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Effect of race on longitudinal central hemodynamics in pregnancy

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KEYWORDS: adaptation; bioreactance; cardiac output; ethnicity; hemodynamics; peripheral vascular resistance; race

CONTRIBUTION

What are the novel findings of this work?
In pregnancy, white women achieve higher cardiac output and lower peripheral vascular resistance than do black and Asian women. In white women, stroke volume and heart rate increase, but in black and Asian women, heart rate increases and stroke volume decreases.

What are the clinical implications of this work?
Race-specific differences in maternal hemodynamic adaptation to pregnancy should be considered in future studies examining the relationship between cardiovascular changes and pregnancy complications, and, on a clinical basis, a lower threshold for hemodynamic assessment during pregnancy should exist for women of black or Asian origin if they demonstrate cardiovascular decompensation.

ABSTRACT

Objective *To compare central hemodynamics between white, black and Asian women in pregnancy.*

Methods *This was a prospective, longitudinal study of maternal central hemodynamics in white, black and Asian women with a singleton pregnancy, assessed using a bioreactance method at 11 + 0 to 13 + 6, 19 + 0 to 24 + 0, 30 + 0 to 34 + 0 and 35 + 0 to 37 + 0 weeks' gestation. At each visit, cardiac output (CO), stroke volume (SV), heart rate (HR), peripheral vascular resistance (PVR) and mean arterial pressure were recorded. Multilevel linear mixed-effects analysis was performed to compare the repeated measures of the cardiac variables between white, black and Asian women, controlling for maternal characteristics, medical history and medication use.*

Results *The study population included 1165 white, 247 black and 116 Asian women. CO increased with gestational age to a peak at 32 weeks and then decreased; the highest CO was observed in white women and the lowest in Asian women. SV initially increased after the first visit but subsequently declined with gestational age in white women, decreased with gestational age in black women and remained static in Asian women. In all three study groups, HR increased with gestational age until 32 weeks and then remained constant; HR was highest in black women and lowest in white women. PVR showed a reversed pattern to that of CO; the highest values were in Asian women and the lowest in white women. The least favorable hemodynamic profile, which was observed in black and Asian women, was reflected in higher rates of a small-for-gestational-age infant.*

Conclusions *There are race-specific differences in maternal cardiac adaptation to pregnancy. White women have the most favorable cardiac adaptation by increasing SV and HR, achieving the highest CO and lowest PVR. In contrast, black and Asian women have lower CO and higher PVR than do white women, with CO increasing through a rise in HR due to declining or static SV.*

INTRODUCTION

Pregnancy represents a model of volume and pressure overload that results in significant alterations in cardiac geometry and function. Optimal maternal cardiac adaptation results in increased blood volume and left ventricular (LV) mass, with concomitant increases in cardiac output (CO), stroke volume (SV) and heart rate (HR), along with

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decreased peripheral vascular resistance (PVR)[1]. Failure to achieve an increase in CO and decrease in PVR has been associated with the development of pre-eclampsia (PE) and delivery of a small-for-gestational-age (SGA) neonate[2–4].

There is extensive evidence of racial disparity in the risks of adverse pregnancy outcomes and long-term development of cardiovascular diseases. In black and Asian pregnant women, compared to white pregnant women, there is an increased risk of miscarriage, stillbirth, PE, SGA and gestational diabetes mellitus (GDM)[5]. Outside of pregnancy, black populations have a higher incidence of hypertension, Type-2 diabetes, end-stage renal failure and mortality from stroke[6–9], while Asian populations have an increased risk of diabetes and mortality from coronary artery disease[10]. In non-pregnant individuals, there are racial differences in cardiovascular function at rest and during exercise, and poorer reserve in black and Asian populations than in white populations[11–14].

The objective of this study was to ascertain the impact of race on maternal cardiac adaptation to pregnancy.

METHODS

Study population

This was a prospective, longitudinal study assessing maternal central hemodynamics in women with a singleton pregnancy attending for routine care in six maternity hospitals in the UK between November 2015 and May 2016. The study was carried out in some of the hospitals participating in an international multicenter study involving routine screening for PE by maternal factors and biomarkers at 11 + 0 to 13 + 6 weeks' gestation[15]; women identified as being at high risk were invited to participate in the ASPRE trial of aspirin *vs* placebo[16]. The study was approved by the NHS Research Ethics Committee (REC reference: 13/LO/1479).

The inclusion criteria were white, black or Asian racial origin, singleton pregnancy resulting in the delivery of a morphologically normal liveborn or stillborn neonate at or after 24 weeks' gestation and attendance for hemodynamic studies for at least three of the four visits. Exclusion criteria were maternal age < 18 years, mixed racial origin, pre-existing maternal cardiac conditions, fetal abnormalities, incomplete follow-up and termination of pregnancy or miscarriage. Maternal demographic characteristics and medical history were recorded and hemodynamic studies were performed at 11 + 0 to 13 + 6, 19 + 0 to 24 + 0, 30 + 0 to 34 + 0 and 35 + 0 to 37 + 0 weeks' gestation. Maternal factors recorded included age, height, weight at each visit, self-reported racial origin (white, black or Asian), method of conception (spontaneous or use of assisted reproductive technology), cigarette smoking during pregnancy, medical history, medication use, parity and obstetric history (nulliparous or parous with or without previous PE and/or SGA).

Assessment of maternal cardiovascular function

Maternal cardiac function was assessed using a non-invasive, bioreactance method (NICOM; Cheetah Medical Ltd, Maidenhead, Berkshire, UK) which has been validated in both pregnant and non-pregnant populations[4,17,18]. The principle of the method is that when an alternating electrical current traverses the thoracic cavity, the bioreactance apparatus uses the simultaneous relative phase shifts to calculate SV. After 15 min of rest, four dual-surface electrodes were applied across the maternal back, and cardiac variables (CO, SV, HR, PVR and mean arterial pressure (MAP)) were recorded with the woman in a sitting position for 10 min at 30-s intervals (20 cycles). The average measurements of the hemodynamic variables in the final 10 cycles were included in the analysis.

Outcomes and definitions

The outcome measure was the effect of maternal racial origin on longitudinal changes in CO, SV, HR, PVR and MAP with advancing gestational age.

The definitions of non-proteinuric gestational hypertension and PE were those of the International Society for the Study of Hypertension in Pregnancy[19]. The diagnosis of a SGA or large-for-gestational-age neonate was based on the finding of birth weight < 10th percentile or > 90th percentile, respectively, according to the Fetal Medicine Foundation reference ranges for gestational age[20]. Neonatal morbidity was defined as the presence of any one or more of respiratory distress syndrome, need for ventilation, intrapartum sepsis, necrotizing enterocolitis or neonatal hypoglycemia.

Statistical analysis

Maternal demographic characteristics, medical history and pregnancy outcomes were compared between racial groups using the chi-square test or Fisher's exact test for categorical variables. Normality of the distribution of numerical data was assessed using the Kolmogorov–Smirnov test. As maternal weight, CO, SV, MAP and PVR were not normally distributed, the distributions were made Gaussian by $\log_{10}$ transformation. The Kruskal–Wallis or one-way ANOVA test with *post-hoc* analysis was used to compare non-normally and normally distributed continuous data, respectively. Data are presented as median (interquartile range) or mean ± SD for non-normally and normally distributed continuous variables, respectively, and as *n* (%) for categorical variables. For the repeated measures analysis of the maternal hemodynamic variables, multilevel linear mixed-effects analysis was performed, controlling for time (the four visits) and factors among maternal demographic characteristics, medical history and medication use, which are known to affect these measurements[21,22]. The fixed-effect component included time, race (white, black or Asian), maternal age, $\log_{10}$ weight, height, parity (nulliparous or

parous with or without previous PE and/or SGA), spontaneous conception, smoking, family history of PE, medical comorbidities including chronic hypertension, autoimmune disease, asthma and diabetes mellitus Type I and Type II, medication use (labetalol, nifedipine/methyldopa, prednisolone, aspirin) and first-order interaction between racial group and time. The likelihood radio (LR) test was used to define the best multilevel model (including only the random slope for time or random intercept *vs* including both the random intercept and slope) and to compare it with the base model (with no random effects). The fixed and random effects of the multilevel models and the estimated marginal means at the four visits are presented.

IBM SPSS Statistics for Windows v. 23 (IBM Corp., Armonk, NY, USA) was used for statistical analysis.

RESULTS

Study population

During the study period, 2024 women were recruited, but 450 were excluded from the analysis because the pregnancy ended in miscarriage or termination ($n = 27$), the woman attended fewer than three of the four visits ($n = 384$) or there was loss to follow-up ($n = 39$). We also excluded women of mixed race ($n = 46$) owing to their small number. The study population comprised 1165 white, 247 black and 116 Asian women. The maternal characteristics at the screening visit and pregnancy outcomes in the three groups are shown and compared in Table 1.

A higher proportion of black and Asian women, compared with white women, were screen positive for PE and participated in the ASPRE trial, but there were no significant differences between the racial groups in the proportion of women allocated to the aspirin *vs* the placebo arm of the trial, and there was no difference in the number of women with PE. Black women were heavier than white women, while Asian women were shorter and lighter. The incidence of smoking was lower in Asian and black women than in white women. There was a higher proportion of nulliparous women in the white group than in the black group, and there were more parous women

Table 1 Demographic characteristics and outcome of 1528 singleton pregnancies according to racial origin

Characteristic	*White* (n = *1165*)	*Black* (n = *247*)	*Asian* (n = *116*)
Maternal age (years)	30.9 ± 5.3	31.4 ± 5.7	31.6 ± 5.3
Maternal weight at booking (kg)	67.9 (60.4–79.1)	77.9 (67.6–90.4)***	61.4 (54.3–69.7)***†††
Maternal height (cm)	165.1 ± 6.3	165.4 ± 6.8	158.6 ± 5.5***†††
Smoker	82 (7.0)	5 (2.0)**	1 (0.9)**
Family history of PE	77 (6.6)	16 (6.5)	2 (1.7)*
Spontaneous conception	1124 (96.5)	245 (99.2)*	113 (97.4)
Nulliparous	642 (55.1)	109 (44.1)**	59 (50.9)
Parous with previous PE and/or SGA	67 (5.8)	28 (11.3)**	10 (8.6)
Chronic hypertension	13 (1.1)	16 (6.5)***	2 (1.7)
Asthma	17 (1.5)	7 (2.8)	1 (0.9)
Pre-existing diabetes	4 (0.3)	4 (1.6)**	1 (0.9)
Autoimmune disease	8 (0.7)	0 (0.0)	1 (0.9)
Use of labetalol	52 (4.5)	28 (11.3)***	4 (3.4)††
Use of nifedipine or methyldopa	12 (1.0)	8 (3.2)**	3 (2.6)
Use of prednisolone	2 (0.2)	1 (0.4)	0 (0.0)
ASPRE trial			
Placebo group	128 (11.0)	67 (27.1)***	21 (18.1)*
Aspirin group	122 (10.5)	64 (25.9)***	19 (16.4)†
Pregnancy outcome			
PE	36 (3.1)	12 (4.9)	3 (2.6)
Gestational hypertension	42 (3.6)	14 (5.7)	3 (2.6)
Gestational diabetes	45 (3.9)	16 (6.5)	12 (10.3)**
Birth before 37 weeks' gestation	41 (3.5)	10 (4.0)	3 (2.6)
Induction of labor	364 (31.2)	72 (29.1)	35 (30.2)
Emergency Cesarean section	192 (16.5)	47 (19.0)	20 (17.2)
Operative delivery for fetal distress	143 (12.3)	33 (13.4)	12 (10.3)
GA at birth (weeks)	40.0 (39.0–40.9)	39.6 (38.9–40.6)**	39.6 (38.6–40.3)**
Neonatal outcome			
Birth weight (g)	3425 ± 540	3232 ± 536***	3153 ± 499***
Birth-weight *Z*-score	−0.020 ± 1.08	−0.407 ± 1.07***	−0.613 ± 1.17***
Birth-weight percentile	51.9 (23.7–76.7)	32.9 (14.2–62.8)***	24.6 (10.9–53.3)
Birth weight < 10th centile	146 (12.5)	44 (17.8)*	28 (24.1)**
Birth weight > 90th centile	122 (10.5)	12 (4.9)	7 (6.0)
Perinatal mortality	4 (0.3)	1 (0.4)	0 (0.0)
Admission to neonatal unit	68 (5.8)	9 (3.6)	8 (6.9)
Neonatal morbidity‡	57 (4.9)	8 (3.2)	5 (4.3)

Data are given as mean ± SD, median (interquartile range) or *n* (%). ‡Includes respiratory distress syndrome, need for ventilation, intrapartum sepsis, necrotizing enterocolitis and neonatal hypoglycemia. Compared with white group: *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$. Compared with black group: †$P < 0.05$; ††$P < 0.01$; †††$P < 0.001$. GA, gestational age; PE, pre-eclampsia; SGA, small-for-gestational age.

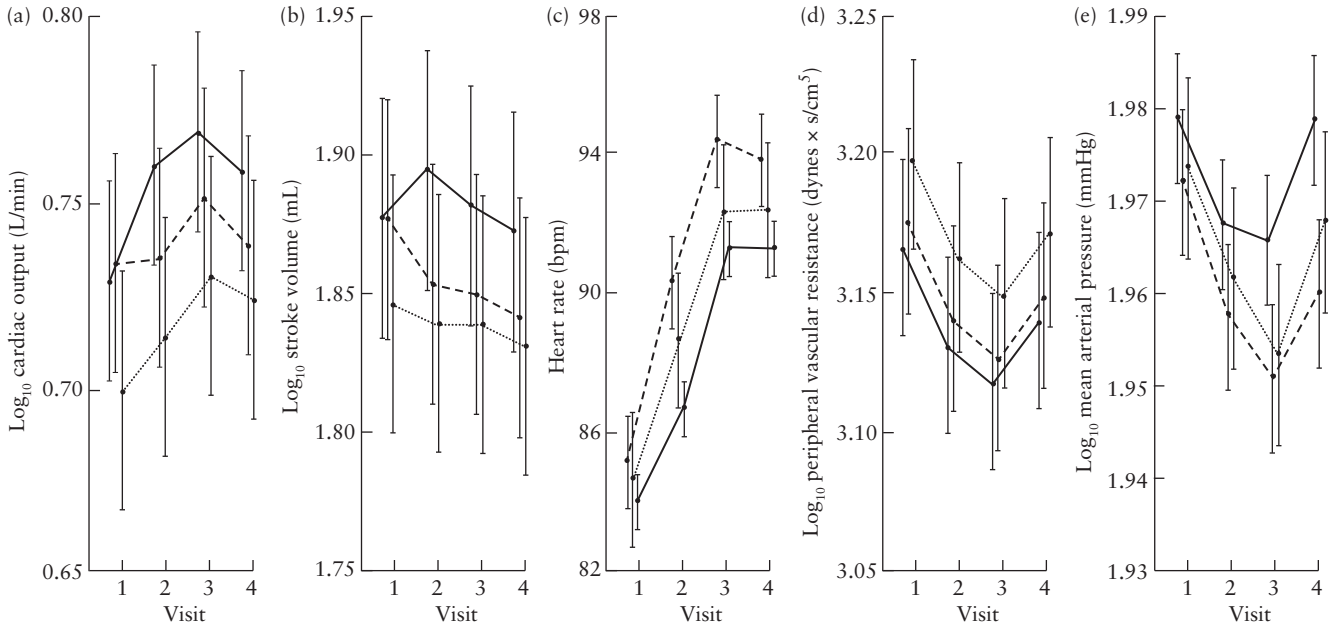

Figure 1 Estimated marginal means with 95% CIs from linear mixed-effects models for $\log_{10}$ cardiac output (a), $\log_{10}$ stroke volume (b), heart rate (c), $\log_{10}$ peripheral vascular resistance (d) and $\log_{10}$ mean arterial pressure (e) at four visits (11 + 0 to 13 + 6 weeks (1), 19 + 0 to 24 + 0 weeks (2), 30 + 0 to 34 + 0 weeks (3) and 35 + 0 to 37 + 0 weeks (4)) in white (——), black (- - -) and Asian (·····) pregnant women, after controlling for demographic characteristics, medical history and medication use.

with a history of PE and/or SGA in the black group. The incidence of chronic hypertension, diabetes mellitus and treatment with antihypertensives was higher in black than in white women. Women of black or Asian origin delivered babies with lower birth weight than did white women. Despite a higher incidence of GDM in Asian than in white women, the former group delivered the smallest infants. On the other hand, the rate of macrosomia was highest in white women, despite a higher proportion of nulliparous women and the lowest incidence of GDM. There were no differences in the rate of PE, emergency Cesarean section, admission to the neonatal unit or neonatal morbidity between the three groups.

Multilevel linear mixed-effects models

The estimated marginal means of the best multilevel models are illustrated in Figure 1. Data on estimated marginal means for all hemodynamic variables are given in Table 2, and data on fixed and random effects of multilevel models for $\log_{10}$CO, HR and $\log_{10}$SV are given in Table S1 and those for $\log_{10}$MAP and $\log_{10}$PVR are given in Table S2. The results of the LR tests for each variable are summarized in Appendix S1.

Maternal demographic characteristics and medical history

Increasing maternal age was associated with a decrease in $\log_{10}$CO and HR and higher $\log_{10}$PVR. Increasing maternal height was associated with higher $\log_{10}$CO and $\log_{10}$SV and lower HR and $\log_{10}$PVR. Increasing $\log_{10}$ maternal weight was associated with higher $\log_{10}$CO, $\log_{10}$SV, HR and $\log_{10}$MAP. Smoking was associated with lower $\log_{10}$MAP. Parous women, irrespective of previous PE and/or SGA, had higher $\log_{10}$CO and lower $\log_{10}$PVR than did nulliparous women, and parous women without previous PE and/or SGA also had higher HR.

Maternal chronic hypertension was associated with higher $\log_{10}$MAP. The use of labetalol or nifedipine/methyldopa was associated with higher $\log_{10}$PVR, and the use of labetalol was also associated with higher $\log_{10}$MAP. The use of prednisolone was associated with higher $\log_{10}$SV, and use of aspirin or placebo, compared with no treatment, was associated with higher $\log_{10}$MAP. Autoimmune disease was associated with lower $\log_{10}$CO and higher $\log_{10}$PVR.

There was no significant contribution in any of the models from spontaneous conception, family history of PE, asthma or diabetes mellitus Type I or II. There was significant interaction between racial group and time for $\log_{10}$CO, $\log_{10}$SV, HR and $\log_{10}$MAP.

Changes with time after controlling for maternal characteristics

Log_{10}CO in all three racial groups increased during the first three visits and declined at the fourth. Black women had consistently lower $\log_{10}$CO than did white women after the first visit, and Asian women had lower $\log_{10}$CO than did white women throughout pregnancy.

Log_{10}SV in white women initially increased after the first visit, but subsequently declined. Log_{10}SV at the first visit was the same in black women as in white women, but at subsequent visits it was lower in black women. In Asian women, $\log_{10}$SV was lower than in

Table 2 Estimated marginal means (95% CI) from multilevel linear mixed-effects models for maternal hemodynamic variables, according to gestational age and racial origin

	Gestational age (weeks)			
Variable	*11 + 0 to 13 + 6*	*19 + 0 to 24 + 0*	*30 + 0 to 34 + 0*	*35 + 0 to 37 + 0*
Log_{10} cardiac output (L/min)				
White	0.727 (0.701–0.754)	0.758 (0.732–0.785)	0.768 (0.741–0.795)	0.757 (0.730–0.784)
Black	0.732 (0.703–0.761)	0.733 (0.704–0.763)**	0.750 (0.721–0.779)*	0.737 (0.708–0.767)**
Asian	0.698 (0.665–0.730)**††	0.713 (0.680–0.745)***	0.729 (0.697–0.761)***	0.723 (0.691–0.755)**
Log_{10} stroke volume (mL)				
White	1.876 (1.833–1.919)	1.894 (1.851–1.937)	1.881 (1.838–1.924)	1.871 (1.828–1.914)
Black	1.875 (1.831–1.920)	1.851 (1.807–1.896)***	1.848 (1.804–1.892)***	1.840 (1.795–1.884)***
Asian	1.844 (1.797–1.892)**†	1.837 (1.790–1.884)***	1.837 (1.790–1.884)***	1.829 (1.782–1.876)***
Heart rate (bpm)				
White	84.046 (83.301–84.793)	86.728 (86.000–87.457)	91.278 (90.547–92.008)	91.248 (90.507–91.988)
Black	85.147 (83.858–86.435)	90.308 (89.013–91.603)***	94.369 (93.050–95.688)***	93.801 (92.469–95.133)***
Asian	84.604 (82.686–86.523)	88.609 (86.706–90.513)	92.295 (90.375–94.214)	92.350 (90.447–94.253)
Log_{10} peripheral vascular resistance (dynes × s/cm^5)				
White	3.167 (3.135–3.199)	3.131 (3.099–3.163)	3.117 (3.085–3.149)	3.139 (3.108–3.171)
Black	3.176 (3.144–3.209)	3.140 (3.107–3.173)	3.126 (3.094–3.159)	3.149 (3.116–3.182)
Asian	3.198 (3.163–3.232)***††	3.162 (3.127–3.196)***††	3.148 (3.113–3.182)***††	3.170 (3.136–3.205)***††
Log_{10} mean arterial pressure (mmHg)				
White	1.979 (1.972–1.986)	1.967 (1.961–1.974)	1.966 (1.959–1.972)	1.978 (1.972–1.985)
Black	1.972 (1.964–1.980)*	1.958 (1.950–1.966)**	1.951 (1.943–1.959)***	1.960 (1.952–1.968)***
Asian	1.973 (1.964–1.983)	1.962 (1.952–1.971)	1.953 (1.944–1.963)**	1.967 (1.958–1.977)**

Variables were controlled for demographic characteristics, medical history and medication use. Compared with white group: *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$. Compared with black group: †$P < 0.05$; ††$P < 0.01$.

white women and it did not change significantly with gestational age.

HR in all three racial groups shared similar incremental trends until the third visit, with a plateau thereafter. In black women, HR was higher than in white women at all four visits. In Asian women, HR was not significantly different from that in white or black women at any of the visits.

Log_{10}PVR and log_{10}MAP in all three racial groups decreased between the first and third visits and subsequently increased. Log_{10}PVR in Asian women was higher than that in white and black women at all four visits and log_{10}MAP in black and Asian women was lower than that in white women at all four visits.

DISCUSSION

Main findings

The results of this study demonstrate that, after adjusting for maternal demographic characteristics and medical history, there are significant differences in cardiac adaptation to pregnancy between women of white, black and Asian racial origin. In all three racial groups, there was an increase in CO with gestational age, which peaked at 32 weeks and subsequently declined at 35–37 weeks; however, CO was higher in white than in black and Asian women. More importantly, the mechanisms by which the women achieved increased CO differed according to race; increased CO in white women was a result of increases in both SV and HR. On the other hand, increased CO in black and Asian women was primarily a result of increased HR, following a decline in or static SV in black and Asian women, respectively. All three groups demonstrated an expected decrease in PVR with gestational age until 32 weeks, with Asian women persistently having the highest PVR, followed by black and then white women, who had the lowest PVR.

Comparison of findings with those of previous studies

Pregnancy represents an excellent model of physiological adaptation to volume overload, comparable to that in trained endurance athletes such as swimmers or runners[23]. Compared with untrained individuals, after indexing for body surface area, central hemodynamics in endurance athletes at rest show increased LV mass, LV end-diastolic dimension and SV, reduced HR[24–27] and normal diastolic and systolic function[14], with no difference in resting CO, PVR and blood pressure[25]. Their hearts undergo eccentric hypertrophy (large dilated cavities and relatively thin walls). These changes are suggestive of modulation of the hemodynamic profile to a more efficient one, basing oxygen transport on SV rather than HR. An efficient energy management system avoids high resting HR because this shortens diastole in each cardiac cycle and results in a reduction in myocardial and coronary perfusion time, impaired ventricular filling and increased myocardial oxygen demand[28]. In normal pregnancy, changes in LV dimensions similar to those seen in athletes have been reported, and the adaptation is efficient since, in the first two trimesters, the increase in CO is due to a 40% increase in SV and only a 10% increase in HR[1].

Previous studies on maternal cardiovascular adaptation in pregnancy either did not report the distribution of women from different racial groups or did not report separately the results for different racial groups. In contrast, studies in non-pregnant populations have reported clear racial differences in cardiovascular risk and hemodynamic adaptation to cardiovascular stress. Asian populations, compared with white populations, have a 2-fold increased risk of coronary artery disease, with a concomitant higher risk of death from cardiovascular disease[10]. Hemodynamic studies have reported that, in Asian individuals at rest, after adjustment for body surface area, HR is 10% higher and SV is 28% lower than in white individuals[29]. A population study of 30 000 subjects in London reported that, compared with white individuals, Asian individuals had impaired longitudinal LV function, greater LV filling pressure and higher rates of concentric remodeling (hearts with thick walls and relatively small cavities), independent of other demographic and clinical parameters[30]. People of African ancestry have a 4-fold increased risk of hypertension compared with that in white populations and a higher risk of mortality associated with stroke[6]. Population studies comparing black and white cohorts at rest have demonstrated that the former had greater LV thickness for equivalent levels of blood pressure and prevalence of concentric hypertrophy[31], higher PVR and lower CO, controlling for body surface area[32]. Furthermore, black compared with white athletes show a disproportionate increase with training in LV mass for a given LV volume, indicating attenuated myocardial relaxation and LV filling[33]. It is therefore possible that black and Asian women enter pregnancy with a less favorable cardiovascular reserve and worse potential to cope with hemodynamic stress than do white women.

Clinical implications

Women of black or Asian race have been shown to have higher rates of SGA compared with that in white women[5,34]. Women who deliver a SGA neonate have been shown to have impaired cardiovascular adaptation to pregnancy. Stott *et al.*[4] studied longitudinal cardiac adaptation in 84 women at high risk owing to a previous hypertensive disorder of pregnancy or chronic hypertension, and found that women who delivered a baby with a birth weight $< 10^{th}$ percentile have static or suppressed CO and SV with persistently raised PVR, with a trend toward higher HR. This characteristic hemodynamic trend further corroborates other studies that show that pregnancies with failed volume response, manifested in suppressed SV, are destined to become SGA[4,35,36]. In our study, the hemodynamic adaptation of black and Asian women was characterized by a similar low-volume high-resistance state, which was reflected in the considerably higher prevalence of a SGA neonate in black and Asian women than in white women. Furthermore, the worse hemodynamic profiles of black and Asian women were in accordance with the finding that a higher proportion of them were screen positive in the ASPRE trial, which further corroborated the findings of our previous study demonstrating that women who screened high risk for preterm PE have impaired cardiovascular adaptation, irrespective of pregnancy outcome[37].

Strengths and limitations

Strengths of our study include, first, examination of the impact of race on maternal central hemodynamics during pregnancy, which has not been reported in previous studies identified by a search of PubMed and EMBASE; second, longitudinal collection of data from a large number of women in each racial group; and, third, adjustment of hemodynamic indices for maternal demographic characteristics and medical history. A limitation of the study is that some of our patients participated in the ASPRE trial of aspirin *vs* placebo. It was for this reason that we controlled for the use of aspirin in the mixed-model analysis, which did not show it to be a significant predictor of maternal hemodynamic variables. An additional limitation is that we did not control for socioeconomic status which, in non-pregnant populations, has been shown to explain to a large extent the differences in adult mortality rate across different races[38,39]. However, we believe that this unavoidable limitation represents a reality in everyday clinical practice that cannot be modified during pregnancy.

Conclusions

We found race-specific differences in maternal hemodynamic adaptation to pregnancy. Such differences should be considered in future studies examining the relationship between cardiovascular changes and pregnancy complications and, on a clinical basis, a lower threshold for hemodynamic assessment during pregnancy should exist for women of black or Asian origin if they demonstrate signs or symptoms of cardiovascular decompensation.

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SUPPORTING INFORMATION ON THE INTERNET

The following supporting information may be found in the online version of this article:

Appendix S1 Multilevel linear mixed-effects models

Table S1 Fixed and random effects of multilevel linear mixed-effects models for maternal cardiac output, stroke volume and heart rate

Table S2 Fixed and random effects of multilevel linear mixed-effects models for maternal peripheral vascular resistance and mean arterial pressure