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Pre-pregnancy stress reactivity and pregnancy outcome

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Summary

Stress has been proposed as a cause of preterm birth (PTB) and small for gestational age (SGA), but stress does not have the same effects on all women. It may be that a woman's reaction to stress relates to her pregnancy health, and previous studies indicate higher reactivity is associated with reduced birthweight and gestational age. The objective of the study was to examine the relationship between pre-pregnancy cardiovascular reactivity to stress and pregnancy outcome. The sample included 917 women in the Coronary Artery Risk Development in Young Adults (CARDIA) Study who had cardiovascular reactivity measured in 1987–1988 and at least one subsequent singleton live birth within an 18-year period. Cardiovascular reactivity was measured using a video game, star tracing, and cold pressor test. Gestational age and birthweight were based on the women's self-report, with PTB defined as birth <37 weeks' gestation and SGA as weight <10th percentile for gestational age. Linear and poisson regression and generalised estimating equations were used to model the relationship between reactivity to stress and birth outcomes with control for confounders. Few associations were seen between reactivity and pregnancy outcomes. Higher pre-pregnancy diastolic blood pressure (adjusted relative risk, 1.14, 95% confidence interval 0.98–1.34) and mean arterial pressure (MAP) reactivity (1.15, 0.98–1.36) were associated with risk of PTB at first pregnancy, while SGA was associated with lower SBP reactivity (0.76, 0.60–0.95). No associations were seen with other measures of reactivity. Contrary to hypothesis, the association between heart rate reactivity and preterm birth in first pregnancy was stronger in whites (aRRs 1.39, 1.03–1.88) than in blacks (1.00, 0.83–1.20; *p* for interaction=0.08). Similar results were found for mean arterial pressure. No strong associations were found between higher pre-pregnancy stress reactivity and SGA or PTB, and stress reactivity did not have a stronger association with birth outcomes in blacks than whites.

Introduction

Preterm birth (PTB) and fetal growth retardation, indicated by low birthweight or small-for-gestational-age (SGA) are major causes of infant morbidity and mortality in this country ^{1,2}. Many studies indicate that higher stress is associated with PTB and SGA,³ but the reported associations have often not been so consistent, with associations often found in only subgroups, using some scales, or due to associations with smoking.^{4–6} Higher levels of

stress have been suggested as a reason for the large disparity in PTB and SGA between black women and women of other races.^{7, 8}

Individuals vary in their physical responses to stress.⁹ Heightened cardiovascular reactivity in response to stressors (i.e. large changes in blood pressure, heart rate, relative to resting levels) has been associated with later development of hypertension^{10–12} and stroke.¹³ In CARDIA, blood pressure reactivity to stressors predicted higher blood pressure and hypertension over 13 years of follow-up.^{14–16} Hyperreactivity was a stronger predictor of risk in blacks than whites, and this racial variation is hypothesised to contribute to disparities in hypertension.^{16, 17} High blood pressure and vascular compromise in pregnancy are major contributors to maternal and neonatal morbidity, including PTB and fetal growth restriction.^{18–20} Three previous studies indicate that increased cardiovascular stress reactivity is also associated with lower birthweight and/or reduced gestational age,^{21, 22} at least in black women.²³ These studies are fairly small, conducted in low-risk populations with minimal ability to assess racial or other differences, and had limited power to control for confounding.

Cardiovascular reactivity is known to be reduced during pregnancy⁹, and pre-pregnancy reactivity may provide a better general picture of a woman's propensity to react and overreact physiologically to stress. Stress reactivity is fairly stable across long time periods.²⁴ In this analysis, we examine the relationship between cardiovascular stress reactivity, PTB, and SGA in participants in a large, prospective cohort study of cardiovascular risk in women of reproductive age. First, we hypothesised that women who reacted more strongly to stress before pregnancy would be more likely to give birth to preterm and SGA infants. Second, given that previous work has indicated stronger reactivity in blacks than whites,^{17, 25, 26} we hypothesized this relationship would explain some of the difference in risk between blacks and whites. Finally, as previous studies have indicated that reactivity is a stronger predictor of both birth outcomes and hypertension in blacks,^{16, 23} we examined the interaction between race and reactivity in predicting birth outcomes.

Materials and methods

The CARDIA Study is a multi-centre, longitudinal, observational study designed to describe the development of risk factors for coronary heart disease in young black and white men and women. In 1985–86, 2,787 women (53% black) aged 18–36 were enrolled at baseline from four geographic areas: Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California.^{27, 28} Participants were re-examined 2, 5, 7, 10, 15 and 20 years after baseline. Among women, retention at 5 years was 84%, at 10 years was 79%, and at 20 years was 72%. Retention was better in older participants than younger; in whites than blacks (88% vs. 81% at year 5) and in those with more education (89% in those with baseline college education or more and 79% in those with less than a high school diploma at exam 5). Blood pressure measurements and a variety of clinical, laboratory and lifestyle measures were obtained at baseline and follow-up exams.^{27, 28}

Blood pressure reactivity was measured at the second exam (1987–1988). The protocol for the reactivity testing has also been described in detail previously.^{15, 29} The testing involved three different tasks: a video game, a mirror image star-tracing task, and a cold pressor task (hand immersion technique). An 8-minute habituation/pre-intervention period was followed by presentation of the video game and star-tracing task in randomised order for 3 minutes each. The cold pressor task, consisting of approximately 45 to 60 seconds of hand immersion in an ice water tank, followed the other two tasks. Only one blood pressure reading was taken during the cold pressor task (initiated after 30 seconds of hand immersion). Blood pressures for the habituation/pre-intervention period, video game, and

the star-tracing task were recorded with an automated blood pressure monitor (2600B Vita-Stat, Spacelabs Medical Inc., Redmond, WA). To minimise the duration of participants' hand immersion, blood pressures during the cold pressor task were recorded manually with a mercury sphygmomanometer.³⁰

2407 women in the study had reactivity measures taken. Women were excluded from this analysis who had hormonal or vascular conditions likely to make their reactivity measurements unrepresentative: women who were pregnant (n=79), breastfeeding (n=57), or were taking blood pressure medications (n=66) at the time of reactivity measures were excluded (there was some overlap among these groups). 1310 of the remaining sample had data on at least one pregnancy during the 20-year follow-up; 1029 had at least one livebirth, and 1008 at least one singleton livebirth. (The etiology of LBW/PTB for multiple births likely differs from singletons and so they were excluded.³¹) 917 women had data on at least one singleton livebirth that took place after the reactivity testing; this corresponded to 1503 total singleton livebirths. 3 women were missing data on gestational age at the first pregnancy and 6 pregnancies were missing data on gestational age. 99 women were missing data on birthweight at first pregnancy and 192 pregnancies were missing data on birthweight. At the 10-year interview, it appears that some women were asked about gestational age but not birthweight (71 women). Time between reactivity measure and first subsequent pregnancy was a mean of 5.2 years, median 4.2 years, range 0.5–17.6 years. Reactivity was not significantly different between dropouts and those who remained in the cohort.

Reactivity was defined as the mean of the three measures taken during the task minus the mean of the pre-intervention measures. Measurements were removed if the diastolic blood pressure (DBP) was implausibly low ($DBP \leq 40$), n=11. Reactivity measures (systolic blood pressure (SBP), DBP, mean arterial pressure ($MAP = DBP + 1/3 (SBP - DBP)$), and heart rate (HR)) were initially examined separately for each task; in later analyses, the video game and star tracing tasks were standardised and averaged for a summary measure. Cold pressor was not included in the summary measure due to the larger amount of missing data and the fact that it yields a different hemodynamic pattern than do the video game and star tracing tasks.

Pregnancy outcome was based on the woman's self-report for each pregnancy. At each follow-up exam, the woman was asked whether she had been pregnant since the previous exam; how the pregnancy had ended; the baby's birthweight; and length of gestation. The outcomes examined were preterm birth (PTB), gestational age at birth <37 weeks, and small-for-gestational-age (SGA), birthweight <10th percentile for gestational age,³² as well as gestational age and birthweight as continuous variables.

T-tests were used to examine the differences in reactivity between those with and without the outcomes. Poisson modeling was used to model relative risk³³ with reactivity measures as continuous measures; median, quartile, and quadratic relationships were also examined, and it was concluded that continuous measures provided an adequate representation of the relationship. Linear and poisson regression models were fit to predict outcome of the first pregnancy (for simplicity and to focus on "incident" outcomes among women with multiple poor outcomes). Generalised estimating equations with an exchangeable correlation matrix were used to model all pregnancies (in order to allow use of all data).

Multivariable models were used to examine confounding. Age at pregnancy, centre, race, education, smoking, parity, use of high blood pressure medication (after visit 2), and body mass index (BMI) were first examined as potential confounders. Centre was considered a possible marker of underlying population or environmental differences that were not captured by other demographic markers. Since for most pregnancies there was not an

interview question that specifically addressed smoking or BMI during the pregnancy, smoking and BMI at the interview prior to the reported pregnancy was the measure used. Any variable that changed the effect estimate for a corresponding outcome at 10% was retained for final models; this was race and centre for PTB, and race, smoking, pre-pregnancy BMI, and blood pressure medication for SGA. Interactions with race were examined using product terms. In addition, we examined the relationship between race and birth outcomes, adjusted for reactivity. If stress reactivity were a major contributor to the race disparity, we would expect the size of the association to be reduced after adjustment.³⁴ Data were also examined limiting to women who reported first pregnancies within 1 (n=64), 2 (n=203), and 5 (n=529) years of reactivity testing; due to the small numbers, continuous gestational age and birthweight were used for these analysis, with adjustment for the same confounders as the PTB and SGA analyses, respectively.

Institutional Review Boards at each participating study centre approved the study. Written, informed consent was obtained from subjects for all study procedures.

Results

Women in this analysis were 20–33 years of age at the stress reactivity visit (table 1). Just less than half were black, and approximately one-quarter were smokers. 21% of women reported at least one preterm birth (13% of pregnancies). Overall reactivity data are provided in table 2. Overall reactivity was higher in older women ($p<0.01$; data not shown). DBP and HR reactivity were higher in blacks ($p<0.01$). SBP and HR reactivity was lower in smokers ($p<0.05$), while cold pressor reactivity was higher in smokers ($p<0.01$).

Few differences in reactivity were seen based on outcome of first pregnancy. Women whose first birth was preterm had higher diastolic blood pressure reactivity and mean arterial pressure reactivity after the video game, and women who gave birth SGA had lower SBP star tracing reactivity. Results were similar when blood pressure measurements were standardised and averaged (table 3). After adjustment, the association between heart rate reactivity and PTB in first pregnancy was stronger in whites (aRRs 1.39, 1.03–1.88) than in blacks (1.00, 0.83–1.20; p for interaction=0.08; table 3); the same was true for mean arterial pressure (aRR 1.35, 1.08–1.68 in whites; 1.04, 0.86–1.26 in blacks; p for interaction=0.14). The association between SBP reactivity and SGA was also higher in whites (1.01, 0.66–1.56) than blacks (0.73, 0.55–0.96). PTB and SGA were also analyzed across all pregnancies using GEE; results were similar with a slightly increased risk of PTB associated with HR reactivity and a reduction of SGA associated with SBP reactivity (supplementary table 1). Results were also similar using gestational age and birthweight as continuous outcomes (supplementary table 2).

The adjusted relative risk for black race and PTB was 2.20 (95% CI 1.57–3.07), while for SGA it was 3.12 (95% CI 1.95–4.98). After adjustment for reactivity variables, the RRs for PTB ranged from 2.17 (95% CI 1.55, 3.03) for average MAP reactivity to 2.57 (95% CI 1.78, 3.70) for cold pressor SBP, and for SGA ranged from 2.74 (1.65, 4.56) for cold pressor DBP reactivity to 3.19 (2.00, 5.08) for average MAP reactivity.

One standard unit of DBP reactivity was associated with an adjusted 135.1g (−7.1, 277.4; $p=0.06$) decrement in birthweight in women giving birth within one year (first pregnancy; adjustment for race, smoking, BMI, and use of blood pressure medications); the corresponding adjusted decrement associated with MAP reactivity was 116.7 (−15.7, 249.1, $p=0.08$). Among women with first pregnancies within two years, one standard unit of HR reactivity was associated with an adjusted 103.4 g decrement in birthweight (−12.6, 219.4,

$p=0.08$); cold pressor SBP reactivity was associated with a 91.3g ($-16.2, 198.8, p=0.10$). No associations were seen with gestational age.

Discussion

We did not find major relationships between pre-pregnancy cardiovascular reactivity and birth outcomes. A few measures – particularly DBP reactivity and MAP reactivity – were associated with minor increases in risk of PTB, but these were not large or statistically robust.

The relatively high risk of PTB and SGA in African-Americans, not attributable to any known risk factor, is one of the most difficult problems in perinatal health. It has been suggested that differences in stress contribute to this disparity,³⁵ with stress reactivity (perhaps influenced by early life hardship) reflecting host susceptibility to stress.⁷ We did not find major indications that this was the case – few interactions with race were seen, and those that were found, while not strong, indicated larger effects in whites than blacks. In addition, adjustment for stress reactivity did not reduce the association between race and the birth outcomes, as would be likely if reactivity were a mediating variable.³⁴

Previous studies have reported a direct association between reactivity during pregnancy and adverse birth outcomes.^{21–23} All three studies reported the strongest associations with diastolic blood pressure reactivity, which is consistent with our analysis. Hatch et al. reported stronger results in black participants, which we did not see.²³ There may be several reasons for the differences between our study and theirs. First, the other studies measured reactivity during pregnancy, while we used pre-pregnancy stress reactivity. There are good reasons to focus on pre-pregnancy health: the lack of successful prevention has led to greater focus on preconception health.^{36–38} Stress reactivity tends to be fairly stable across long time periods,²⁴ is reduced during pregnancy,³⁹ and has predicted other outcomes in non-pregnant adults after long periods of follow-up.¹⁴ Nonetheless, it is possible that reactivity in the pregnancy is more physiologically relevant, perhaps through having direct and immediate effects on the fetal environment. Our results for birthweight were stronger when limited to pregnancies occurring within a year or two of the measurement. Finally, the results of previous studies may not be particularly robust. Two samples were quite small ($n=40$ ²¹ and $n=70$ ²²) and the third found relationships only in black women,²³ and measured reactivity only in a lower-risk subset.²³

Strengths of our study include the standardised protocol and unusual opportunity to measure a woman's health before pregnancy. We believe that examining a woman's health across her life, not during pregnancy, is essential for understanding pregnancy health. Limitations include self-report of birth outcomes. However, data indicate that women report birthweight and gestational age of their children accurately^{40–43}, and our results were not different when limited to very preterm births (data not shown), which are reported more accurately than overall preterm births (Catov et al., in review). In particular, the proportion of reported PTB is fairly high, suggesting there may have been some misclassification of gestational age. The proportion of black women reporting PTB is particularly high, so the lack of effect seen in this group could be partially due to misclassification. Another limitation is the variable time interval from the reactivity test to the beginning of pregnancy, although analysis indicated little correlation between the interval between reactivity testing and pregnancy outcome. In addition, we did not have information on stressors during the pregnancy. It is possible that reactivity is important only against a background of high stress.

In conclusion, we did not find strong associations between stress reactivity and low birthweight or preterm birth. However, our results suggest that reactivity may be associated

with perinatal outcomes in whites, but not blacks. Future studies may need to concentrate on measurements closer to pregnancy, focus on other aspects of stress response, or look at responses to stressors more directly relevant to the woman.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Iyasu S, Tomashek K, Barfield W. Infant mortality and low birth weight among black and white infants - United States, 1980–2000. *MMWR Surveillance Summary*. 2002; 51:589–592.
2. MacDorman, MF.; Callaghan, WM.; Mathews, TJ.; Hoyert, DL.; Kochanek, KD. Trends in preterm-related infant mortality by race and ethnicity: United States, 1999–2004. U. S. Department of Health and Human Services; 2007. [updated 2007 October 15, 2008; cited 2009 September 1]; Available from
3. Hobel CJ, Goldstein A, Barrett ES. Psychosocial stress and pregnancy outcome. *Clinical Obstetrics and Gynecology*. 2008; 51:333–348. [PubMed: 18463464]
4. Dole N, Savitz DA, Hertz-Picciotto I, Siega-Riz AM, McMahon MJ, Buekens P. Maternal Stress and Preterm Birth. *American Journal of Epidemiology*. 2003; 147:14–24. [PubMed: 12505886]
5. Neggers Y, Goldenberg R, Cliver S, Hauth J. The relationship between psychosocial profile, health practices, and pregnancy outcomes. *Acta Obstetrica Gynecologica Scandinavica*. 2006; 85:277–285.
6. St-Laurent J, De Wals P, Moutquin JM, Niyonsenga T, Noiseux M, Czernis L. Biopsychosocial determinants of pregnancy length and fetal growth. *Paediatric and Perinatal Epidemiology*. 2008; 22:240–248. [PubMed: 18426519]
7. Hogue CJ, Bremner JD. Stress model for research into preterm delivery among black women. *American Journal of Obstetrics and Gynecology*. 2005; 192:S47–55. [PubMed: 15891712]
8. Kuzawa CW, Sweet E. Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *American Journal of Human Biology*. 2009; 21:2–15. [PubMed: 18925573]
9. de Weerth C, Buitelaar JK. Physiological stress reactivity in human pregnancy--a review. *Neuroscience and Biobehavioral Review*. 2005; 29:295–312.
10. Kaushik RM, Mahajan SK, Rajesh V, Kaushik R. Stress profile in essential hypertension. *Hypertension Research*. 2004; 27:619–624. [PubMed: 15750254]
11. Rose KM, North K, Arnett DK, Ellison RC, Hunt SC, Lewis CE, et al. Blood pressure and pulse responses to three stressors: associations with sociodemographic characteristics and cardiovascular risk factors. *Journal of Human Hypertension*. 2004; 18:333–341. [PubMed: 14739908]
12. Steptoe A, Marmot M. Impaired cardiovascular recovery following stress predicts 3-year increases in blood pressure. *Journal of Hypertension*. 2005; 23:529–536. [PubMed: 15716693]
13. Everson SA, Lynch JW, Kaplan GA, Lakka TA, Sivenius J, Salonen JT. Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke*. 2001; 32:1263–1270. [PubMed: 11387485]
14. Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, et al. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation*. 2004; 110:74–78. [PubMed: 15210592]

15. Markovitz JH, Raczynski JM, Wallace D, Chettur V, Chesney MA. Cardiovascular reactivity to video game predicts subsequent blood pressure increases in young men: The CARDIA study. *Psychosomatic Medicine*. 1998; 60:186–191. [PubMed: 9560868]
16. Knox SS, Hausdorff J, Markovitz JH. Reactivity as a predictor of subsequent blood pressure: racial differences in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Hypertension*. 2002; 40:914–919. [PubMed: 12468579]
17. Lepore SJ, Revenson TA, Weinberger SL, Weston P, Frisina PG, Robertson R, et al. Effects of social stressors on cardiovascular reactivity in Black and White women. *Annals of Behavioral Medicine*. 2006; 31:120–127. [PubMed: 16542126]
18. Committee on Technical Bulletins of the American College of Obstetricians and Gynecologists. ACOG technical bulletin. Hypertension in pregnancy. Number 219--January 1996. *International Journal of Gynaecology and Obstetrics*. 1996; 53:175–183. [PubMed: 8735301]
19. Thorp JM. Placental vascular compromise: Unifying the etiologic pathways of perinatal compromise. *Current Problems in Obstetrics and Gynecology*. 2001; 24:197–220.
20. Lorentzen B, Henriksen T. Plasma lipids and vascular dysfunction in preeclampsia. *Seminars in Reproductive Endocrinology*. 1998; 16:33–39. [PubMed: 9654605]
21. McCubbin JA, Lawson EJ, Cox S, Sherman JJ, Norton JA, Read JA. Prenatal maternal blood pressure response to stress predicts birth weight and gestational age: a preliminary study. *American Journal of Obstetrics & Gynecology*. 1996; 175:706–712. [PubMed: 8828438]
22. Gomez Ponce de Leon R, Gomez Ponce de Leon L, Coviello A, De Vito E. Vascular maternal reactivity and neonatal size in normal pregnancy. *Hypertension in Pregnancy*. 2001; 20:243–256. [PubMed: 12044333]
23. Hatch MC, Berkowitz G, Janevic T, Sloan R, Lapinski R, James T, et al. Race, cardiovascular reactivity, and preterm delivery among active-duty military women. *Epidemiology*. 2006; 17:178–182. [PubMed: 16477258]
24. Veit R, Brody S, Rau H. Four-year stability of cardiovascular reactivity to psychological stress. *Journal of Behavioral Medicine*. 1997; 20:447–460. [PubMed: 9415855]
25. Richman LS, Bennett GG, Pek J, Siegler I, Williams RB Jr. Discrimination, dispositions, and cardiovascular responses to stress. *Health Psychology*. 2007; 26:675–683. [PubMed: 18020838]
26. Anderson NB. Racial differences in stress-induced cardiovascular reactivity and hypertension: current status and substantive issues. *Psychological Bulletin*. 1989; 105:89–105. [PubMed: 2648440]
27. Cutter GR, Burke GL, Dyer AR, Friedman GD, Hilner JE, Hughes GH, et al. Cardiovascular risk factors in young adults. The CARDIA baseline monograph. *Control Clinical Trials*. 1991; 12:1S–77S.
28. Friedman GD, Cutter GR, Donahue RP, Hughes GH, Hulley SB, Jacobs DR Jr, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. *Journal of Clinical Epidemiology*. 1988; 41:1105–1116. [PubMed: 3204420]
29. Markovitz JH, Raczynski JM, Lewis CE, Flack J, Chesney M, Chettur V, et al. Lack of independent relationships between left ventricular mass and cardiovascular reactivity to physical and psychological stress in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *American Journal of Hypertension*. 1996; 9:915–923. [PubMed: 8879349]
30. Markovitz JH, Tucker D, Lewis CE, Sanders PW, Warnock DG. Inverse relationship of urinary cyclic GMP to blood pressure reactivity in the CARDIA study: vasodilatory regulation of sympathetic vasoconstriction. *Coronary Artery Risk Development in Young Adults*. *Psychosomatic Medicine*. 1998; 60:319–326. [PubMed: 9625219]
31. Moutquin JM. Socio-economic and psychosocial factors in the management and prevention of preterm labour. *BJOG*. 2003; 110 (Suppl 20):56–60. [PubMed: 12763113]
32. Alexander GR, Kogan MD, Himes JH. 1994–1996 U.S. singleton birth weight percentiles for gestational age by race, Hispanic origin, and gender. *Matern Child Health J*. 1999; 3:225–231. [PubMed: 10791363]
33. Spiegelman D, Hertzmark E. Easy SAS calculations for risk or prevalence ratios and differences. *American Journal of Epidemiology*. 2005; 163:199–200. [PubMed: 15987728]

34. Hafeman DM. "Proportion explained": a causal interpretation for standard measures of indirect effect? *American Journal of Epidemiology*. 2009; 170:1443–1448. [PubMed: 19850625]
35. Paul K, Boutain D, Agnew K, Thomas J, Hitti J. The relationship between racial identity, income, stress and C-reactive protein among parous women: implications for preterm birth disparity research. *Journal of the National Medical Association*. 2008; 100:540–546. [PubMed: 18507206]
36. Iams JD, Romero RA, Culhane J, Goldenberg RL. Primary, secondary, and tertiary interventions to reduce the morbidity and mortality of preterm birth. *Lancet*. 2008; 371:164–175. [PubMed: 18191687]
37. Ashton DM, Lawrence HC 3rd, Adams NL 3rd, Fleischman AR. Surgeon General's Conference on the Prevention of Preterm Birth. *Obstetrics and Gynecology*. 2009; 113:925–930. [PubMed: 19305340]
38. Posner SF, Broussard DL, Sappenfield WM, Streeter N, Zapata LB, Peck MG. Where are the data to drive policy changes for preconception health and health care? *Womens Health Issues*. 2008; 18:S81–86. [PubMed: 19059552]
39. de Weerth C, Buitelaar JK. Physiological stress reactivity in human pregnancy--a review. *Neuroscience Biobehavioral Review*. 2005; 29:295–312.
40. Troude P, L'Helias LF, Raison-Boulley AM, Castel C, Pichon C, Bouyer J, et al. Perinatal factors reported by mothers: do they agree with medical records? *European Journal of Epidemiology*. 2008; 23:557–564. [PubMed: 18560979]
41. Adegboye AR, Heitmann B. Accuracy and correlates of maternal recall of birthweight and gestational age. *Bjog*. 2008; 115:886–893. [PubMed: 18485168]
42. Jaspers M, de Meer G, Verhulst FC, Ormel J, Reijneveld SA. Limited validity of parental recall on pregnancy, birth, and early childhood at child age 10 years. *Journal of Clinical Epidemiology*. 2010; 63:185–191. [PubMed: 19682856]
43. Seidman DS, Slater PE, Ever-Hadani P, Gale R. Accuracy of mothers' recall of birthweight and gestational age. *British Journal of Obstetrics and Gynaecology*. 1987; 94:731–735. [PubMed: 3663531]

Table 1

Description of study population (limited to women with singletons born after exam 2, n=917). All data are at time of blood pressure reactivity testing (exam 2) unless otherwise noted

	Blacks (n=419)		Whites (n=498) ^a	
	N	%	N	%
Age in years				
18–25	206	49.2%	135	27.1%
25–30	158	37.7%	238	47.8%
30–34	55	13.1%	125	25.1%
Education				
high school or less	156	37.2%	85	17.1%
some college	185	44.2%	121	24.3%
college	62	14.8%	182	36.6%
>college	16	3.8%	110	22.1%
Smoking				
never smoker	273	65.5%	286	57.8%
former smokers	32	7.7%	97	19.6%
smoker	112	26.9%	112	22.6%
Marital status				
married	91	21.9%	199	40.0%
living with partner	47	11.3%	67	13.5%
widowed/separated/divorced	30	7.2%	27	5.4%
never married	248	59.6%	205	41.2%
Body mass index category (kg/m ²)				
<=18.5	23	5.5%	27	5.4%
18.5–25	221	52.9%	384	77.1%
>25–30	92	22.0%	56	11.2%
>30	82	19.6%	31	6.2%
Total gravidity (baseline+follow-up)				
1	48	11.5%	55	11.0%
2	90	21.5%	154	30.9%
3	100	23.9%	135	27.1%
4+	181	43.2%	154	30.9%
Any preterm birth baby during follow-up				
Yes	125	29.8%	69	13.9%
No	294	70.2%	429	86.1%
Any low birthweight baby during follow-up				
Yes	69	16.5%	27	5.4%
No	350	83.5%	471	94.6%
	mean	std	mean	std
Baseline systolic blood pressure (mmHg)	104.6	8.8	101.3	8.8
Baseline diastolic blood pressure (mmHg)	65.9	9.0	64.0	8.4

	Blacks (n=419)		Whites (n=498) ^a	
	N	%	N	%
Gestational age at first pregnancy	38.6	3.25 ^b	39.4	2.4 ^c
Birthweight at first pregnancy	3168.0	722.2 ^d	3451.4	532.9 ^e

^a all differences across race p<0.01

^b median gestational age 40, 25th percentile 37, 75th percentile 40, minimum 23, maximum 48

^c median gestational age 40, 25th percentile 38, 75th percentile 41, minimum 23, maximum 45

^d median 3231.9, 25th percentile 2863.3, 75th percentile 3600.5, minimum 539, maximum 5018

^e median 3472.9, 25th percentile 3175.2, 75th percentile 3798.9, minimum 567, maximum 4649

Table 2

Blood pressure reactivity among women in the CARDIA study with later singleton livebirths

	N	mean	std	
pre-intervention				
SBP	913	106.7	9.5	
DBP	913	63.4	9.8	
HR	912	71.8	9.4	
MAP	917	77.4	8.6	
		median	25th percentile	75th percentile
video game				
SBP difference	913	8.3	4.1	13.4
DBP difference	854	8.5	4.3	12.7
HR difference	853	4.0	1.0	7.8
MAP difference	858	8.7	4.8	12.3
star tracing				
SBP difference	739	10.2	5.3	15.3
DBP difference	854	9.6	5.8	14.3
HR difference	851	5.7	2.0	9.8
MAP difference	859	10.0	6.3	14.5
cold pressor				
SBP difference	916	21.5	14.5	30.5
DBP difference	687	26.3	17.3	35.0

* SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; MAP, mean arterial pressure

Table 3

Association (relative risk) between reactivity and outcome of first pregnancy after reactivity testing, stratified by race

	preterm birth ^b									
	overall			white			black			p for interaction
	RR	95% CI	p	RR	95% CI	p	RR	95% CI	p	
summary SBP reactivity ^a	1.07	[0.90, 1.27]	0.42	1.29	[0.94, 1.77]	0.12	0.99	[0.81, 1.22]	0.95	0.18
summary DBP reactivity	1.13	[0.96, 1.32]	0.13	1.34	[1.05, 1.70]	0.02	1.04	[0.88, 1.25]	0.63	0.17
summary MAP reactivity	1.14	[0.97, 1.34]	0.11	1.35	[1.08, 1.68]	0.01	1.04	[0.86, 1.26]	0.68	0.14
summary HR reactivity	1.08	[0.92, 1.26]	0.37	1.39	[1.03, 1.88]	0.03	1.00	[0.83, 1.20]	0.96	0.08
cold pressor SBP	0.94	[0.80, 1.11]	0.47	0.93	[0.67, 1.30]	0.68	0.94	[0.78, 1.15]	0.56	0.95
cold pressor DBP	0.96	[0.82, 1.12]	0.60	0.92	[0.70, 1.22]	0.56	0.98	[0.81, 1.17]	0.79	0.73
	small-for-gestational-age ^c									
	overall			white			black			p for interaction
	RR	95% CI	p	RR	95% CI	p	RR	95% CI	p	
summary SBP reactivity ^a	0.76	[0.60, 0.95]	0.02	1.01	[0.66, 1.56]	0.96	0.73	[0.55, 0.96]	0.03	0.21
summary DBP reactivity	0.93	[0.75, 1.15]	0.49	0.93	[0.67, 1.30]	0.68	0.92	[0.70, 1.21]	0.55	0.95
summary MAP reactivity	0.82	[0.66, 1.02]	0.07	0.83	[0.57, 1.21]	0.33	0.83	[0.63, 1.10]	0.19	0.99
summary HR reactivity	0.88	[0.68, 1.14]	0.35	1.22	[0.73, 2.04]	0.44	0.77	[0.59, 1.01]	0.06	0.14
cold pressor SBP	1.15	[0.88, 1.49]	0.30	1.45	[0.93, 2.25]	0.10	1.03	[0.78, 1.36]	0.85	0.22
cold pressor DBP	0.99	[0.79, 1.25]	0.95	1.15	[0.79, 1.69]	0.47	1.03	[0.78, 1.36]	0.85	0.64

^aSBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; MAP, mean arterial pressure; RR, relative risk; CI, confidence interval^bPreterm birth models are adjusted for center.^cSmall for gestational age defined as birthweight <10th percentile for gestational age. Small for gestational age models are adjusted for smoking, race, pre-pregnancy body mass index, and use of blood pressure medications during follow-up.