



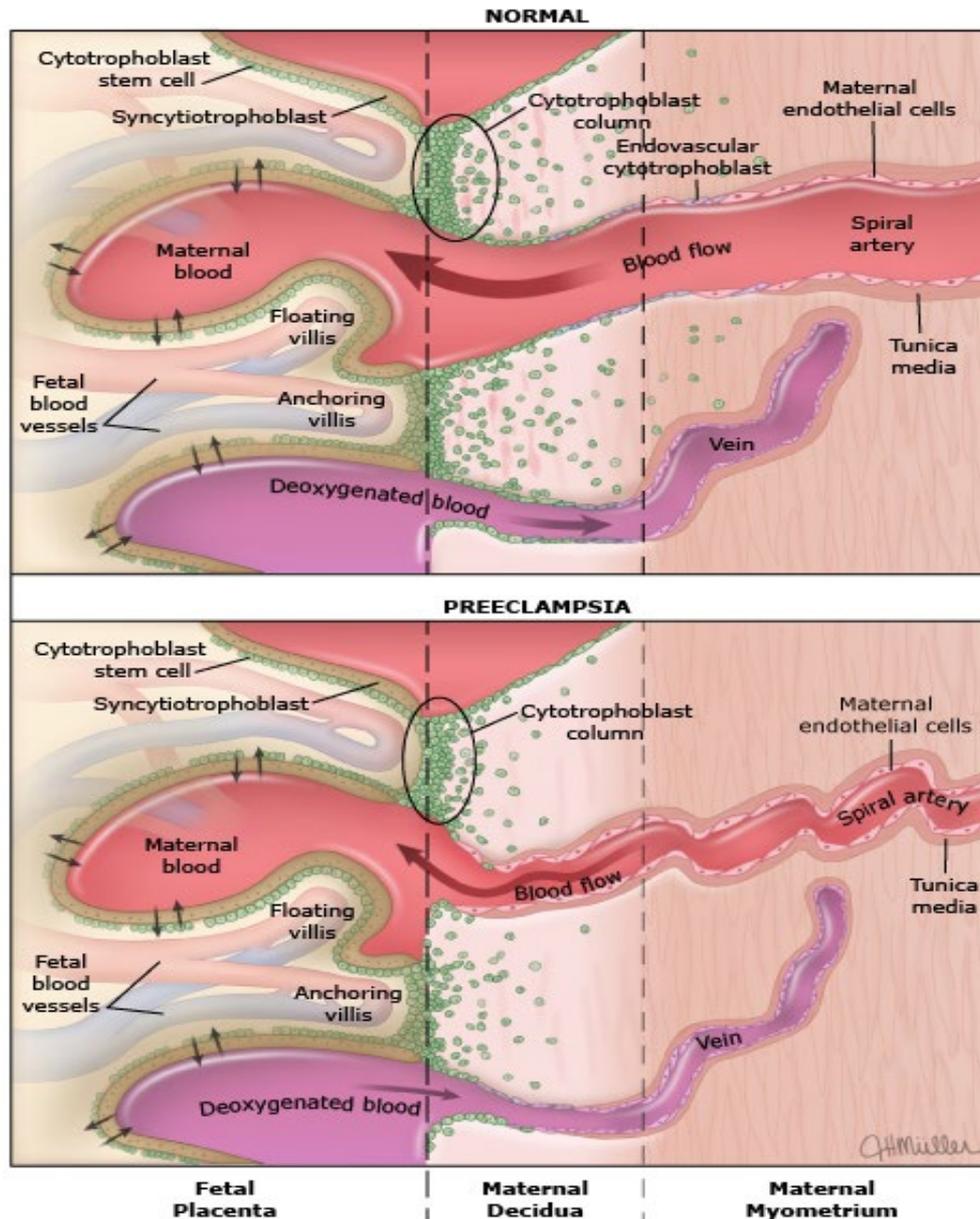
**Utilization of Self-Monitored Blood Pressure Kits to Support
Perinatal Hypertension Management:
The Cuff Kit Project**

Karen L Florio DO MPH

Outline

- **Updates on pathophysiology, management, treatment and prevention of hypertensive disorders of pregnancy**
- **Disparities in outcomes**
- **Validity of HBPM**
 - **Antenatal and postpartum**
- **Appropriate patient selection for HBPM and potential barriers**
- **The Missouri Cuff Kit Project**

Abnormal placentation in preeclampsia

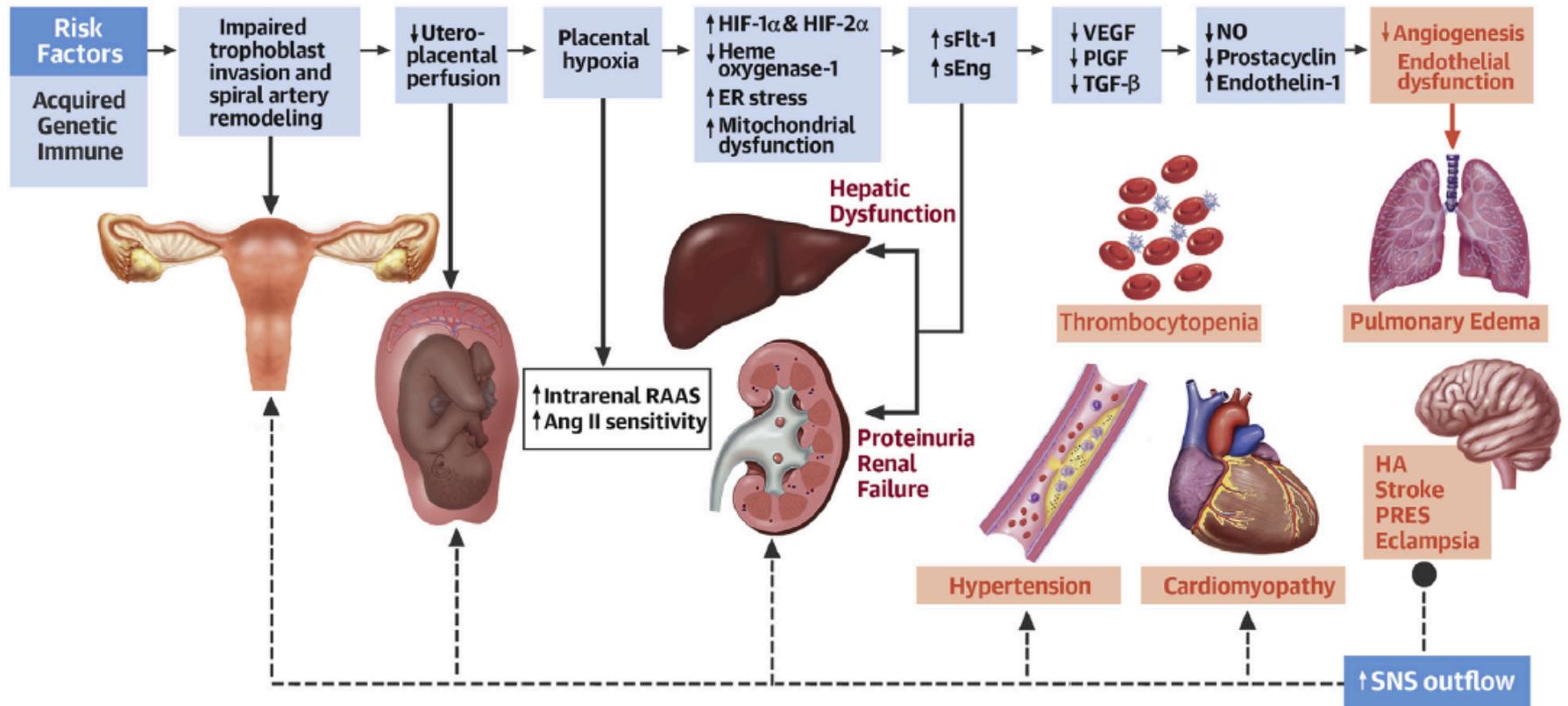


But why....

- Defective trophoblastic differentiation
- Placental hypoperfusion
- Decidual pathology

Exchange of oxygen, nutrients, and waste products between the fetus and mother depends on adequate placental perfusion by maternal vessels. In normal placental development, invasive cytotrophoblasts of fetal origin invade the maternal spiral arteries, transforming them from small-caliber resistance vessels to high-caliber capacitance vessels capable of providing placental perfusion adequate to sustain the growing fetus. During the process of vascular invasion, the cytotrophoblasts differentiate from an epithelial phenotype to an endothelial phenotype, a process referred to as "pseudovasculogenesis" or "vascular mimicry" (upper panel). In preeclampsia, cytotrophoblasts fail to adopt an invasive endothelial phenotype. Instead, invasion of the spiral arteries is shallow, and they remain small caliber, resistance vessels (lower panel).

CENTRAL ILLUSTRATION Pathogenesis of Preeclampsia



Ives, C.W. et al. J Am Coll Cardiol. 2020;76(14):1690-702.

Acquired, genetic, and immune risk factors contribute to early placental dysfunction (Stage 1). Placental dysfunction results in release of anti-angiogenic factors, leading to later multiorgan dysfunction (Stage 2). **Solid arrows** represent progression of disease. **Dashed arrows** represent SNS effect on respective organs. Ang II = angiotensin II; ER = endoplasmic reticulum; HA = headache; HIF = hypoxia-inducible transcription factor; HIF = hypoxia-inducible transcription factor; NO = nitric oxide; PlGF = placental growth factor; PRES = posterior reversible encephalopathy syndrome; RAAS = renin-angiotensin-aldosterone system; sEng = soluble endoglin; sFlt = soluble fms-like tyrosine kinase; SNS = sympathetic nervous system; TGF = transforming growth factor; VEGF = vascular endothelial growth factor.

TABLE 2 Diagnostic Criteria for Preeclampsia

Always necessary. . .

Hypertension

- SBP \geq 140 mm Hg or DBP \geq 90 mm Hg on two occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)
- SBP \geq 160 mm Hg or DBP \geq 105 mm Hg on one occasion

. . .And 1 of the following

Proteinuria

- \geq 300 mg per 24-h urine
- Protein/creatinine ratio of \geq 30 mg/g
- Dipstick reading of 2+ (urinary protein \geq 300 mg)

OR any 1 of the following (in the absence of other renal disease)

Thrombocytopenia

- Platelet count $<$ 100,000/L

Renal insufficiency

- Serum creatinine concentration \geq 1.1 mg/dL

Impaired liver function

- Elevated concentration of aspartate aminotransferase or alanine aminotransferase
- Severe persistent right upper quadrant or epigastric pain unresponsive to medications

Pulmonary edema

- Diagnosed by physical examination

Neurological signs

- New-onset headache unresponsive to medication
- Visual disturbances

Fetal growth restriction*

- Estimated fetal weight $<$ 10th percentile

Adapted from ACOG Practice Bulletin

ACOG = American College of Obstetrics and Gynecology; DBP = diastolic blood pressure; SBP = systolic blood pressure

Box 3. Preeclampsia with Severe Features

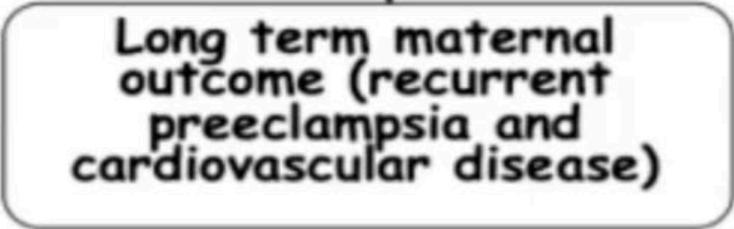
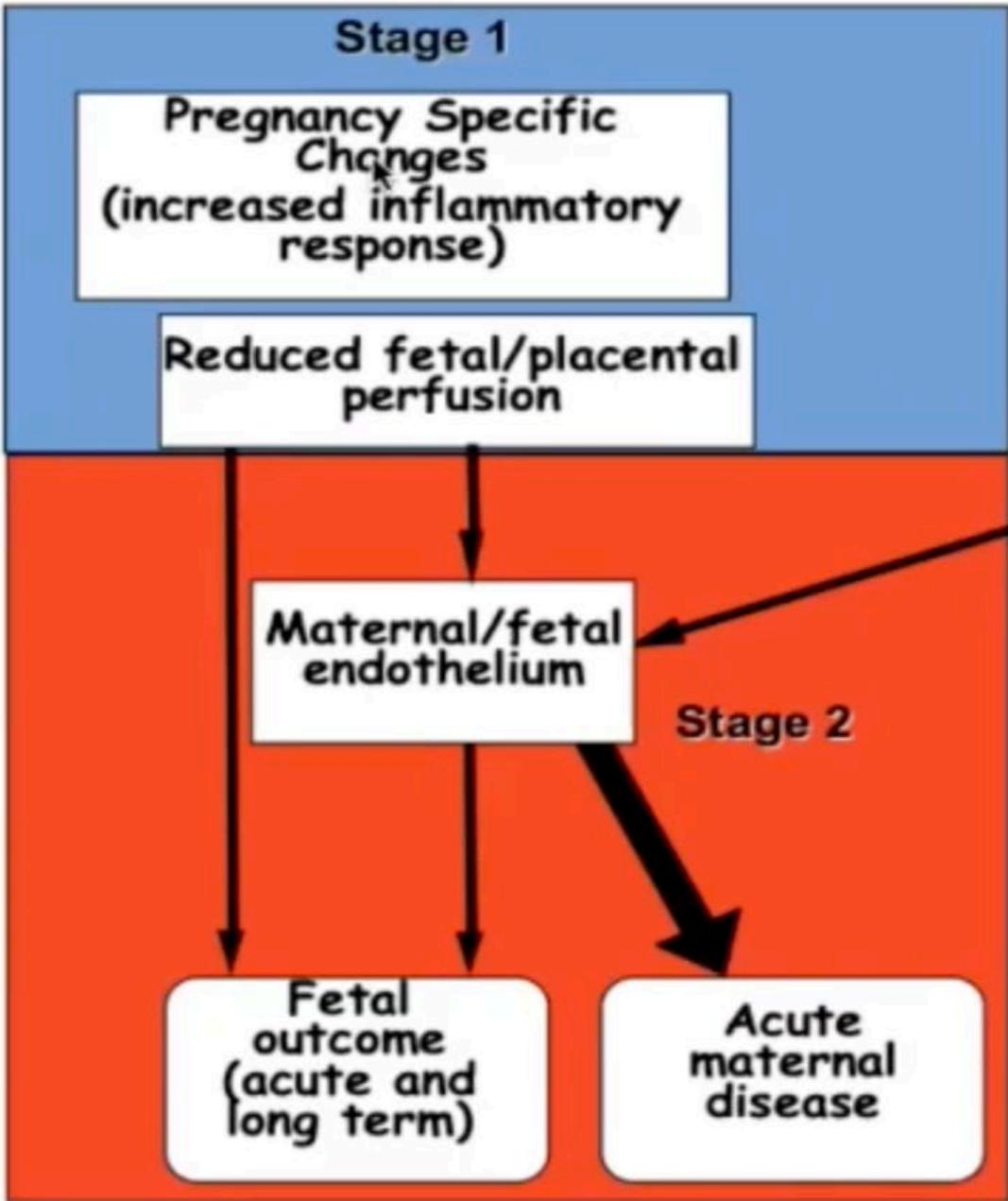
- Systolic blood pressure of 160 mm Hg or more, or diastolic blood pressure of 110 mm Hg or more on two occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)
- Thrombocytopenia (platelet count less than $100,000 \times 10^9/L$)
- Impaired liver function that is not accounted for by alternative diagnoses and as indicated by abnormally elevated blood concentrations of liver enzymes (to more than twice the upper limit normal concentrations), or by severe persistent right upper quadrant or epigastric pain unresponsive to medications
- Renal insufficiency (serum creatinine concentration more than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease)
- Pulmonary edema
- New-onset headache unresponsive to medication and not accounted for by alternative diagnoses
- Visual disturbances

with previously normal BP

of other renal disease

symptoms

* ACOG definition. Adapted from the American College of Obstetrics and Gynecology Committee on Practice Bulletins—Gynecology. Committee Opinion No. 549: Management of Preeclampsia and Eclampsia. Obstet Gynecol. 2013;121(5):1122-1131.



Contributors to Abnormal Placentation

- Immunologic factors
 - Oocyte donor IVF (4x higher risk)
- Genetic component
 - Family history (2-5x higher risk)
 - Partners of men who were product of PEC or whose previous partner was PEC
 - Mutations in cardiac genes (i.e. Corin and *TTN*)
- Trisomy 13 fetus
 - Genes for sFlt-1 and Flt-1
- Environmental factors
 - BMI > 40 (aOR 6.04)
 - Low calcium intake
- Inflammation (i.e. obesity and diabetes) and complement activation

Risk Factors for HDP

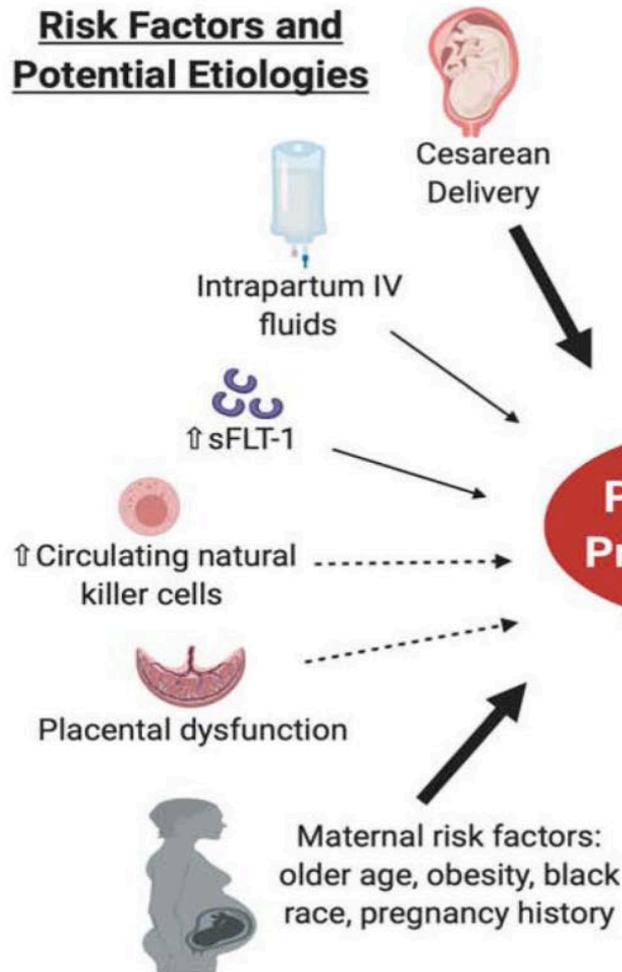
TABLE 2 | Major predisposing risk factors for the development of preeclampsia.

Risk factor	OR or RR (95% CI)
Antiphospholipid antibody syndrome	9.7 (4.3–21.7)
Renal disease	7.8 (2.2–28.2)
Prior preeclampsia	7.2 (5.8–8.8)
Systemic lupus erythmatosis	5.7 (2.0–16.2)
Nulliparity	5.4 (2.8–10.3)
HIV+ HAART treatment	5.6 (1.7–18.1)
HIV positive (untreated)	4.9 (2.4–10.1)
Chronic hypertension	3.8 (3.4–4.3)
Diabetes Mellitus	3.6 (2.5–5.0)
Multiple Gestation	3.5 (3.0–4.2)
Strong family history of cardiovascular disease (heart disease or stroke in ≥ 2 first degree relatives)	3.2 (1.4–7.7)
Obesity	2.5 (1.7–3.7)
Family history of preeclampsia in first degree relative	2.3–2.6 (1.8–3.6)
Advanced maternal age (> 40) for multips	1.96 (1.34–2.87)
Advanced maternal age (> 40) for nulliparas	1.68 (1.23–2.29)

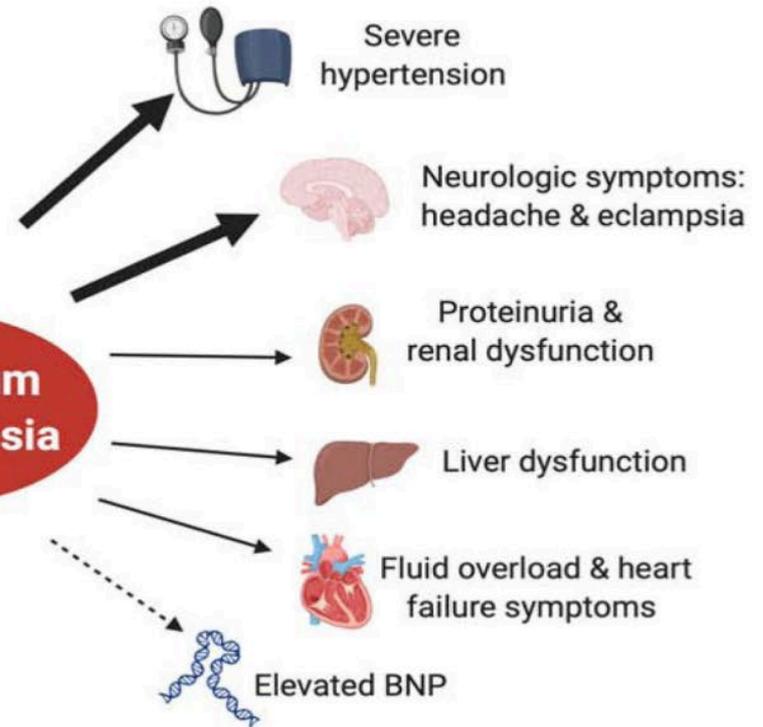
What about Postpartum Pre-eclampsia

HAUSPURG and JEYABALAN

Risk Factors and Potential Etiologies



Clinical Manifestations



Screening for Pre-Eclampsia: USA

Table 1. Clinical Risk Assessment for Preeclampsia^a

Risk level	Risk factors	Recommendation
High ^b	<ul style="list-style-type: none"> • History of preeclampsia, especially when accompanied by an adverse outcome • Multifetal gestation • Chronic hypertension • Pregestational type 1 or 2 diabetes • Kidney disease • Autoimmune disease (ie, systemic lupus erythematosus, antiphospholipid syndrome) • Combinations of multiple moderate-risk factors 	Recommend low-dose aspirin if the patient has ≥ 1 of these high-risk factors
Moderate ^c	<ul style="list-style-type: none"> • Nulliparity • Obesity (ie, body mass index >30) • Family history of preeclampsia (ie, mother or sister) • Black persons (due to social, rather than biological, factors)^d • Lower income^d • Age 35 years or older • Personal history factors (eg, low birth weight or small for gestational age, previous adverse pregnancy outcome, >10-year pregnancy interval) • In vitro conception 	<p>Recommend low-dose aspirin if the patient has ≥ 2 moderate-risk factors</p> <p>Consider low-dose aspirin if the patient has 1 of these moderate-risk factors</p>
Low	Prior uncomplicated term delivery and absence of risk factors	Do not recommend low-dose aspirin

^a Includes only risk factors that can be obtained from the patient medical history.

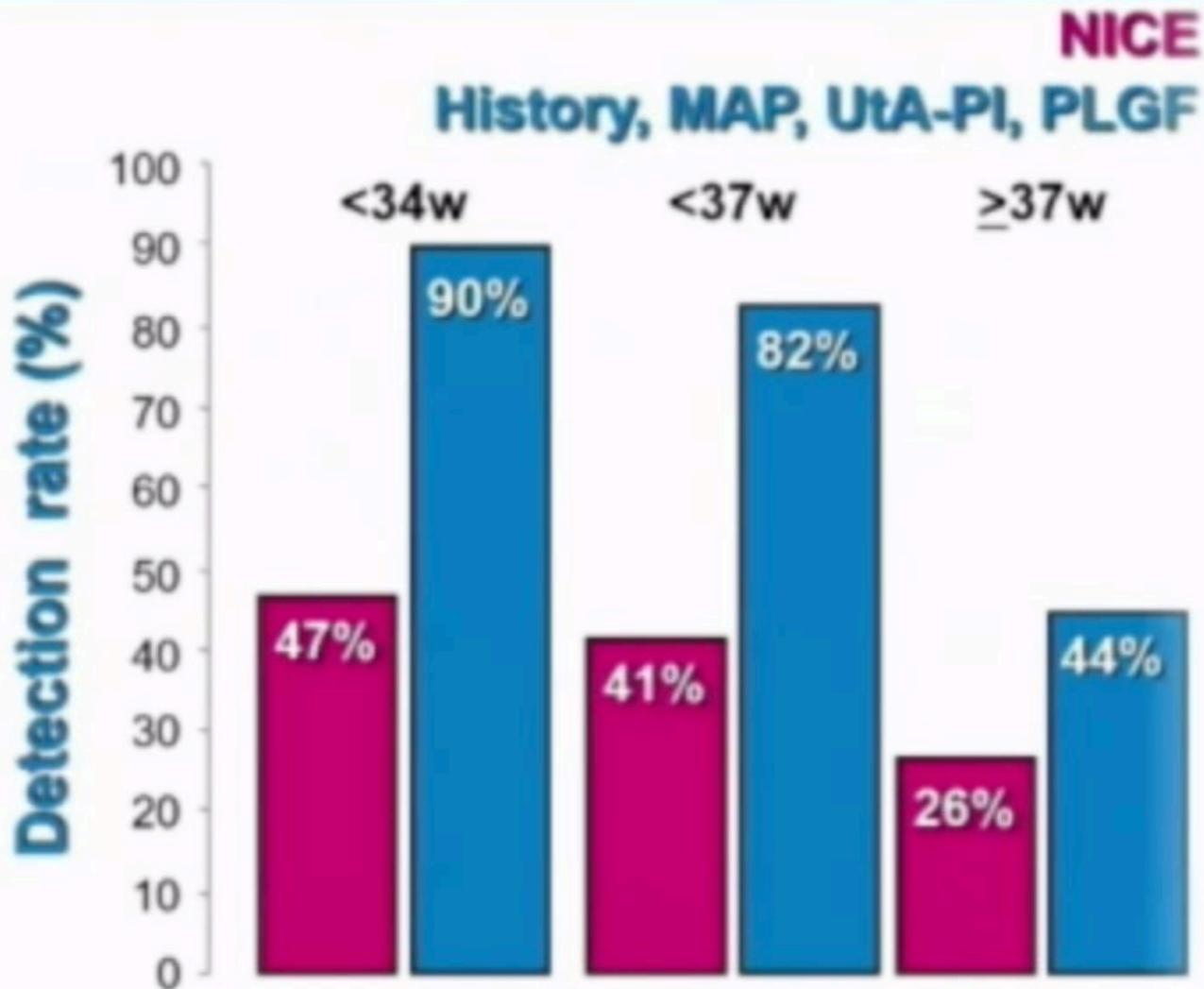
^b Includes single risk factors that are consistently associated with the greatest risk for preeclampsia. Preeclampsia incidence would likely be at least 8% in a population of pregnant individuals having 1 of these risk factors.

^c These factors are independently associated with moderate risk for preeclampsia,

some more consistently than others. A combination of multiple moderate-risk factors may place a pregnant person at higher risk for preeclampsia.

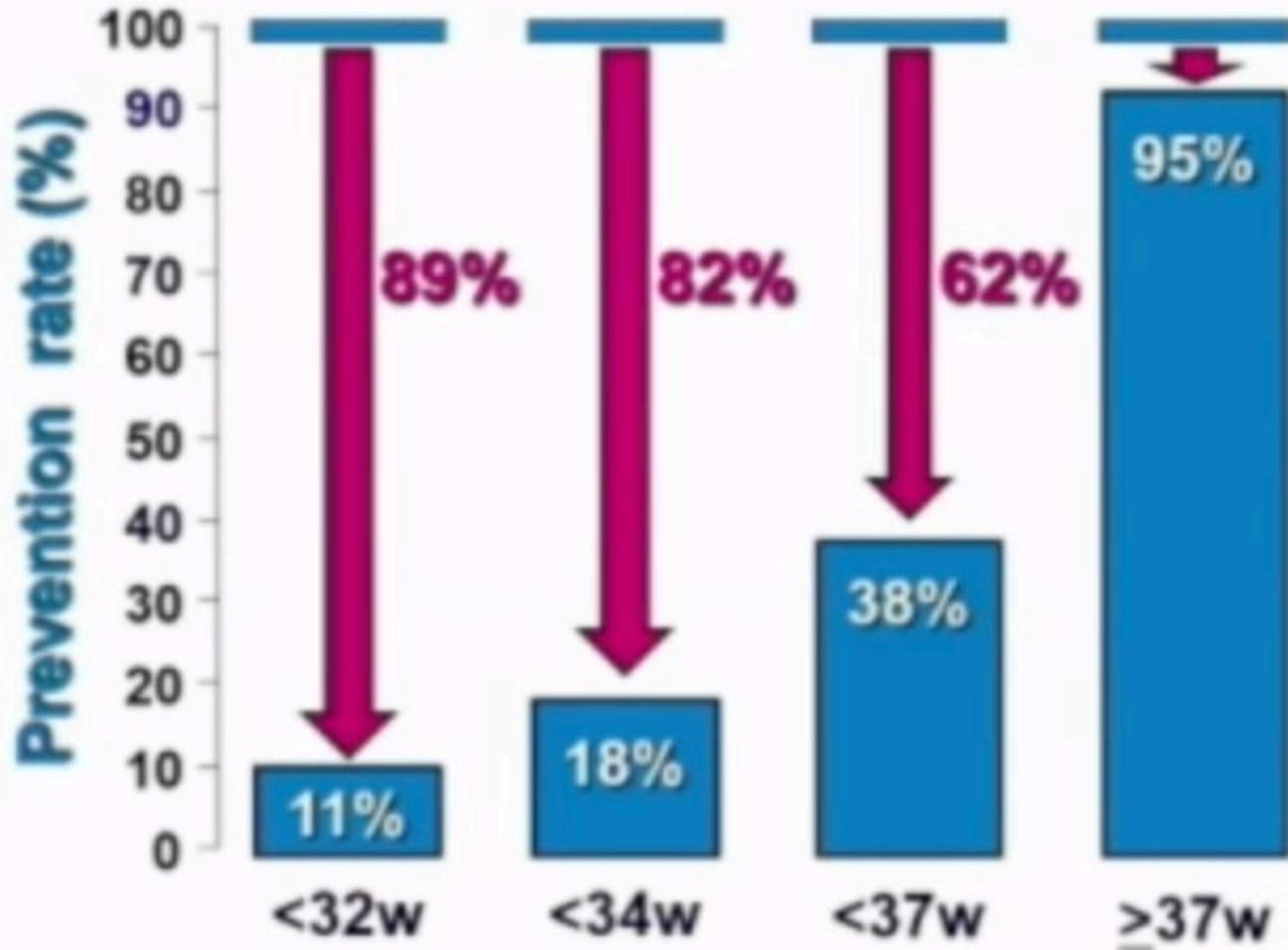
^d These factors are associated with increased risk due to environmental, social, and historical inequities shaping health exposures, access to health care, and the unequal distribution of resources, not biological propensities.

Screening for Pre-eclampsia: European



Tan *et al.* Comparison of diagnostic accuracy of early screening for pre-eclampsia by NICE guidelines and a method combining maternal factors and

Prevention of Pre-Eclampsia



So Why Don't We Use European Screening?

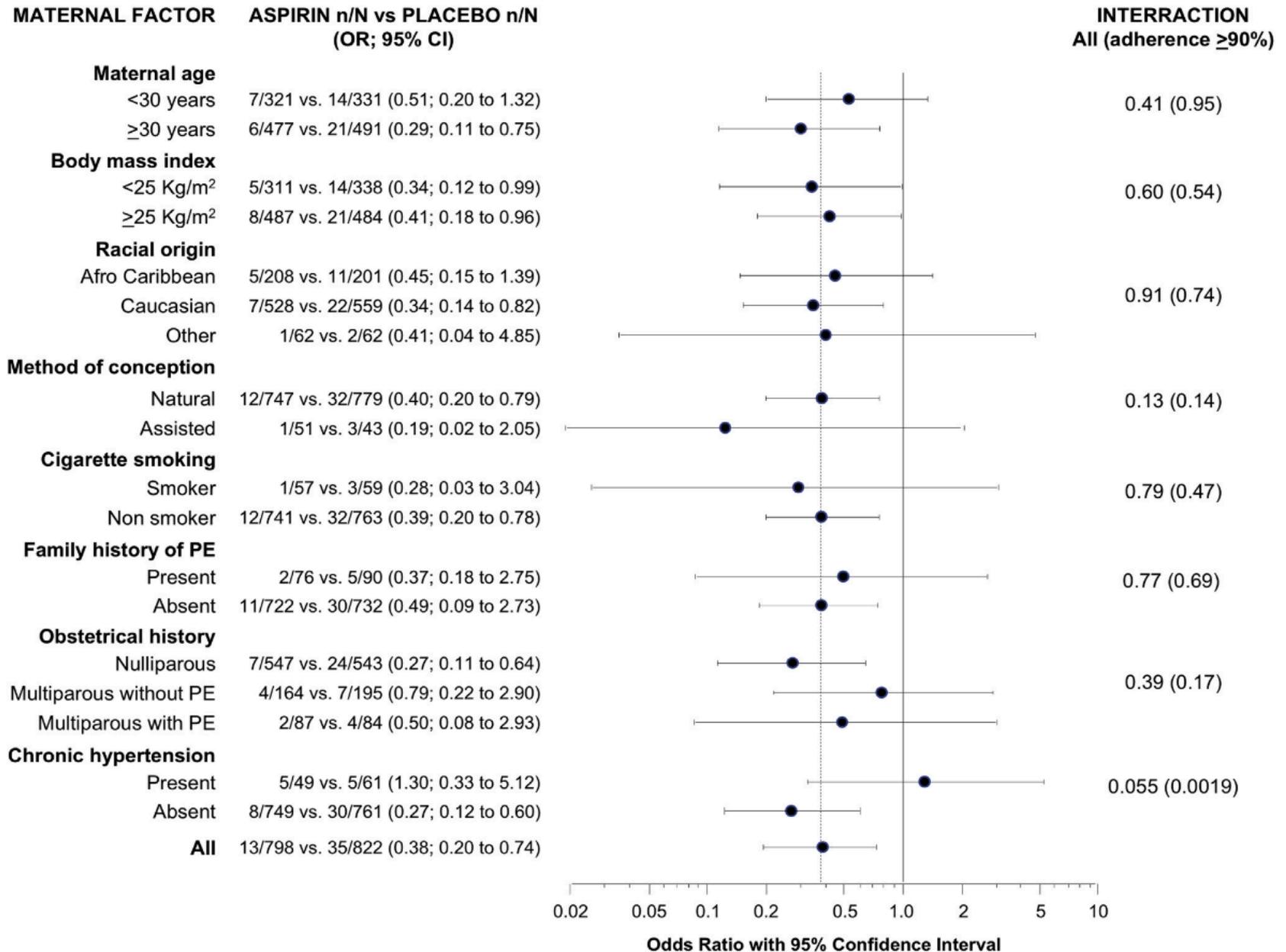
Table 2. Outcomes and Preeclampsia-Associated Costs of Each Strategy (Per 100,000 Women)

	No Aspirin	Ultrasound or Biomarker Measures	USPSTF Screen	Universal Aspirin
Total cases of preeclampsia	4,234	3,780	3,818	3,472
Preterm	1,320	829	873	515
Term	2,914	2,951	2,945	2,957
Additional cases of preeclampsia	762	308	346	—
Cases of gastrointestinal bleeding	0	4	6	20
Aspirin-exacerbated respiratory disease	0	90	134	480
Total cost (\$)	38,967,706	39,433,876	28,229,050	20,217,325
Incremental cost (\$)	18,750,381	19,216,551	8,011,725	—

USPSTF, U.S. Preventive Services Task Force.

FIGURE 1

Effect of aspirin on preterm preeclampsia according to maternal factor



Short Term Morbidity from HDP

- Increased for both antepartum (6.9%) and postpartum (12.1%) HDP
- People with pre-eclampsia (aOR 1.96), severe pre-eclampsia (aOR 3.46) and eclampsia (aOR 12.46) have significant cardiovascular morbidity associated with delivery hospitalization
- Risk for cHTN is 30-40% at 2-7 years
- Higher risk for hospitalizations (13.7% v 11.4%)

Long-term Maternal Risks from HDP

- Cardiovascular disease
 - Preeclampsia predictive of future cardiovascular and cerebrovascular disease up to 9-fold
 - Risks are equal to that of *smoking or obesity*
 - Can occur as early as 3-5 years after delivery
 - Related to both severity and number of episodes
 - Higher with early onset, severe disease or associated growth restriction (*similar risk to someone with diabetes*)
 - Permanent arterial changes
 - Lifestyle interventions after preeclampsia can reduce risk by 4-13%, *but never back to baseline*

Long-term Maternal Risks from HDP

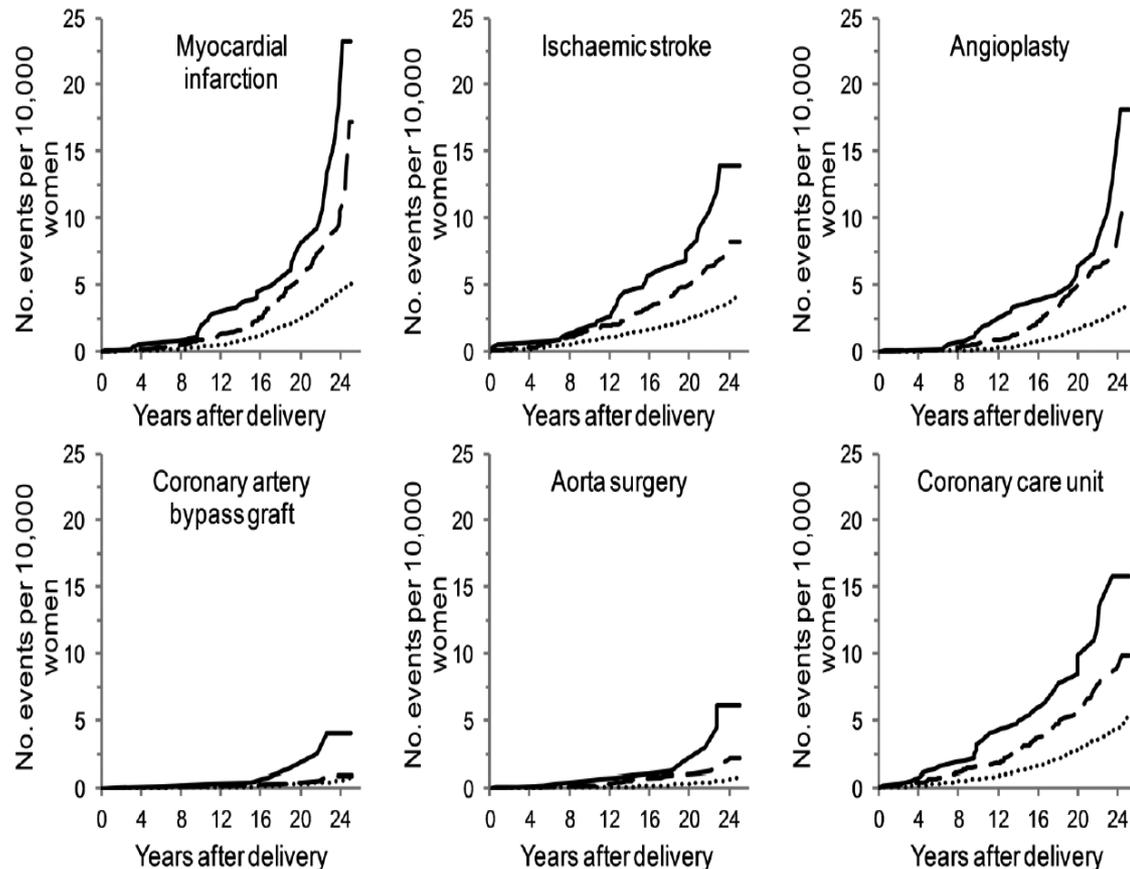


Figure 1 Cumulative incidence of cardiovascular events for recurrent, non-recurrent and no pre-eclampsia among women with two deliveries or more. Solid line, recurrent pre-eclampsia; dashed line, non-recurrent pre-eclampsia; dotted line, no pre-eclampsia.

Preeclampsia and future cardiovascular health: A systematic review and meta-analysis

Wu P et al. *Circulation Outcomes* 2017

Table 1. Sensitivity Analysis With Regard to Duration of Follow-Up

Outcomes		<1 y	1–10 y	>10 y
Cardiovascular disease death	Adjusted	...	2.30 (1.65–3.20), n=1	2.21 (1.73–2.81), n=3
Coronary heart disease	Adjusted	3.10 (1.56–6.15), n=1	3.78 (0.43–77.30), n=2	1.46 (0.95–2.25), n=3
	Unadjusted	2.09 (1.64–2.66), n=3
Coronary heart disease death	Adjusted	2.10 (1.25–3.51), n=4
Heart failure	Adjusted	4.10 (2.90–5.80), n=1	8.42 (4.39–16.17), n=2	1.60 (0.73–3.50), n=1
	Unadjusted	...	4.27 (2.09–8.71), n=1	2.73 (1.30–5.74), n=2
Stroke	Adjusted	2.22 (1.73–2.85), n=2	3.56 (0.52–24.28), n=2	1.18 (0.95–1.46), n=2
	Unadjusted	1.60 (1.47–1.74), n=1

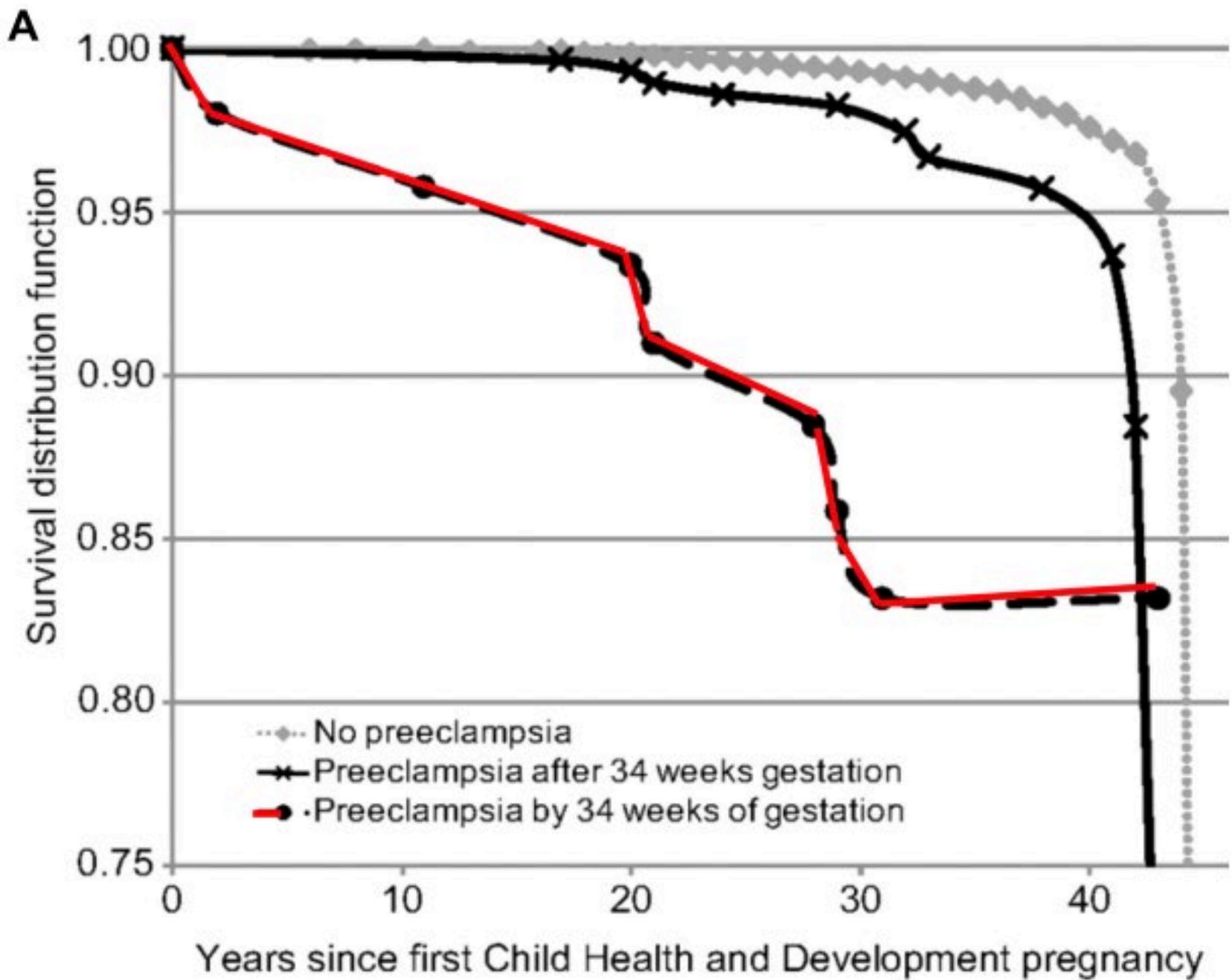
Values are represented as risk ratio (95% CI). CI indicates confidence interval.

Risk Factors for Long-Term Complications

Table 2. Sensitivity Analysis With Regard to Age, Pregestational Body Mass Index or Weight, Pregestational Smoking, Pregestational Diabetes Mellitus or Gestational Diabetes Mellitus, and Pregestational Hypertension

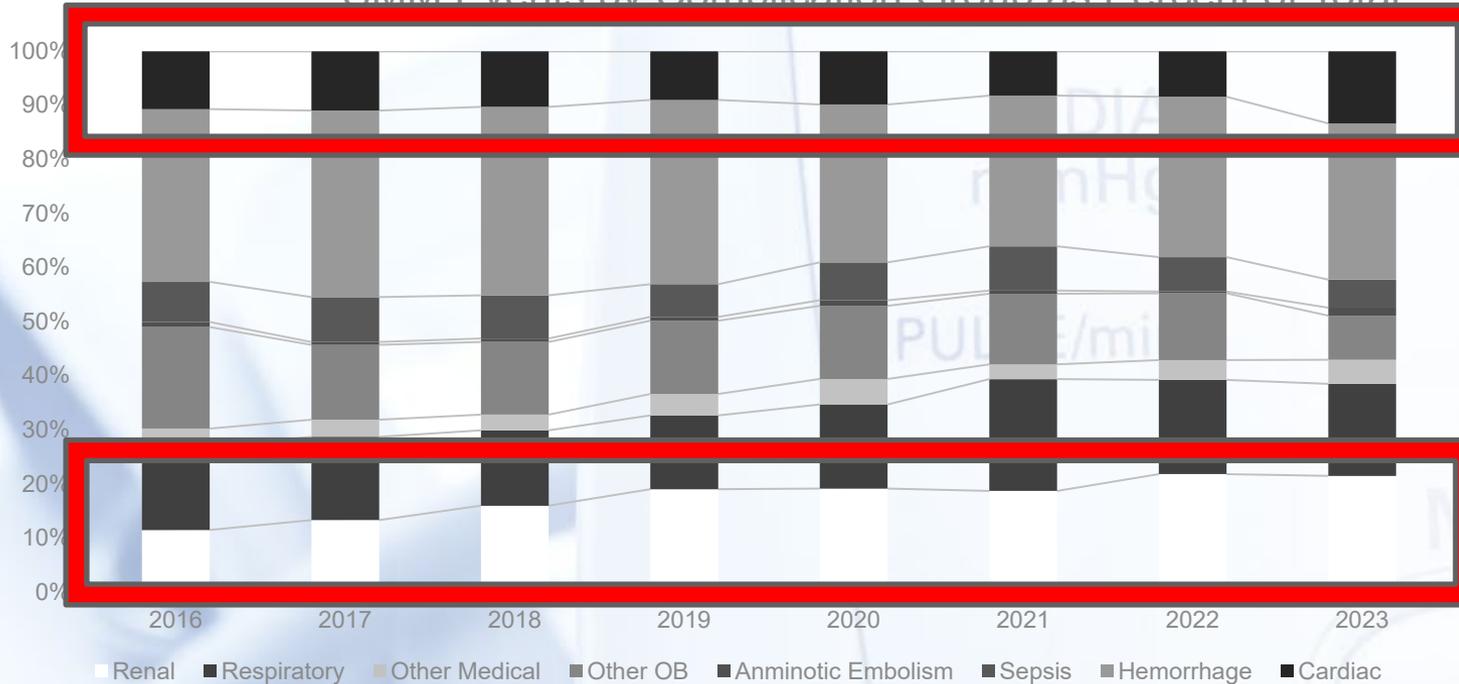
Outcomes	Age	BMI/Weight	Diabetes Mellitus/GDM	Smoking	Hypertension
Cardiovascular mortality	2.21 (1.83–2.66), n=4
Coronary heart disease	3.13 (1.45–6.75), n=5	1.84 (1.23–2.74), n=3	2.16 (1.03–4.52), n=2	1.56 (1.11–2.20), n=4	3.84 (0.81–18.16), n=3
Coronary heart disease death	2.63 (1.74–3.98), n=3
Heart failure	3.89 (1.83–8.26), n=3	2.74 (1.10–6.83), n=2	3.89 (1.83–8.26), n=3
Stroke	2.04 (1.60–2.60), n=5	1.94 (1.42–2.65), n=3	2.46 (1.11–5.43), n=3	1.64 (1.12–2.40), n=4	...

Values are represented as risk ratio (95% CI). BMI indicates body mass index; CI, confidence interval; and GDM, gestational diabetes mellitus.



SMM Events by Complication Group

SMM Events by Complication Group as Percent of Total



2022 Represents

- 21% increase in overall SMM Incidence since 2016
- Driven by
 - 130% increase in Renal
 - 37% increase in Respiratory
 - 30% increase in Other Medical

Disparities in HDP

B Adjusted prevalence of hypertensive disorders of pregnancy

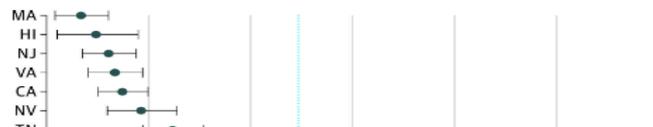
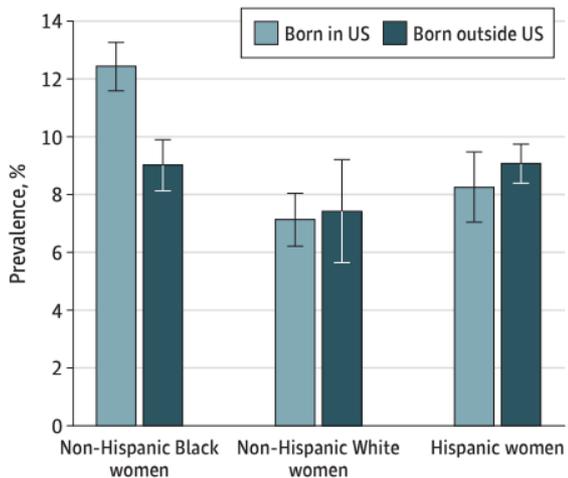
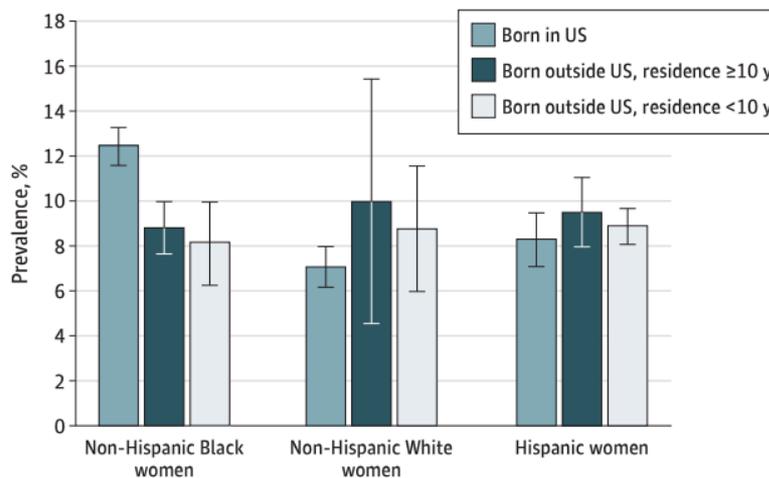


Figure 2. Age-Adjusted Prevalence of Preeclampsia by Nativity and Duration of US Residence Stratified by Race and Ethnicity

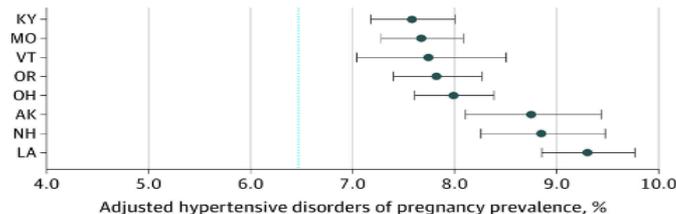
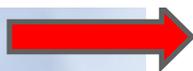
A Stratified by nativity



B Stratified by nativity and duration of US residence

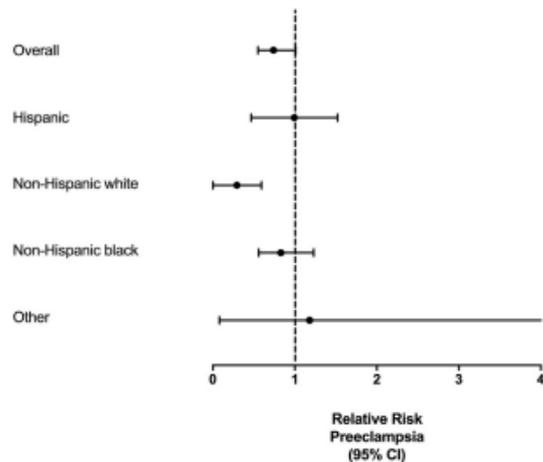


Error bars indicate SEs.



Aspirin Prevention by Race

FIGURE 2
Forest plot of outcomes by ethnicity and race, Low-Risk Aspirin (LRA) study

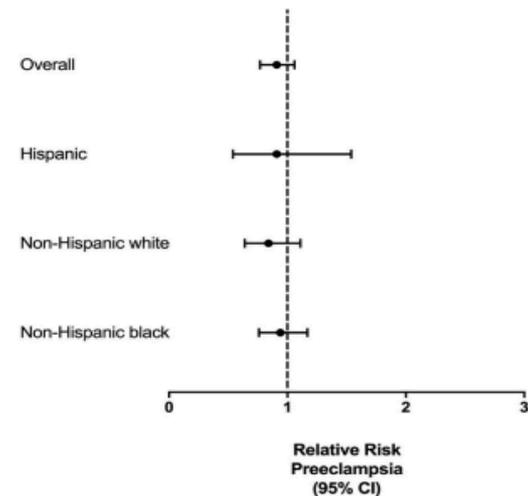


The efficacy of aspirin for prevention among subjects at a low risk of the occurrence of preeclampsia was observed to be significant only among non-Hispanic white women and not among non-Hispanic black or Hispanic women.

CI, confidence interval.

Tolcher et al. *Impact of ethnicity and race on aspirin for preeclampsia prevention. AJOG MFM 2020.*

FIGURE 3
Forest plot of outcomes by ethnicity and race, High-Risk Aspirin (HRA) study



There was no significant impact of aspirin in the prevention of preeclampsia among subjects at high risk, including when stratified by race or ethnicity.

CI, confidence interval.

Tolcher et al. *Impact of ethnicity and race on aspirin for preeclampsia prevention. AJOG MFM 2020.*

TABLE 1

Outcomes by ethnicity and race in the Low-Risk Aspirin (LRA) study among women receiving aspirin or placebo for preeclampsia prevention (n = 3134) (Hispanic [n = 1018], non-Hispanic white [n = 559], non-Hispanic black [n = 1533], other [n = 24])

Outcome	Aspirin group (n=1570)	Placebo group (n=1564)	P value ^a	RR (95% CI)	P value
Preeclampsia, n (%)	69 (4.395)	94 (6.010)	.070	0.740 (0.550–1.010)	.052
Hispanic	23 (4.536)	26 (5.088)		0.900 (0.520–1.560)	.716
Non-Hispanic white	3 (1.090)	17 (5.986)		0.190 (0.060–0.630)	.007
Non-Hispanic black	42 (5.405)	50 (6.614)		0.830 (0.560–1.230)	.347
Other	1 (5.051)	1 (7.692)		1.100 (0.000–10.700)	.500
GA at delivery, mean (SD)	38.600 (3.200)	38.800 (2.900)	—	—	.427
Hispanic	38.800 (2.800)	39 (2.400)			.177
Non-Hispanic white	39.100 (2.600)	39.100 (2.300)			.429
Non-Hispanic black	38.400 (3.600)	38.500 (3.300)			.905
Other	38.600 (1.500)	39.500 (2.600)			.089
Preterm delivery ^b , n (%)	157 (10.000)	146 (9.335)	.890	1.090 (0.880–1.340)	.450
Hispanic	47 (9.270)	33 (6.458)		1.450 (0.950–2.230)	.090
Non-Hispanic white	17 (6.182)	23 (8.099)		0.780 (0.430–1.420)	.420
Non-Hispanic black	93 (11.969)	89 (11.772)		1.030 (0.780–1.350)	.840
Other	0	1 (7.692)		—	—
Abruption, n (%)	11 (0.701)	2 (0.128)	.880	5.560 (1.240–25.060)	.025
Hispanic	2 (0.394)	0		—	—
Non-Hispanic white	5 (1.818)	0		—	—
Non-Hispanic black	4 (0.515)	2 (0.257)		1.980 (0.360–10.790)	.428
Other	0	0		—	—
SGA infant, n (%)	73 (4.650)	90 (5.754)	.490	0.820 (0.610–1.110)	.203
Hispanic	15 (2.959)	23 (4.501)		0.670 (0.350–1.260)	.212
Non-Hispanic white	12 (4.364)	10 (3.521)		1.270 (0.560–2.900)	.563
Non-Hispanic black	45 (5.792)	56 (7.407)		0.800 (0.550–1.160)	.237
Other	1 (9.091)	1 (7.692)		1.180 (0.080–16.780)	.902
Stillbirth, n (%)	13 (0.828)	5 (0.320)	.250	2.620 (0.940–7.350)	.066
Hispanic	1 (0.197)	1 (0.196)		1.030 (0.060–13.340)	.986
Non-Hispanic white	1 (0.364)	1 (0.352)		1.050 (0.070–16.640)	.974
Non-Hispanic black	11 (1.416)	3 (0.397)		3.610 (1.010–12.890)	.048
Other	0	0		—	—
Neonatal death, n (%)	4 (0.255)	7 (0.448)	.890	0.580 (0.170–1.970)	.379
Hispanic	2 (0.391)	2 (0.391)		1.030 (0.150–7.250)	.979
Non-Hispanic white	0	1 (0.352)		—	—
Non-Hispanic black	2 (0.265)	4 (0.529)		0.490 (0.090–2.680)	.413
Other	0	—		—	—

CI, confidence interval; GA, gestational age; RR, relative risk; SD, standard deviation; SGA, small for gestational age.

^a Test of interaction between ethnicity and aspirin use for each outcome; ^b Preterm delivery considered at <37 wk.

Tolcher et al. Impact of ethnicity and race on aspirin for preeclampsia prevention. AJOG MFEM 2020.

TABLE 2

Outcomes by ethnicity and race in the High-Risk Aspirin (HRA) study among women receiving aspirin or placebo for preeclampsia prevention (n = 2539) (Hispanic [n = 269], non-Hispanic white [n = 832], non-Hispanic black [n = 1426], other [n = 12])

Outcome	Aspirin group (n=1273)	Placebo group (n=1266)	P value ^a	RR (95% CI)	P value
Hispanic	21 (15.909)	25 (18.248)		0.91 (0.54–1.54)	.72
Non-Hispanic white	73 (18.114)	92 (21.445)		0.84 (0.64–1.11)	.22
Non-Hispanic black	136 (18.605)	137 (19.712)		0.94 (0.76–1.17)	.58
Other	1 (1.000)	0		—	—
GA at delivery, mean (SD)	36.20 (3.90)	35.90 (4.40)	—	—	.43
Hispanic	36.20 (3.50)	35.80 (4)	—	—	.28
Non-Hispanic white	36.30 (3.80)	35.80 (4.30)			.23
Non-Hispanic black	36.20 (4.10)	36 (4.60)			.82
Other	37.40 (2)	39.60 (0.90)			.10
Preterm delivery ^b , n (%)	502 (39.434)	532 (42.022)	.26	0.94 (0.86–1.03)	.19
Hispanic	51 (38.636)	64 (46.715)		0.86 (0.65–1.13)	.29
Non-Hispanic white	165 (40.943)	195 (45.455)		0.90 (0.76–1.05)	.17
Non-Hispanic black	284 (38.851)	273 (39.281)		0.99 (0.87–1.12)	.85
Other	2 (28.571)	0		—	—
Abruption, n (%)	15 (1.178)	22 (1.738)	.62	0.68 (0.35–1.30)	.24
Hispanic	1 (0.758)	2 (1.460)		0.55 (0.05–5.87)	.61
Non-Hispanic white	5 (1.241)	6 (1.399)		0.88 (0.27–2.87)	.84
Non-Hispanic black	9 (1.231)	14 (2.014)		0.61 (0.27–1.40)	.24
Other	—	0		—	—
SGA infant, n (%)	101 (7.934)	92 (7.267)	.47	1.09 (0.83–1.43)	.55
Hispanic	7 (5.303)	7 (5.109)		1.09 (0.39–3.01)	.87
Non-Hispanic white	30 (7.444)	25 (5.828)		1.26 (0.75–2.10)	.38
Non-Hispanic black	64 (8.755)	60 (8.633)		1.08 (0.72–1.41)	.96
Other	0	0		—	—
Stillbirth, n (%)	21 (1.650)	32 (2.528)	.76	0.65 (0.38–1.13)	.12
Hispanic	1 (0.758)	3 (2.190)		0.36 (0.04–3.41)	.37
Non-Hispanic white	5 (1.241)	7 (1.632)		0.76 (0.24–2.37)	.63
Non-Hispanic black	15 (2.052)	21 (3.022)		0.68 (0.35–1.30)	.24
Other	0	1 (20.000)		—	—
Neonatal death, n (%)	21 (1.650)	23 (1.817)	.28	0.91 (0.51–1.63)	.75
Hispanic	3 (2.273)	3 (2.190)		1.08 (0.22–5.25)	.92
Non-Hispanic white	6 (1.489)	4 (0.932)		1.59 (0.45–5.58)	.47
Non-Hispanic black	12 (1.642)	16 (2.302)		0.71 (0.34–1.49)	.37
Other	0	0		—	—

CI, confidence interval; GA, gestational age; RR, relative risk; SD, standard deviation; SGA, small for gestational age.

^a Test of interaction between ethnicity and aspirin use for each outcome; ^b Preterm delivery considered at <37 wk.

Tolcher et al. Impact of ethnicity and race on aspirin for preeclampsia prevention. AJOG MFM 2020.

Social Determinants of Suboptimal Cardiovascular Health Among Pregnant Women in the United States

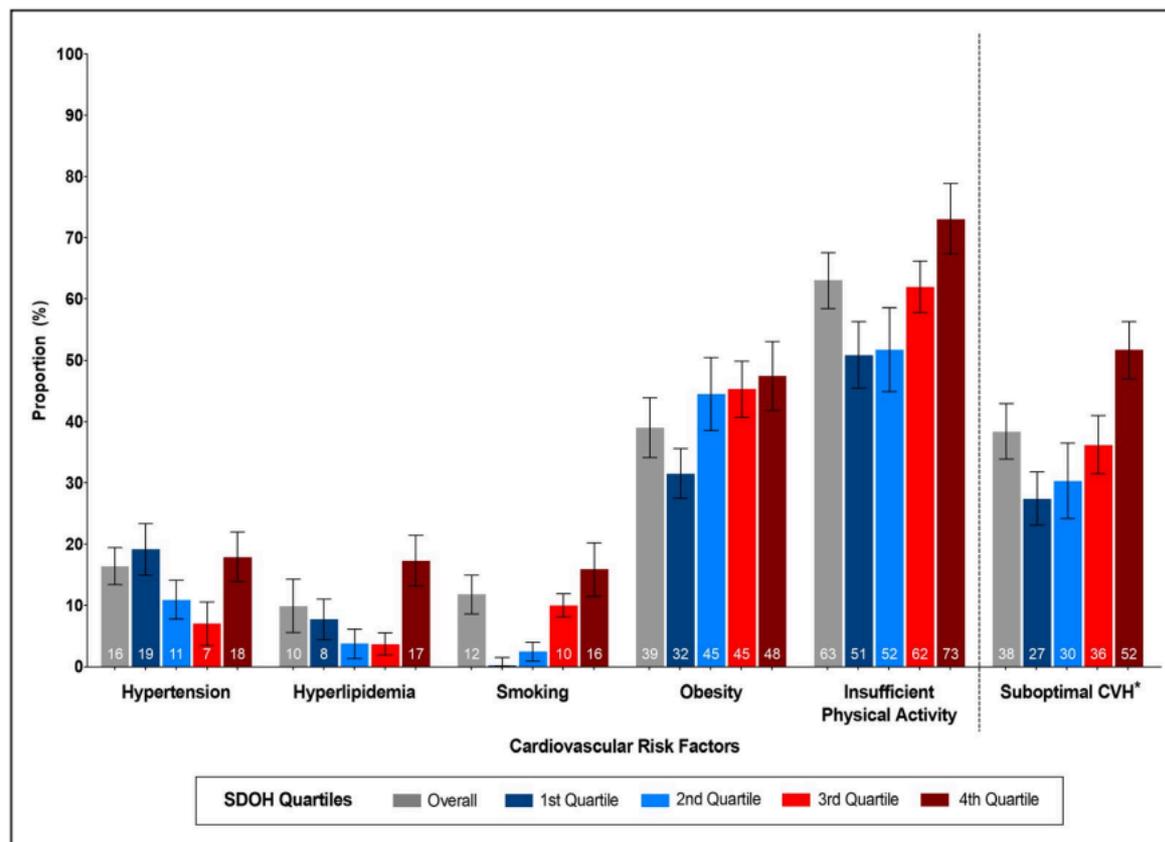


Figure 2. Age-adjusted prevalence of cardiovascular risk factors and suboptimal CVH among pregnant women in the United States, overall and by SDOH quartiles.

*Suboptimal CVH defined as ≥ 2 cardiovascular risk factors (hypertension, diabetes, hyperlipidemia, smoking, obesity, and insufficient physical activity). CVH indicates cardiovascular health; and SDOH, social determinants of health.

SDoH and Pre-eclampsia

FIGURE 2

A

Subgro

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[†]Odds Ratio

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FIGURE 2 Continued

B

Subgroup	Event Rate (per 100,000)	P interaction (Race×Income)	OR (95% CI)
All Patients	2436991		
Acute Coronary Syndrome			
Low Income			
Black	42	<0.01	1.45 (1.06 to 1.99)
Hispanic	7		0.41 (0.22 to 0.70)
White(Reference)	23		
High Income			
Black	55		2.45 (1.48 to 4.21)
White(Reference)	14		
Stroke			
Low Income			
Black	257	<0.01	1.92 (1.69 to 2.18)
Hispanic	198		1.43 (1.23 to 1.65)
White(Reference)	134		
Medium Income			
Black	351		1.69 (1.52 to 1.87)
Hispanic	235		1.46 (1.32 to 1.63)
Asian or Pacific Islander	191		1.51 (1.22 to 1.84)
White(Reference)	157		
High Income			
Black	258		1.67 (1.37 to 2.03)
White(Reference)	151		
Acute Kidney Injury			
Low Income			
Black	950	<0.01	2.06 (1.92 to 2.22)
Hispanic	567		1.37 (1.25 to 1.49)
Asian or Pacific Islander	906		1.96 (1.61 to 2.41)
White(Reference)	408		
Medium Income			
Black	946		2.14 (2.01 to 2.27)
Hispanic	540		1.21 (1.13 to 1.30)
Asian or Pacific Islander	784		1.53 (1.35 to 1.72)
White(Reference)	426		
High Income			
Black	991		2.29 (2.06 to 2.55)
Hispanic	495		1.14 (1.01 to 1.29)
Asian or Pacific Islander	874		1.72 (1.52 to 1.94)
White(Reference)	453		
Heart Failure			
Medium Income			
Hispanic	279	<0.01	1.77 (1.33 to 2.34)
White(Reference)	266		
High Income			
Black	464		0.48 (0.34 to 0.69)
White(Reference)	223		

% CI)

.59 to 2.91)

.22 to 2.41)

.42 to 3.87)

.21 to 2.09)

.11 to 2.98)

.65 to 4.49)

.01 to 2.88)

.02 to 5.11)

.22 to 1.52)

.10 to 2.15)

.10 to 1.36)

.75 to 0.96)

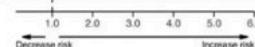
.59 to 0.95)

.52 to 0.89)

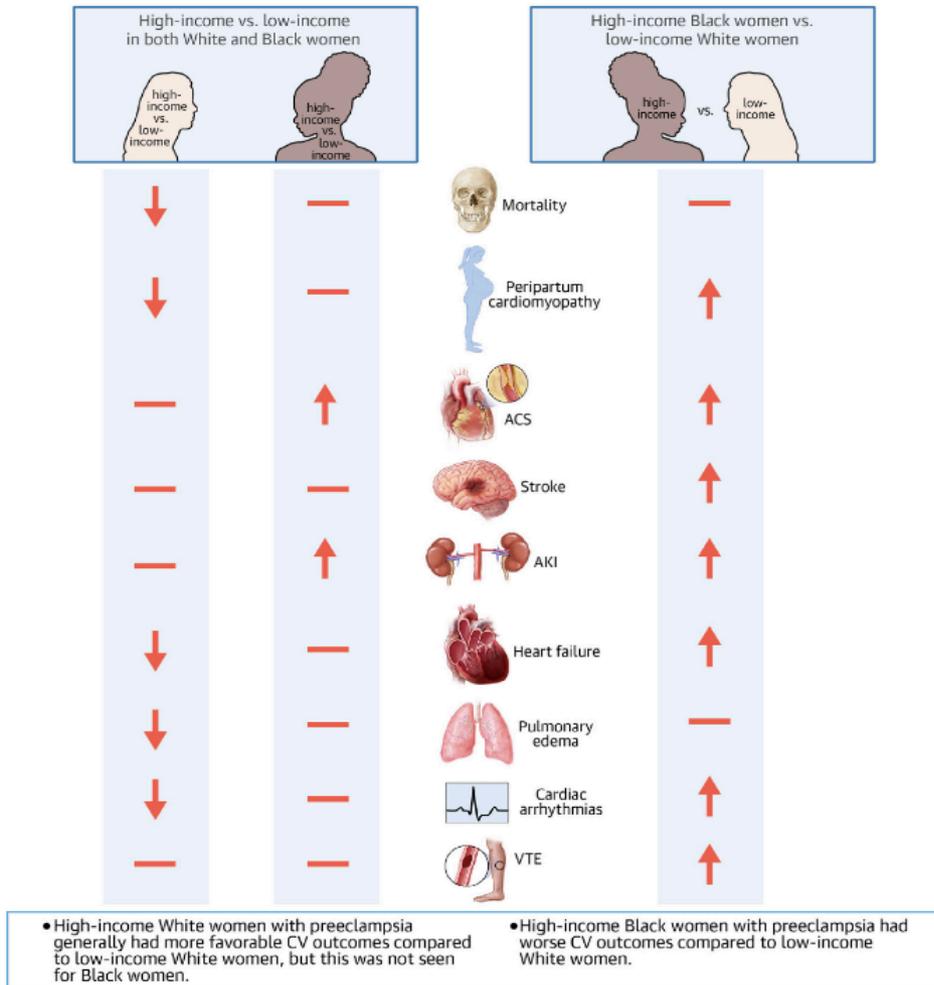
ome, stroke,

mbolism.

[†]Odds Ratio (OR) 95% Confidence Interval (CI) based on logistic regression model adjusted for: age, region, hypertension, diabetes, hyperlipidemia, heart failure, chronic kidney disease, coronary artery disease, obesity, smoking, multiple gestation and cesarean delivery
All event rates are per 100,000 preeclampsia hospitalizations during delivery



CENTRAL ILLUSTRATION: Racial and Socioeconomic Disparities in Cardiovascular Outcomes of Preeclampsia Hospitalizations in the United States 2004-2019: A Propensity-Matched Analysis



Zahid S, et al. *JACC Adv.* 2022;1(3):100062.

WEB OF CAUSATION

STRUCTURAL and SOCIAL DETERMINANTS: IMPACT ON HEALTH

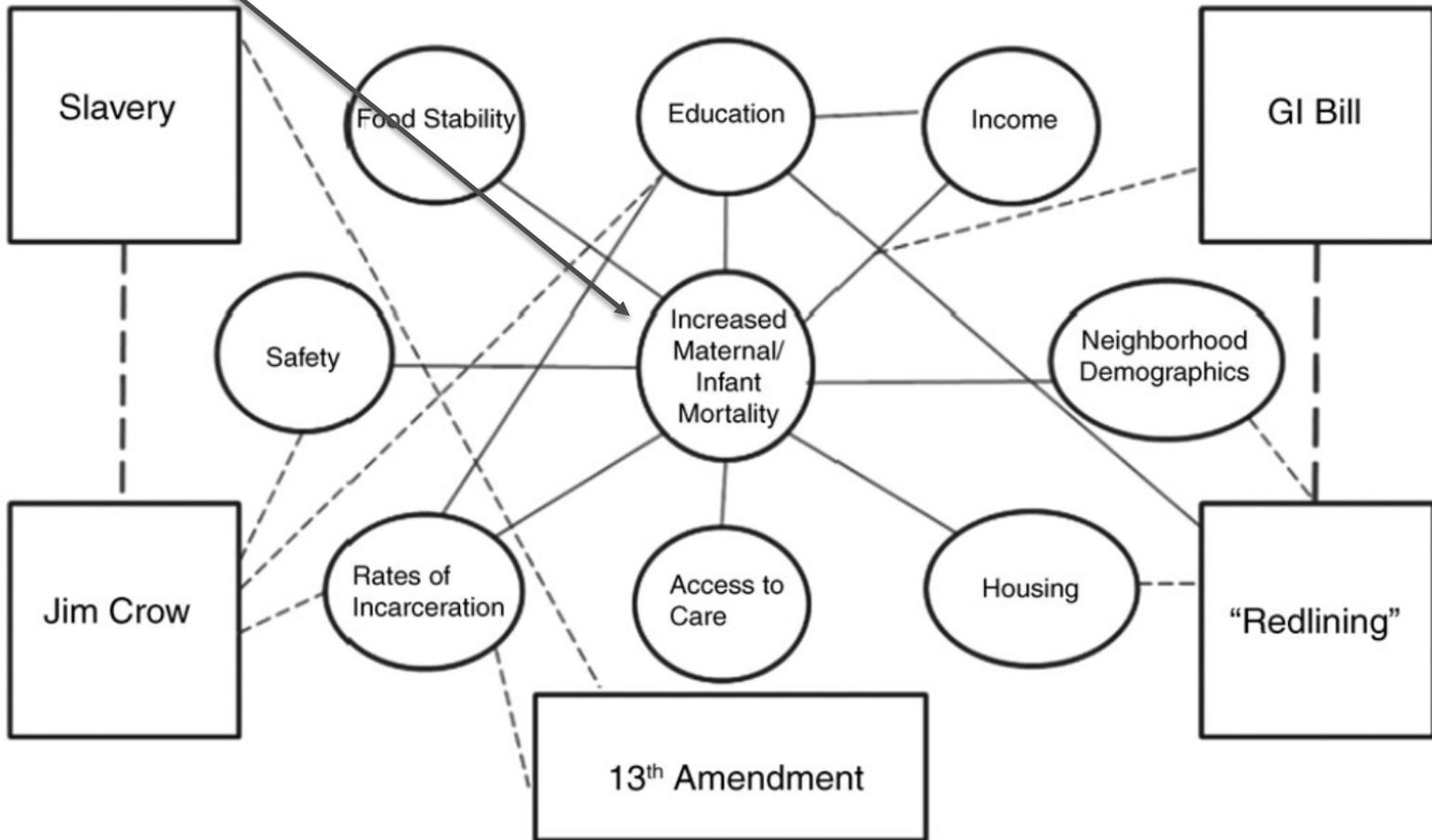
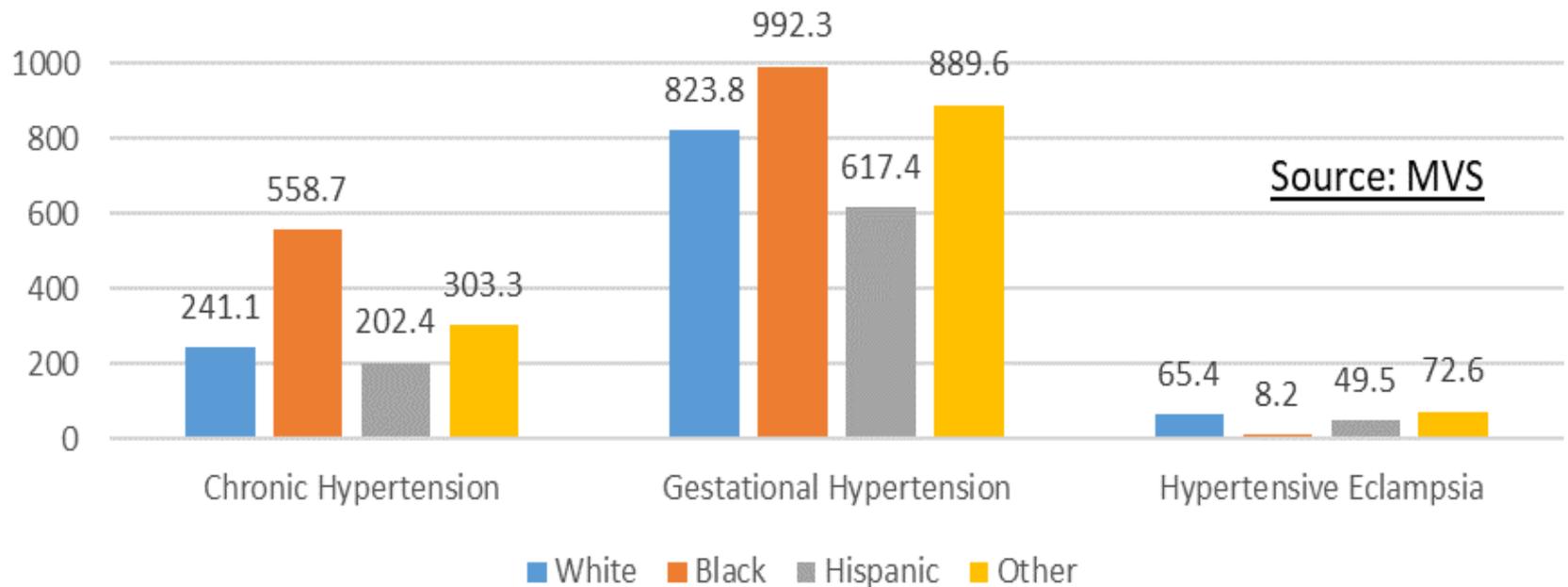


FIG. 1. ROOTT Theoretical Framework.¹⁵ This figure depicts the theoretical framework developed by ROOTT¹⁵ used to identify structural and social determinants of maternal and infant mortality in the United States. Structural determinants are those depicted in *boxes* connected by *dashed lines*, which in turn shape the distribution of social determinants (those depicted in *circles* and connected by *solid lines*). The multiple and interconnected pathways between structural and social determinants lead to increased maternal and infant mortality rates and socially defined inequities in these outcomes. ROOTT, Restoring Our Own Through Transformation.

Missouri Data on HDP and Disparities

**Figure 5: Hypertension During Pregnancy by Race
Ratio per 10,000 2017-2019**



Disparities in Missouri

- 1 in 4 mothers did not start prenatal care until after first trimester
 - 40% of Black mothers
- Higher rates of obesity in Missouri (25.7%)
 - 34.6% for Black mothers
- Black mothers make up 15.3% of all live births, but 29.3% of all SMM
- Less paid maternity leave (30.3% for White mothers, 19.6% for Black mothers)

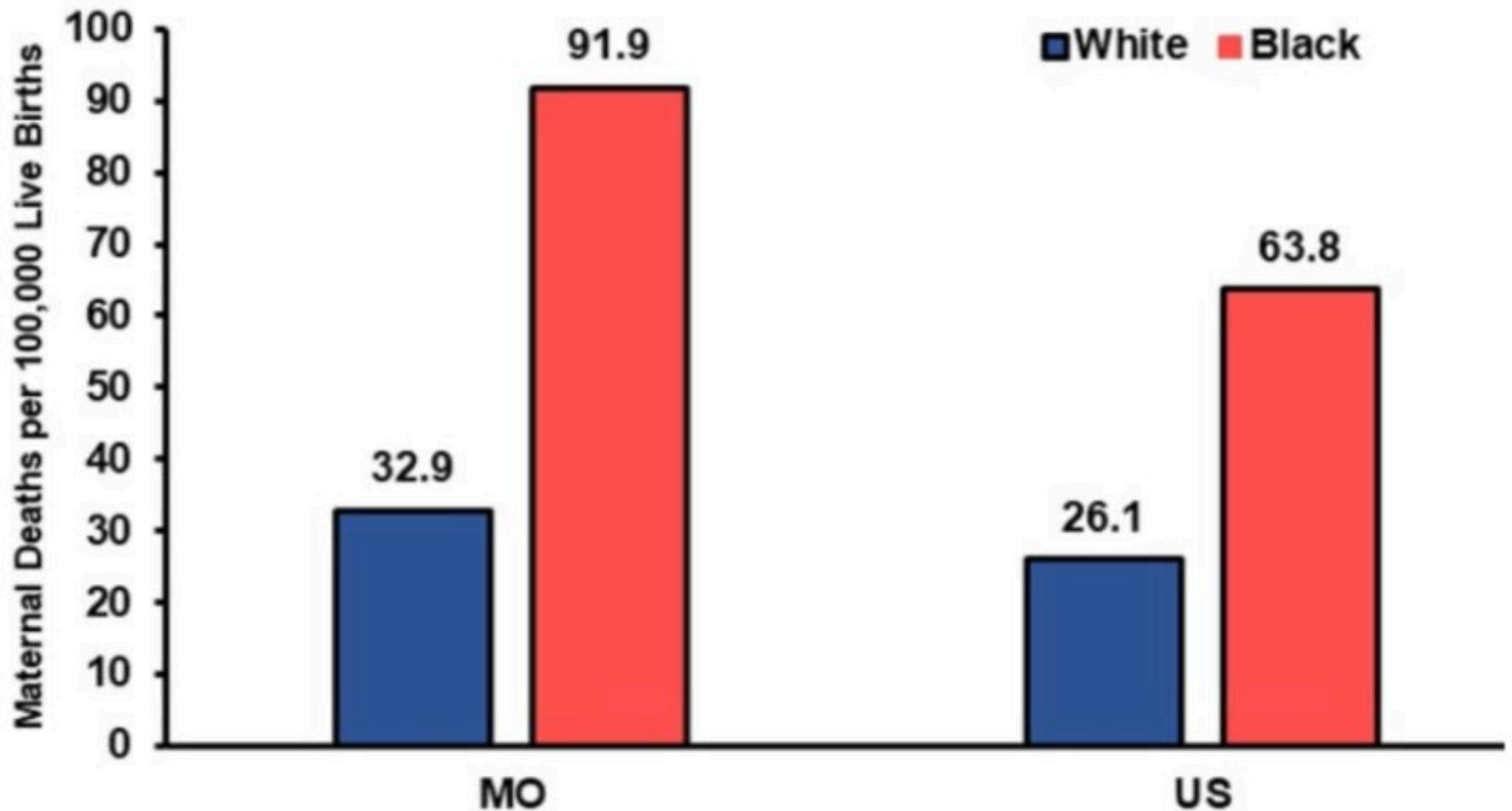
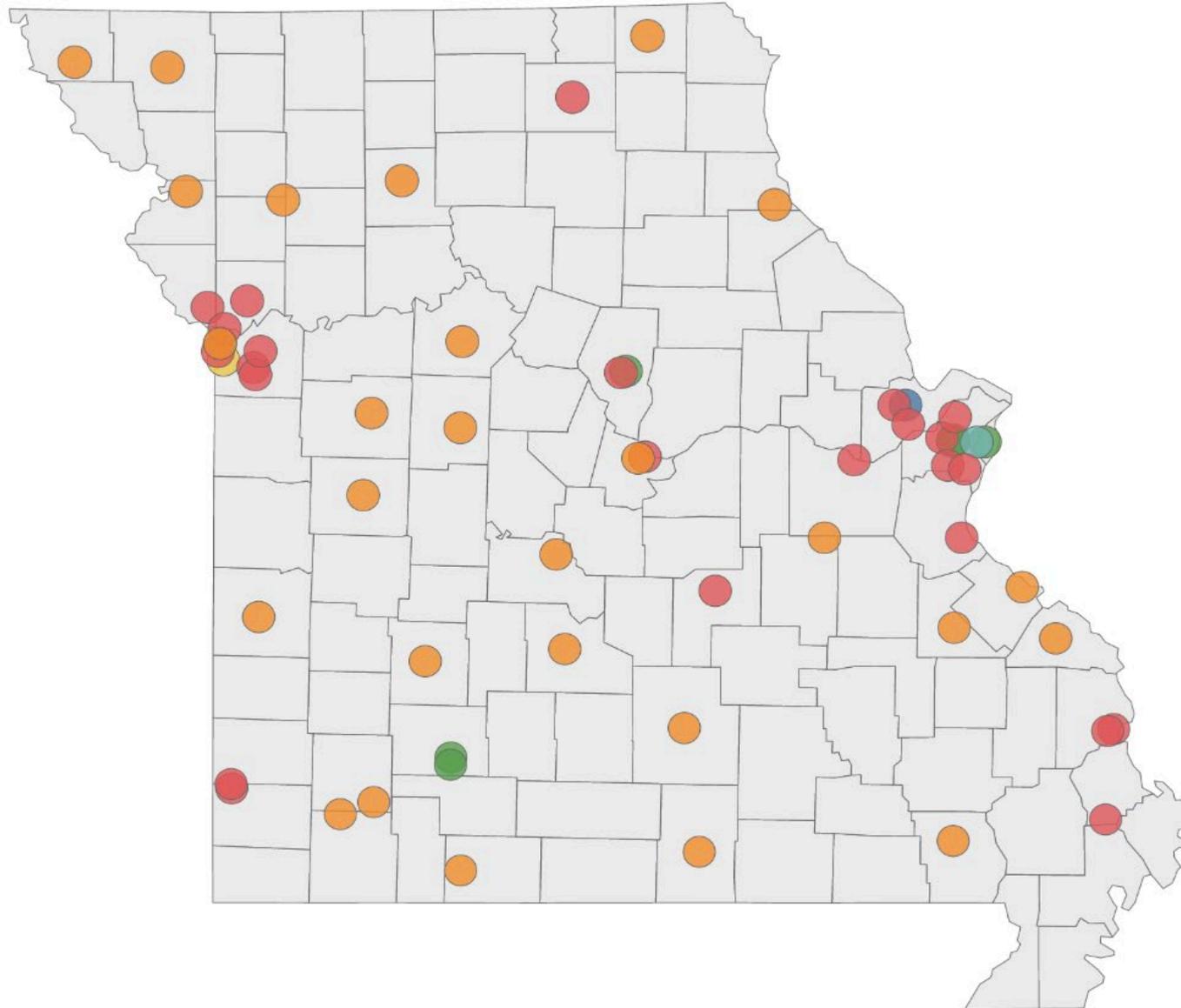


Figure 2. Maternal mortality rates by race for Missouri and the United States. Adopted from Academy Health Rankings.⁴

Maternal Level of Care



Select a Map

Maternal Care

Miles of Radius

5



- Level 1
- Level 2
- Level 3
- Level 4
- Birth Center
- Pending

Hold CTRL to select multiple levels

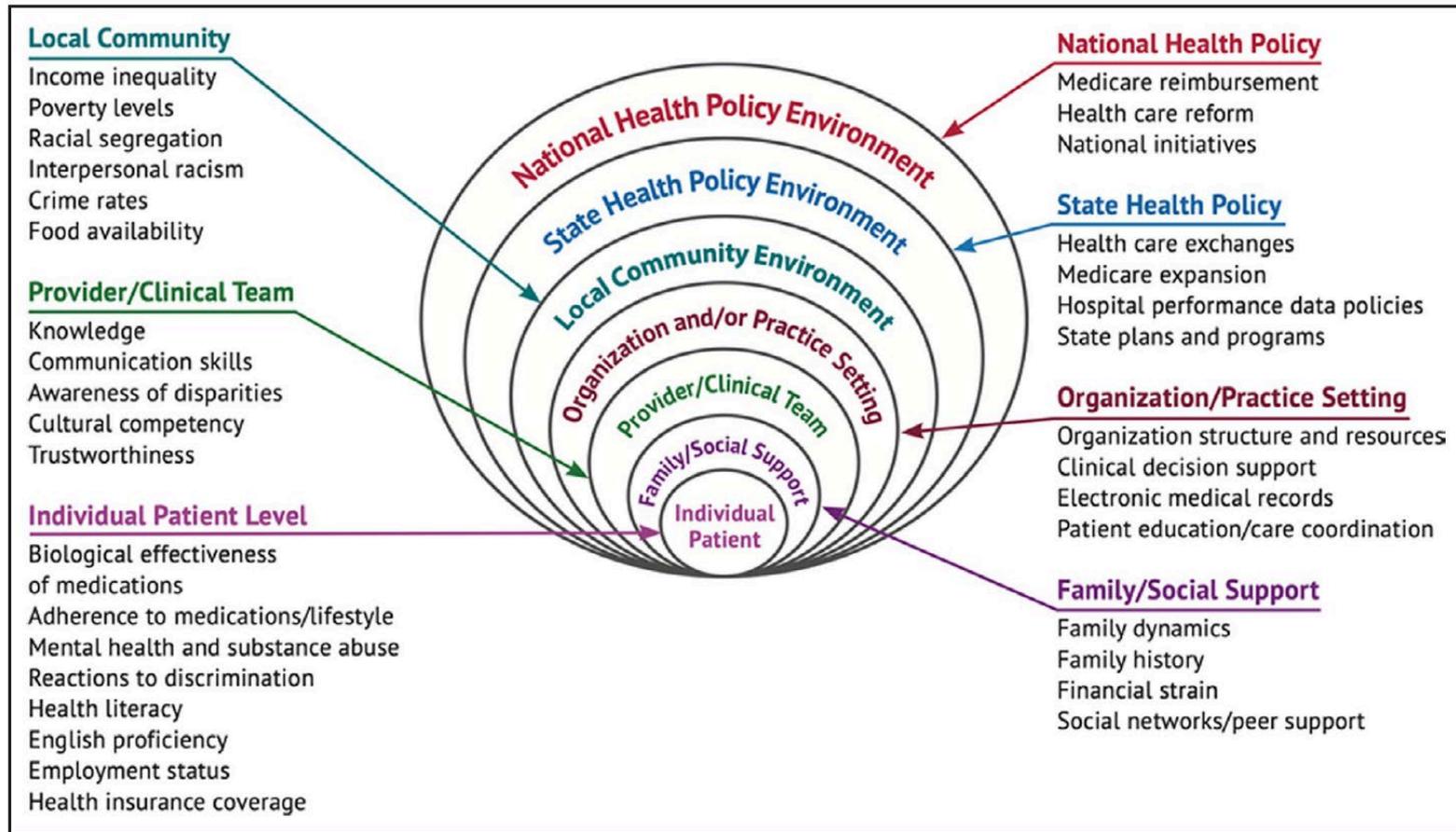
Select For More Information

Maternal LOC

Neonatal LOC

Hospital Designations

Reasons for Inequities in HTN Control





**So how do we reduce disparities
to improve outcomes for birthing
people with HDP??**

Validity of Home Blood Pressure Monitoring

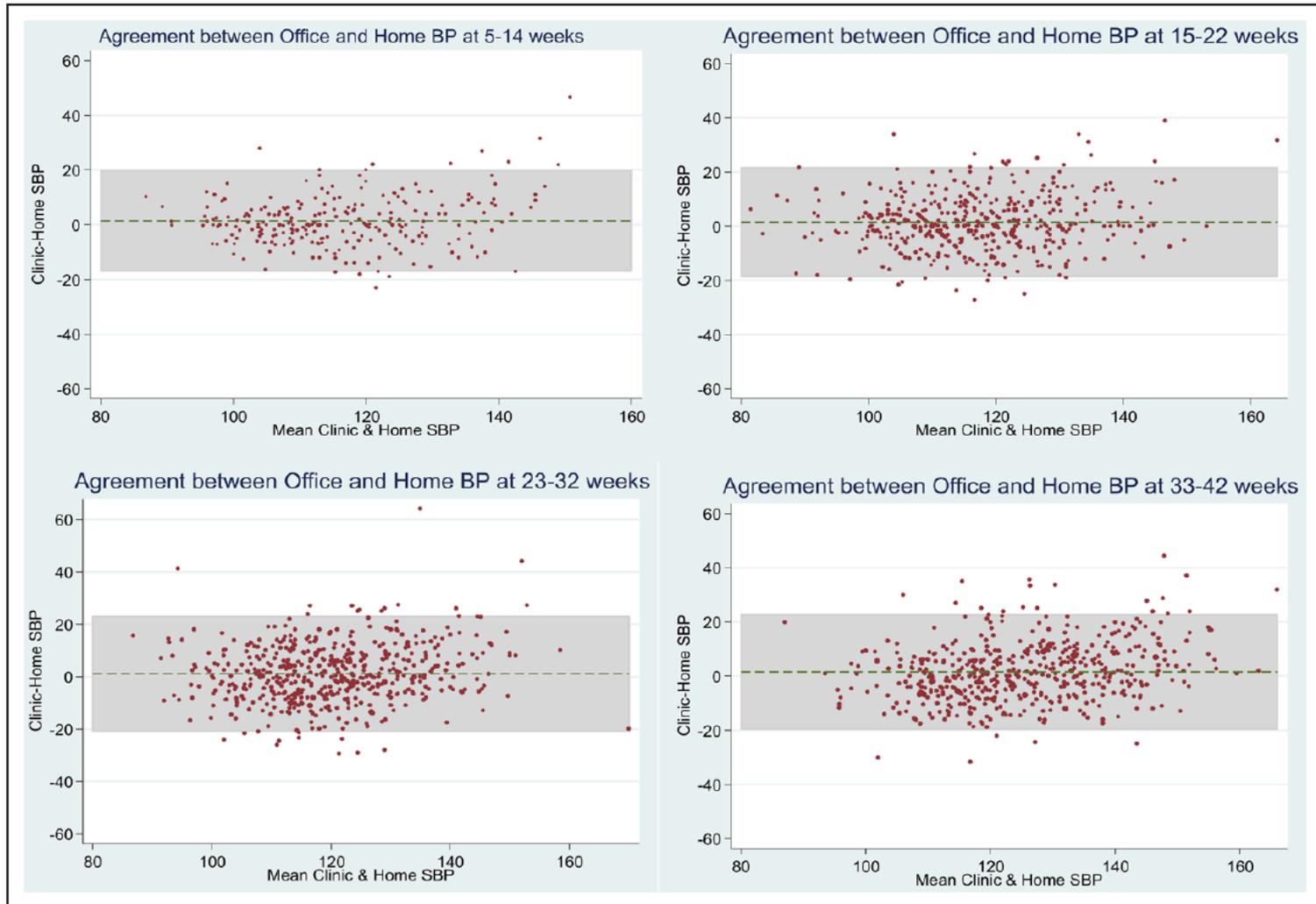


Figure 3. Agreement between clinic and self-monitored blood pressure (BP) readings during pregnancy. Bland-Altman plots were used to examine the influence of mean BP on the clinic-self difference. The mean clinic and self-monitored readings were plotted against clinic-self monitored readings (complete cases). At 5 to 14 wk, there was a mean difference of 1.403, 6.8% (17 of 250) readings were outside limits of agreement, and 95% limits of agreement were -16.943, 19.750. At 15 to 22 wk, a mean difference of 1.550 was observed, 6.26% (27 of 431) readings were outside limits of agreement, and the 95% limits of agreement were -18.576, 21.677. At 23 to 32 wk gestation, there was a mean difference of 1.067, 4.82% (25 of 519) readings were outside limits of agreement, and the 95% limits of agreement were -20.736, 22.871. At 33 to 42 wk gestation, there was a mean difference of 1.494, 4.66% (22 of 472) readings were outside limits of agreement, and 95% limits of agreement were -19.429, 22.417. Diastolic plots are shown in Figure S6.

Use in the Antepartum Period

BUMP 1 and 2 RCT Trials

- Does not necessarily lead to earlier diagnosis of PEC
- Does not necessarily lead to better maternal or fetal outcomes
- Showed equivalency in detection of HTN

Table 2. Primary Outcome: Mean Blood Pressure for Participants With Chronic Hypertension and Gestational Hypertension

	Self-monitoring	Usual care	Adjusted mean difference (95% CI)	P value
Chronic hypertension				
Primary outcome available, No. (%) ^a	229 (98.3)	215 (97.3)		
Blood pressure, mean (SD), mm Hg				
Systolic ^b	133.8 (10.3)	133.6 (11.1)	0.03 (-1.73 to 1.79) ^c	.97
Diastolic	84.0 (7.4)	84.3 (7.9)	-0.03 (-1.28 to 1.22)	.96
Gestational hypertension				
Primary outcome available, No. (%) ^a	187 (94.9)	190 (95.5)		
Blood pressure, mean (SD), mm Hg				
Systolic	137.6 (12.1)	137.2 (10.8)	-0.03 (-2.29 to 2.24) ^d	.98
Diastolic	86.1 (7.8)	86.3 (7.7)	-0.35 (-1.77 to 1.06)	.63

^a Statistical comparisons completed when >2% event rate for self-monitoring vs usual care. Log-Poisson generalized linear mixed-effects model with robust standard errors adjusted for randomized group and parity as fixed effects; and site as a random effect. Level of significance $P < .05$.

^c One or more of the following: eclampsia, transient ischemic attack or stroke,

involvement (creatinine ≥ 50 $\mu\text{mol/L}$), and hematological improvement (platelets $< \times 100^9/\text{L}$).

^d Estimated median difference (95% CI) derived from quantile regression adjusted for randomized arm, parity, and site.

Use in the Antepartum Period

Table 1 Demographic characteristics at inclusion and diagnoses in hypertensive pregnant women using home blood-pressure monitoring (HBPM) and in hypertensive controls managed according to local protocol

Table 2 Hospital care and monitoring required per patient in hypertensive pregnant women using home blood-pressure monitoring (HBPM)

Table 4 Pregnancy outcome and adverse maternal, fetal and neonatal events for hypertensive pregnant women using home blood-pressure monitoring (HBPM) and hypertensive controls managed according to local protocol

<i>Parameter</i>	<i>HBPM (n = 108)</i>	<i>Controls (n = 58)</i>	<i>P*</i>
GA at delivery (weeks)	39.0 (37.6–40.3)	39.3 (38.0–40.6)	0.395
Birth weight (g)	3211.0 (2693.8–3595.0)	3100.0 (2846.3–3550.0)	0.730
Neonatal unit admission	12 (11.1)	11 (19.0)	0.163
Steroid administration	11 (10.2)	4 (6.9)	0.481
Magnesium sulfate administration	3 (2.8)	5 (8.6)	0.094
Adverse maternal outcome†	1 (0.9)	2 (3.4)	0.245
Adverse fetal outcome†	27 (25.0)	14 (24.1)	0.902
Adverse neonatal outcome†	6 (5.6)	3 (5.2)	0.979

Data are given as median (interquartile range) or *n* (%). *Comparisons between study groups by chi-square and Fisher's exact test for categorical variables and Mann–Whitney *U*-test for continuous variables. †Adverse outcomes defined in main text. GA, gestational age. Data are given as median (interquartile range). Comparisons between HBPM and control group by Mann–Whitney *U*-test. DAD, day assessment unit.

general practitioner and out-of-hours triage. BP, blood pressure; GA, gestational age; HDU, high-dependency unit; PE, pre-eclampsia.

Data are given as median (interquartile range) or *n* (%). *Comparisons between study groups by chi-square and Fisher's exact test for categorical variables and Mann–Whitney *U*-test for continuous variables. †At commencement of blood-pressure monitoring.

Use in the Postpartum Period

TABLE

Outpatient outcomes in telehealth vs standard outpatient care participants

	Telehealth (<i>n</i> =214)		Standard outpatient care (<i>n</i> =214)		<i>P</i> value	RR (95% CI)	Adjusted <i>P</i> value	Adjusted RR (95% CI)
Healthcare utilization through 6 wk								
Hypertension-related hospital readmissions ^a , <i>n</i> (%)	1	(0.5)	8	(3.7)	.037	0.13 (0.02–0.99)	.045	0.12 (0.01–0.96)
Hypertension-related emergency or triage room visits ^a , <i>n</i> (%)	11	(4.6)	13	(6.0)	.831	0.76 (0.38–1.85)	.808	0.81 (0.36–1.80)
Number of blood pressure reviews within 10 days of delivery ^a , <i>n</i> (%)	202	(94.4)	129	(60.3)	<.001	1.56 (1.39–1.76)	<.001	1.59 (1.36–1.77)
6 wk study endpoint								
Number of participants on antihypertensive treatment regimes ^a , <i>n</i> (%)	57	(26.6)	37	(17.3)	.027	1.54 (1.06–2.23)	.866	1.03 (0.74–1.44)

Data are expressed as mean, median (interquartile range), or *n* (%).

CI, confidence interval; RR, relative risk; SD, standard deviation.

^a Adjusted for the delivery mode, insurance status, antihypertension medication use at the time of hospital discharge, and the total number of postpartum admission days.

Hoppe. Telehealth with remote blood pressure monitoring for postpartum hypertension. *Am J Obstet Gynecol* 2020.

Compliance with SMBP

WHEN THE READINGS ARE RANKED AS "GREEN", THE SOFTWARE INDICATES THAT
« YOUR BLOOD PRESSURE IS WITHIN NORMAL RANGE ».
WHAT DO YOU THINK OF THE SOFTWARE'S ANALYSIS?

N = 82

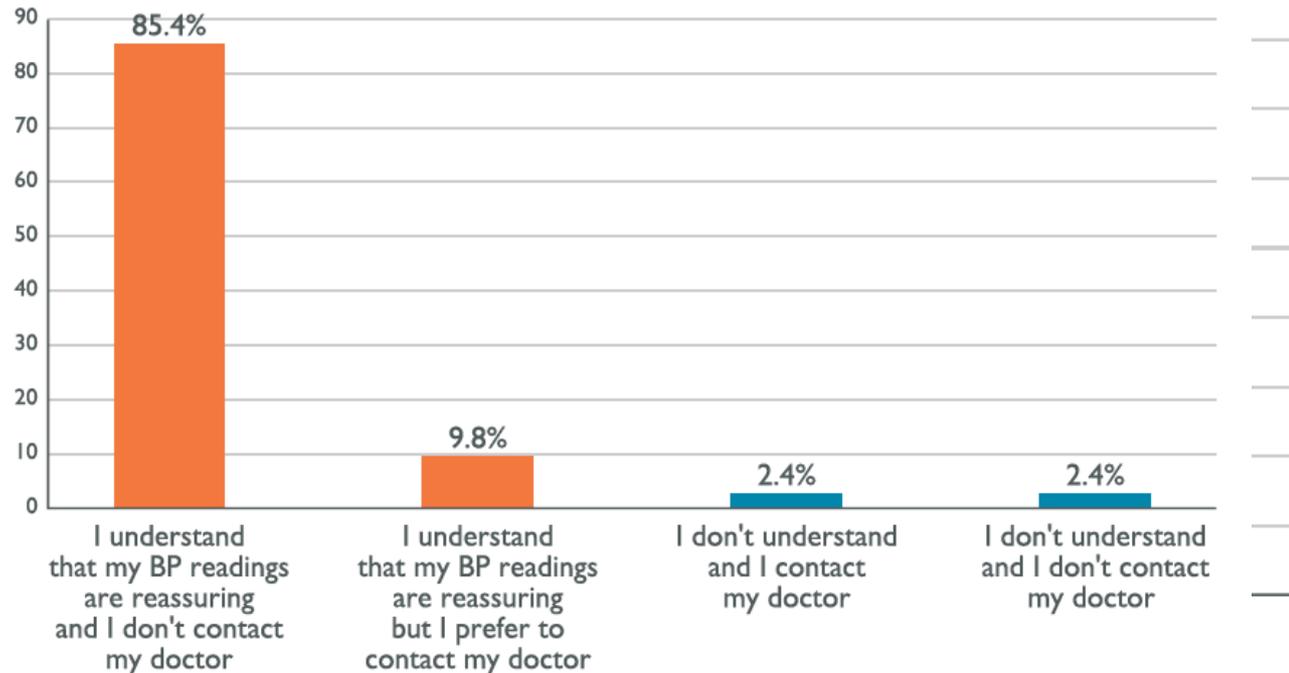


Figure 6 Participants' reaction toward green ranked readings.

Figure 5 Women's opinions regarding the Hy-result system's usefulness.

Patient Satisfaction with SMBP

Table 3 Univariate logistic regression results

Domain	Question	Variable	Odds Ratio (OR)	Standard Error (SE)	95% CI
Burden of care	To what extent do you prefer going to the hospital or clinic instead of using the mHealth technology at home?	No significant variables			
		Gestational hypertension	12	17.4	0.71–204
	How much would you recommend the mHealth technology to other women in your situation?	Preeclampsia without severe features	36*	54.7	1.86–701
		All other preeclampsia	24*	31	1.9–294
		Starting medication after discharge	4.1*	2.9	1.0–16.4
		Non-Hispanic White	7.6 ⁺	7.1	1.2–47.4
Satisfaction	How enjoyable are the mHealth devices to use?	All other preeclampsia	12.7*	13	1.7–94.2
		Chronic hypertension	8.8 ⁺	11	0.77–101
	Overall how satisfied are you with the mHealth devices?	All other preeclampsia	19*	26	1.3–283
		Maternal BMI	0.94*	0.03	0.88–0.99
		Infant discharging with mother	4.4 ⁺	3.48	0.90–21
		Starting medication after discharge	3.1 ⁺	1.9	0.94–10

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, + $p < 0.1$

Cost Effectiveness of SMBP

Table 3. Summary of reviewed studies.

Study	Time	Conditions	Intervention vs control	Cost-effectiveness
Ahmed et al ³⁰	Prenatal	HDP	Tight control vs less tight control of HDP	+
Barton et al ³¹	Prenatal	HDP	Outpatient monitoring vs antepartum hospitalization	+
Brooten et al ³²	Prenatal	HDP	Half of prenatal care in home by nurse specialists vs usual prenatal care	+
Buyse et al ³³	Prenatal	HDP	Telemonitoring vs in-hospitalized monitoring	+
Drost et al ³⁴	Postpartum	History of HDP	Annual hypertension screening in primary care vs usual care	+
Dunlop et al ³⁵	Prenatal	HDP	Day care management vs inpatient care	+
Harrison et al ³⁶	Prenatal	HDP	In-home care vs in-hospital antenatal care	-
Kim et al ³⁷	Postpartum	History of GDM	screening strategy for preventing T2DM	+
Kolu et al ³⁸	Prenatal	Risk factors for GDM including history of GDM	gestational lifestyle intervention during pregnancy vs routine care	-
Lagerweij et al ³⁹	Postpartum	History of HDP	Early preventive cardiovascular disease risk screening followed by risk-based lifestyle interventions vs no screening	-
Laanssens et al ⁴⁰	Prenatal	HDP	Remote monitoring vs conventional care	+
Mallampati et al ⁴¹	Prenatal	Risk factors for pre-eclampsia including history of HDP	Low-dose aspirin prophylaxis program vs routine care	+
Marseille et al ⁴²	Prenatal + postpartum	Risk factors for GDM including history of GDM and found GDM	Screening followed by antenatal care (diet and exercise counseling, glucose control medications and monitoring) and postpartum care (metformin or lifestyle management) vs routine care	+
Moss et al ⁴³	Prenatal	GDM	Treating mild GDM vs routine pregnancy care	+
Ohno et al ⁴⁴	Prenatal	GDM	Treating mild GDM vs routine pregnancy care	+
Poncet et al ⁴⁵	Prenatal	Risk factors for GDM including history of GDM	Screening high-risk women with 50 g OGTT vs screening all pregnant women with 50 g or 75 g OGTT	+
Simon et al ⁴⁶	Prenatal	HDP	Administration of magnesium sulfate vs placebo	+
Todorova-Ananieva ⁴⁷	Postpartum	History of GDM	Prophylactic program (advice of dietary regimen, reduction of body weight and lifestyle alteration) for preventing T2DM	+
van Baaren et al ⁴⁸	Postpartum	History of HDP	Preventive screening on cardiovascular risk factors followed by subsequent antihypertension medication vs no follow-up	+
Vijgen et al ⁴⁹	Prenatal	HDP	Induction of labor vs expectant monitoring	+
Werner et al ⁵⁰	Prenatal	Risk factors for pre-eclampsia including history of HDP	Low-dose aspirin prophylaxis program vs routine care	+
Xydopoulos et al ⁵¹	Prenatal	HDP	Home blood pressure monitoring vs traditional monitoring	+

Note. "+" means cost-effective and "-" means not cost-effective. GDM indicates gestational diabetes mellitus; HDP, hypertensive disorders of pregnancy; OGTT, oral glucose tolerance test; T2DM, type 2 diabetes mellitus.

Figure 1. Study framework

Early Life
Prior HDP/

Prena

GDM indicates gestational diabe

Later in Life

Long-term outcomes
Cardiovascular diseases
Hypertension
Diabetes
Stroke

Barriers to SMBP and Needed Policy Change

Process for SMBP implementation and multi-level barriers at each step

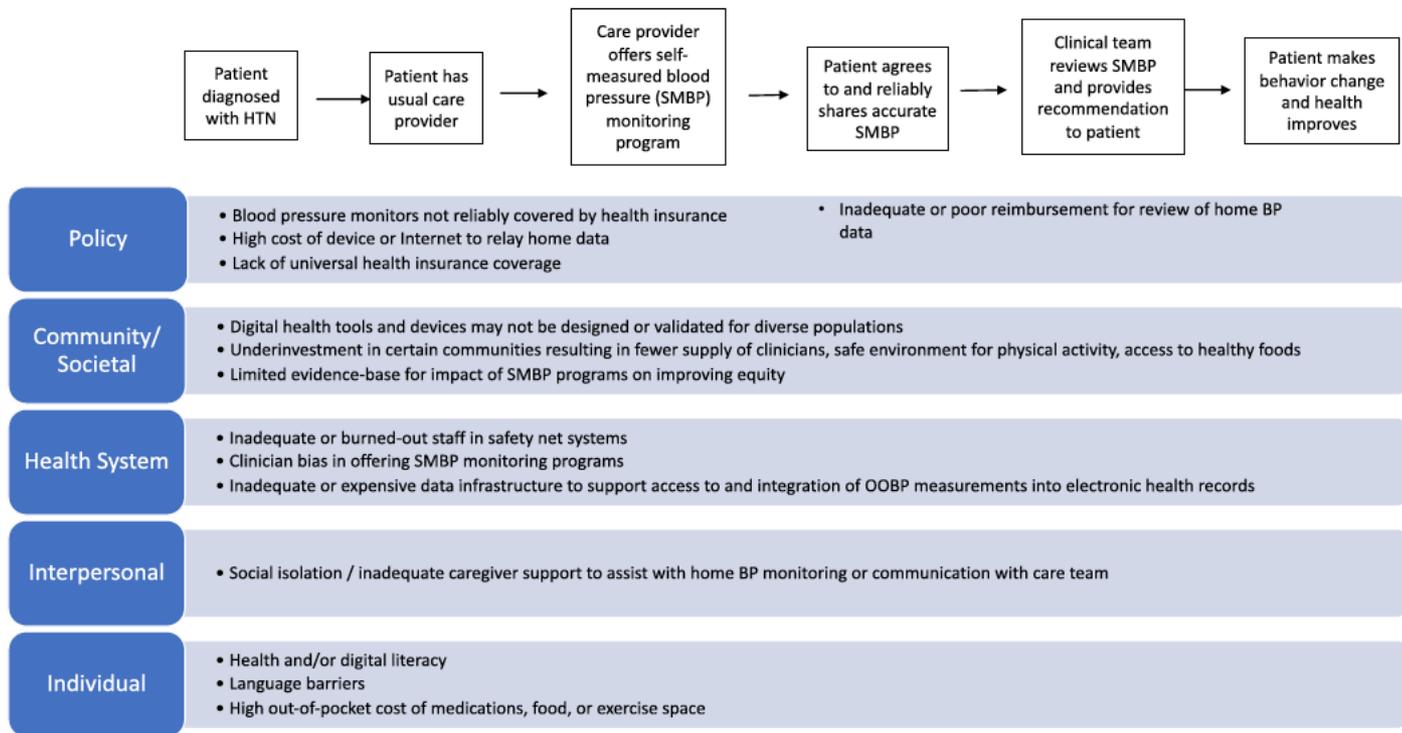


Fig. 1 Multi-level barriers to process for SMBP implementation

The Cuff Kit Project

- Grant #1 from the Missouri Foundation for Health
 - **Focus on equity, decreasing disparities and elevating the community voice**
 - **Must also partner with community organizations (not just hospitals)**
 - **Distributed ~3000 cuff kits to vulnerable, at-risk maternal populations**
- Grant #2 from MO DHSS COVID-19 Health Equity Funding
 - **Distributed ~4400 cuff kits to vulnerable, at-risk maternal populations**
 - **Research on efficacy underway**
- Partnership with the Preeclampsia Foundation
- Distribution of blood pressure kits to postpartum birthing people with pre-eclampsia



The Cuff Kit Project

This program is supported by the Centers for Disease Control and Prevention of the U.S. Department of Health and Human Services (HHS) as part of a financial assistance award totaling \$35,569,951 with 100 percent funded by CDC/HHS. The contents are those of the author(s) and do not necessarily represent the official views of, nor an endorsement, by CDC/HHS, or the U.S. Government. The program has received a portion of this funding from the Department of Health and Senior Services, Office of Rural Health and Primary Care to expand efforts to address health disparities caused by COVID-19

Future Collaborative Work

- Roll out of Cardiovascular bundle in 2024
- Ask Me, Hear Me Campaign



MISSOURI DEPARTMENT OF
**HEALTH &
SENIOR SERVICES**

Pregnancy-Associated Mortality Review



Summary

- Pre-eclampsia is a dynamic, multisystem disorder with gross disparities amongst racial groups that requires prompt recognition and management to improve outcomes
- Missouri has one of the worst disparity ratios for APO secondary to HDP and it will take a multipronged approach to improve outcomes, including the use of home BP monitoring
- The Cuff Kit Project has the potential to diminish disparities, improve outcomes and reduce cost

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