, incomplete adjustment for maternal and socioeconomic confounders, and potential selection bias in cohort recruitment. Outcome assessment

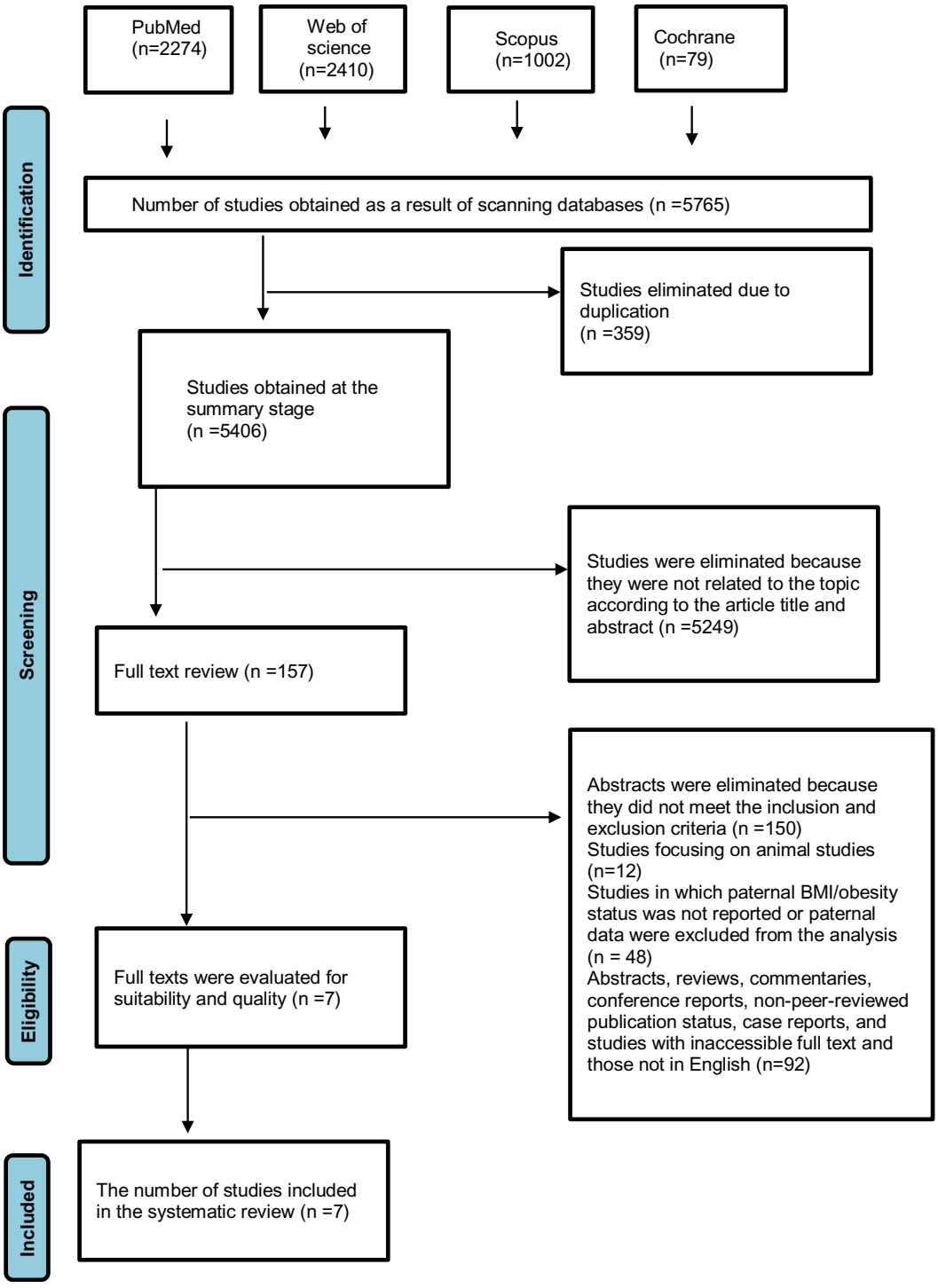

Figure 1. PRISMA flow chart of the study selection process for the systematic review.

was generally based on clinical records, reducing the likelihood of differential misclassification for birth outcomes.

In a prospective cohort study conducted in China by Zhang et al. (2025), 412 small-for-gestational-age (SGA) infants were assessed. Fathers were categorized into four groups: underweight ($n = 46$), normal weight ($n = 188$), overweight ($n = 124$), and obese ($n = 54$). The children of these fathers were followed for 36 months, and their physical development (height and weight measurements) was monitored. The study found a U-shaped relationship between paternal preconception body mass index (BMI) extremes (<18.5 or ≥28 kg/m^2) and child outcomes ($p < 0.05$). Compared with children of fathers with normal BMI (18.5–24.9 kg/m^2), paternal obesity was associated with an increased risk of childhood obesity and multi-domain neurodevelopmental disorders. These findings suggest that paternal BMI may be an important determinant of child health and neurodevelopment. Additionally, the study reported no significant differences between groups regarding maternal conditions, including gestational hypertension, preeclampsia, diabetes, hypothyroidism, connective tissue disorders, umbilical cord complications, and placental abnormalities.[17]

Table 2. Characteristics, main findings, and methodological quality of studies included

Author (Year)/ country	Study design and sample	Outcomes assessed	Results	Conclusion	Adjustment variables	Quality (JBI)
Zhang et al., 2025/China.[17]	Prospective cohort, 412 small-for-gestational-age (SGA) infants	Paternal Body Mass Index (BMI); child physical growth and neurodevelopment	A U-shaped relationship was observed between paternal BMI and child outcomes ($p < 0.05$). Paternal obesity increased the risk of obesity and multiple neurodevelopmental disorders in children, whereas paternal underweight was associated with lower birth weight and severe SGA.	Paternal BMI was found to be an important determinant of child neurodevelopment and obesity	Maternal age 32.7 ± 3.8 Paternal age 34.2 ± 4.5 Maternal BMI Normal (55.3%) Obesity (11.1%)	8
Lin et al., 2023/China.[7]	Prospective cohort, 29.518 mother father infant triads	Paternal BMI; child physical growth and neurodevelopment	Paternal overweight and obesity increased the risk of Hypertensive disorders of pregnancy (HDCP), cesarean section, excessive gestational weight gain (GWG), and macrosomia. It was also associated with asthma, hand-foot-and-mouth disease (HFMD), dental caries, and obesity in children.	Paternal BMI affects both pregnancy complications and long-term child health	Adjusted for maternal age, paternal age, maternal BMI, paternal education, annual family income, parity, and lifestyle factors.	9
Lin et al., 2022/China.[18]	Prospective cohort, 7.683 singleton pregnancies	Paternal BMI	It was found that in the paternal obesity group, the incidences of SGA, macrosomia, preeclampsia, cesarean delivery, and postpartum hemorrhage were higher.	Paternal obesity was associated with adverse fetal growth patterns and pregnancy outcomes.	Maternal age 29.79 ± 3.9 Paternal age 30.97 ± 4.0 Maternal BMI Normal (73%) Obesity (2.3%) Obesity Paternal education high school and below: 58.25% Maternal education high school and below: 50.86% Obesity Gravity <2:54.99% The proportion of those with a low socio-economic status is 7.39 %.	8
Guo et al., 2022/China.[19]	Retrospective cohort, 4.79 million couples	Paternal BMI	Paternal overweight and obesity increased the risk of Large for Gestational Age (LGA). Similar trends were observed with maternal BMI.	Abnormal preconception BMI in either parent is a modifiable risk factor for SGA/LGA.	Maternal and paternal age (LMP), height, ethnicity, education, residence, alcohol use, smoking and passive smoking, maternal BMI, hypertension, diabetes, parity, and adverse pregnancy history.	8
Sun et al., 2022/China.[20]	Prospective cohort, 34.104 triads	Paternal BMI	Paternal pre-pregnancy overweight and obesity were associated with increased risks of preterm birth and LBW.	Paternal obesity is an important preconceptional risk factor.	Adjusted for maternal age, paternal age, maternal BMI, smoking status, alcohol consumption, parity, education level, and socioeconomic variables.	9
Wei et al., 2022/China.[21]	Prospective cohort, A total of 34,104 pregnant women with singleton pregnancies	Maternal and Paternal BMI	Maternal and paternal pre-pregnancy overweight/obesity increased the risk of LBW, VLBW and ELBW. Combined parental overweight/obesity further increased these risks.	Parental preconception BMI is a determinant of low birth weight. There were statistically significant differences across four maternal BMI groups for maternal age, ethnicity, educational level, parity, history of adverse pregnancy outcomes, gestational	Adjusted for maternal and paternal age, ethnicity, education, parity, adverse pregnancy history, gestational weight gain, pregnancy complications, smoking and alcohol use.	9

(*Continued*)

Table 2. (*Continued*)

Author (Year)/ country	Study design and sample	Outcomes assessed	Results	Conclusion	Adjustment variables	Quality (JBI)
				weight gain recommendation rage, pregnancy complications in this pregnancy, and husband smoking status before wife pregnancy (all $p < 0.05$).		
Retnakaran et al., 2021/ Canada.[22]	Prospective observational cohort, 1.292 couples	Paternal BMI	Paternal BMI was positively associated with birth weight, but its effect was limited (explaining 0.7% of the variance). Maternal BMI was found to be a more dominant factor.	Paternal BMI affects fetal growth.	Maternal age 25 ± 3 Paternal age 26 ± 3 Maternal BMI Normal (20.1 ± 2.4) Obesity (20.6 ± 2.6) Paternal BMI Normal (22.3 ± 2.7) Obesity (22.0 ± 2.6)	8

In a prospective study in China by Lin et al. (2023) including 29,518 participants, the mean paternal age was 35.1 ± 4.9 years, and the mean maternal age was 33.0 ± 4.4 years. Based on paternal BMI classifications, 996 fathers (3.4%) were underweight, 16,028 (54.3%) had a normal BMI, 9761 (33.1%) were overweight, and 2.733 (9.3%) were obese. Significant associations were found between paternal BMI groups and paternal age, paternal education, maternal age, maternal BMI, and annual family income. Cesarean delivery, gestational weight gain (GWG) above recommended limits, and macrosomia were significantly higher in the paternal overweight and obese groups compared with the normal BMI group. HDCP were also more frequent in the paternal obesity group. Multivariable logistic regression analyses showed that paternal obesity significantly increased the risks of HDCP, cesarean delivery, excessive GWG, and macrosomia. Moreover, paternal obesity was associated with long-term adverse child health outcomes, including asthma, hand-foot-and-mouth disease (HFMD), dental caries, and obesity. In the overweight paternal group, higher rates of cesarean delivery, excessive GWG, and childhood asthma, HFMD, and obesity were also observed. These findings indicate that preconception paternal obesity is an important risk factor for both pregnancy complications and child health.[7]

In a large-scale retrospective cohort study by Guo et al. (2022) in China, over 4.7 million mother–father–infant triads from the National Free Preconception Health Examination Project were evaluated. Among live births, 5.44% were SGA and 10.73% were LGA. After adjusting for confounding factors, paternal overweight and obesity were found to increase the risk of LGA infants. Similar patterns were observed with maternal preconception BMI and SGA/LGA outcomes. Overall, abnormal preconception BMI in either parent was identified as a preventable and significant risk factor for SGA and LGA infants.[19]

In a prospective cohort study conducted in China by Lin et al. (2022), 7683 singleton pregnancies were evaluated. Grouping according to paternal BMI, 201 were underweight, 3813 were normal weight, 2913 were overweight, and 756 were obese. In the paternal obesity group, incidences of preeclampsia, SGA, macrosomia, cesarean delivery, and postpartum hemorrhage were significantly higher than in the normal BMI group. In the overweight group, rates of macrosomia and cesarean delivery were also significantly elevated. As paternal BMI increased, fetal ultrasound parameters and neonatal and placental weights also increased ($p < 0.05$), but in the obese group, this increase plateaued, and differences between groups were no longer significant.[18]

Sun et al. (2022) conducted a large cohort study including 34,104 mother–father–infant triads, finding a significant association between paternal obesity and adverse perinatal outcomes. Compared with fathers with normal BMI, paternal overweight and obesity significantly increased the risks of preterm birth and low birth weight (LBW), and these associations remained significant after adjusting for maternal and paternal confounders. These findings support paternal obesity as an important preconception risk factor affecting not only fetal growth but also gestational duration and birth weight.[20]

In a prospective cohort study by Wei et al. (2022) in central China, including 34,104 mother–father–infant triads, the relationship between parental preconception BMI and LBW, very low birth weight (VLBW), and extremely low birth weight (ELBW) was evaluated. Maternal preconception overweight and obesity were associated with significant increases in LBW, VLBW, and ELBW risk, while paternal preconception overweight and obesity significantly increased LBW and VLBW risk. Additionally, when both parents were overweight or obese before pregnancy, the risk of LBW, VLBW, and ELBW was further elevated.[21]

Sun et al. (2022) and Wei et al. (2022) were derived from the same prospective cohort conducted at Hunan Provincial Maternal and Child Health Care Hospital, including 34,104 couples recruited between 2013 and 2019. However, the studies addressed different research questions and outcomes. Sun et al. investigated the association between paternal pre-pregnancy BMI and preterm birth as well as LBW, whereas Wei et al. examined the effects of both maternal and paternal BMI on low birth weight and its subtypes (VLBW and ELBW). Therefore, both studies were retained in the review, but the shared cohort was acknowledged when interpreting the findings.

In a prospective observational cohort study in Canada by Retnakaran et al. (2021), 1292 pregnant women and their partners were evaluated. The mean birth weight was 3294 ± 450 g, with 11.4% of neonates being LGA and 7.3% SGA. After adjusting for preconception maternal and paternal factors and pregnancy and delivery variables, each unit increase in maternal preconception

BMI was associated with a 42.2 g increase in birth weight, while each unit increase in paternal preconception BMI was associated with a smaller 10.7 g increase. Maternal BMI accounted for 6.2% of the variance in birth weight, while paternal BMI explained only 0.7%. Independent predictors of LGA were maternal preconception BMI, maternal age, and gestational weight gain, while paternal BMI showed no independent association with LGA or SGA. This study also confirmed that the effect of paternal BMI on birth weight is more limited compared with maternal factors.[22]

Discussion

This systematic review provides a comprehensive evaluation of current evidence on the role of paternal obesity in fetal development, pregnancy complications, and early childhood outcomes within the framework of the DOHaD theory. Overall, the reviewed studies indicate that preconception paternal BMI is significantly associated with fetal growth patterns, pregnancy complications, and, in some cases, childhood health outcomes. The studies included in this review highlight that, alongside maternal factors, paternal obesity also contributes to fetal development and pregnancy outcomes.

The heterogeneity observed across studies may be explained by differences in study populations, study designs, adjustment strategies, and BMI classification systems, as Chinese studies generally applied WGOC criteria whereas international studies mostly used WHO criteria.

Most of the included studies reported that paternal overweight and obesity were significantly associated with adverse perinatal outcomes, including macrosomia, cesarean delivery, hypertensive disorders of pregnancy, preterm birth, and LBW.[7,18,21] Notably, large-scale prospective cohort studies by Lin et al. demonstrated that the effects of paternal obesity on these pregnancy outcomes persisted even after adjusting for important confounders, such as maternal age, maternal BMI, socioeconomic status, and lifestyle factors.[7,18] These findings support the notion that paternal obesity may be an important risk factor for adverse pregnancy outcomes.

Large-scale retrospective cohort studies are consistent with these findings. Guo et al. (2022) reported that paternal overweight and obesity significantly increased the risk of LGA infants.[19] Similarly, Zhang et al. (2025) showed that paternal BMI above the normal range was associated with childhood obesity and neurodevelopmental problems.[17] These findings suggest that the effects of paternal obesity are not limited to the perinatal period but may persist into early childhood. However, some studies reported that the effect of paternal BMI is more limited compared with maternal BMI. In their prospective cohort study in Canada, Retnakaran et al. (2021) found that paternal BMI explained only a small proportion of the variance in birth weight, whereas maternal BMI was a stronger determinant.[22] Nevertheless, Wei et al. (2022) reported that the effect of paternal BMI became more pronounced in the presence of maternal overweight, obesity, or excessive gestational weight gain, affecting fetal growth and the risk of low birth weight.[21] These findings highlight the importance of jointly considering the weight status of both parents during the preconception period.

Although the mechanisms underlying the effects of paternal obesity on pregnancy and childhood outcomes are not yet fully understood, current evidence suggests a potential role for epigenetic regulation. Noor et al. (2019) demonstrated that paternal BMI was associated with genome-wide DNA methylation patterns in umbilical cord blood at birth, and these epigenetic changes could persist into childhood.[23] Similarly, Houfflyn et al. (2017) suggested that paternal obesity might influence fetal development through sperm-derived epigenetic modifications.[12]

In addition to residual confounding, the direction of potential bias should be considered when interpreting the findings. In most included studies, paternal obesity is likely correlated with unmeasured or incompletely measured lifestyle and socioeconomic factors, including dietary patterns, physical inactivity, and household-level health behaviors. As a result, observed associations may be partially inflated due to positive confounding, leading to an overestimation of the true independent effect of paternal BMI. Conversely, adjustment for maternal BMI and shared environmental factors may also attenuate the observed associations, potentially underestimating the biological contribution of paternal metabolic status. Measurement error in paternal BMI, particularly when self-reported, further increases the likelihood of non-differential misclassification, which may bias effect estimates toward the null. The net direction of bias is therefore uncertain and likely varies across studies depending on study design and adjustment strategies. Overall, these methodological limitations highlight that causal inference regarding paternal obesity should be interpreted with caution.

An important methodological issue across the included studies is residual confounding. Paternal obesity is closely associated with shared household factors such as diet quality, physical activity, smoking, and socioeconomic status. Although several studies adjusted for maternal BMI and parental age, the extent and consistency of adjustment varied substantially. Consequently, some reported associations may partially reflect shared environmental influences rather than a purely paternal biological effect.

Limitations

This systematic review has several limitations. First, the majority of the included studies were conducted in China, which may limit the generalizability of the findings to other geographic regions and ethnic populations. The study designs were primarily prospective and retrospective cohort studies, and the absence of randomized controlled trials prevents drawing definitive causal inferences. Meta-analysis was not performed due to heterogeneity in study design, BMI classifications, outcome definitions, and follow-up durations. Additionally, methods for assessing paternal BMI (self-reported vs. measured) and BMI cut-off values varied across studies, which may limit the comparability of the results. Potential publication bias should also be considered. Because only seven studies met the inclusion criteria, a formal assessment using funnel plots or statistical tests was not appropriate. Nevertheless, studies reporting null associations may be underrepresented in the published literature. Regarding exposure assessment, most included studies relied on self-reported pre-pregnancy BMI, which may have introduced exposure misclassification due to recall bias or underreporting of body weight. Only a limited number of studies used objectively measured anthropometric data. In addition, residual confounding remained a potential source of bias because adjustment strategies varied across studies and some important confounders, such as socioeconomic status, lifestyle factors, and dietary habits, were not consistently controlled.

Conclusion

This systematic review suggests that paternal obesity may play a clinically relevant role. The findings support the notion that

preconception care should not focus solely on maternal health, but also incorporate paternal weight management and metabolic health into reproductive health policies. Future large-scale studies are needed to clarify the effects of paternal obesity further and to help translate the DOHaD approach into clinical practice.

Data availability statement. The data that support the findings of this study are available from the corresponding author, TGE, upon reasonable request.

Acknowledgements. The authors thank all researchers whose studies were included in this review.

Author contribution. Tuba Güner Emül: Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Data curation, Conceptualization. Emine Kaplan Serin: Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization.

Financial support. This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Competing interests. The authors declare no conflicts of interest.

Ethical standard. Ethical approval was not required because this study is a systematic review based exclusively on previously published studies.

References

1. Barker DJP, Eriksson JG, Forsen T, Osmond C. Fetal origins of adult disease: strength of effects and biological basis. *Int J Epidemiol*. 2002; 31(6), 1235–1239. https://doi.org/10.1093/ije/31.6.1235.
2. İmre KE, Akyol A. Developing the developmental origins of health and disease (DOHaD) awareness scale to assess an education module for improving dietary behavior among college students. *Peer J*. 2024; 12, e.18669.
3. Gluckman PD, Hanson MA, Low FM. Evolutionary and developmental mismatches are consequences of adaptive developmental plasticity in humans and have implications for later disease risk. *Philosophical Transactions of The Royal Society London B Biological Sciences*. 2019; 374(1770), 20180109. https://doi.org/10.1098/rstb.2018.0109
4. Patro B, Liber A, Zalewski B, Poston L, Szajewska H, Koletzko B. Maternal andpaternal body mass index and offspring obesity: a systematic review. *Ann NutrMetab*. 2013; 63(1–2), 32–41.
5. Flanagan EW, Most J, Altazan AD, Boyle KE, Redman LM. A role for the early pregnancy maternal milieu in the intergenerational transmission of obesity. *Obesity Silver Spring Md*. 2021; 29(11), 1780–1786. https://doi.org/10.1002/oby.23283.
6. O'Brien CM, Louise J, Deussen A, Grivell R, Dodd JM. The effect of maternal obesity on fetal biometry, body composition, and growth velocity. *J Matern Fetal Neonatal Med*. 2020; 33(13), 2216–2226. https://doi.org/10.1080/14767058.2018.1543658.
7. Lin Y, Chen Z, Qian Q, *et al*. Effects of paternal obesity on maternal neonatal outcomes and long-term prognosis in adolescents. *Front Endocrinol*. 2023; 14, 1114250.
8. Blüher M. Obesity: global epidemiology and pathogenesis. *Nat Rev Endocrinol*. 2019; 15(5), 288–298. https://doi.org/10.1038/s41574-019-0176-8.
9. World Health Organization. 2022. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
10. McPherson JA, Smid MC, Smiley S, Stamilio DM. Association of maternal obesity with child cerebral palsy or death. *Am J Perinatol*. 2017; 34(6), 563–557. https://doi.org/10.1055/s-0036-1594015.
11. Salas-Huetos A, Maghsoumi-Norouzabad L, James ER, *et al*. Male adiposity, sperm parameters and reproductive hormones: an updated systematic review and collaborative meta-analysis. *Obes Rev*. 2021; 22(1), e13082.
12. Houfflyn S, Matthys C, Soubry A. Male obesity: epigenetic origin and effects in sperm and offspring. *Curr Mol Biol Rep*. 2017; 3(4), 288–296. DOI: https://doi.org/10.1007/s40610-017-0083-5.
13. Gette F, Aziz Ali S, Ho MSP, *et al*. Long-term health outcomes of preterm birth: a narrative review. *Front Pediatr*. 2025; 13, 1565897. https://doi.org/10.3389/fped.2025.1565897.
14. Centre for Reviews and Dissemination. *Systematic Reviews: CRD's Guidance for Undertaking Reviews in Health Care. University of York, 2008 Published by CRD*, 2008. York Publishing Services Ltd, University of York. ISBN 978-1-900640-47-3.
15. Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. PRISMA2020: an R package and Shiny app for producing PRISMA. 2020-compliant flow diagrams, with interactivity for optimised digital transparency and open synthesis. *Campbell Syst Rev*. 2022; 18, e1230. https://doi.org/10.1002/cl2.1230.
16. Barker TH, Stone JC, Sears K, *et al*. The revised JBI critical appraisal tool for the assessment of risk of bias for randomized controlled trials. *JBI Evid Synth*. 2023; 21(3), 494–506. https://doi.org/10.11124/JBIES-22-00430.
17. Zhang Y, Shao S, Qin J, *et al*. Associations between paternal body mass index and neurodevelopmental–physical outcomes in small-for-gestational-age children. *Diagnostics*. 2025; 15, 2133. https://doi.org/10.3390/diagnostics15172133.
18. Lin J, Gu W, Huang H. Effects of paternal obesity on fetal development and pregnancy complications: a prospective clinical cohort study. *Front Endocrinol*. 2022; 13, 826665. https://doi.org/10.3389/fendo.2022.826665.
19. Guo T, Yang Y, Jia J, *et al*. Preconception paternal/ maternal body mass index and risk of small/large for gestational age infant in over 4.7 million Chinese women aged 20–49 years: a population-based cohort study in China. *Br J Nutr*. 2022; 129, 1–28.
20. Sun M, Zhang S, Chen L, *et al*. Association between paternal pre-pregnancy body mass index with preterm birth and low birth weight. *Front Pediatr*. 2022; 10, 955544.
21. Wei J, Wang T, Shu J, *et al*. Parental pre-pregnancy body mass index and risk of low birth weight in ospring: a prospective cohort study in central China. *Front Public Health*. 2022; 10, 1036689. https://doi.org/10.3389/fpubh.2022.1036689.
22. Retnakaran R, Wen SW, Tan H, *et al*. Paternal weight prior to conception and infant birthweight: a prospective cohort study. *Nutr Diabetes*. 2021; 11(1), 28.
23. Noor N, Cardenas A, Rifas-Shiman SL, *et al*. Association of periconception paternal body mass index with persistent changes in DNA methylation of offspring in childhood. *JAMA Netw Open*. 2019; 2(12), e1916777–e1916777.