

Maternal Pregnancy Complications and Child Growth Trajectories from Birth to Age Five: A Population-Based Cohort Study of 548,318 Mother–Child Pairs

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Research Article

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Abstract

Purpose

To examine whether gestational diabetes, hypertensive disorders of pregnancy (HDP) and maternal anemia, together with nutritional and psychosocial factors, are associated with weight- and height-for-age trajectories from birth to five years.

Methods

We analyzed a population-based registry of 548,318 mother–child pairs (northeastern Iran, 2017–2025) with growth measurements at 15 visits from 3 days to 5 years. WHO weight-for-age and height-for-age z-scores (ZWFA, ZHFA) were modelled with cluster-robust regression including condition $\times$ age interactions, adjusted for pre-pregnancy BMI, folic acid, Edinburgh Postnatal Depression Scale (EPDS) score and sociodemographic factors. Robustness was assessed with split-sample validation, regression-to-the-mean simulation, multiple imputation, mixed-effects models and a complete-follow-up analysis.

Results

HDP ($n = 10,428$) and gestational diabetes ($n = 3,220$) were associated with lower z-scores in early infancy (ZWFA $\beta = -0.23$, 95% CI -0.26 to -0.21 , and -0.20 , -0.25 to -0.15 , respectively), with deficits narrowing by age five (condition $\times$ age interactions, FDR-adjusted $p < 0.0001$). These interactions replicated across independent halves of the cohort and were 8–24 SD larger than expected from regression to the mean. Diabetes interactions lost significance among children with complete follow-up. Anemia associations were not robust. BMI and maternal education were positively associated with growth; EPDS showed little association.

Conclusion

HDP and, less robustly, gestational diabetes were associated with early growth deficits that narrowed during the first years of life. Closer growth monitoring of exposed infants in early infancy may be warranted; confirmatory studies are needed.

What is Known:

- Gestational diabetes and hypertensive disorders of pregnancy (HDP) alter fetal growth.
- Evidence on how these exposures shape growth trajectories up to age five is limited and inconsistent.

What is New:

- In 548,318 mother–child pairs, HDP and gestational diabetes were linked to early weight and length deficits that narrowed by age five.
- The HDP pattern was robust to regression-to-the-mean, imputation and mixed-model checks, supporting closer growth monitoring in early infancy.

Introduction

Gestational diabetes, hypertensive disorders of pregnancy (HDP) and anemia are common pregnancy complications and established risk factors for adverse perinatal outcomes. Because growth in the first years of life predicts later metabolic, neurodevelopmental and human-capital outcomes [1], attention within the developmental origins of health and disease framework has extended beyond the perinatal period to how these exposures shape postnatal growth trajectories.

Offspring of mothers with gestational diabetes are often born large, yet their postnatal growth is heterogeneous: birth size and adiposity have fallen in more recently managed cohorts [2], infants born smaller after metformin treatment show accelerated postnatal growth [3], and reviews describe age-specific and heterogeneous alterations in offspring growth [4, 5]. Maternal anemia has been associated with stunting, reduced birth length and fetal growth restriction, although findings are not fully consistent [6–8]. Evidence after preeclampsia and other HDP is mixed, partly because these disorders co-occur with fetal growth restriction and preterm birth [9, 10]; one cohort found that preeclampsia-exposed infants were smaller at birth but had caught up by 2 years [11].

Maternal nutritional status, including body mass index (BMI) and folic acid supplementation, also influences offspring growth [12–14]. Antenatal depression has been linked to preterm birth and low birthweight [15, 16], but associations with later child growth are inconsistent [17–24], and many studies rely on unvalidated psychosocial proxies rather than validated instruments.

Existing cohorts typically have few postnatal measurements and examine exposures individually. Using a population-based registry with measurements at 15 ages from birth to 5 years, we examined the associations of gestational diabetes, HDP and anemia, nutritional factors and a validated measure of antenatal depressive symptoms with WHO-standardized weight-for-age and height-for-age trajectories, and tested whether early differences narrow over time.

Methods

Study design and data source

This population-based retrospective cohort study used Sina, the maternal–child health information system of Mashhad University of Medical Sciences (MUMS), northeastern Iran, which serves approximately 5.5 million people in 17 counties of Razavi Khorasan province. Data are recorded during routine antenatal and child-health surveillance in the public primary health care network (health houses, health posts and comprehensive health centres). All pregnancies recorded between Iranian years 1396 and 1403 (approximately 2017–2025) were included irrespective of maternal nationality (non-Iranian mothers, predominantly Afghan, <5%), yielding 548,318 mother–child pairs with growth measurements scheduled at 15 visits (3, 15 and 30 days; 2, 4, 6, 7, 9, 12, 15, 18 and 24 months; and 3, 4 and 5 years). Each mother contributed one record, and no family or sibling identifier was available. The MUMS Research Ethics Committee approved the study (IR.MUMS.MEDICAL.REC.1401.689) and waived individual consent for this de-identified secondary analysis.

Exposures and covariates

Gestational diabetes (onset after 20 weeks) was diagnosed by a one-step 75-g oral glucose tolerance test using IADPSG/Iranian Ministry of Health criteria (fasting ≥ 92 , 1-hour ≥ 180 or 2-hour ≥ 153 mg/dL). Anemia was defined by trimester-specific WHO hemoglobin thresholds (<11 g/dL in the first and third trimesters; <10.5 g/dL in the second).

The registry field labelled preeclampsia captured only postpartum-onset cases (n=19) and could not be analyzed; we therefore defined HDP as chronic hypertension predating pregnancy (n=9,095) or new-onset gestational hypertension after 20 weeks (n=3,606), both at $\geq 140/90$ mmHg (combined n=10,428; 1.9%). Because proteinuria was not recorded, HDP is broader than preeclampsia.

Covariates were self-reported pre-pregnancy BMI (10–60 kg/m²), total folic acid tablets consumed, the antenatal Edinburgh Postnatal Depression Scale (EPDS; validated Persian version; mean score across assessments and ever ≥ 13), a domestic violence score adapted from the WHO Multi-country Study instrument, a 0–12 Healthy Lifestyle Score across six domains (the latter two unvalidated), maternal age (12–55 years), maternal education (ordinal 0–10), parity, residency type (metropolis, suburb, smaller city, rural; an urbanicity/socioeconomic proxy), child sex, multiple birth and physician-recorded prematurity. A food insecurity score (Persian adaptation of the USDA module) was available for 14.5% of mothers and was used only in sensitivity models. Gestational age was not recorded.

Growth outcomes

Weight-for-age and height-for-age z-scores (ZWFA, ZHFA) were computed from raw measurements using the sex-specific LMS tables of the WHO Child Growth Standards [25], applying length-for-age references before and height-for-age references from 24 months, in line with the national measurement protocol. LMS parameters were linearly interpolated for visits at 3, 15 and 30 days. Z-scores beyond ± 6 were set to missing.

Statistical analysis

ZWFA and ZHFA were modelled separately by ordinary least squares (OLS) regression with standard errors clustered by child. Models included the three conditions, age (linear and quadratic), condition $\times$ age and condition $\times$ age² interactions, and all covariates. Food insecurity was added in sensitivity models. Benjamini–Hochberg false discovery rate (FDR) correction was applied across non-intercept coefficients within each model; Bonferroni correction and joint Wald tests for each condition were also applied. Coefficients are reported with 95% confidence intervals (CI) and standardized coefficients ($\beta \times$ predictor SD). Clustering was required given high within-child correlation (intraclass correlation 0.56–0.64).

Robustness checks comprised restricted cubic splines for age; quantile regression; exclusion of influential observations; alternative implausible-value thresholds; split-sample validation (two halves of $\sim 179,000$ children); a parametric simulation (30 replicates) testing whether regression to the mean and measurement noise [26] could produce the observed interactions under constant effects; inverse-cluster-size weighting for informative visit frequency [27]; multiple imputation by chained equations (five datasets, Rubin's rules) [28, 29]; a random-intercept, random-slope mixed model [30] in a random subsample of 150,000 children; and a sensitivity analysis restricted to children measured at both 3 days and 5 years to assess selective attrition. Details are provided in Online Resource 1. Because no prospective power calculation was performed, retrospective minimum detectable effect sizes were calculated (Online Resource 1). Analyses used Python 3.12 (pandas, NumPy, SciPy) with two-sided $\alpha=0.05$. The analysis was not pre-registered and is exploratory.

Results

Sample characteristics

Of 548,318 mother–child pairs, 3,220 (0.6%) mothers had gestational diabetes, 10,428 (1.9%) HDP and 2,182 (0.4%) anemia. Mean maternal age was 32.4 years (SD 6.6); 428,313 mothers had at least one antenatal EPDS assessment (mean score 2.86; 2.9% ever ≥ 13). Mothers with diabetes or HDP were older, had higher BMI and parity and lower education, and their infants were more often preterm (14.4% and 10.6% vs 5.2%; Table 1). The primary models included approximately 358,150 children and 2.79 million observations; observations per visit ranged from about 290,000 at 2–4 months to 33,195 at 5 years (Table 2). The complete-case sample (65.3%) differed from excluded children on all baseline characteristics (Online Resource 1).

Table 1. Baseline characteristics by gestational diabetes, anemia, and HDP status (unadjusted comparison).

Characteristic	No diabetes	Diabetes	p_FDR	No anemia	Anemia	p_FDR	No HDP	HDP	p_FDR
N	545,098	3,220		546,136	2,182		537,890	10,428	
Maternal age, years, mean (SD)	32.4 (6.6)	37.0 (6.3)	<.0001	32.4 (6.6)	33.4 (6.7)	<.0001	32.3 (6.6)	35.1 (6.9)	<.0001
Maternal education, 0–10, mean (SD)	4.2 (2.2)	3.9 (2.2)	<.0001	4.2 (2.2)	4.0 (2.1)	<.001	4.2 (2.2)	3.8 (2.0)	<.0001
Pre-pregnancy BMI, mean (SD)	25.1 (4.8)	29.0 (5.3)	<.0001	25.1 (4.9)	24.5 (5.1)	<.0001	25.1 (4.8)	28.6 (5.7)	<.0001
Parity, mean (SD)	0.9 (1.1)	1.6 (1.4)	<.0001	0.9 (1.1)	1.2 (1.2)	<.0001	0.9 (1.1)	1.5 (1.3)	<.0001
Folic acid tablets, total, mean (SD)	7.1 (17.8)	10.6 (22.4)	<.0001	7.1 (17.8)	7.8 (19.5)	0.103	7.0 (17.7)	10.2 (22.5)	<.0001
Birthweight, kg, mean (SD)	3.2 (0.5)	3.3 (0.7)	<.0001	3.2 (0.5)	3.2 (0.5)	0.048	3.2 (0.5)	3.1 (0.6)	<.0001
Female sex, n (%)	265,343 (48.7%)	1,591 (49.4%)	0.470	265,872 (48.7%)	1,062 (48.7%)	1.000	261,899 (48.7%)	5,035 (48.3%)	0.416
Preterm birth, n (%)	28,381 (5.2%)	464 (14.4%)	<.0001	28,716 (5.3%)	129 (5.9%)	0.241	27,736 (5.2%)	1,109 (10.6%)	<.0001
Current multiple birth, n (%)	15,369 (2.8%)	86 (2.7%)	0.687	15,385 (2.8%)	70 (3.2%)	0.360	15,077 (2.8%)	378 (3.6%)	<.0001

Table 2. Number of observations contributing to the main models, by visit age.

Visit age	N (ZWFA model)	N (ZHFA model)
3 days	120,118	120,009
15 days	80,034	79,972
30 days	271,338	271,252
2 mo	294,030	293,822
4 mo	289,968	289,812
6 mo	265,351	265,250
7 mo	214,592	214,518
9 mo	212,009	211,954
12 mo	259,673	259,671
15 mo	150,269	150,279
18 mo	256,311	256,327
24 mo	145,569	145,567
3 yr	107,947	107,948
4 yr	84,972	84,973
5 yr	33,195	33,198

Maternal conditions and growth trajectories

HDP showed the largest association: z-scores were lower in early infancy (ZWFA β -0.231, 95% CI -0.255 to -0.206; ZHFA β -0.208, -0.234 to -0.181), with positive linear and negative quadratic age interactions (all $p_{\text{FDR}} < 0.0001$) indicating a narrowing gap (Table 3). HDP was significant as a block (Wald $\chi^2(3) = 388.8$ for ZWFA and 276.6 for ZHFA; $p < 10^{-15}$) and all terms survived Bonferroni correction. Gestational diabetes showed a similar but smaller pattern (ZWFA β -0.200, -0.245 to -0.155; ZHFA β -0.195, -0.244 to -0.146; diabetes $\times$ age β 0.181 and 0.153; $p_{\text{FDR}} < 0.0001$), with the deficit narrowing over the first 2–3 years. Anemia main effects were not significant (p_{FDR} 0.668 and 0.117); its linear age interactions reached FDR significance (p_{FDR} 0.022 and 0.044), but anemia did not reach joint significance and did not survive Bonferroni correction. The diabetes $\times$ HDP interaction was not significant (355 mothers with both; $p \geq 0.26$), and the diabetes–anemia overlap ($n = 28$) was too small for inference.

Table 3. Cluster-robust regression coefficients with 95% confidence intervals (FDR-corrected p-values and standardized effect sizes also reported) for maternal medical conditions and their interaction with age, main models (N = 2,785,238–2,785,782 observations; 358,144–358,147 children).

Term	ZWFA β (95% CI)	ZWFA std. β	ZWFA p_FDR	ZHFA β (95% CI)	ZHFA std. β	ZHFA p_FDR
Diabetes	-0.2000 (-0.2451, -0.1549)	-0.017	<.0001	-0.1950 (-0.2440, -0.1460)	-0.016	<.0001
Diabetes $\times$ Age	0.1810 (0.1222, 0.2398)	0.020	<.0001	0.1530 (0.0942, 0.2118)	0.017	<.0001
Diabetes $\times$ Age ²	-0.0260 (-0.0397, -0.0123)	-0.009	<.001	-0.0200 (-0.0337, -0.0063)	-0.007	.007
HDP	-0.2308 (-0.2553, -0.2063)	-0.033	<.0001	-0.2075 (-0.2344, -0.1806)	-0.029	<.0001
HDP $\times$ Age	0.2291 (0.2007, 0.2574)		<.0001	0.2094 (0.1797, 0.2392)		<.0001
HDP $\times$ Age ²	-0.0392 (-0.0451, -0.0332)		<.0001	-0.0348 (-0.0411, -0.0286)		<.0001
Anemia	-0.0170 (-0.0699, 0.0359)	-0.001	.668	-0.0490 (-0.1078, 0.0098)	-0.003	.117
Anemia $\times$ Age	0.0790 (0.0163, 0.1417)	0.007	.022	0.0730 (0.0064, 0.1396)	0.007	.044
Anemia $\times$ Age ²	-0.0160 (-0.0297, -0.0023)	-0.005	.023	-0.0130 (-0.0267, 0.0007)	-0.004	.083
BMI	0.02500 (0.02441, 0.02559)	0.123	<.0001	0.01400 (0.01341, 0.01459)	0.067	<.0001
Folate	0.00040 (0.00020, 0.00060)	0.008	<.0001	0.00040 (0.00020, 0.00060)	0.007	<.0001
Domestic violence	-0.00490 (-0.00627, -0.00353)	-0.010	<.0001	-0.00760 (-0.00917, -0.00603)	-0.016	<.0001
Lifestyle	0.00240 (0.00103, 0.00377)	0.005	.002	0.00780 (0.00623, 0.00937)	0.016	<.0001
Maternal age	-0.00090 (-0.00423, 0.00243)	-0.006	.755	0.00960 (0.00588, 0.01332)	0.064	<.0001
Maternal age ²	-0.000100 (-0.000159, -0.000041)	-0.055	<.0001	-0.000200 (-0.000259, -0.000141)	-0.085	<.0001
Maternal education	0.01410 (0.01253, 0.01567)	0.028	<.0001	0.01870 (0.01694, 0.02046)	0.037	<.0001
EPDS, mean score	0.00020 (-0.00098, 0.00138)	0.000	.863	0.00250 (0.00113, 0.00387)	0.007	<.001
EPDS, ever $\geq$ 13	0.00460 (-0.01539, 0.02459)	0.001	.764	-0.02180 (-0.04336, -0.00024)	-0.003	.059

Note. Age in years, centered at birth. Standardized β = coefficient $\times$ SD(predictor); outcomes are already in SD units (z-scores). HDP = hypertensive disorders of pregnancy (n = 10,428), a composite of two registry fields not requiring proteinuria and therefore broader than a strict clinical preeclampsia diagnosis (see Methods). EPDS = Edinburgh Postnatal Depression Scale (validated instrument); domestic violence and lifestyle scores are unvalidated proxies.

Robustness

Condition $\times$ age interactions replicated in independent halves of the cohort (e.g., ZWFA HDP $\times$ age β 0.217 and 0.242; diabetes $\times$ age 0.151 and 0.175). Under simulated regression to the mean and measurement noise, null interaction coefficients centred near zero, and the observed interactions were 8.4–24.1 SD above these null distributions. HDP- and diabetes-exposed children had slightly more visits than unexposed children (7.54 and 7.39 vs 7.05), but inverse-cluster-size weighting left estimates materially unchanged (e.g., ZWFA HDP $\times$ age β 0.256). Multiple imputation (539,872 children) gave modestly attenuated but significant estimates for HDP (ZWFA β -0.208; HDP $\times$ age 0.202) and diabetes (β -0.159; diabetes $\times$ age 0.143), with anemia non-significant. The random-slope model confirmed the HDP and diabetes findings (e.g., ZWFA HDP $\times$ age β 0.223; diabetes $\times$ age 0.113; $p < 10^{-15}$) but, unlike OLS, also showed significant anemia $\times$ age interactions (ZWFA β 0.107, $p < 0.001$; ZHFA β 0.098, $p = 0.004$). Spline models, which fitted better than the quadratic (lower AIC/BIC), reproduced the diabetes pattern. Among children with complete follow-up, the diabetes and anemia interactions were no longer significant and the diabetes direction reversed; HDP was not examined in this analysis.

Nutritional, socioeconomic and psychosocial factors

Pre-pregnancy BMI had the largest standardized association among these factors (ZWFA 0.123; ZHFA 0.067), and folic acid intake and maternal education (standardized β 0.028 and 0.037) were also positively associated with both outcomes (all $p_{\text{FDR}} < 0.0001$). Maternal age showed an inverted-U association. The validated EPDS score was not associated with ZWFA ($p_{\text{FDR}} > 0.76$) and only weakly with ZHFA (standardized β 0.007), whereas the unvalidated domestic violence (-0.010 to -0.016) and lifestyle scores (0.005 to 0.016) showed stronger associations. Prematurity (standardized β -0.18 to -0.23), multiple birth (-0.07 to -0.09) and rural residence (-0.06 to -0.13) were the strongest covariate correlates.

Discussion

In this registry of 548,318 mother–child pairs, HDP and gestational diabetes were each associated with lower weight- and height-for-age in early infancy, followed by narrowing of the gap over the first years of life. The HDP association was the largest and most robust, persisting across split-sample validation, regression-to-the-mean simulation, multiple imputation and random-slope models. The diabetes association was attenuated among children with complete follow-up, and the anemia association depended on model specification.

HDP is an established cause of placental insufficiency and fetal growth restriction, which plausibly explains the early deficit. The subsequent narrowing agrees with catch-up growth by 2 years after preeclampsia [11] and with mixed evidence from smaller cohorts [9, 10]. Because our HDP definition combines chronic and gestational hypertension without proteinuria, the findings apply to HDP broadly rather than to preeclampsia specifically.

For gestational diabetes, lower adjusted z-scores in early infancy are consistent with reduced birth size in recent, actively managed cohorts [2], and the subsequent narrowing resembles accelerated growth after smaller birth size [3]. The deficit was roughly twice as large at the 10th as at the 90th percentile, suggesting that already smaller infants are most affected. However, the loss of the interaction among children with complete follow-up indicates that selective attrition may partly explain the apparent catch-up. The anemia association was fragile across checks, in keeping with inconsistent prior evidence [6]. The validated EPDS showed little association with growth, whereas unvalidated proxies suggested stronger effects; this is consistent with inconsistent literature on maternal depression and child growth [17, 18, 24] and underlines the value of validated measurement.

Given the sample size, small effects reached statistical significance; the medical-condition effects (about 0.2 SD) are modest relative to the -2 SD threshold for growth faltering, but may matter at population level. For diabetes, the spline-predicted gap was largest in the first 6 months (about -0.14 to -0.16 SD) and close to zero by 12–15 months, suggesting that infants of mothers with gestational diabetes could be flagged for additional weight and length checks at the existing 2- and 6-month visits. A comparable window has not yet been estimated for HDP, although its larger effect suggests similar priority; HDP has also not yet been examined in the complete-follow-up, spline and quantile analyses, so its effect size should not be regarded as settled. Children of less-educated and rural mothers were smaller throughout the trajectory, independent of medical conditions, so clinical measures should be paired with broader socioeconomic and rural equity interventions. Food insecurity and developmental screening data were disproportionately missing for rural and lower-resource families, indicating that the groups at greatest risk may also be least visible in routine surveillance.

Strengths include population-wide coverage, 15 measurement ages, WHO z-scores computed from raw data, objective exposure criteria, multiple-testing correction and extensive robustness checks. Limitations are important. Associations are observational, and within-mother comparison was impossible because each mother contributed one record. Gestational age was unavailable, so small-for-gestational-age infants could not be distinguished from preterm infants. HDP subtypes could not be separated. Psychosocial scores and residency type are unvalidated proxies. The complete-case sample was not representative of the full cohort, food insecurity was largely missing, and attrition at older ages may bias trajectory estimates. EPDS data were merged by record order, although consistency checks supported correct alignment. The registry's urban/rural split (74.7%/25.3%) differs from official estimates (80–85% urban), suggesting more complete capture of rural births. The mixed model was fitted to a subsample. Finally, the analysis was iterative and not pre-registered, so the true false-positive risk exceeds within-model corrected p-values, and findings may not generalize beyond similar primary-care settings.

Future studies should recover gestational age (e.g., from ultrasound records) to separate growth restriction from prematurity, apply inverse-probability-of-attrition weighting to test whether catch-up persists after accounting for selective follow-up, use data sources with repeat pregnancies or quasi-experimental designs to strengthen causal inference, and replace residency type with a validated socioeconomic index. The anemia association should be replicated in an independent sample before further investigation.

Conclusion

In this large regional registry, HDP and, less robustly, gestational diabetes were associated with early growth deficits that narrowed over the first years of life. These exploratory findings generate specific hypotheses for confirmatory studies with gestational age data, family linkage and modelling of attrition, and support closer growth monitoring of exposed infants in early infancy.

Abbreviations

BMI, body mass index; CI, confidence interval; EPDS, Edinburgh Postnatal Depression Scale; FDR, false discovery rate; HDP, hypertensive disorders of pregnancy; MUMS, Mashhad University of Medical Sciences; OGTT, oral glucose tolerance test; OLS, ordinary least squares; WHO, World Health Organization; ZHFA, height-for-age z-score; ZWFA, weight-for-age z-score

Declarations

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Competing interests

The author declares no competing interests.

Author contributions

Veda Vakili: Conceptualization, Methodology, Formal analysis, Data curation, Investigation, Writing – original draft, Writing – review & editing, Supervision.

Ethics approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the MUMS Research Ethics Committee (IR.MUMS.MEDICAL.REC.1401.689).

Consent to participate

As this study used de-identified secondary registry data, the requirement for individual informed consent was waived by the MUMS Research Ethics Committee.

Consent for publication

Not applicable (no individual person's data, images, or identifiable information are presented).

Clinical trial number

Not applicable.

Data availability

The dataset analyzed in this study was derived from a regional maternal–child health registry maintained by Mashhad University of Medical Sciences (MUMS). The data are not publicly available due to institutional data-governance restrictions protecting patient confidentiality, but are available from the corresponding author upon reasonable request and subject to approval by the registry’s data custodian and, where applicable, the MUMS Research Ethics Committee.

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