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Xi Wang
Shanxi Bethune Hospital
Muye Han
hancugb@gmail.com
Taiyuan University of Science and Technology

Research Article

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Preconception diabetes and hypertension and severe maternal-neonatal morbidity among non-Hispanic Black women in the United States: a national retrospective cohort study of risk pathways

Xi Wang[1] and Muye Han[2*]

[1]Department of Gynaecology and Obstetrics, Shanxi Bethune Hospital, Taiyuan, China.
[2]College of Computer Science and Technology, Taiyuan University of Science and Technology, Taiyuan, China.

*Corresponding author(s). E-mail(s): hancugb@gmail.com;
Contributing authors: Dingyichen4552@126.com;

Abstract

Background: Non-Hispanic Black women in the US face approximately three-fold higher risk of pregnancy-related death and twice the risk of severe maternal morbidity (SMM) than White women; preconception metabolic disease is modifiable but its contribution to severe maternal-neonatal morbidity among Black women is unquantified.

Methods: In this national retrospective cohort study, we analysed 2022–2023 National Center for Health Statistics Linked Birth/Infant Death files (7 281 110 live births); the 2023 cohort was primary; 2022 served for temporal replication. Analyses focused on non-Hispanic Black women ($\boldsymbol{n = 491\,753}$), with non-Hispanic White women ($\boldsymbol{n = 1\,787\,833}$) as reference. Outcomes were neonatal critical illness (CRITICAL), SMM proxies, and a maternal-neonatal composite. Exposures were pre-gestational diabetes (PGDM), pre-gestational hypertension (PGHTN), gestational hypertension and diabetes, assisted reproductive technology (ART), and advanced maternal age. DAGs defined adjustment sets; we estimated adjusted risk differences by g-computation, assessed confounding with E-values, decomposed pathways by exploratory interventional mediation to locate modifiable windows, and applied double/debiased machine learning. Hypothetical policy scenarios estimated avoidable cases.

Results: Black women had higher prevalence of PGDM (1.69% vs 0.98%), PGHTN (5.77% vs 3.11%), Medicaid (64.4% vs 27.6%), late or no prenatal care (10.4% vs 4.7%), infant death (1.07% vs 0.44%), CRITICAL (15.69% vs 12.45%), and the composite endpoint (16.63% vs 13.65%). Among Black women, PGDM had the largest adjusted composite-endpoint risk difference (16.25 percentage points), then gestational hypertension (12.13) and PGHTN (10.08). ART showed a 12.53-percentage-point difference, reported as an association. Preterm birth mediated 63.4% of the PGHTN effect and 61.8% of the PGDM effect, with gestational disease, cesarean and multiple gestation modelled as mediator–outcome confounders; multiple gestation mediated 41.7% of the ART association. Deprivation explained 24.5% of the Black-White composite disparity; prenatal-care access accounted for 4.4%.

Conclusions: Among the six exposures, PGDM had the largest adjusted composite-endpoint risk difference (16.25 percentage points), and most of that difference operated after conception through

preterm birth and related mechanisms; the composite endpoint was dominated by its neonatal component. Healthcare access and deprivation explained only part of the racial disparity, leaving structural and care-quality mechanisms largely unexplained; avoidable-case estimates were highly sensitive to assumed effectiveness and are best read as illustrative.

Keywords: Adjusted risk differences; Preconception diabetes and hypertension; Neonatal critical illness; Mediation analysis; Temporal validation

Background

Non-Hispanic Black women in the United States are roughly three times as likely to experience pregnancy-related death compared with White women; this persistent disparity has not narrowed over four decades and cannot be fully explained by differences in education, household income, or insurance coverage alone [1, 2, 3, 4]. Severe maternal morbidity (SMM) demonstrates comparable inequities: CDC 21-indicator surveillance identifies approximately two-fold higher SMM rates among Black women, with excess risk concentrated in life-threatening events including eclampsia, sepsis, and cardiac arrest [5, 6]. Within our 2023 study cohort, first-year infant mortality was 1.07% among Black women versus 0.44% among White women (RR 2.44), and CRITICAL neonatal morbidity prevalence was 15.69% versus 12.45%. These perinatal inequities are rooted in structural determinants. Racial disparities in pregnancy outcomes are thought to arise from systemic racism operating via residential segregation, neighbourhood-level socioeconomic deprivation, unequal insurance access, substandard hospital care, and implicit provider bias [7, 8, 9, 10, 11]. In our cohort, Medicaid financed 64.4% of births among Black women compared with 27.6% among White women; late or absent prenatal care affected 10.4% versus 4.7%; and 6.2% versus 2.4% of individuals received ≤ 2 prenatal visits. However, the contribution of preconception metabolic disease to severe maternal-neonatal morbidity among Black women has not been quantified. We therefore estimated adjusted risk differences for six prespecified exposures on severe maternal-neonatal morbidity among non-Hispanic Black women, decomposed the mediating pathways, and quantified the Black-White disparity attributable to modifiable factors.

Methods

Study design and data sources

We conducted a national retrospective cohort study using the 2022 and 2023 National Center for Health Statistics (NCHS) Linked Birth/Infant Death public-use files [12, 13], totalling 7,281,110 live birth records. Causal-model outcomes were derived from birth-certificate variables; linked infant-death records were used only for descriptive reporting and were not included as outcomes in the causal models. The 2023 cohort formed the primary analytic sample, with the 2022 cohort reserved for temporal replication. Reporting adhered to the STROBE guidelines [14]; the completed STROBE checklist is provided in the supplementary materials. This secondary analysis used de-identified public-use data and was exempt from institutional review under 45 CFR 46.104(d)(4). No *a priori* sample-size calculation was performed; the sample size was determined by national data availability, yielding narrow confidence intervals for all pre-specified effect estimates.

Study population and race/ethnicity framework

Primary analyses focused on non-Hispanic Black women (n = 491,753, 2023), with non-Hispanic White women (n = 1,787,833) serving as the reference group. Hispanic, non-Hispanic Asian and other racial-ethnic groups were analysed descriptively only. Records with unknown maternal race (n = 33,232, 0.9%) were excluded from primary analyses and retained solely within the study flow diagram. Race-ethnicity was defined using the standard MRACEHISP composite variable. Race was conceptualised as a socially constructed structural proxy rather than a biological trait and was not included as a node in the causal directed

acyclic graph (DAG); all causal modelling was stratified by racialised group.

Outcome, exposure, and mediator definitions

Four birth-certificate-derived perinatal outcomes were defined for analysis: (1) **CRITICAL**, a composite neonatal acute morbidity endpoint including neonatal intensive care unit admission, assisted ventilation (either immediately after delivery or for more than six hours), surfactant administration or neonatal seizures; (2) **SMM**, a five-item severe maternal morbidity measure comprising blood transfusion, third- or fourth-degree perineal laceration, uterine rupture, unplanned hysterectomy and intensive care unit admission; (3) **Severe-SMM**, a restricted four-item SMM definition excluding perineal laceration; (4) a composite primary endpoint defined as either SMM or CRITICAL morbidity. This birth-certificate-based proxy differs from the standard CDC 21-indicator hospital-coded SMM definition. Infant death was derived from the linked death record and reported descriptively, including unadjusted risk ratios. The 2023 cohort file (released January 2026) links deaths occurring in 2023 (n = 17,316) and in 2024 (n = 2,456; deaths within 365 days of birth among the 2023 cohort, 12.4% of all linked deaths), providing complete first-year follow-up comparable to the 2022 cohort. Six prespecified exposures were examined: two pre-gestational conditions (pre-gestational diabetes mellitus [PGDM], pre-gestational hypertension [PGHTN]), two pregnancy-onset conditions (gestational hypertension, gestational diabetes mellitus [GDM]), assisted reproductive technology (ART), and advanced maternal age. Mediator variables included multiple gestation, preterm birth, socioeconomic deprivation (low educational attainment or unmarried status) and inadequate prenatal care (late or no prenatal care). NCHS coding enforces strict mutual exclusivity between pre-gestational and gestational forms of the same disease (P(gestational disease | pre-gestational disease) = 0), so gestational hypertension and gestational diabetes were analysed only among women without the corresponding pre-gestational condition.

Causal framework and statistical analysis

A temporally structured DAG was developed to encode plausible perinatal causal pathways and derive minimal back-door adjustment sets for each exposure (Figure 1) [15, 16, 17]. Medicaid status, recorded only at delivery, was excluded as a post-exposure variable unsuitable for confounding adjustment. The final DAG structure, the full variable roster across nine temporal tiers, and the minimal back-door covariate adjustment sets for each exposure are provided in Supplementary Tables S1 and S2; the complete DAG edge list is included in the analysis-code repository. Adjustment sets were minimal back-door sets identified by greedy search under d-separation; for PGDM this comprised advanced maternal age, obesity, pre-gestational hypertension and smoking, with analogous sets for the other exposures. Because these minimal sets exclude sociodemographic variables (WIC receipt, low education, unmarried status, foreign-born nativity) that enter the DAG upstream of obesity and smoking, we additionally evaluated an extended set adding all four variables as a baseline-sensitivity check (Supplementary Table S3).

Average causal effects (ACEs) were estimated via g-computation on the risk-difference scale, selected for direct interpretability in terms of preventable population-level cases. Logistic outcome models were standardised to the empirical covariate distribution under exposed and unexposed conditions, with 95% confidence intervals generated via 200 bootstrap resamples. E-values were calculated to quantify robustness against unmeasured confounding [18]. Exploratory mediation analyses employed randomised interventional direct and indirect effects to locate modifiable pathways rather than to claim mechanism [19, 20]. This framework accommodates exposure-induced mediator-outcome confounding in metabolic pathway models and non-manipulable racial-group exposures in disparity decomposition, both of which violate natural effect identification criteria. All total, direct and indirect effects were estimated on the risk-difference scale, and the proportion mediated was computed as the indirect-to-total risk-difference ratio. For pathways whose adjustment sets contain no post-exposure covariates, the mediator was randomly assigned according to its

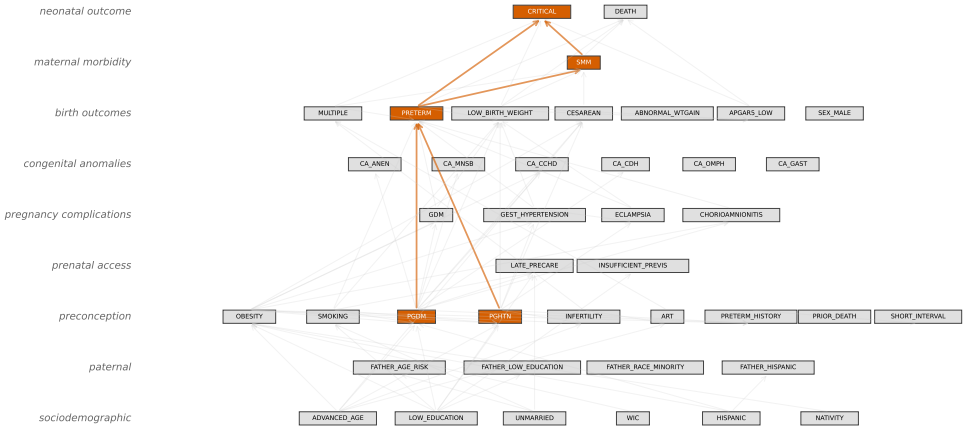


Fig. 1 Temporally structured DAG (41 nodes, 9 tiers). The DAG posits that sociodemographic factors (tier 1) precede paternal factors (tier 2), which precede preconception exposures (tier 3, including metabolic disease), prenatal-access factors (tier 4, including late prenatal care), pregnancy complications (tier 5), congenital anomalies (tier 6), birth outcomes (tier 7), maternal morbidity (tier 8), and the neonatal outcome (tier 9); the highlighted path represents the hypothesised metabolic→preterm/SMM→critical pathway.

conditional distribution under the counterfactual exposure level, $P(M \mid X{=}x', C_{\text{base}})$, marginalised over the empirical covariate distribution. For the metabolic pathways, the DAG-derived mediator-outcome confounder set additionally contains post-exposure covariates L (gestational diabetes, gestational hypertension, eclampsia, multiple gestation, cesarean delivery); to accommodate these exposure-induced mediator-outcome confounders, we fitted conditional models $P(L_j \mid X, C_{\text{base}})$ for each post-exposure covariate and evaluated the interventional expectations by Monte Carlo simulation, drawing L from its distribution under the relevant counterfactual exposure level and marginalising over the empirical distribution of the baseline covariates (the joint distribution of L approximated as the product of its marginal conditional models). Because NCHS coding enforces strict mutual exclusivity between pre-gestational and gestational disease, every simulated exposure-L combination lies within the observed support. Percentile bootstrap confidence intervals (200 resamples for Black women and for the disparity decompositions, 100 for White women in the modifiable-preconception pathways) were used for all components and proportions. Two families of pathways were analysed. First, three modifiable preconception pathways were decomposed within Black and White women: ART → multiple gestation → CRITICAL; PGDM → preterm birth → CRITICAL; and PGHTN → preterm birth → CRITICAL; the outcome for all three was the CRITICAL neonatal endpoint. Each was adjusted for the union of the DAG-derived back-door sets for the exposure-outcome, exposure-mediator and mediator-outcome relations (Supplementary Table S4); for the metabolic pathways this union adds the mediator-outcome confounders gestational disease (GDM, gestational hypertension, eclampsia), cesarean delivery and multiple gestation, several of which are downstream of the exposure, whereas the ART pathway was adjusted for advanced maternal age only (the infertility-treatment marker is deliberately not adjusted because ART is a subset of infertility treatment). Second, Black-White disparity pathways were decomposed with racialised group as the exposure (a proxy for structural racism), late or no prenatal care and socioeconomic deprivation (low education or unmarried) as mediators, and the composite (SMM or CRITICAL) morbidity or metabolic-disease endpoints as outcomes. These disparity decompositions were adjusted for advanced maternal age ($\geq$35) and foreign-born nativity, with additional deprivation adjustment for the prenatal-care pathway. Unlike the main

g-computation models, which used the minimal back-door set for each exposure (e.g., advanced maternal age, obesity, pre-gestational hypertension and smoking for PGDM; Supplementary Table S2), the modifiable-preconception mediation sets are supersets that additionally include mediator-outcome confounders, whereas the disparity decomposition uses a parsimonious age and nativity set because racialised group is exogenous to the DAG. Modifiable-preconception proportions represent the proportion of the total effect mediated; disparity-decomposition proportions represent descriptive fractions of the racial disparity attributable to each mediator and are not interpreted as causal effects of racial category.

Heterogeneity, sensitivity, and replication

Heterogeneity in PGDM and PGHTN effects among Black women was evaluated using `CausalForestDML` with Neyman-orthogonal scoring and five-fold cross-fitting [21] on the complete-case analytical sample of 189,564 Black women. The confounder set W matched the g-computation back-door adjustment sets for each exposure (advanced maternal age, obesity, pre-gestational hypertension and smoking for PGDM; advanced maternal age, obesity, pre-gestational diabetes and smoking for PGHTN). The heterogeneity feature set X comprised the four prespecified subgroup indicators not already contained in W (education below high school, college completion, WIC receipt, foreign-born nativity); age $\geq$35 and obesity were themselves confounders in W and were therefore evaluated as effect modifiers only through the interaction tests, not as forest heterogeneity features. Nuisance models were fitted by random forests (100 trees, maximum depth 4, minimum leaf size 500). Primary heterogeneity assessment relied on treatment-by-subgroup interaction testing within linear probability models with robust standard errors, supplemented by conditional average treatment effect estimates. ART was excluded from heterogeneity modelling owing to low population prevalence (0.82%). Medicaid was omitted from DML features because it is recorded at delivery. Missingness was low overall and comparable across racial groups (e.g., obesity 2.73% in Black vs 1.55% in White women; low maternal education 1.09% vs 0.75%). Marital status was the exception: it was blank for 11.1% of records (Black 3.7%, White 5.8%) owing to institutional non-reporting by some states, and such records were excluded from the marital-status denominator rather than coded as unmarried. Complete-case analysis was used for primary models; comprehensive missing-data evaluations and mode-imputation sensitivity checks are described in the Methods; alternative DAG specification analyses are reported in the supplementary materials. Temporal replication involved recalculation of crude and DAG-adjusted ACEs across the 2022 cohort, with Spearman rank correlation used to evaluate cross-year consistency of effect rankings.

Policy scenario burden estimation

Scenario modelling estimated potentially avoidable cases among Black women using $N \times$ prevalence $\times$ effectiveness $\times$ risk difference. Effectiveness was varied across four pre-specified tiers (20%, 40%, 60%, 80%) as a sensitivity parameter. Four scenarios were evaluated: metabolic risk-difference reduction, prenatal care access, preterm-birth-related burden reduction, and racial disparity reduction target. Each scenario used a distinct risk-difference quantity, as detailed in Supplementary Table S5: the metabolic scenario combined PGDM and PGHTN into a single composite exposure (prevalence 6.94%), avoiding double-counting of women with both conditions, and its average causal effect on each outcome was estimated by g-computation under the union of the PGDM and PGHTN back-door sets (excluding the exposure components themselves). Burden estimates use the population-averaged ACE as an approximation of the effect among exposed women, under the assumption that effect heterogeneity is small relative to overall scenario uncertainty. Outputs are illustrative hypothetical burden estimates, not predicted outcomes of real-world interventions.

Computational environment

All statistical analyses were undertaken using Python 3.12, with full package specifications and reproducibility protocols documented in Supplementary Table S6 and the supplementary materials. A fixed random seed (42) was used for all stochastic procedures.

Results

Study population and baseline disparities

Among 3,605,081 live births in 2023, 491,753 (13.6%) were to non-Hispanic Black women (Figure 2; Table 1). After complete-case restriction, the analytic sample for each exposure model ranged from 449,657 to 491,157, and composite-endpoint (SMM or CRITICAL) events ranged from 70,335 to 81,676; per-exposure model sample sizes, exposed counts and event counts are reported in Supplementary Table S7. Table 1 summarises maternal demographic, prepregnancy comorbidity, healthcare access, and pregnancy characteristics stratified by maternal race/ethnicity. Black women had higher prevalence of PGDM (1.69% vs 0.98%; RR 1.72), PGHTN (5.77% vs 3.11%; RR 1.85), and obesity (42.1% vs 29.3%; RR 1.44). Medicaid coverage was 64.4% vs 27.6%; late or no prenatal care 10.4% vs 4.7%; and $\leq$2 prenatal visits 6.2% vs 2.4%. ART use was lower in Black women (0.82% vs 2.40%; RR 0.34). Infant death (1.07% vs 0.44%; RR 2.44), CRITICAL (15.69% vs 12.45%; RR 1.26), and the composite endpoint (16.63% vs 13.65%; RR 1.22) were higher in Black women. CRITICAL comprised NICU admission in 13.19%, assisted ventilation in 7.56% (immediate, $\leq$6 h) and 2.63% (>6 h), surfactant administration in 0.69%, and neonatal seizures in 0.04% (Table 1). The four-item severe-SMM proxy was 1.04% vs 0.71% (RR 1.47), with higher rates of transfusion (7.6 vs 5.6 per 1,000), intensive care unit admission (2.8 vs 1.6), uterine rupture (0.58 vs 0.40), and unplanned hysterectomy (0.57 vs 0.47). The lower five-item SMM prevalence in Black women (1.46% vs 1.56%) reflects their lower perineal laceration rate (4.3 vs 8.7 per 1,000); the four-item severe-SMM proxy, which excludes laceration, was higher in Black women. In singleton births, the four-item severe-SMM proxy was 0.95% vs 0.66% (RR 1.44) and the composite endpoint 15.10% vs 12.53% (RR 1.20) (Supplementary Table S8).

Adjusted risk differences in Black women

Crude risk differences and DAG-adjusted average causal effects (ACEs) for the composite outcome across prespecified exposures are summarised in Table 2. Table 3 presents ACE estimates with corresponding 95% confidence intervals for both composite and severe SMM outcomes. Estimated ACEs on the composite endpoint were largest for PGDM (16.25 pp; 95% CI 15.17–17.33), followed by ART (12.53 pp; 11.30–13.90; association), gestational hypertension (12.13 pp; 11.81–12.48), PGHTN (10.08 pp; 9.53–10.60), gestational diabetes (5.19 pp; 4.75–5.68), and advanced maternal age (3.76 pp; 3.48–4.03) (Table 3). For severe SMM, the largest estimates were ART (1.80 pp; association), PGHTN (0.94 pp), and PGDM (0.53 pp; 95% CI 0.35–0.99), an order of magnitude smaller than the composite ACEs and confirming that the composite estimates are dominated by the neonatal component. Adjustment modestly attenuated most crude differences: PGDM 21.94 to 16.25 pp (crude RR 2.37 to marginal RR 2.01), PGHTN 13.29 to 10.08 pp (1.85 to 1.64), and gestational hypertension 12.43 to 12.13 pp (1.88 to 1.86) among women without PGHTN. Advanced maternal age required no adjustment and had identical crude and standardised estimates. E-values ranged from 1.78 to 3.44 (PGDM 3.44; PGHTN 2.66; ART 2.91). Compared with White women, estimated effects in Black women were larger for ART (+3.4 pp; 12.53 vs 9.14; association), advanced maternal age (+2.1 pp), and gestational hypertension (+1.7 pp), and smaller for PGDM (−2.1 pp); PGHTN was similar (+0.6 pp). In singleton births, severe-SMM effects were PGHTN 0.71 pp (0.56–0.98), PGDM 0.35 pp (0.22–0.72), and ART 0.16 pp (0.10–1.81; association); composite effects for PGDM and PGHTN were 16.27 and 9.30 pp (Supplementary Table S9). The attenuation was markedly larger for ART (1.80 to 0.16 pp, 91%) than for PGHTN (0.94 to 0.71 pp) and PGDM (0.53 to 0.35 pp), consistent with multiple gestation being a major pathway for ART-related maternal morbidity. Extending the back-door sets with the full sociodemographic baseline (WIC receipt, low education, unmarried status, foreign-born nativity) left the composite ACEs essentially unchanged (PGDM 16.15 [15.03–17.37] vs 16.25 pp; PGHTN 10.21 [9.64–10.84] vs 10.08 pp; Supplementary Table S3), despite the smaller effective sample imposed by state-level non-reporting of marital status.

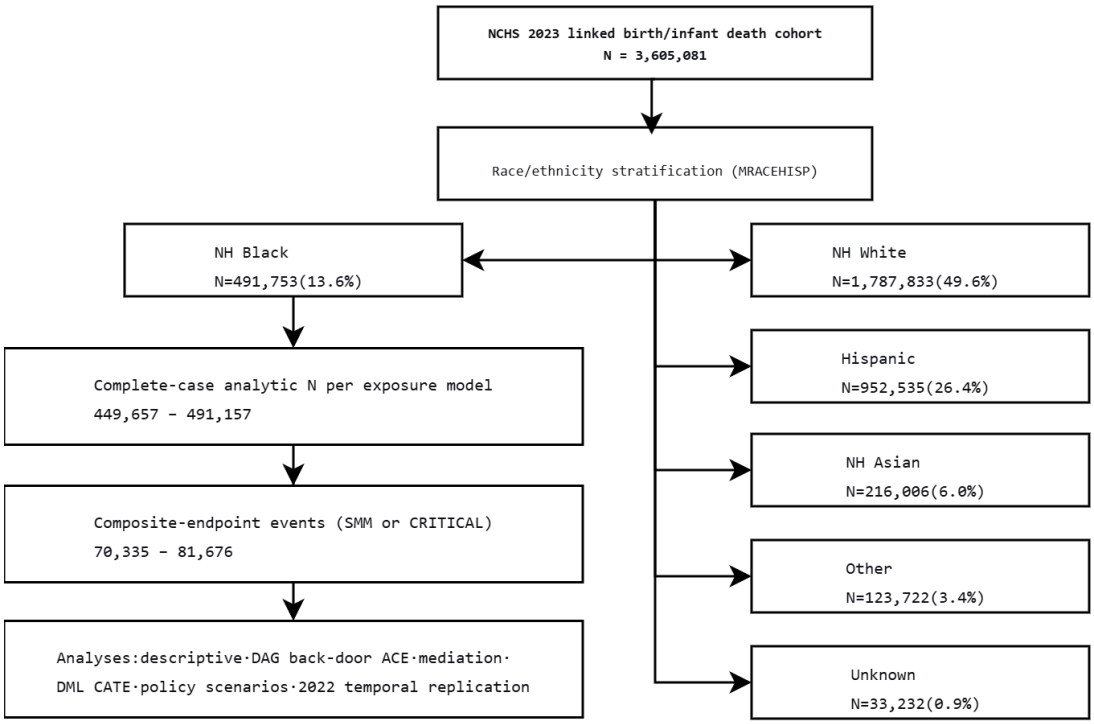


Fig. 2 Cohort selection stratified by maternal race/ethnicity from the 2023 NCHS linked birth and infant death cohort. The full cohort included $N = 3\,605\,081$ birth records. Records were stratified by maternal race/ethnicity (MRACEHISP). The source population comprised non-Hispanic Black women ($N = 491\,753$). After complete-case restriction, the analytic sample for each exposure model ranged from $N = 449\,657$ to $N = 491\,157$, with composite-endpoint (SMM or CRITICAL) events ranging from 70,335 to 81,676. Analyses included descriptive statistics, DAG-based back-door average causal effect estimation, interventional mediation, DML conditional average treatment effect heterogeneity, hypothetical policy burden scenarios, and temporal replication using the 2022 cohort. NH, non-Hispanic.

Exploratory mediation

Within Black women, multiple gestation mediated 41.7% (95% CI 37.3–47.0) of the ART effect on CRITICAL (indirect 4.84 pp [4.44–5.24]; total 11.61 pp [10.20–12.80]). The indirect effect through preterm birth, with gestational disease, cesarean delivery and multiple gestation modelled as mediator–outcome confounders, explained 63.4% (61.0–65.5) of the PGHTN effect (indirect 6.53 pp [6.26–6.80]; total 10.30 pp [9.87–10.84]) and 61.8% (59.2–65.3) of the PGDM effect (indirect 10.63 pp [10.00–11.30]; total 17.20 pp [15.99–18.16]). Corresponding proportions in White women were ART 32.7% (31.2–34.2), PGHTN 50.2% (48.2–51.9), and PGDM 61.1% (59.4–63.0); ART total effect in White women was 7.39 pp (association). In disparity-pathway decompositions on the composite (SMM or CRITICAL) morbidity endpoint, prenatal care access net of deprivation accounted for 4.4% (3.4–5.7) of the Black-White disparity. Deprivation accounted for 24.5% (19.9–28.0) of the composite-morbidity disparity and 11.4% (7.6–13.6) of the metabolic-disease disparity (Supplementary Table S4).

Heterogeneity within Black women

DML ATEs were 18.9 pp for PGDM and 10.7 pp for PGHTN, differing modestly from the g-computation composite ACEs in Table 2 (16.25 and 10.08 pp), reflecting the different modelling assumptions of the two estimators. Interaction tests (Benjamini-Hochberg FDR across 12 tests) identified three significant modifiers: obesity (-6.1 pp, $q = 0.017$) and foreign nativity (-6.3 pp, $q = 0.023$) for PGDM, and age $\geq$35 ($+2.5$ pp, $q = 0.036$) for PGHTN (Supplementary Table S10, Supplementary Figure S1). Education and WIC

showed no significant interaction (all $p > 0.20$). No subgroup had a null or reversed estimated effect.

Policy scenario burden estimation

Across the effectiveness ladder (Table 4), the hypothetical composite-endpoint burden among Black women ranged from 92 to 369 cases for prenatal care access, 880 to 3,520 for metabolic risk-difference reduction, 2,931 to 11,726 for racial disparity reduction target, and 7,014 to 28,056 for preterm-birth-related burden reduction. These are illustrative burden ranges under assumed effectiveness, not projected effects of named programmes.

Temporal replication

Crude risk differences were stable between 2022 and 2023 (PGDM 22.10 vs 22.04 pp; PGHTN 13.03 vs 13.51 pp; the replication RDs are computed on the complete-case sample for the temporal analysis and therefore differ marginally from the Table 2 model-sample values of 21.94 and 13.29 pp). DAG-adjusted ACEs also replicated (PGDM 16.8 vs 16.3 pp; PGHTN 9.8 vs 10.1 pp). ART was the least stable (10.6 vs 12.5 pp). ACE rank-order concordance was $\rho = 0.943$ ($p = 0.005$), with ART and gestational hypertension swapping second and third positions between 2022 and 2023 (Supplementary Figure S2). A constrained-network comparison is reported in the Supplementary Information.

Discussion

This study centred Black women in a nationwide analysis of adjusted risk differences and modifiable pathways for severe maternal-neonatal morbidity. Preconception metabolic disease carried the largest adjusted risk difference on the composite endpoint (PGDM 16.3 pp), ahead of ART (12.5 pp), gestational hypertension (12.1 pp) and PGHTN (10.1 pp); E-values of 2.66–3.44 indicate that an unmeasured confounder would need roughly three-fold associations with both exposure and outcome to overturn these estimates. Most of the metabolic effect operated after conception: preterm birth and related post-conception mechanisms explained 63.4% of the PGHTN effect and 61.8% of the PGDM effect, and multiple gestation explained 41.7% of the ART effect; the PGHTN proportion was larger in Black than in White women (63.4% vs 50.2%). Prenatal care access, net of deprivation, accounted for 4.4% of the modelled Black-White composite-morbidity disparity, and socioeconomic deprivation for 24.5%. The PGDM result should be read through the estimate ladder. The adjusted risk difference (16.3 pp) was smaller than the crude difference (21.9 pp) after adjustment for the DAG-derived back-door set (advanced maternal age, obesity, smoking, and co-occurring pre-gestational hypertension), but it remained the largest single exposure effect after adjustment. The composite endpoint is dominated by neonatal CRITICAL (prevalence 15.69% vs 12.45%; crude RR 1.26), so the headline number reflects neonatal more than maternal morbidity. Roughly three-fifths of the metabolic effects operated through post-conception mechanisms including preterm birth. The larger PGHTN proportion in Black women (63.4% vs 50.2%) is consistent with a weathering interpretation, in which chronic exposure to structural racism concentrates cardiometabolic risk earlier and magnifies downstream consequences [10, 22]. The observational pattern cannot confirm this mechanism, but it is consistent with it. Prenatal care access, net of deprivation, accounted for only 4.4% of the Black-White composite-morbidity disparity, while socioeconomic deprivation accounted for 24.5%. Most of the gap remains unexplained by measured mediators. Qualitative and provider-focused work points to implicit bias, colour-evasion, and care quality as contributors that administrative data cannot capture [23]. Access markers, although sharply stratified, explain only part of the disparity.

Policy implications

The ART finding (1.8 pp on severe SMM; 12.5 pp on composite morbidity; 9.1 pp in White women) is reported as an association and is best read as a marker of who reaches fertility care, not as a modifiable exposure; it points to inequity in access rather than to a target for clinical intervention. The results with clearer policy weight follow the pathway we identify: metabolic disease before pregnancy, then preterm birth, then neonatal morbidity. The two residual

scenarios in Table 4 correspond to the intervention classes discussed below: preterm-birth-related burden reduction (community perinatal support) and racial disparity reduction target (anti-bias training). Preconception metabolic care is the upstream lever, but its reach depends on coverage and control before pregnancy; community perinatal support reaches women who are already pregnant. In our data the post-conception share was larger than the preconception share for both metabolic exposures, which argues for funding both, with antenatal care as the nearer-term target [24, 25]. Community perinatal support is bounded by workforce supply and reimbursement, which vary by state [26, 27], and its modelled case count rests on the crude preterm-birth association, giving it a weaker evidential basis than the metabolic scenario. Medicaid expansion and 12-month postpartum coverage matter by keeping women connected to care across this sequence [28]. Anti-bias training and trust-building address the unexplained residual, and their effect on birth outcomes is less well quantified [23, 29]. Across all scenarios, avoided cases depend on effectiveness, coverage, uptake, and feasibility, none of which is observed in the NCHS data.

Implications for obstetric practice

The mediation results point to the antenatal and intrapartum window. Because roughly three-fifths of the metabolic-disease effect operated through post-conception mechanisms (gestational disease, multiple gestation, cesarean delivery and preterm birth), preconception optimisation alone cannot close the morbidity gap: the women who enter pregnancy with PGDM or PGHTN become the case-load of antenatal and intrapartum care, and it is in that window that most of the modifiable effect accrues. For obstetric teams, the two changes most likely to matter are earlier referral of women with pre-gestational disease to high-risk antenatal care and a planned delivery window for women with chronic hypertension [24, 25]; cervical-length screening, progesterone and postpartum metabolic follow-up are plausible complements, but their effect on the composite endpoint is not estimated here. The 41.7% of the ART effect carried by multiple gestation points the same way: management of multiple gestations, including fetal-growth surveillance and planned timing of delivery, is where ART-related maternal risk is modifiable in practice. Socioeconomic deprivation accounted for 24.5% of the composite disparity and prenatal-care access a further 4.4%; most of the gap remained unexplained by measured mediators. The implication is that expanding insurance and access is necessary but not sufficient, and the residual points toward what happens inside the delivery room. Hospital-level quality—standardised obstetric emergency drills for haemorrhage and eclampsia, timely ICU transfer, team-based communication—and provider-level bias are the plausible remaining levers, none of which the NCHS data can measure. Improving care quality is therefore not an alternative to social policy; it is where the remaining disparity is most likely to be concentrated.

Relevance to Sustainable Development Goal 3

The infant-mortality disparity (1.07% vs 0.44%) and neonatal CRITICAL disparity (15.69% vs 12.45%) bear on SDG target 3.2; the maternal-morbidity gradient bears on target 3.1. Identifying preconception metabolic disease and preterm birth as the principal identifiable pathways suggests where progress might realistically come from for the group carrying the largest burden.

Strengths and limitations

Strengths include national scale, explicit temporal ordering, centring of Black women, transparent separation of crude and adjusted estimates, formal interaction tests, and temporal replication. The main limitations are structural. The DAG was specified *a priori* from published literature, and all ACE estimates rely on this causal specification. The constrained-network comparison in the Supplementary Information provides a data-informed check rather than a comprehensive DAG sensitivity analysis. Back-door adjustment sets were derived from the DAG for each exposure and are listed in full in Supplementary Table S2; for PGDM the set comprised advanced maternal age, obesity, pre-gestational hypertension, and smoking, with analogous sets for the other exposures. Alternative DAGs with a direct PGDM–PGHTN edge produced point estimates of 19.50 pp (PGDM) and 11.51 pp (PGHTN),

without reversal of effect direction (Supplementary Table S11). Residual unmeasured shared causes of these two conditions may confound both ACEs, and E-values only approximately quantify this potential bias. We do not directly measure structural racism; we only observe racialised group membership, not segregation, discrimination, or provider bias, so disparity decompositions are descriptive and do not represent causal estimates of racism. Outcome ascertainment is another limitation: birth-certificate SMM variables undercount cases relative to the full CDC 21-indicator definition, and CRITICAL outcome recording varies by hospital resources. As Black women are more likely to receive care in under-resourced hospitals, some of the observed excess risk may stem from differential ascertainment rather than true differences in incidence. Other limits follow from coding and design. The public-use birth file does not include parity, gravidity, or prior obstetric history, so these preconception characteristics are not represented in the DAG or in any adjustment set; residual confounding from unmeasured obstetric history cannot be excluded, and the reported E-values quantify the confounding strength required to overturn each estimate. Mutually exclusive coding of pregestational and gestational disease means gestational disease cannot be identified as a mediator. The 2022 cohort is a replication from the same registry, not an independent validation cohort. Complete-case and mode-imputed estimates were similar (PGDM 16.25 vs 16.47 pp, PGHTN 10.08 vs 10.47 pp, and ART 12.53 vs 12.55 pp on the composite endpoint; Supplementary Table S12), but residual bias from non-random missingness cannot be fully excluded. Mediation assumes no unmeasured confounding of the mediator–outcome relationship. For the metabolic pathways (PGDM and PGHTN through preterm birth), the DAG-derived mediator–outcome confounder set includes gestational disease, cesarean delivery and multiple gestation, several of which occur after the exposure; we accommodated these exposure-induced mediator–outcome confounders by simulating post-exposure covariates from their conditional distributions under the counterfactual exposure level (Methods). Because the temporal position of cesarean delivery relative to preterm birth is uncertain (cesarean rates are higher after preterm birth), a sensitivity analysis excluding cesarean delivery from the post-exposure set gave PGDM and PGHTN proportions of 51.5% and 54.2% in Black women (Supplementary Table S4); the metabolic-pathway proportions should therefore be interpreted as the share of the effect operating through post-conception mechanisms broadly, rather than through preterm birth alone. These three modifiable-preconception pathways were analysed on the CRITICAL neonatal endpoint only, so their total effects (e.g., PGDM 17.20 pp on CRITICAL) are not directly comparable to the composite-endpoint ACEs in Table 2 (e.g., PGDM 16.25 pp). By contrast, the Black-White disparity decomposition used the composite (SMM or CRITICAL) morbidity outcome, so the 24.5% and 4.4% proportions refer to the composite disparity rather than the CRITICAL endpoint. Mediated proportions should be interpreted as conditional on each pathway's confounder set and specific to the stated outcome. G-computation assumes a correctly specified outcome model. Every effect estimate is model-based and model-conditional rather than an observed intervention effect.

Conclusions

Across 7.28 million US birth records, preconception metabolic disease had the largest absolute risk difference of the six prespecified exposures for severe maternal-neonatal morbidity among Black women, largely through preterm birth. Healthcare access and socioeconomic deprivation accounted for additional shares of the disparity; the remainder points to structural and care-quality mechanisms not captured in these data. Avoidable-case estimates were highly sensitive to assumed effectiveness and are best read as illustrative ranges rather than as projections.

List of abbreviations

ACE average causal effect
ART assisted reproductive technology
CDC Centers for Disease Control and Prevention
CI confidence interval
CRITICAL neonatal acute-morbidity composite (study outcome)
DAG directed acyclic graph
DML double/debiased machine learning

FDR false discovery rate
GDM gestational diabetes mellitus
NCHS National Center for Health Statistics
PGDM pre-gestational diabetes mellitus
PGHTN pre-gestational hypertension
pp percentage points
RR risk ratio
SDG Sustainable Development Goal
SMM severe maternal morbidity
STROBE Strengthening the Reporting of Observational Studies in Epidemiology
WIC Special Supplemental Nutrition Program for Women, Infants, and Children

Declarations

Ethics approval and consent to participate

This was a secondary analysis of completely de-identified, publicly available NCHS linked birth/infant-death cohort files. No direct identifiers or private clinical records were accessed, and the study involved no interaction, recruitment, or intervention with human participants. The analysis was exempt from institutional review board approval and informed consent under 45 CFR 46.104(d)(4).

Consent for publication

Not applicable.

Availability of data and materials

The NCHS linked birth/infant-death cohort public-use files are publicly available from the CDC. The derived analysis dataset can be regenerated from the public source files using the accompanying pipeline.

Code availability

The complete analysis code (Python scripts 01–09, seeded for reproducibility) is provided as Additional file 3 (analysis-code archive). All analyses used the publicly available NCHS data; the derived dataset can be regenerated from the public source files with this code.

Registration and pre-specified analysis plan

This study was not prospectively registered, as it is a secondary analysis of publicly available, de-identified vital-statistics data and not a clinical trial. The analytic plan—the temporally structured DAG (41 binary variables across nine temporal tiers), the six pre-specified exposures, the primary composite morbidity outcome, and the DAG-derived back-door adjustment sets—was fixed in the analysis code (`dag_spec.py`, `config.py`) before effect estimation and is recorded in the code (see Code availability). The constrained-network temporal comparison (Supplementary Table S13) and the policy-scenario effectiveness ladders are reported as sensitivity and descriptive analyses and are not presented as pre-specified confirmatory tests.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

The first author defined the clinical variables, reviewed the literature, and led the writing. The corresponding author designed the analysis, wrote the computational pipeline, and carried out the formal analysis. Both authors interpreted the results and approved the final manuscript.

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Additional files

Additional file 1. Supplementary Information: Supplementary Tables S1–S13 and Supplementary Figures S1–S2 (temporally structured DAG and variable dictionary; DAG-derived back-door adjustment sets; sensitivity and robustness analyses; interventional mediation and disparity decomposition; policy-scenario input parameters; software environment; per-model sample sizes; DML heterogeneity and interaction tests; extended-baseline, alternative-DAG, complete-case versus mode-imputed, and constrained-network sensitivity comparisons; DML CATE heterogeneity dot plot; temporal replication of 2022 versus 2023).
Additional file 2. Completed STROBE checklist for cohort studies, with section references.
Additional file 3. Analysis-code archive (Python scripts 01–09 with fixed random seed, analysis library, DAG specification and configuration), as described under Code availability.

Characteristic	NH Black	NH White	Hispanic	NH Asian	Other
Live births, n	491,753	1,787,833	952,535	216,006	123,722
Infant death	1.07%	0.44%	0.48%	0.34%	0.70%
CRITICAL (neonatal)	15.69%	12.45%	10.51%	10.64%	13.38%
NICU admission	13.19%	9.43%	8.84%	8.65%	10.34%
Assisted ventilation (immediate, ≤6 h)	7.56%	7.14%	4.65%	4.86%	7.46%
Assisted ventilation (>6 h)	2.63%	2.33%	1.53%	1.32%	2.49%
Surfactant administration	0.69%	0.53%	0.40%	0.29%	0.62%
Neonatal seizures	0.04%	0.04%	0.03%	0.02%	0.04%
SMM (5-item proxy)	1.46%	1.56%	1.19%	2.71%	1.65%
Severe-SMM (4-item)	1.04%	0.71%	0.68%	0.74%	1.02%
Composite morbidity	16.63%	13.65%	11.41%	12.91%	14.59%
Composite severe morbidity (4-item)	16.26%	12.89%	10.96%	11.13%	14.03%
PGDM	1.69%	0.98%	1.38%	1.52%	1.72%
PGHTN	5.77%	3.11%	2.07%	1.97%	3.81%
GDM	6.68%	7.49%	8.69%	15.76%	9.09%
Gestational hypertension	11.76%	10.91%	8.23%	6.84%	11.12%
Obesity	42.14%	29.30%	35.49%	13.63%	37.69%
Smoking	2.73%	4.43%	0.77%	0.19%	5.75%
Medicaid	64.43%	27.64%	58.38%	22.09%	52.18%
Late/no prenatal care	10.43%	4.70%	9.78%	4.97%	9.52%
≤2 prenatal visits	6.23%	2.43%	4.87%	2.02%	5.63%
Advanced maternal age (≥35)	19.60%	21.35%	17.80%	35.26%	16.10%
Low education (≤HS)	49.68%	27.56%	56.70%	16.23%	44.79%
Unmarried	69.66%	27.22%	54.27%	11.78%	56.98%
WIC	46.46%	19.53%	49.29%	16.92%	37.32%
ART	0.82%	2.40%	0.74%	4.48%	1.12%
Cesarean delivery	36.97%	31.13%	32.06%	34.21%	30.63%
Preterm birth	14.65%	9.44%	10.11%	9.08%	11.34%
Low birthweight	14.86%	7.06%	7.94%	9.47%	9.31%

SMM = severe maternal morbidity (birth-certificate-based proxy); PGDM = pre-gestational diabetes mellitus; PGHTN = pre-gestational hypertension; GDM = gestational diabetes mellitus; WIC = Special Supplemental Nutrition Program for Women, Infants, and Children; ART = assisted reproductive technology. Preterm birth is based on the obstetric estimate of gestation (<37 wk). Percentages are among records with non-missing values; "unmarried" is among records with non-blank marital status (blank for 3.7% of Black and 5.8% of White women, see Methods). Totals exclude 33,232 records with unknown maternal race (0.9% of all 2023 births; see Methods). Composite severe morbidity (4-item) = severe SMM (4-item) or CRITICAL morbidity.

Table 1 Baseline characteristics by race/ethnicity (2023 cohort). Infant death reflects complete first-year follow-up (deaths occurring in 2023 and 2024).

Exposure	Crude RD	Crude RR	OR†	RR‡	ACE	E-value
PGDM	21.94	2.37	2.51	2.01	16.25	3.44
PGHTN	13.29	1.85	1.87	1.64	10.08	2.66
ART	14.94	1.91	2.07	1.76	12.53	2.91
Gestational hypertension	12.43	1.88	2.17	1.86	12.13	3.12
Gestational diabetes	5.82	1.37	1.42	1.33	5.19	2.00
Advanced maternal age	3.76	1.24	1.29	1.24	3.76	1.78

Table 2 Estimate ladder: from crude association to adjusted estimate (composite morbidity, Black women).

RD = risk difference; RR = risk ratio; OR = odds ratio; ACE = average causal effect (g-computation standardisation); RD and ACE are in percentage points (pp); † conditional (logistic); ‡ marginal (standardised). Crude RD/RR are computed on the complete-case (model) sample so that crude and adjusted estimates share the same denominator. For most exposures, adjustment modestly attenuated the crude association (confounding by sociodemographic factors); for ART the infertility-treatment marker is not adjusted (ART is a subset of any infertility treatment), so only advanced age is adjusted and the estimate is attenuated only modestly (14.94 → 12.53 pp); for gestational hypertension and gestational diabetes the crude and adjusted estimates are computed among women without the corresponding pre-gestational disease; and for advanced maternal age (no adjustment required) the crude and standardised estimates coincide. Conditional OR and marginal RR are distinct association metrics. OR is numerically larger than RR by definition, even for unadjusted estimates, and does not imply a stronger effect.

Exposure	Outcome	ACE (pp)	95% CI
Pre-gestational diabetes	Composite morbidity	16.25	15.17–17.33
ART (association)	Composite morbidity	12.53	11.30–13.90
Gestational hypertension	Composite morbidity	12.13	11.81–12.48
Pre-gestational hypertension	Composite morbidity	10.08	9.53–10.60
Gestational diabetes	Composite morbidity	5.19	4.75–5.68
Advanced maternal age	Composite morbidity	3.76	3.48–4.03
ART (association)	Severe SMM	1.80	1.27–2.29
Pre-gestational hypertension	Severe SMM	0.94	0.74–1.09
Pre-gestational diabetes	Severe SMM	0.53	0.35–0.99

Table 3 Average causal effects (ACE) among Black women (2023).

ACE = average causal effect estimated by g-computation; numerically identical to the adjusted risk difference in the abstract. pp = percentage points. Gestational hypertension and gestational diabetes are redefined among women without the corresponding pre-gestational disease, because the NCHS codes are strictly mutually exclusive (P(gestational disease | pre-gestational disease) = 0). ART is reported as an association, not a causal effect (ART is a subset of any infertility treatment, so the infertility-treatment marker is not adjusted). Severe SMM (4-item proxy) occupies the same causal position in the DAG as the SMM composite and uses the same DAG-derived adjustment set as SMM for each exposure (Supplementary Table S2). For White women, ACEs (composite) were: PGDM 18.30, PGHTN 9.48, gestational hypertension 10.46, ART 9.14 (95% CI 8.72–9.60), GDM 4.01, advanced age 1.66 pp.

Scenario	Severe SMM	CRITICAL	Composite
Metabolic risk-difference reduction	62–247	869–3,474	880–3,520
Prenatal care access	3–11	96–382	92–369
Preterm-birth-related burden reduction	266–1,062	7,073–28,291	7,014–28,056
Racial disparity reduction target	324–1,297	3,193–12,770	2,931–11,726

Table 4 Model-estimated potentially avoidable cases among Black women (2023): ranges across effectiveness assumptions.

Scenario burden estimates only (N × prevalence × effectiveness × risk difference), with effectiveness varied as an assumption across a uniform 20/40/60/80% ladder for all four scenarios; the metabolic scenario uses a single DAG-adjusted ACE of a composite "PGDM or PGHTN" exposure (so women with both conditions are not double-counted), adjusted for the union of the PGDM and PGHTN back-door sets excluding the exposure components themselves (Supplementary Table S5), and no literature "control" rate is imported because "eliminating" the metabolic risk difference is not the same as achieving glycaemic or blood-pressure control in the existing population; values rounded to nearest case. Each column is an independent outcome-specific burden estimate; the composite column can be marginally smaller than the CRITICAL column because the exposure reduces maternal morbidity occurring without concurrent neonatal illness.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- supplementary.pdf
- analysiscodeclean.zip
- STROBE.pdf