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Maternal age and parity influences on health outcomes: a multivariable regression analysis of mothers and infants

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Abstract

Background Adverse pregnancy and birth outcomes remain significant public health challenges, particularly in low- and middle-income countries (LMICs). Maternal age and parity are recognised as key factors, yet their combined influence on maternal and infant outcomes is less understood, especially in LMICs.

Objectives We investigated the combined effects of maternal age and parity on maternal health risks, including body mass index (BMI), gestational diabetes mellitus (GDM), and hypertension, as well as infant birth outcomes, namely birth weight, length, and gestational age, in an urban South African cohort.

Methods This study used data from 830 pregnant women (aged 18–44) enrolled in the Soweto First 1000 Days (S1000) longitudinal cohort. Group comparisons were conducted using ANOVA, chi-square, or Kruskal-Wallis tests. Multivariable Linear and logistic regression models assessed associations between age-parity and outcomes, adjusting for sociodemographic factors. Analyses were conducted in StataSE 18; $p < .05$ was considered significant.

Results Mothers > 23 years, ≥ 1 child had higher BMI (28.6 kg/m^2 , $p < .001$) and increased likelihood of hypertension (44.1% , $p < .001$), and GDM (7.4% , $p = .012$). Nulliparous women showed greater gestational weight gain (0.39 kg/week , $p < .001$) and an increased likelihood of having low birth weight (2960 g vs. 3185 g , $p = .002$), small for gestational age (SGA) (22.9% , $p = .009$), and shorter birth length infants ($z = -0.29$, $95\% \text{ CI: } [-0.57 \text{ to } -0.01]$, $p = .04$). Infants of mothers ≤ 23 years, ≥ 1 child had higher birth weight ($\beta = 0.60$, $95\% \text{ CI: } [0.32-0.88]$; $p < .001$) and length z -scores ($95\% \text{ CI: } [0.01-0.97]$, $p = .046$). Hypertension ($\beta = -0.99$, $95\% \text{ CI: } [-1.52 \text{ to } -0.45]$, $p < .001$) and GDM ($\beta = -0.57$, $95\% \text{ CI: } [-1.10 \text{ to } -0.04]$, $p = .036$) were associated with shorter gestational age.

Conclusion Maternal age and parity were associated with distinct risks to maternal and infant health. These findings support the need for more targeted, risk-based antenatal strategies in LMICs.

Keywords Maternal age, Parity, Pregnancy risk factors, Birth outcomes, South africa

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Introduction

Maternal and infant health outcomes are key surveillance indicators of national health and well-being. Preventable adverse outcomes, such as pre-term birth, low birth weight as well as maternal and infant mortality remain a significant global health challenge. Recent estimates indicate 13.4 million preterm births [1], 19.8 million low birth weight infants [2], and 4.5 million deaths, comprising 0.29 million maternal deaths, 1.9 million stillbirths and 2.3 million newborn deaths worldwide [3]. Maternal age and a woman's number of pregnancies (i.e., parity) are increasingly recognised as key factors influencing birth outcomes. In high-income countries, maternal age at first birth has risen, now averaging 30 years or older [4], and parity has declined from more than 3.3 children per mother in 1960 to approximately 1.5 children per woman in 2022 [5]. In contrast, low- and middle-income countries (LMICs) experience higher rates of adolescent pregnancies, with an estimated 21 million girls between the ages of 15 and 19 becoming pregnant annually [6].

Maternal age and parity extremes are both linked with adverse infant outcomes. Young mothers (≤ 19 years) experience risks such as maternal-fetal nutritional competition, which limits optimal fetal growth and is linked to low birth weight and small-for-gestational-age (SGA) infants [7–10]. Pelvic underdevelopment in young women may lead to cephalo-pelvic disproportion [11, 12], leading to obstructed and/or prolonged labour and increasing the risk of maternal or perinatal mortality or morbidity [13, 14]. Risks such as obesity and poor diets that tend to be more prevalent in older mothers (≥ 35 years), can result in excessive birth weight and greater fat deposition in the fetus due to over nutrition [15–17]. Similarly, metabolic disorders, such as hypertension and diabetes, often experienced by older mothers, increase the risk of maternal cardiovascular complications, pre-eclampsia, and gestational diabetes, leading to intra-uterine growth restriction (IUGR), premature birth, and increased rates of caesarean delivery [18–20]. This dual risk is demonstrated in an LMIC-based study, where both advanced and young maternal age were associated with preterm birth, low birth weight, stunting at two years, and adverse long-term effects [21].

Parity further influences pregnancy and birth risks. Nulliparous mothers, who lack prior reproductive experience, encounter a higher risk of obstructed labour, caesarean delivery and an increased likelihood of having low birth weight infants [22]. Conversely, multiparity (≥ 5 deliveries) is associated with better birth outcomes due to prior childbirth experience and improved physiological adaptations [23]. However, excessive parity seemingly diminishes these benefits, with high multiparity linked to preterm birth, low birth weight, and placental abruption. Uterine perforation and reduced myometrial strength

[24], placental abnormalities [25, 26] as well as nutritional deficiencies due to repeated pregnancies [27] may operate as possible underlying factors for the increased risks.

Despite extensive research, evidence remains limited on infant growth outcomes and anthropometric measures at birth and during early infancy [28]. Moreover, existing studies often fail to account for potential confounding factors, such as socioeconomic status (SES) and rely on cross-sectional data. Addressing these gaps is crucial for developing targeted interventions that optimise growth patterns, which are critical for long-term health and development, ultimately improving maternal and infant health outcomes.

We investigated whether maternal age at the time of the first childbirth and parity are associated with both maternal pregnancy and infant health outcomes within the Soweto First 1000 Days (S1000) Cohort. We hypothesise a U-shaped relationship, whereby extremes in maternal age and parity are associated with an increased risk of adverse maternal and infant health outcomes.

Methods

This study is a secondary use of the data from the Soweto First 1000 Days (S1000) cohort, a longitudinal pregnancy study, undertaken at the SAMRC/Wits Developmental Pathways for Health Research Unit (DPHRU) at Chris Hani Baragwanath Academic Hospital (CHBAH), in Soweto, Johannesburg. Soweto is a major urban area with a historically low-income status and a population of approximately 1.6 million, predominantly Black African individuals, and high burden of non-communicable diseases, such as diabetes, hypertension, and elevated cholesterol levels [29]. The primary aim of the S1000 study was to conduct an in-depth investigation into the relationship between maternal health conditions (e.g., obesity, gestational hypertension, gestational diabetes, HIV), biological and physiological factors (e.g., physical activity, dietary patterns) and fetal and infant growth and developmental outcomes through the first two years postpartum. The current study focuses on the maternal factors that impact pregnancy and infant outcomes.

Participants

The study participants consisted of 830 pregnant women drawn from the S1000 cohort between the ages of 18 and 44 (median age of 29), residing in the Soweto region and attending antenatal care at CHBAH. The inclusion criteria required participants to be 18 years and not enrolled in school, with no maximum age limit applied. Women under 18 years were excluded given that they are still undergoing growth and development, and pregnancy during adolescence involves physiological adaptations that may not be generalisable to the adult population.

Including this age group could introduce biological heterogeneity, complicating interpretation of findings, and participation may pose additional psychosocial and ethical risks, including sensitive disclosures, potential stigma, and the burden of study procedures, that could outweigh the benefits, particularly as the study was not designed to generate adolescent-focused evidence. Participants for the broader S1000 study were gradually recruited between 2014 and 2016 through the hospital's fetal medicine unit. Eligibility criteria included self-reported Black South African women living in Soweto or the surrounding area, with natural conception, ideally less than 14 weeks but no more than 20 weeks pregnant. Participants were required to have no diagnosis of epilepsy, Type-1 diabetes or cancer and no intellectual disabilities at the time of recruitment. Participants with Type-1 diabetes were excluded due to the clinical management these conditions require during pregnancy. Other forms of diabetes, such as chronic diabetes (e.g., Type-2), overt diabetes diagnosed only prior to the study pregnancy and not during study visits, and gestational diabetes mellitus (GDM) were included. Individuals with intellectual disabilities were excluded based on their limited capacity for providing informed consent. Data for the S1000 study were collected at six time points during pregnancy, including < 14 weeks; 14–18 weeks; 19–23 weeks; 24–28 weeks; 29–33 weeks and 34–38 weeks, and at the time of delivery. All women were notified about the nature and aims of the broader S1000 study and provided written informed consent for participant and data use. Ethical approval for the large-scale study was obtained from the Human Ethics Research Committee of the University of the Witwatersrand's Research Ethics Committee (Medical) (ethical clearance number: M120524).

Measures

Maternal health and socio-demographic variables

At the baseline visit (<14 weeks gestation), trained research staff administered questionnaires to collect maternal demographics (enrolment age, education, smoking status), pregnancy-related information, and SES. The questionnaire items used in this study have been compiled into a separate document, which has been submitted as a supplementary file. Parity was defined as the number of previous births at ≥ 24 weeks, including still births. Participants were asked to report on prior pregnancy-related conditions (e.g., anaemia, respiratory, cardiac, kidney disease, hypertension, and gestational diabetes). Self-reported HIV status was obtained at each pregnancy visit, which was validated against medical records. An 11-item self-report asset index taken from the Demographic and Health Surveys (DHS) (available at: www.measuredhs.com) was used to determine SES, with participants scored according to the number of assets

they owned. Higher scores were indicative of a higher SES, with scores ranging from 0 to 11.

Maternal anthropometry and pregnancy outcomes

Maternal height at baseline was measured using a wall-mounted Stadiometer (Holtain, UK) and weight at each pregnancy visit was obtained using a digital scale. Baseline weight (<14 weeks gestational age) and height were used to calculate BMI and women were categorised as underweight (<18 kg/m²), normal (18.5 < 25 kg/m²) or overweight (≥ 25 kg/m²). Gestational weight gain (kg/week) was calculated as the difference between baseline and final pregnancy weights divided by the number of weeks between the two.

Blood pressure (mm Hg) during the pregnancy at recruitment was measured using an Omron 6 automated machine (Kyoto, Japan), with hypertension defined as a systolic measure of ≥ 140 and/or a diastolic measure of ≥ 90 . Hypertension was determined from both self-reported history on recruitment, measurements at booking as well as first detection during the study pregnancy. This approach captures both pre-existing hypertension and hypertension arising during pregnancy, providing an indication of chronic and early-onset pregnancy-related hypertensive conditions. Haemoglobin levels (g/dL) were measured using a HemoCue. Women were considered anaemic if they had a haemoglobin level below 11.0 g/dL using values from the baseline or second visit (14–18 weeks gestational age) if women were unavailable for the baseline visit. Urine dipsticks detected glucosuria and proteinuria. Gestational Diabetes Mellitus (GDM) was determined using a two-hour 75-gram oral glucose tolerance test (OGTT). In accordance with the WHO's 2013 criteria [30], the presence of GDM was determined if any of the following thresholds were met: fasting plasma glucose of 5.1–6.9 mmol/l, or one-hour plasma glucose of ≥ 10.0 mmol/l or two-hour plasma glucose of 8.5–11.0 mmol/l.

Delivery outcomes and infant anthropometry

Gestational age at delivery was calculated from the time between delivery date and baseline ultrasound scan, added to the gestational age at enrolment, which was measured in days using the crown-to-rump length. Newborn birth weight and length were assessed using a calibrated SECA Baby Scale (SECA) and a Harpenden Infantometer (Holtain, London, UK), respectively. Trained nurses administered these measures, and the newborns were assessed within 24 h of delivery. If conducting this assessment within this time frame was not feasible (e.g., infant admitted to the hospital for observation), it was done within 48 h. The International Newborn Size at Birth Application tool [31] was used to determine the birth weight centiles and weight-to-length

z-scores based on newborn gender and gestational age at delivery (total days). The following criteria were used to categorise newborns based on birth weight. Specifically, infants were classified as SGA if their birth weight fell below the 10th percentile, appropriate-for-gestational-age (AGA) if their birth weight was between the 10th and 90th percentiles, and large-for-gestational-age (LGA) if their birth weight exceeded the 90th percentile [32]. Additionally, low birth weight was defined as less than 2.5 kg, while macrosomia was defined as a birth weight greater than 4.0 kg.

Data analysis

Data obtained from 830 mother-newborn pairs were analysed using StataSE version 18. Maternal age-parity groups (nulliparous mothers; mothers ≤ 23 years, ≥ 1 child; and mothers > 23 years, ≥ 1 child) were created using parity status and maternal age at recruitment. The groups served as a proxy for maternal age at first childbirth. While a maternal age cutoff of 35 years is commonly accepted, the cutoff age of 23 was chosen pragmatically to ensure balanced group sizes for analysis while approximating the timing of first childbirth within our sample. This cutoff age aligns with an expanded definition, which classifies individuals aged 10–24 years as adolescence or “young people” [33, 34], making the group of mothers ≤ 23 years, ≥ 1 child broadly indicative of adolescent and young maternal childbearing, and those > 23 years, ≥ 1 child representative of older maternal childbearing. Descriptive statistics were used to compare maternal, pregnancy and infant characteristics across these age-parity groups using one-way ANOVA (normally distributed) or Kruskal-Wallis tests (skewed distributed) for continuous variables and chi-squared tests for categorical variables. Post hoc pairwise comparisons followed significant omnibus results.

Associations between maternal age-parity groups and infant outcomes were examined using multivariable linear (continuous variables) and logistic regression models (categorical variables). Infant outcomes included birth weight z-scores, birth weight category (SGA vs. LGA), birth length z-scores, and gestational age at delivery (in weeks). Maternal age-parity was the primary exposure variable and was entered as a three-level categorical variable (nulliparous = reference). For each birth outcome, a series of nested models was analysed to examine the associations between maternal age-parity and the outcome of interest, while incrementally adjusting for additional factors. Each analysis included six models. Model 1 including the unadjusted association between age-parity and the respective outcome variable, and Model 2 adjusted for key socio-demographic covariates (neonatal sex, HIV status, SES and maternal level of education). Models 3 to 5 sequentially introduced maternal health

conditions, BMI (Model 3), gestational diabetes mellitus (GDM; Model 4), and hypertension (Model 5), as explanatory variables. The final model (Model 6) included all maternal health variables and socio-demographic covariates simultaneously to assess fully adjusted associations. All models were tested for multicollinearity and influential outliers. Regression diagnostics were performed to assess model assumptions, including normality of residuals (for linear models) and goodness-of-fit (for logistic models). Statistical significance was set at $p < .05$ for all analyses.

Results

Participant characteristics

Maternal sociodemographic, anthropometry, pregnancy outcomes and birth characteristic stratified by age-parity groups are presented in Table 1. The mean maternal age was 29.81 years (SD = 5.89), with a median age of 29 years (IQR: 25–34). Nulliparous women were more likely to have tertiary education ($p < .001$) and had a higher SES than mothers > 23 years, ≥ 1 child ($p = .009$). HIV prevalence was higher among mothers > 23 years, ≥ 1 child compared to nulliparous women ($p < .001$). Significant group differences were observed for maternal weight and BMI, but not height. Mothers > 23 years, ≥ 1 child (70.9 kg, IQR: 60.6–81.7) had a higher median weight than nulliparous women (65.9 kg, IQR: 57.3–77.4; $p < .001$) and higher BMI at recruitment (28.6 kg/m², IQR: 24.4–32.2), with more classified as overweight ($p < .001$). No significant height differences occurred ($p = .165$), indicating weight as the main driver of BMI variation. Gestational weight gain per week was higher in nulliparous women (0.4 kg/week, IQR: 0.3–0.5) than mothers > 23 years, ≥ 1 child (0.3 kg/week, IQR: 0.2–0.4; $p < .001$).

Differences in maternal pregnancy outcomes at baseline were also identified. Significant differences were found in hypertension, with a higher prevalence of chronic ($p = .007$) and pregnancy hypertension ($p < .001$) in mothers > 23 years, ≥ 1 child. Similarly, GDM was significantly more prevalent in mothers > 23 years, ≥ 1 child compared to nulliparous women ($p = .012$), whereas the converse occurred for respiratory disease ($p = .001$).

For delivery outcomes, there was a marginally significant difference in gestational age at delivery, with infants from nulliparous women having greater gestational age (39, IQR: 38–40) compared to mothers > 23 years, ≥ 1 child (38, IQR: 37–39). Infants of nulliparous women had significantly lower median birth weight (2960, IQR: 2581.25–3228.75) compared to those of mothers ≤ 23 years, ≥ 1 child (3185, IQR: 2935–3342.5, $p = .002$), which corresponds to low birth weight being more common among the nulliparous group ($p = .009$). Small for gestational age infants comprised 17.2% of the sample and were most common among nulliparous women (22.9%;

Table 1 Maternal sociodemographic, anthropometry, pregnancy and delivery outcomes by maternal age-parity group

Variable	Total (n = 830)	Nulliparous (n = 231)	Mothers ≤ 23 years, ≥ 1 child (n = 54)	Mothers > 23 years, ≥ 1 child (n = 545)	p
Individual					
Maternal Age (mean, SD)	29.81 (5.89)	26.07 (4.98)	22.22 (0.9)	32.15 (5.02)	< 0.001***
Maternal Age (median, IQR; max)	29 (25–34); 44	24 (23–28); 44; n = 231	22 (22–23); 23; n = 54	32 (28–36); 43; n = 545	< 0.001***
Level of Education					< 0.001***
Up to Secondary Education	621 (74.8)	150 (64.9)	43 (79.6)	428 (78.5)	
Tertiary Education	209 (25.2)	81 (35.1)	11 (20.4)	117 (21.5)	
HIV status					< 0.001***
Negative	563 (67.8)	182 (78.8)	39 (72.2)	342 (62.8)	
Positive	267 (32.2)	49 (21.2)	15 (27.8)	203 (37.3)	
Smoking Status; n = 829					0.328
Nonsmoker	758 (91.4)	206 (89.6)	48 (88.9)	504 (92.5)	
Smoker	71 (8.6)	24 (10.4)	6 (11.1)	41 (7.5)	
Household					
SES (median, IQR; max)	6 (5–6); 9	6 (5–7); 9; n = 231	5.5 (5–7); 9; n = 54	5 (5–6); 9; n = 545	0.009**
Maternal Anthropometry					
Weight, kg (mean, SD)	71.26 (16.2)	67.94 (15.43)	69.03 (13.91)	72.89 (16.51)	< 0.001***
Weight, kg (median, IQR; max)	69.1 (59.7–80.6); 158.8	65.9 (57.3–77.35); 137.1	66.95 (60.3–75.2); 111.8	70.9 (60.6–81.7); 158.8	< 0.001***
Height, cm (mean, SD)	158.77 (6.69)	159.22 (7.71)	158.09 (5.42)	158.64 (6.34)	0.165
Height, cm (median, IQR; max)	158.5 (154.8–162.6); 193.6	159.2 (155.3–163.05); 193.6	157 (154.8–160.83); 173.9	158.4 (154.5–162.4); 180.2	0.165
BMI at recruitment, kg/m ² (< 14 weeks) (median, IQR; max)	27.62 (23.79–31.44); 60.58	26.28 (22.23–29.68); 53.62	26.59 (23.99–30.86); 43.81	28.57 (24.41–32.21); 60.58	< 0.001***
Maternal BMI Category					< 0.001***
Underweight (< 18.5)	276 (33.3)	94 (40.7)	21 (38.9)	161 (29.5)	
Normal (18.5 < 25)	272 (32.8)	85 (36.8)	17 (31.5)	170 (31.2)	
Overweight (25 ≤)	282 (34)	52 (22.5)	16 (29.6)	214 (39.3)	
Gestational Weight, kg/week (median, IQR; max); n = 829	0.35 (0.23–0.47); 2.2	0.39 (0.26–0.54); 1.01; n = 230	0.38 (0.28–0.51); 0.94; n = 54	0.33 (0.21–0.44); 2.2; n = 545	< 0.001***
Maternal Pregnancy Factors at Baseline Pregnancy Visit 1					
Anaemia; n = 814					0.181
No	802 (98.5)	217 (97.3)	53 (98.2)	532 (99.1)	
Yes	12 (1.5)	6 (2.7)	1 (1.9)	5 (0.9)	
Cardiac Disease; n = 827					0.555
No	817 (98.8)	226 (99.1)	54 (100)	537 (98.5)	
Yes	10 (1.2)	2 (0.88)	0	8 (1.5)	
Chronic Hypertension (pre-pregnancy diagnosis); n = 822					0.007**
No	749 (91.1)	214 (94.7)	53 (98.2)	482 (88.9)	
Yes	73 (8.9)	12 (5.3)	1 (1.9)	60 (11.1)	
Hypertension (during pregnancy); n = 815					< 0.001***
No	729 (89.5)	208 (93.7)	54 (100)	467 (86.6)	
Yes	86 (10.6)	14 (6.3)	0	72 (13.7)	
Respiratory disease; n = 823					0.001**
No	790 (96)	208 (92)	54 (100)	528 (97.2)	
Yes	33 (4)	18 (8)	0	15 (2.8)	
Kidney Disease; n = 826					0.661
No	818 (99)	225 (98.7)	54 (100)	539 (99.1)	
Yes	8 (1)	3 (1.3)	0	5 (0.9)	
Proteinuria; n = 792					0.323

Table 1 (continued)

Variable	Total (n=830)	Nulliparous (n=231)	Mothers ≤ 23 years, ≥ 1 child (n=54)	Mothers > 23 years, ≥ 1 child (n=545)	p
No	779 (98.4)	214 (99.1)	53 (100)	512 (98)	0.986
Yes	13 (1.6)	2 (0.9)	0	11 (2.1)	
Type 2 Diabetes; n = 826					0.986
No	813 (97.9)	226 (97.8)	53 (98.2)	534 (98)	
Yes	17 (2.1)	5 (2.2)	1 (1.8)	11 (2)	0.012*
Gestational Diabetes Mellitus; n = 741					
No	658 (88.8)	195 (94.2)	42 (89.4)	421 (86.5)	0.012*
Yes	83 (11.2)	12 (5.8)	5 (10.6)	66 (13.6)	
Delivery Outcomes					0.032*
Gestational age at delivery, weeks (median, IQR; max); n = 814	39 (37–40); 42	39 (38–40); 42; n = 226	39 (38–39.5); 41; n = 51	38 (37–39); 42; n = 537	
Preterm Delivery	129 (15.9)	36 (15.9)	5 (9.8)	88 (16.4)	0.469
Birth Status; n = 800					0.746
Alive	788 (98.5)	217 (98.6)	51 (100)	520 (98.3)	0.537
Antepartum Death	5 (0.6)	2 (0.9)	0	3 (0.6)	
Intrapartum Death	7 (0.88)	1 (0.5)	0	6 (1.1)	
Neonatal Sex; n = 828					0.183
Male	430 (51.9)	118 (51.3)	32 (59.3)	280 (51.5)	
Female	398 (48.1)	112 (48.7)	22 (40.7)	264 (48.5)	0.002**
Mode of Delivery; n = 814					
Vaginal	340 (41.8)	106 (46.9)	20 (39.2)	214 (39.9)	0.009**
Caesarean Section	474 (58.2)	120 (53.1)	31 (60.8)	323 (60.1)	
Infant Anthropometry					0.026*
Infant Birth Weight, g (median, IQR; max); n = 791	3030 (2675–3300); 4500	2960 (2581.25–3228.75); 4165; n = 218	3185 (2935–3342.5); 3935; n = 51	3040 (2720–3327.5); 4500; n = 522	
Birth Weight Category; n = 791					< 0.001***
Small for Gestational Age (SGA)	136 (17.2)	50 (22.9)	6 (11.8)	80 (15.3)	
Appropriate for Gestational Age	599 (75.7)	161 (73.9)	42 (82.4)	396 (75.9)	0.04*
Large for Gestational Age	56 (97.1)	7 (3.2)	3 (5.9)	46 (8.8)	
Low Birth Weight	135 (17.1)	43 (19.7)	1 (2)	91 (17.4)	0.04*
Birth Length, cm (median, IQR; max); n = 790	48.5 (46.7–50.2); 64.2	48.7 (46.7–50.38); 58; n = 218	49 (48–51.05); 61; n = 51	48.4 (46.6–50); 64.2; n = 521	
Birth Weight Z-score (median, IQR; max); n = 791	−0.32 (−1–0.38) 3.86	−0.59 (−1.24–0.11); 3.09; n = 218	−0.13 (−0.6–0.65); 1.58; n = 51	−0.28 (−0.91–0.5); 3.86; n = 522	0.04*
Birth Length Z-score (median, IQR; max); n = 790	−0.25 (−1.07–0.77); 5.34	−0.27 (−1.08–0.72); 4.1; n = 218	0.41 (−0.63–1.33); 4.53; n = 51	−0.29 (−1.1–0.7); 5.34; n = 521	

$p=.009$), while LGA was most prevalent among mothers > 23 years, ≥ 1 child (8.8%). A higher median birth length was observed among infants born to mothers ≤ 23 years, ≥ 1 child (49, IQR: 48.0–51.1) compared to mothers > 23 years, ≥ 1 child (48.4, IQR: 46.6–50, $p=.026$). Similarly, birth length z-scores differed significantly across groups, with infants born to mothers ≤ 23 years, ≥ 1 child having higher median z-score (0.41, IQR: −0.63 to 1.33), compared to those of mothers > 23 years, ≥ 1 child (−0.29, IQR: −1.10 to −0.70; $p=.040$).

Maternal Age-Parity Influence on Infant Outcomes

Results from the linear regression models assessing the relationship between maternal age-parity and infant birth weight z-scores are presented in Table 2. Findings from the unadjusted model (M1) show that both mothers ≤ 23 years, ≥ 1 child ($\beta = 0.52$, 95% CI [0.21–0.83], $p=.001$) and mothers > 23 years, ≥ 1 child ($\beta = 0.35$, 95% CI [0.19–0.51], $p<.001$) had significantly higher birth weight z-scores compared to nulliparous mothers, with these associations remaining significant across all subsequent models. Birth weight z-scores remained significantly higher in

Table 2 Linear regression analyses of the association between age-parity groups and maternal Body Mass Index, gestational diabetes mellitus and hypertension on continuous birth weight z-scores

[illegible]

Table 2 (continued)

	M1 (n = 791)			M2 (n = 779)			M3 (n = 779)			M4 (n = 690)			M5 (n = 777)			M6 (n = 689)		
	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p
Up to Secondary Education	Ref			Ref			Ref			Ref			Ref			Ref		
Higher education	0.1	-0.07; 0.28	0.239	0.2	-0.3; 0.69	0.429	0.06	-0.12; 0.24	0.527	0.11	-0.07; 0.28	0.223	0.08	-0.1; 0.26	0.363			
Model 1 (M1) = Unadjusted Linear regression of age-parity and birth weight z-scores																		
Model 2 (M2) = Linear regression of age-parity and birth weight z-scores, adjusted for neonatal gender, HIV status, SES, and level of education																		
Model 3 (M3) = Linear regression including age-parity and birth weight z-scores, with BMI category added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 4 (M4) = Linear regression including age-parity and birth weight z-scores, with GDM added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 5 (M5) = Linear regression including age-parity and birth weight z-scores, with hypertension added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 6 (M6) = Fully adjusted linear regression model including age-parity, birth weight z-scores, and all maternal health factors (BMI, GDM, and hypertension), adjusted for neonatal gender, HIV status, SES, and maternal education																		

CI Confidence Interval, BMI/Body Mass Index, GDM Gestational Diabetes Mellitus, HIV Human Immunodeficiency Virus, SES Socioeconomic Status

the mothers ≤ 23 years, ≥ 1 child ($\beta = 0.52$, 95% CI [0.20–0.83], $p < .001$) and mothers > 23 years, ≥ 1 child ($\beta = 0.33$, 95% CI [0.16–0.50], $p < .001$) groups after including BMI in Model 3. Additionally, both normal ($\beta = 0.21$, 95% CI [0.04–0.39], $p = .018$) and overweight ($\beta = 0.35$, 95% CI [0.18–0.53], $p < .001$) BMI categories were associated with significantly higher birth weight z-scores compared to underweight status. In Model 4, GDM was not significantly associated with birth weight z-scores ($p = .231$), and hypertension in Model 5 was not significantly associated with birth weight z-scores ($p = .263$). In the fully adjusted model including all variables and covariates (M6), the association between age-parity and birth weight z-scores remained significant both for mothers ≤ 23 years, ≥ 1 child ($\beta = 0.60$, 95% CI [0.27–0.93], $p < .001$) and mothers > 23 years, ≥ 1 child ($\beta = 0.38$, 95% CI [0.2–0.55], $p < .001$). Maternal BMI continued to show a significant positive influence on birth weight in this model, with normal ($\beta = 0.19$, 95% CI [0.01–0.38], $p = .038$) and overweight ($\beta = 0.39$, 95% CI [0.20–0.57], $p < .001$) mothers having significantly higher birth weight z-scores.

The logistic regression analysis is presented in Table 3, which assessed the odds of delivering a LGA infant versus SGA infant across age-parity groups. Model 1 showed that mothers > 23 years, ≥ 1 child had significantly higher odds of delivering LGA infants compared to nulliparous women (OR = 4.11, 95% CI [1.72–9.81], $p = .001$), whereas the association for mothers ≤ 23 years, ≥ 1 child was not statistically significant (OR = 3.57, 95% CI [0.75–17.07], $p = .118$). Adjustment for neonatal sex, HIV status, SES, and education in Model 2 revealed a slight strengthening of the association for both mothers ≤ 23 years, ≥ 1 child (OR = 4.36, 95% CI [0.83–22.94], $p = .082$) and mothers > 23 years, ≥ 1 child (OR = 5.47, 95% CI [2.17–13.76], $p < .001$); however, the results for mothers ≤ 23 years, ≥ 1 child remained non-significant. When BMI was introduced in Model 3, the odds of LGA remained significantly high for mothers > 23 years, ≥ 1 child (OR = 3.99, 95% CI [1.55–10.28], $p = .004$). Normal-weight mothers (vs. underweight) had significantly increased odds of LGA (OR = 5.40, 95% CI [1.65–17.72], $p = .005$), with even greater odds observed in overweight mothers (OR = 11.34, 95% CI [3.55–36.24], $p < .001$). The association between mothers > 23 years, ≥ 1 child and LGA further increased when GDM was introduced in Model 4 (OR = 8.04, 95% CI [2.58–25.05], $p < .001$); however, GDM itself was not significantly associated with LGA (OR = 1.12, 95% CI [0.34–3.73], $p = .851$). Hypertension was not associated with LGA in Model 5 (OR = 1.07, 95% CI [0.43–2.67], $p = .882$), and the association between mothers > 23 years, ≥ 1 child status and LGA remained significant (OR = 6.12, 95% CI [2.28–16.47], $p < .001$). In Model 6, mothers > 23 years, ≥ 1 child continued to show significantly higher odds of delivering LGA infants

Table 3 Logistical regression analyses of the association between age-parity groups and maternal Bodu Mass Index, gestational diabetes mellitus and hypertension on categorical birth weight (SGA vs. LGA)

	M1 (n = 192)			M2 (n = 189)			M3 (n = 189)			M4 (n = 167)			M5 (n = 189)			M6 (n = 167)		
	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
Age-Parity																		
Nulliparous	Ref			Ref			Ref			Ref			Ref			Ref		
Mothers < 23 years, ≥ 1 child	3.57	0.72; 17.61	0.118	4.36	0.83; 22.94	0.082	2.11	0.39; 11.4	0.387	8.97	1.09; 73.88	0.042*	5.06	0.93; 27.44	0.06	5.63	0.6; 52.78	0.131
Mothers ≥ 23 years, ≥ 1 child	4.11	1.72; 9.81	0.001**	5.47	2.17; 13.76	<0.001***	3.99	1.55; 10.28	0.004**	8.04	2.58; 25.05	<0.001***	6.12	2.28; 16.47	<0.001***	7.95	2.12; 29.88	0.002***
BMI																		
Underweight (< 18.5)	Ref						Ref									Ref		
Normal (18.5 < 25)							5.4	1.65; 17.72	0.005**							7.98	1.65; 38.55	0.01*
Overweight (25 ≤)							11.34	3.55; 36.24	<0.001***							16.31	3.45; 77	<0.001***
GDM																		
No										Ref						Ref		
Yes										1.12	0.34; 3.73	0.851				1.1	0.28; 4.29	0.888
Hypertension (during pregnancy);																		
No																		
Yes													Ref			Ref		
													1.07	0.43; 2.67	0.882	0.78	0.28; 2.15	0.627
Neonatal Sex																		
Male				Ref			Ref			Ref			Ref			Ref		
Female				1.04	0.54; 2.02	0.769	1.03	0.51; 2.09	0.93	1.08	0.52; 2.25	0.837	1.12	0.57; 2.21	0.736	1.2	0.54; 2.66	0.663
HIV Status																		
Negative				Ref			Ref			Ref			Ref			Ref		
Positive				0.5	0.24; 1.04	0.063	0.59	0.27; 1.31	0.197	0.47	0.21; 1.07	0.072	0.54	0.26; 1.12	0.099	0.62	0.26; 1.52	0.295
SES				0.86	0.67; 1.1	0.233	0.79	0.6; 1.04	0.089	0.9	0.66; 1.24	0.525	0.89	0.69; 1.15	0.374	0.91	0.65; 1.28	0.603
Level of Education																		

Table 3 (continued)

	M1 (n = 192)			M2 (n = 189)			M3 (n = 189)			M4 (n = 167)			M5 (n = 189)			M6 (n = 167)		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Up to Secondary Education				Ref			Ref			Ref			Ref			Ref		
Higher Education				2	0.9; 4.39	0.09	1.89	0.8; 4.44	0.145	1.77	0.73; 4.29	0.208	2.16	0.96; 4.83	0.062	1.63	0.61; 4.36	0.33
Model 1 (M1): Unadjusted logistic regression of age-parity and categorical birth weight																		
Model 2 (M2): Logistic regression of age-parity and categorical birth weight, adjusted for neonatal gender, HIV status, SES, and level of education																		
Model 3 (M3): Logistic regression including age-parity and categorical birth weight, with BMI category added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 4 (M4): Logistic regression including age-parity and categorical birth weight, with GDM added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 5 (M5): Logistic regression including age-parity and categorical birth weight, with hypertension added, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 6 (M6): Fully adjusted logistic regression model including age-parity, categorical birth weight, and all maternal health factors (BMI, GDM, and hypertension), adjusted for neonatal gender, HIV status, SES, and maternal education																		
OR Odds Ratio, CI Confidence Interval, BMI Body Mass Index, GDM Gestational Diabetes Mellitus, HIV Human Immunodeficiency Virus, SES Socioeconomic Status																		

(OR = 7.95, 95% CI [2.12–29.88], $p = .002$). Neither GDM nor hypertension showed independent significant associations with birth weight category in the fully adjusted model. BMI retained its significant effect, with normal-weight (OR = 7.98, 95% CI [1.64–38.77], $p = .010$) and overweight (OR = 16.31, 95% CI [3.99–66.71], $p < .001$) mothers showing higher odds of LGA compared to underweight counterparts. Across all models, none of the covariates reached statistical significance.

The linear regression models presented in Table 4 include the results for birth length z-scores. Mothers ≤ 23 years, ≥ 1 child had infants with significantly higher birth length z-scores compared to nulliparous mothers ($\beta = 0.49$, 95% CI [0.01–0.97], $p = .046$) in the unadjusted model (Model 1). While this association remained significant across all models, the inclusion of BMI (Model 3), GDM (Model 4), and hypertension (Model 5) did not significantly alter the strength or direction of the observed association for mothers < 23 years, ≥ 1 child. Moreover, none of the maternal factors themselves were significantly associated with birth length z-scores. Socio-economic status was significantly associated with birth length z-scores in Model 2 ($\beta = 0.12$, 95% CI [0.03–0.21], $p = .012$), Model 3 ($\beta = 0.11$, 95% CI [0.02–0.20], $p = .018$), Model 4 ($\beta = 0.13$, 95% CI [0.03–0.22], $p = .011$), Model 5 ($\beta = 0.12$, 95% CI [0.03–0.21], $p = .009$), and Model 6 ($\beta = 0.12$, 95% CI [0.02–0.22], $p = .016$).

Table 5 presents the results from the Linear regression models for the gestational age outcome. The unadjusted Model 1 showed no statistically significant association between maternal age-parity group and gestational age at delivery. These associations remained non-significant after adjusting for covariates in Model 2. Across Models 3 to 6, the associations between maternal age-parity and gestational age remained non-significant. Hypertension was independently associated with shorter gestation in Model 5 ($\beta = -1.03$, 95% CI [-1.59 to -0.48], $p < .001$) and Model 6 ($\beta = -0.99$, 95% CI [-1.52 to -0.45], $p < .001$). Additionally, GDM showed a modest but statistically significant negative association with gestational age in Model 4 ($\beta = -0.61$, 95% CI [-1.13 to -0.08], $p = .025$) and remained significant in Model 6 ($\beta = -0.57$, 95% CI [-1.10 to -0.04], $p = .036$). These findings suggest that while maternal age and parity do not independently influence gestational age at delivery, hypertension and GDM are key risk factors associated with shorter gestation. Maternal BMI category was not significantly associated with gestational age in any model, and none of the covariates showed significant associations.

Discussion

Key findings

This study investigated the combined influence of maternal age and parity on both pregnancy outcomes

Table 4 Linear regression analyses of the association between age-parity groups and maternal Body Mass Index, gestational diabetes mellitus and hypertension on continuous birth length z-scores

M1 (n = 790)			M2 (n = 778)			M3 (n = 778)			M4 (n = 689)			M5 (n = 776)			M6 (n = 688)		
β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p
Age-Parity																	
Nulliparous	Ref		Ref			Ref			Ref			Ref			Ref		
Mothers < 23 years, ≥ 1 child	0.49	0.01; 0.97	0.046*	0.51	0.03; 0.99	0.038*	0.5	0.02; 1	0.041*	0.64	0.13; 1.2	0.014*	0.5	0.01; 0.98	0.044*	0.62	0.11; 1.13
Mothers ≥ 23 years, ≥ 1 child	-0.09	-0.34; 0.16	0.49	-0.04	-0.29; 0.22	0.772	-0.06	-0.32; 0.2	0.647	-0.006	-0.27; 0.26	0.968	-0.01	-0.27; 0.25	0.937	-0.01	-0.29; 0.27
BMI																	
Underweight (< 18.5)	Ref		Ref			Ref									Ref		
Normal (18.5 < 25)						0.18	-0.09; 0.45	0.202							0.17	-0.11; 0.46	0.234
Overweight (25 ≤)						0.18	-0.1; 0.45	0.205							0.19	-0.1; 0.48	0.196
GDM																	
No									Ref						Ref		
Yes									-0.1	-0.46; 0.27	0.605				-0.11	-0.48; 0.26	0.557
Hypertension (during pregnancy);																	
No												Ref					
Yes												-0.28	-0.64; 0.09	0.138	-0.2	-0.58; 0.17	0.298
Neonatal Sex																	
Male						Ref			Ref			Ref			Ref		
Female						-0.04	-0.26; 0.18	0.703	-0.05	-0.27; 0.17	0.679	-0.04	-0.27; 0.19	0.756	-0.05	-0.27; 0.18	0.734
HIV Status																	
Negative						Ref			Ref			Ref			Ref		
Positive						-0.08	-0.32; 0.16	0.512	-0.07	-0.31; 0.16	0.54	-0.07	-0.31; 0.18	0.609	-0.08	-0.32; 0.16	0.649
SES						0.12	0.03; 0.21	0.012*	0.11	0.02; 0.2	0.018*	0.13	0.03; 0.22	0.011*	0.12	0.03; 0.21	0.009**
Level of Education															0.12	0.02; 0.22	0.016*

Table 4 (continued)

	M1 (n = 790)			M2 (n = 778)			M3 (n = 778)			M4 (n = 689)			M5 (n = 776)			M6 (n = 688)		
	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p

Up to Secondary Education

Higher education

Model 1 (M1) = Unadjusted Linear regression of age-parity birth length z-scores

Model 2 (M2) = Linear regression of age-parity and birth length z-scores adjusted for neonatal gender, HIV status, SES and level of education

Model 3 (M3) = Linear regression including age-parity and birth length z-scores, with BMI category added, adjusted for neonatal gender, HIV status, SES, and maternal education

Model 4 (M4) = Linear regression including age-parity and birth length z-scores, with GDM added, adjusted for neonatal gender, HIV status, SES, and maternal education

Model 5 (M5) = Linear regression including age-parity and birth length z-scores, with hypertension added, adjusted for neonatal gender, HIV status, SES, and maternal education

Model 6 (M6) = Fully adjusted linear regression model including age-parity, birth length z-scores, and all maternal health factors (BMI, GDM, and hypertension), adjusted for neonatal gender, HIV status, SES, and maternal education

CI Confidence Interval, BMI Body Mass Index, GDM Gestational Diabetes Mellitus, HIV Human Immunodeficiency Virus, SES Socioeconomic Status

(BMI, GDM and hypertension), and birth outcomes (birth weight and length z-scores, SGA, LGA and gestational age) in a South African urban cohort. Overall, we found evidence partially in support of our hypothesised U-shaped relationship, whereby both extremes of maternal age and parity were expected to be associated with variations in outcomes. Nulliparous women had lower infant birth weight (2960 g vs. 3185 g, $p < .001$), higher rates of SGA births (22.9% vs. 15.3%, $p < .001$), and lower BMI (26.28 vs. 28.57 kg/m², $p < .001$). In contrast, mothers > 23 years, ≥ 1 child had higher BMI (26.3 vs. 28.6 kg/m², $p < .001$), and higher prevalence of hypertension (13.7% vs. 6.3%, $p < .001$) and GDM (13.6% vs. 5.8%, $p = .012$), as well as a greater proportion of LGA infants (8.8% vs. 3.2%, $p = .009$). While median BMI values for the nulliparous and mothers > 23 years, ≥ 1 child groups are not classified as obese, associations with birth outcomes were found in our study, particularly SGA among nulliparous women and LGA among mothers > 23 years, ≥ 1 child. Mothers ≤ 23 years, ≥ 1 child delivered infants with higher birth weight and length z-scores compared to nulliparous women (2960 g vs. 3185, $p < .001$), suggesting a protective effect of reproductive experience when combined with younger maternal age. These findings address a notable gap in the literature by simultaneously examining multiple maternal risk factors, thereby offering insights into reproductive risk factors in LMICs and informing targeted antenatal interventions based on age and parity.

Contextualisation of findings

Group difference analysis revealed significant differences across maternal age-parity groups. In line with global and South African trends [35, 36], nulliparous women were more likely to have completed higher levels of education. Mothers > 23 years, ≥ 1 child had a higher prevalence of HIV, consistent with prior research [37–39], which is possibly due to prolonged sexual activity and greater cumulative exposure to HIV risk factors [40]. Significant group differences also occurred for maternal anthropometric measures. Mothers > 23 years, ≥ 1 child had moderately higher weight and BMI compared to nulliparous women, and a greater proportion classified as overweight, which is associated with increased risk of metabolic complications, gestational diabetes, and LGA deliveries. These findings correspond with studies showing factors contributing to age-related weight-gain in women, such as poor-quality diets, decreased physical activity, hormonal changes, cumulative weight, retention and metabolic changes associated with successive pregnancies [41]. Despite having a higher BMI in early pregnancy, multiparous women had moderately lower gestational weight gain per week compared to nulliparous women, consistent with findings from a large retrospective cohort [42].

Table 5 Linear regression analyses of the association between age-parity groups and maternal body mass index, gestational diabetes mellitus and hypertension on continuous gestational age (weeks)

[illegible]

Table 5 (continued)

	M1 (n = 814)			M2 (n = 801)			M3 (n = 801)			M4 (n = 712)			M5 (n = 798)			M6 (n = 710)		
	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p	β	95% CI	p
Up to Secondary Education	Ref			Ref			Ref			Ref			Ref			Ref		
Higher education	0.29	-0.11; 0.69	0.155	0.29	-0.11; 0.69	0.155	0.3	-0.1; 0.7	0.145	0.25	-0.14; 0.65	0.211	-0.34	-0.07; 0.75	0.099	0.32	-0.9; 0.71	0.122
Model 1 (M1) = Unadjusted Linear regression of age-parity gestational age																		
Model 2 (M2) = Linear regression of age-parity and gestational age adjusted for neonatal gender, HIV status, SES and level of education																		
Model 3 (M3) = Linear regression including age-parity, gestational age, and BMI category, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 4 (M4) = Linear regression including age-parity, gestational age, and GDM, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 5 (M5) = Linear regression including age-parity, gestational age, and hypertension, adjusted for neonatal gender, HIV status, SES, and maternal education																		
Model 6 (M6) = Fully adjusted linear regression model including age-parity, gestational age, and all maternal health factors (BMI, GDM, and hypertension), adjusted for neonatal gender, HIV status, SES, and maternal education																		
CI Confidence Interval, BMI Body Mass Index, GDM Gestational Diabetes Mellitus, HIV Human Immunodeficiency Virus, SES Socioeconomic Status																		

This greater weight gain in nulliparous women is likely due to a combination of factors, such as their body's initial physiological adaptation to pregnancy as well as potential behavioural and psychological influences [43, 44]. Gestational weight gain patterns have important implications, with both inadequate and excessive gain being associated with pregnancy outcomes. Insufficient weight gain is associated with increased risks of IUGR, low birth weight, and preterm birth, which may translate to SGA infants [45] who are more prone to complications such as hypoglycaemia [46], temperature instability, and respiratory distress [47], as well as developmental delays [48], and long-term health conditions, including metabolic and cardiovascular diseases [49]. Conversely, excessive weight gain increases the likelihood of LGA infants [50], gestational diabetes, hypertensive disorders [51, 52], labour complications such as dystocia, as well as an increased rate of caesarean section deliveries [50].

Partly in line with our hypothesis, we found that parity at one extreme (i.e., nulliparity) was largely associated with lower birth weight, as well as a higher rate of SGA births, which have important implications given that SGA is associated with neonatal morbidity [45–47, 53], developmental delays [48], and long-term health conditions, including metabolic and cardiovascular diseases [49]. Our findings align with research demonstrating first pregnancy complications associated with uterine inexperience. Without prior gestational experience, the uterus of nulliparous women has yet to undergo adaptive remodelling and vascular changes [54–57], which may lead to suboptimal maternal–fetal resource allocation [54, 58, 59]. Further evidence in support of uterine priming, whereby prior pregnancies facilitate physiological adaptations that promote fetal growth, is our finding that multiparity, independent of maternal age, was significantly associated with birth outcomes. Infants from mothers ≤ 23 years, ≥ 1 child and mothers > 23 years, ≥ 1 child groups had infants with moderately higher birth weight scores than nulliparous women. However, only infants born to mothers ≤ 23 years, ≥ 1 child had moderately higher birth length z-scores, which have significant implications given that greater birth length is associated with better growth trajectories [60]. These findings suggest that linear growth (i.e., birth length) is more sensitive to maternal age, which is supported by prior research. In particular, studies suggest that linear growth depends more on placental and vascular function, which often declines with maternal age. Vascular ageing, characterised by increased arterial stiffness, reduced blood flow to the uterus, and impaired endothelial function, can limit the placental capacity to support sustained linear growth [61, 62].

Similar to age and parity, maternal BMI was also largely linked to infant birth weight, with normal and

overweight mothers delivering infants with higher birth weight z-scores and having increased odds of LGA births compared to underweight women. Moreover, maternal BMI partially explained the association between age-parity, particularly in mothers >23 years, ≥ 1 child, and birth weight and LGA. These findings are consistent with existing evidence not only showing that older and multiparous women tend to have higher BMI but also that higher maternal BMI promotes greater fetal growth and fat deposition due to increased nutrient transfer [41, 63–65]. Despite the moderate effect, the associated risks of LGA births, such as delivery complications and increased likelihood of future metabolic disease, underscore the importance of these findings [50–52].

In contrast to birth weight and linear growth outcomes, maternal age-parity was not significantly associated with gestational age at delivery in our study. However, gestational age was independently and moderately associated with maternal GDM and hypertension, both linked to shorter gestations. Shorter gestational age and preterm birth are associated with higher rates of mortality and morbidity, such as cerebral palsy, respiratory conditions, and increased need for intensive neonatal care [66]. These results are possibly attributed to metabolic disturbances linked to elevated maternal blood glucose levels, and vascular dysfunction associated with hypertension, both of which contribute to placental insufficiency, triggering early delivery, spontaneously or through medical intervention [67–69].

Together, these findings suggest a dual burden of age and parity, particularly for mothers >23 years, ≥ 1 child. While prior pregnancies may enhance physiological readiness for childbirth, supporting fetal growth, age-related increases in BMI and metabolic risk seemingly offset these benefits. In particular, these conditions increase the likelihood of LGA births and associated complications such as obstructed labour, perineal trauma, and postpartum haemorrhage [28, 50, 51]. In contrast, young nulliparous women, particularly those with low BMI, may be at higher risk of SGA births.

Implications

Our findings suggest that maternal age and parity together influence pregnancy and birth outcomes. This highlights the limitations of age-based risk frameworks, suggesting that solely relying on maternal age in prenatal risk assessment may be insufficient and potentially overlook important reproductive and metabolic risk factors.

Research implications of our study include highlighting the need to incorporate more detailed reproductive histories, for example age at first birth, intervals between pregnancies and cumulative parity, to better understand how these factors influence maternal and fetal outcomes. Additionally, the findings demonstrate the value of

investigating how underlying mechanisms, such as placental function, vascular and metabolic adaptations, and inflammation, influence the relationship between maternal age, parity, and birth outcomes, which may clarify causal pathways.

The findings emphasise the need for antenatal care to go beyond standard maternal age screening but also account for reproductive history (i.e., parity) and emerging chronic disease risks, such as GDM and hypertension. In particular, they underscore the importance of more tailored antenatal interventions. For example, nutritional counselling and fetal growth monitoring can be applied for underweight, nulliparous women, whereas weight management support and proactive metabolic screening may be beneficial for overweight, older multiparous women. These findings can, therefore, guide healthcare providers in optimising resource allocation, facilitating earlier identification and intervention for at-risk women, and improving maternal and infant outcomes.

Finally, this study highlights the need for refined antenatal care guidelines in South Africa and other LMICs. While maternal age, parity, and chronic disease risk factors are recorded in existing frameworks such as the Basic Antenatal Care (BANC) the Road to Health Card (RTHC), this study emphasises the need to improve the application of these data. In particular, our findings underscore the importance of risk stratification models that not only document information but also uses it to improve risk identification and targeted interventions. This goal aligns with the South Africa's Reproductive, Maternal, Newborn, Child, and Adolescent Health (RMNCAH) policy framework and the WHO 2020 antenatal guidelines [70], which prioritises personalised, context-specific risk-based models of antenatal care. More effective use of data on key factors identified in this study, namely maternal age, parity, and chronic disease risk, within existing approaches can improve risk stratification by better identifying women who are more likely to experience adverse outcomes, thereby enabling the earlier detection and more targeted intervention.

Limitations and future research

Our study has several limitations that need to be considered when interpreting the findings. Age at recruitment and parity were used as a proxy for estimating the age at first childbirth, assuming regular birth intervals, with a cutoff age of 23 applied rather than the widely accepted threshold of 35 years. This approach may lead to misclassification of maternal age-parity groupings, limit our ability to distinguish the effects of early childbearing from those of higher parity on birth outcomes and, therefore, not fully capture the complexity of maternal-age related risks. Due to the inclusion criteria of 18 years and the absence of older mothers in the sample, this study does

not capture the extremes of maternal age. This is a notable limitation given that both adolescent and advanced maternal age are associated with adverse maternal and infant outcomes. The recruitment through clinic attendance may introduce selection bias, limiting the representativeness of the sample. While BMI, GDM, and hypertension were included in the analysis of age-parity groups and infant outcomes, there remains the potential for various other residual confounders not accounted for in this study that may have influenced the observed associations (e.g., nutritional status, substance use, environmental exposures). Due to the cross-sectional nature of the study, causality between maternal age, parity, and infant outcomes cannot be inferred. Finally, missing birth record data and the specific urban, South African context of the cohort limit the generalisability of our findings to other populations and settings. Specifically, our findings may not be applicable to rural communities, mothers accessing private healthcare, or populations in high-income countries with different healthcare systems.

Future research should focus on incorporating more detailed reproductive histories (i.e., age at first birth, total number of pregnancies, and the interval between pregnancies), to provide clarity on how timing and frequency of childbirth influence maternal and fetal outcomes. Biological and physiological indicators should be considered in future studies to provide an understanding of the underlying processes that link maternal age, parity, and metabolic risk to birth outcomes. Additionally, future studies should include maternal age extremes to provide a more comprehensive assessment of the influence of maternal age and parity on infant outcomes.

Conclusion

This study provides important information on the combined influence of maternal age, parity, and metabolic risk factors on pregnancy and birth outcomes in a South African urban cohort. The findings reveal distinct vulnerabilities across maternal age-parity groups. Multiparity was associated with higher birth weight, partly explained by higher BMI in older mothers, while linear growth appeared more sensitive to maternal age, favouring younger multiparous women. Parity likely influences birth outcomes through structural mechanisms, namely placental and uterine adaptations, rather than through metabolic or hypertensive pathways. However, exploring these mechanisms was beyond the scope of this study and, therefore, warrants further research. Nulliparous women, despite higher SES and education, had infants with lower birth weight, higher rates of SGA, and lower BMI. Hypertension and GDM were both associated with shorter gestational age. These findings underscore the need for more holistic approaches to antenatal care in

LMICs, where limited access to healthcare and resources can exacerbate existing risks.

Abbreviations

Abbreviation	Full Term
AGA	Appropriate for gestational age
BANC	Basic Antenatal Care
BMI	Body mass index
CHBAH	Chris Hani Baragwanath Academic Hospital
DHS	Demographic and Health Surveys
DPHRU	Developmental Pathways for Health Research Unit
GDM	Gestational diabetes mellitus
IUGR	Intrauterine Growth Restriction
LMIC(s)	Low- and middle-income country (countries)
LGA	Large for gestational age
OGTT	Oral glucose tolerance test
RMNCAH	Reproductive, Maternal, Newborn, Child and Adolescent Health
RTHC	Road to Health Card
S1000	Soweto First 1000 Days
SAMRC	South African Medical Research Council
SGA	Small for gestational age
SES	Socioeconomic status
WHO	World Health Organization

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12884-025-08194-8>.

Supplementary Material 1.

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Authors' contributions

S.A.N and L.N conceptualised the study. S.A.N and L.N developed research methods and implemented the study. S.A analysed the data and M.L, J.B. and U.T. assisted in the analysis plan and/or interpretation of the findings. S.A wrote the manuscript which has been reviewed and approved by all co-authors.

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Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving research study participants were approved by the University of the Witwatersrand's Research Ethics Committee (Medical) for data collection (M120524) and data analysis (M180949). Written informed consent was obtained from all participants. This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving research study participants were approved by the University of the Witwatersrand's Research Ethics Committee (Medical) (ethical clearance number: M120524). Written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

- Ohuma EO, Moller AB, Bradley E, Chakwera S, Hussain-Alkhateeb L, Lewin A. National, regional, and global estimates of preterm birth in 2020, with trends from 2010: a systematic analysis. *The Lancet*. 2023;402: 1261–1271. [https://doi.org/10.1016/S0140-6736\(23\)00878-4](https://doi.org/10.1016/S0140-6736(23)00878-4).
- Okwaraji YB, Krasevec J, Bradley E, Conkle J, Stevens GA, Gatica-Domínguez G. National, regional, and global estimates of low birthweight in 2020, with trends from 2000: a systematic analysis. *Lancet*. 2024;403:1071–80. [https://doi.org/10.1016/S0140-6736\(23\)01198-4](https://doi.org/10.1016/S0140-6736(23)01198-4).
- World Health Organization. Improving maternal and newborn health and survival and reducing stillbirth: progress report 2023. Geneva: World Health Organization; 2023.
- Organisation for Economic Co-operation and Development. SF2.3: Age of Mothers at Childbirth and Age-Specific Fertility. 2023 [Accessed 8th July 2025]. https://webfs.oecd.org/els-com/Family_Database/SF_2_3_Age_mothers_childbirth.pdf [Accessed 8th July 2025].
- Organisation for Economic Co-operation and Development. Society at a Glance 2024: OECD Social Indicators. 2024 [Accessed 2nd July 2025]. <https://doi.org/10.1787/918d8db3-en>. [Accessed 2nd July 2025].
- Sully E, Biddlecom A, Darroch J, Riley T, Ashford L. Adding It Up: Investing in Sexual and Reproductive Health 2019. 2020 [Accessed 2nd June 2025]. <https://www.guttmacher.org/report/adding-it-up-investing-in-sexual-reproductive-health-2019> [Accessed 2nd June 2025].
- Endalamaw A, Engeda EH, Ekubagewargies DT, Belay GM, Tefera MA. Low birth weight and its associated factors in Ethiopia: a systematic review and meta-analysis. *Ital J Pediatr*. 2018;44: 1–12. <https://doi.org/10.1186/s13052-018-0586-6>.
- Akseer N, Keats EC, Thurairajah P, Cousins S, Bétran AP, Oaks BM. Characteristics and birth outcomes of pregnant adolescents compared to older women: An analysis of individual level data from 140,000 mothers from 20 RCTs. *EClinicalMedicine*. 2022; <https://doi.org/10.1016/j.eclinm.2022.101309>.
- Dahlui M, Azahar N, Oche OM, Aziz NA. Risk factors for low birth weight in Nigeria: evidence from the 2013 Nigeria Demographic and Health Survey. *Glob Health Action*. 2016;9(1). <https://doi.org/10.3402/gha.v9.28822>.
- Marvin-Dowle K, Kilner K, Burley VJ, Soltani H. Impact of adolescent age on maternal and neonatal outcomes in the Born in Bradford cohort. *BMJ Open*. 2018;8(3). <https://doi.org/10.1136/bmjopen-2017-016258>.
- Moraes AN, Likwa RN, Nzala SH. A retrospective analysis of adverse obstetric and perinatal outcomes in adolescent pregnancy: the case of Luapula Province, Zambia. *Matern Health Neonatol Perinatol*. 2018;4(1):20. <https://doi.org/10.1186/s40748-018-0088-y>.
- Socolov DG, Iorga M, Carauleanu A, Ilea C, Blidaru I, Boiculescu L, et al. Pregnancy during Adolescence and Associated Risks: An 8-Year Hospital-Based Cohort Study (2007–2014) in Romania, the Country with the Highest Rate of Teenage Pregnancy in Europe. *BioMed Res Int*. 2017;2017(1):9205016. <https://doi.org/10.1155/2017/9205016>.
- Alkire BC, Vincent JR, Burns CT, Metzler IS, Farmer PE, Meara JG. Obstructed Labor and Caesarean Delivery: The Cost and Benefit of Surgical Intervention. *PLOS ONE*. 2012;7(4). <https://doi.org/10.1371/journal.pone.0034595>.
- Ayenew AA. Incidence, causes, and maternofetal outcomes of obstructed labor in Ethiopia: systematic review and meta-analysis. *Reprod Health*. 2021;18:1–14. <https://doi.org/10.1186/s12978-021-01103-0>.
- Marshall NE, Abrams B, Barbour LA, Catalano P, Christian P, Friedman JE, et al. The importance of nutrition in pregnancy and lactation: lifelong consequences. *Am J Obstet Gynecol*. 2022;226(5):607–32.
- George LA, Uthlaut AB, Long NM, Zhang L, Ma Y, Smith DT. Different levels of overnutrition and weight gain during pregnancy have differential effects on fetal growth and organ development. *Reprod Biol Endocrinol*. 2010;8(1). <http://doi.org/10.1186/1477-7827-8-75>.
- Wrottesley SV, Prioreschi A, Kehoe SH, Ward KA, Norris SA. A maternal “mixed, high sugar” dietary pattern is associated with fetal growth. *Matern Child Nutr*. 2020;16(2). <https://doi.org/10.1111/mcn.12912>.
- Gongora MC, Wenger NK. Cardiovascular complications of pregnancy. *Int J Mol Sci*. 2015;16(10):23905–28. <https://doi.org/10.3390/ijms161023905>.
- Mandić-Marković V, Dobrijević Z, Robajac D, Miljuš G, Šunderić M, Penezić A. Biochemical Markers in the Prediction of Pregnancy Outcome in Gestational Diabetes Mellitus. *Medicina (Mex)*. 2024;60(8). <https://doi.org/10.3390/medicina60081250>.
- Ryan D. Obesity in women: a life cycle of medical risk. *Int J Obes*. 2007;31(2):S3–7. <https://doi.org/10.1038/sj.ijo.0803729>.
- Fall CH, Sachdev HS, Osmond C, Restrepo-Mendez MC, Victora C, Martorell R, et al. Association between maternal age at childbirth and child and adult outcomes in the offspring: a prospective study in five low-income and middle-income countries (COHORTS collaboration). *Lancet Glob Health*. 2015;3(7):e366–77. [https://doi.org/10.1016/S2214-109X\(15\)00038-8](https://doi.org/10.1016/S2214-109X(15)00038-8).
- Lee AC, Mullany LC, Tielsch JM, Katz J, Khatri SK, Leclerc SC. Community-based stillbirth rates and risk factors in rural Sarlahi. *Nepal Int J Gynecol Obstet*. 2011;113(3):199–204. <https://doi.org/10.1016/j.ijgo.2010.12.015>.
- Lin L, Lu C, Chen W, Li C, Guo VY. Parity and the risks of adverse birth outcomes: a retrospective study among Chinese. *BMC Pregnancy Childbirth*. 2021;21(1):257. <https://doi.org/10.1186/s12884-021-03718-4>.
- Hefler L, Lemach A, Seebacher V, Polteraue S, Tempfer C, Reinthaller A. The intraoperative complication rate of nonobstetric dilation and curettage. *Obstet Gynecol*. 2009;113(6): 1268–1271. <https://doi.org/10.1097/AOG.0b013e3181a66f91>.
- Dai J, Shi Y, Wu Y, Guo L, Lu D, Chen Y. The interaction between age and parity on adverse pregnancy and neonatal outcomes. *Front Med*. 2023;10. <https://doi.org/10.3389/fmed.2023.1056064>.
- Martinelli KG, Garcia EM, Neto Et Dos S, Gama S. Advanced maternal age and its association with placenta praevia and placental abruption: a meta-analysis. *Cad Saude Publica*. 2018;34. <https://doi.org/10.1590/0102-311X00206116>.
- Zgliczynska M, Kosinska-Kaczynska K. Micronutrients in multiple pregnancies: the knowns and unknowns: A systematic review. *Nutrients*. 2021;13(2). <https://doi.org/10.3390/nu13020386>.
- Kozuki N, Lee AC, Silveira MF, Sania A, Vogel JP, Adair L, et al. The associations of parity and maternal age with small-for-gestational-age, preterm, and neonatal and infant mortality: a meta-analysis. *BMC Public Health*. 2013;13(3):S2. <https://doi.org/10.1186/1471-2458-13-S3-S2>.
- Lopes Ibanez-Gonzalez D, Norris SA. Chronic Non-Communicable Disease and Healthcare Access in Middle-Aged and Older Women Living in Soweto, South Africa. *PLOS ONE*. 2013;8(10):e78800. <https://doi.org/10.1371/journal.pone.0078800>.
- World Health Organization. Diagnostic Criteria and Classification of Hyperglycaemia First Detected in Pregnancy. 2013 [Accessed 2nd October 2025]. http://iris.who.int/bitstream/handle/10665/85975/WHO_NMH_MND_13.2_eng.pdf?sequence=1 [Accessed 2nd October 2025].
- INTERGROWTH-21st. The International Newborn Size at Birth Standards Application. 2017 [Accessed 9th November 2024]. <http://intergrowth21.ndog.ox.ac.uk/> [Accessed 9th November 2024].
- Cunningham F, Leveno K, Bloom S, Dashe J, Hoffman B, Casey B, et al. Williams obstetrics. 25th edn New York: McGraw-Hill Education/Medical; <http://accessmedicine.mhmedical.com/book.aspx?bookid=1918#158894346> [Accessed 15th November 2024].
- Sawyer SM, Azzopardi PS, Wickremaratne D, Patton GC. The age of adolescence. *Lancet Child Adolesc Health*. 2018;2(3):223–8. [https://doi.org/10.1016/S2352-4642\(18\)30022-1](https://doi.org/10.1016/S2352-4642(18)30022-1).
- World Health Organization. Outcome 2: Factsheets on Priority Health Areas and Case Studies Developed by Adolescents and Youth in the South East Asia Region. 2021 [Accessed 20th August 2025]. https://cdn.who.int/media/docs/default-source/country-profiles/mca/gym-outcome-2_22-march.pdf [Accessed 20th August 2025].
- Biney E, Amoateng A, Acheampong, and Ewemojoe O. Patterns of fertility in contemporary South Africa: Prevalence and associated factors. *Tong K wai (ed.) Cogent Social Sciences*. 2021;7(1): 1858575. <https://doi.org/10.1080/23311886.2020.1858575>.
- Statistics South Africa. Recorded live births, 2023. 2024 [Accessed 2nd June 2025]. <https://www.statssa.gov.za/publications/P0305/P03052023.pdf> [Accessed 2nd June 2025].
- Hoque M, Hoque ME, Van Hal G, Buckus S. Prevalence, incidence and seroconversion of HIV and Syphilis infections among pregnant women of South

- Africa. *South Afr J Infect Dis*. 2021;36(1). <https://doi.org/10.4102/sajid.v36i1.296>.
38. Mabunda SA, Sigovana K, Chitha W, Apalata T, Nomatshila S. Socio-demographic associations of HIV among women attending antenatal care in selected rural primary care facilities in South Africa's Eastern Cape province. *BMC Infect Dis*. 2021;21:1–11. <https://doi.org/10.1186/s12879-020-05744-7>.
39. Zuma K, Simbayi L, Zungu N, Moyo S, Jooste ME. The HIV epidemic in South Africa: key findings from 2017 national population-based survey. *Int J Environ Res Public Health*. 2022;19(13). <https://doi.org/10.3390/ijerph19138125>.
40. Baruwa OJ. Associations between lifetime pregnancy and sexual risk behaviors among 15–24-year-old adolescent girls and young women in South Africa: Secondary analyses of the 2016 Demographic Health Survey. *PLOS Glob Public Health*. 2024;4(6). <https://doi.org/10.1371/journal.pgph.0003317>.
41. Hurtado MD, Saadedine M, Kapoor E, Shufelt CL, Faubion SS. Weight gain in midlife women. *Curr Obes Rep*. 2024;13(2): 352–363. <https://doi.org/10.1007/s13679-024-00555-2>.
42. Chen C, Lei Z, Xiong Y, Ni M, He B, Gao J. Gestational weight gain of multiparas and risk of primary preeclampsia: a retrospective cohort study in Shanghai. *Clin Hypertens*. 2023;29(1). <https://doi.org/10.1186/s40885-023-00254-5>.
43. Samura T, Steer J, Michelis LD, Carroll L, Holland E, Perkins R. Factors associated with excessive gestational weight gain: review of current literature. *Glob Adv Health Med*. 2016;5(1):87–93. <https://doi.org/10.7453/gahmj.2015.094>.
44. Restall A, Taylor RS, Thompson JM, Flower D, Dekker GA, Kenny LC. Risk factors for excessive gestational weight gain in a healthy, nulliparous cohort. *J Obes*. 2014;2014(1). <https://doi.org/10.1155/2014/148391>.
45. Victor A, de França da Silva Teles L, Aires IO, de Carvalho LF, Luzia LA, Artes R, et al. The impact of gestational weight gain on fetal and neonatal outcomes: the Araraquara Cohort Study. *BMC Pregnancy and Childbirth*. 2024;24(1): 320.
46. Wang LY, Wang LY, Wang YL, Ho CH. Early neonatal hypoglycemia in term and late preterm small for gestational age newborns. *Pediatr Neonatol*. 2023;64(5):538–46. <https://doi.org/10.1016/j.pedneo.2022.09.021>.
47. Ralph JL, Silva SG, Dail RB, Brandon DH. Body temperature instability and respiratory morbidity in the very low birth weight infant: a multiple case, intensive longitudinal study. *BMC Pediatrics*. 2020;20(1): 485. <https://doi.org/10.1186/s12887-020-02351-y>.
48. Naz S, Hoodbhoy Z, Jaffar A, Kaleem S, Hasan BS, Chowdhury D, et al. Neurodevelopment assessment of small for gestational age children in a community-based cohort from Pakistan. *Arch Dis Childhood*. 2023;108(4):258–63.
49. D'Agostin M, Morgia CDS, Vento G, Nobile S. Long-term implications of fetal growth restriction. *World J Clin Cases*. 2023;11(13):2855. <https://doi.org/10.12998/wjcc.v11.i13.2855>.
50. Mochhoury L, Laamiri F, Marc I, Chebabe M. The impact of maternal weight gain during pregnancy on perinatal outcomes. *S Afr J Child Health*. 2024;18(1):28–32.
51. Hedderson MM, Gunderson EP, Ferrara A. Gestational Weight Gain and Risk of Gestational Diabetes Mellitus. *Obstet Gynecol*. 2010;115(3). https://journals.lww.com/greenjournal/fulltext/2010/03000/gestational_weight_gain_and_risk_of_gestational.18.aspx.
52. Kamiyama Y, Ogawa K, Morisaki N, Arata N, Wada S. Association between gestational weight gain and chronic disease risks in later life. *Sci Reports*. 2024;14(1): 659. <https://doi.org/10.1038/s41598-023-50844-4>.
53. Haksari EL, Hakimi M, Ismail D. Neonatal mortality in small for gestational age infants based on reference local newborn curve at secondary and tertiary hospitals in Indonesia. *BMC Pediatr*. 2023;23(1):214. <https://doi.org/10.1186/s12887-023-04023-z>.
54. Tian Y, Yang X. A review of roles of uterine artery doppler in pregnancy complications. *Front Med*. 2022;9. <https://doi.org/10.3389/fmed.2022.813343>.
55. Fodera DM, Russell SR, Jackson JL, Fang S, Chen X, Vink J. Material properties of nonpregnant and pregnant human uterine layers. *J Mech Behav Biomed Mater*. 2024;151. <https://doi.org/10.1016/j.jmbbm.2023.106348>.
56. Wu Y, Li M, Zhang J, Wang S. Unveiling uterine aging: Much more to learn. *Ageing Res Rev*. 2023;86. <https://doi.org/10.1016/j.arr.2023.101879>.
57. Khalil A, Thilaganathan B. Role of uteroplacental and fetal Doppler in identifying fetal growth restriction at term. *Antenatal Fetal Surveill*. 2017;38: 38–47. <https://doi.org/10.1016/j.bpobgyn.2016.09.003>.
58. Shakuntala C, Jyoti Y, Pradeep B. Uterine artery resistance index in first trimester and maternal neonatal outcome. *Asian Pac J Trop Dis*. 2012;2(6):481–4. [https://doi.org/10.1016/S2222-1808\(12\)60105-4](https://doi.org/10.1016/S2222-1808(12)60105-4).
59. Gómez O, Figueras F, Martínez M, del Río M, Palacio M, Eixarch E, et al. Sequential changes in uterine artery blood flow pattern between the first and second trimesters of gestation in relation to pregnancy outcome. *Ultrasound Obstet Gynecol*. 2006;28(6):802–8. <https://doi.org/10.1002/uog.2814>.
60. Krebs NF, Hambidge KM, Westcott JL, Garcés AL, Figueroa L, Tshetu AK, et al. Birth length is the strongest predictor of linear growth status and stunting in the first 2 years of life after a preconception maternal nutrition intervention: the children of the Women First trial. *Am J Clin Nutrition*. 2022;116(1):86–96.
61. Ling HZ, Garcia Jara P, Nicolaides KH, Kametas NA. Effect of maternal age on cardiac adaptation in pregnancy. *Ultrasound Obstet Gynecol*. 2021;58(2): 285–292. <https://doi.org/10.1002/uog.23614>.
62. Lean SC, Heazell AEP, Dilworth MR, Mills TA, Jones RL. Placental Dysfunction Underlies Increased Risk of Fetal Growth Restriction and Stillbirth in Advanced Maternal Age Women. *Sci Reports*. 2017;7(1): 9677. <https://doi.org/10.1038/s41598-017-09814-w>.
63. Kelly AC, Powell TL, Jansson T. Placental function in maternal obesity. *Clin Sci*. 2020;134(8): 961–984. <https://doi.org/10.1042/CS20190266>.
64. Parlee SD, MacDougall OA. Maternal nutrition and risk of obesity in offspring: The Trojan horse of developmental plasticity. *Modulation Adipose Tissue Health Dis*. 2014;1842(3):495–506. <https://doi.org/10.1016/j.jbbadis.2013.07.007>.
65. Gul R, Iqbal S, Anwar Z, Ahd SG, Ali SH, Pirzada S. Pre-pregnancy maternal BMI as predictor of neonatal birth weight. *PLOS ONE*. 2020;15(10):e0240748. <https://doi.org/10.1371/journal.pone.0240748>.
66. Ismail AQT, Boyle EM, Pillay T, Boyle EM, Modi N, Rivero-Arias O, et al. Clinical outcomes for babies born between 27–31 weeks of gestation: Should they be regarded as a single cohort? *J Neonatal Nursing*. 2023;29(1):27–32. <https://doi.org/10.1016/j.jnn.2022.04.003>.
67. Preda A, Iliescu DG, Comănescu A, Zorilă GL, Vladu IM, Forțoiu MC, et al. Gestational Diabetes and Preterm Birth: What Do We Know? Our Experience and Mini-Review of the Literature. *J Clin Med*. 2023;12(14). <https://doi.org/10.3390/jcm12144572>.
68. Cao X, Zu D, Liu Y. Effects of interaction between gestational hypertension and history of preterm birth on the risk of preterm birth: an analysis based on the national vital statistics system database. *Med Sci Monit: Int Med J Exp Clin Res*. 2022;28:e935094-1. <https://doi.org/10.12659/MSM.935094>.
69. Huangxiaoyu Li BZ Binbin Yin, Nan Jiang. Effect of Combined Gestational Diabetes Mellitus and Preeclampsia on Pregnancy Outcomes. *CEOG*. 2025;52(2): 27065-null. <https://doi.org/10.31083/CEOG27065>.
70. World Health Organization. WHO antenatal care recommendations for a positive pregnancy experience. Nutritional interventions update: Multiple micronutrient supplements during pregnancy. Geneva: World Health Organization; 2020.

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