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Original Article

Effect of age, parity, and race on the incidence of pregnancy associated hypertension and eclampsia in the United States

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ABSTRACT

Purpose: To describe the incidence of pregnancy associated hypertension and eclampsia from adolescence through the fifth decade of life, including the effect of parity and race, in the United States.

Methods: Data were evaluated from the National Center for Health Statistics (vital statistics section). The data were stratified by maternal age group, parity (G1, first pregnancy; G2+, second or higher pregnancy), and racial group.

Results: The incidence of pregnancy associated hypertension (PAH) decreased with increased age in late adolescence in the G2+ group but not the G1 group (total and all racial groups). The incidence of PAH was significantly greater for non-Hispanic black or non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .02$) except age $\leqslant$15 years (G2+ group) and 45–54 years (both G1 and G2+ groups). The incidence of eclampsia decreased with increased age in late adolescence in the G2+ group (total and all racial groups) and the G1 group (total and non-Hispanic black groups). The incidence of eclampsia was significantly greater for non-Hispanic black than non-Hispanic white and for non-Hispanic white than Hispanic groups for all age groups except age $\leqslant$15 years in the G2+ group. The incidence of PAH and eclampsia increased substantially in both G1 and G2+ groups in the fifth decade of life (total and all racial groups).

Conclusions: The incidence of PAH (G2+ group) and eclampsia (G1 and G2+ groups) decreased with increased age during adolescence and increased in the fifth decade (G1 and G2+ groups).

Introduction

The hypertensive disorders of pregnancy include gestational hypertension (nonproteinuric preeclampsia), mild preeclampsia, severe preeclampsia, and eclampsia. There is increased maternal and fetal morbidity and mortality with increased severity of maternal gestational hypertensive syndromes [1–7]. Young maternal age is a risk factor for adverse outcomes including preterm birth, low fetal birth weight, fetal growth restriction, late fetal death, and infant mortality [8]. However, there is controversy about the relation between young maternal age and gestational hypertensive disorders. Most studies were limited because of insufficient sample size, were not stratified by parity, or did not exclude subjects with pregestational hypertension [1,2,9].

Insulin resistance increases during puberty and subsequently resolves [10,11]. Normal pregnancy is characterized by increasing insulin resistance throughout gestation. However, insulin resistance and metabolic syndrome before pregnancy and during early gestation are risk

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factors for gestational hypertension and preeclampsia [12–16]. Clarification of the risk for the gestational hypertensive disorders immediately after puberty may add further evidence to the role of insulin resistance in determining the risk for pregnancy associated hypertension (PAH).

There are >4 million annual births in the United States. The National Vital Statistics System is a data collection program of the National Center for Health Statistics in the United States. Data are provided from original birth certificates and vital registration systems that are maintained and operated by all states and territories. The National Center for Health Statistics promotes a uniform national database by compiling the data and working closely with the states and territories to develop standardized certificates, reports for data collection, and methods of data preparation.

The National Center for Health Statistics public database includes 2 categories for hypertensive disorders of pregnancy: (1) pregnancy associated hypertension (PAH), which includes gestational hypertension, preeclampsia, and eclampsia; and (2) eclampsia as an individual category. Gestational hypertension, preeclampsia, and eclampsia have similar risk factors and are important markers for the future development of metabolic syndrome and cardiovascular diseases [15–21]. Therefore, PAH may be a useful clinical indicator. Eclampsia is an extreme manifestation of the maternal hypertensive syndrome. The database includes information about parity, birth order, preexisting medical conditions, race, and ethnicity.

The purpose of the present study was to describe the incidence of PAH and eclampsia from adolescence through the fifth decade of life, including the effect of parity, race, and ethnicity, in the United States.

Methods

Database

The incidence of PAH for first (G1) and second or higher (G2+) pregnancies was calculated using data from the National Center for Health Statistics vital statistics section (public access section). PAH and eclampsia were selected as the health characteristics of interest. Women were excluded from the study because of (1) prepregnancy hypertension, (2) diabetes (prepregnancy or gestational diabetes in the index pregnancy), or (3) absence of information available about diabetes status and hypertension status (Table 1). The data were stratified by maternal age group, parity, and racial group. Parity was determined through the evaluation of the "Total Birth Order" variable in the database. Total Birth Order is defined as the total number of pregnancies the mother has reported, including the index delivery. The data for PAH were further stratified in a sub-study by exclusion of women who reported tobacco use or for whom no information was available regarding tobacco use.

Data analysis

The incidence of PAH was calculated for each maternal age category for both G1 and G2+ groups (G1, first pregnancy; G2+, second or higher pregnancy). The G2+ pregnancy category included data for all pregnancies except the first pregnancy. Data about PAH from 4 years (2004, 2005, 2007, and 2008) were combined for statistical analysis. The incidence of eclampsia was similarly calculated with data from 13 years (1997 through 2009) and combined for statistical analysis. Furthermore, the data were stratified by racial groups including Hispanic, non-Hispanic white, and non-Hispanic black. The incidence of PAH was evaluated in women who self-reported no tobacco use during the reported pregnancy.

For both PAH and eclampsia, statistical significance was evaluated with Spearman's rank correlation test for ordinal trends and with the Z test for difference of proportions [22,23]. Statistical significance was defined by $P \leqslant .05$. A table for PAH and eclampsia was created to emphasize the trend in the adolescent women. Graphs for PAH and eclampsia were created to emphasize the overall incidence trend for all age groups stratified by race.

This study was approved as an Exemption from IRB Review by the Olive View-UCLA Education and Research Institute IRB.

Results

The sample size was substantially larger for eclampsia than PAH (Table 1). The incidence of PAH decreased with increased age in late adolescence in the G2+ group but not in the G1 group (total and all racial groups) (Table 2). In the total adolescent group (ages ⩽15–19), the incidence of PAH was significantly lower in the G2+ than G1 group

Table 1
Total number of subjects in the study of pregnancy associated hypertension and eclampsia in the United States.[a]

Subjects	PAH[b]	PAH in nonsmokers[b]	Eclampsia[c]
Total subjects reported	16,814,328	16,814,328	53,188,835
Subjects excluded	4,069,296[d]	11,966,723[e]	9,119,696[d]
Total subjects analyzed	12,745,032	4,847,605	44,069,139

[a] Data reported as number of subjects. *Abbreviation:* PAH, pregnancy associated hypertension.
[b] Data from years 2004, 2005, 2007, and 2008 (year 2006 excluded because it was a transition year for diabetes reporting).
[c] Data from years 1997–2009.
[d] Exclusions: subjects who had prepregnancy hypertension; diabetes mellitus (including pregestational and gestational diabetes); or absence of information available about diabetes status, or hypertension status.
[e] Exclusions: subjects who had prepregnancy hypertension; diabetes mellitus (including pregestational and gestational diabetes); tobacco use in pregnancy; absence of information available about diabetes status, hypertension status, or tobacco use in pregnancy.

Table 2
Incidence of pregnancy associated hypertension in adolescents in the United States.[a]

Race	Age (y)	G1			G2+		
		No. subjects with PAH	Total No. subjects in age group	Incidence of PAH (%)	No. subjects with PAH	Total No. subjects in age group	Incidence of PAH (%)
Total							
	⩽15	3958	87215	4.54	169	4869	3.47
	16	6194	139929	4.43	529	17762	2.98
	17	10414	232146	4.49	1468	52868	2.78
	18	15504	332022	4.67	3188	122576	2.60
	19	19921	405654	4.91	5969	228071	2.62
	$P \leqslant$ [b]			NS			.0002
Hispanic							
	⩽15	1290	35783	3.61	65	2194	2.96
	16	2048	57390	3.57	182	7688	2.37
	17	3057	86427	3.54	440	21646	2.03
	18	3789	108963	3.48	807	44951	1.80
	19	4334	120509	3.60	1334	75024	1.78
	$P \leqslant$ [b]			NS			.0001
Non-Hispanic white							
	⩽15	1094	22361	4.89	40	925	4.32
	16	2228	45046	4.95	155	4325	3.58
	17	4472	89714	4.98	506	15634	3.24
	18	7612	147497	5.16	1382	43445	3.18
	19	10932	199899	5.47	2716	92694	2.93
	$P \leqslant$ [b]			NS			.002
Non-Hispanic black							
	⩽15	1574	29071	5.41	64	1750	3.66
	16	1918	37493	5.12	192	5749	3.34
	17	2885	56005	5.15	522	15588	3.35
	18	4103	75562	5.43	999	34180	2.92
	19	4655	85246	5.46	1919	60353	3.18
	$P \leqslant$ [b]			NS			.02

[a] Data reported as number or % subjects. Data from years 2004, 2005, 2007, and 2008 (year 2006 excluded because it was a transition year for diabetes reporting). *Abbreviations:* G1, first pregnancy; G2+, second or higher pregnancy; PAH, pregnancy associated hypertension.
[b] Change in incidence with age. NS, not significant ($P > .05$).

($P < .0001$). The incidence of PAH was significantly lower in the G2+ than G1 group when stratified by race (ages 16–19) ($P < .0001$). The incidence of PAH was also significantly lower in G2+ than G1 women in the third through the fourth decade of life but similar in the 2 groups in the fifth decade of life (Fig. 1) ($P < .0001$). The incidence of PAH increased substantially in both G1 and G2+ groups in the fifth decade of life (total and all racial groups) ($P \leqslant .0001$) (Fig. 1). The incidence of PAH was significantly greater for the non-Hispanic black or non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .02$) except age ⩽15 years (G2+ group) and 45–54 years (both G1 and G2+ groups), which had low birth numbers (Fig. 1). G2+ women who declined tobacco use demonstrate a similar declining incidence of PAH in late adolescence ($P \leqslant .0003$) (Fig. 2).

The incidence of eclampsia decreased with increased age in late adolescence in the G2+ group (total and all racial groups) and the G1 group (total and non-Hispanic black groups) (Table 3). In the total adolescent group (ages ⩽15–19), the incidence of eclampsia was significantly lower in the G2+ than G1 group ($P < .0002$) (Table 3). The incidence of eclampsia was significantly lower in the G2+ than G1 group when stratified by race (ages 16–19) ($P < .0001$). The incidence of eclampsia increased in both G1 and G2+ groups in the fifth decade of life (total and all racial groups) ($P \leqslant .0001$) (Fig. 3). The incidence of eclampsia was significantly greater for the non-Hispanic black than non-Hispanic white and for non-Hispanic white than Hispanic groups for all age groups except age ⩽15 years in the G2+ group, which had low birth numbers (Fig. 3).

Discussion

The present epidemiologic study showed a decreasing incidence of PAH and eclampsia with increased age during adolescence in the G2+ group (Tables 2 and 3), and both G1 and G2+ groups had increased incidence of both PAH and eclampsia in the fifth decade of life (Figs. 1 and 3). These patterns noted were similar in all racial groups. Exclusion of tobacco users did not change the age-related decrease in the incidence of PAH in adolescence for G2+ women (Fig. 2).

PAH is a clinically important outcome measure [16–21,24–26]. The various clinical categories of PAH may have a similar cause and vary in severity of disease. Women who initially have early gestational hypertension are at an increased risk of developing preeclampsia and eclampsia during pregnancy [24]. Gestational hypertension (diastolic

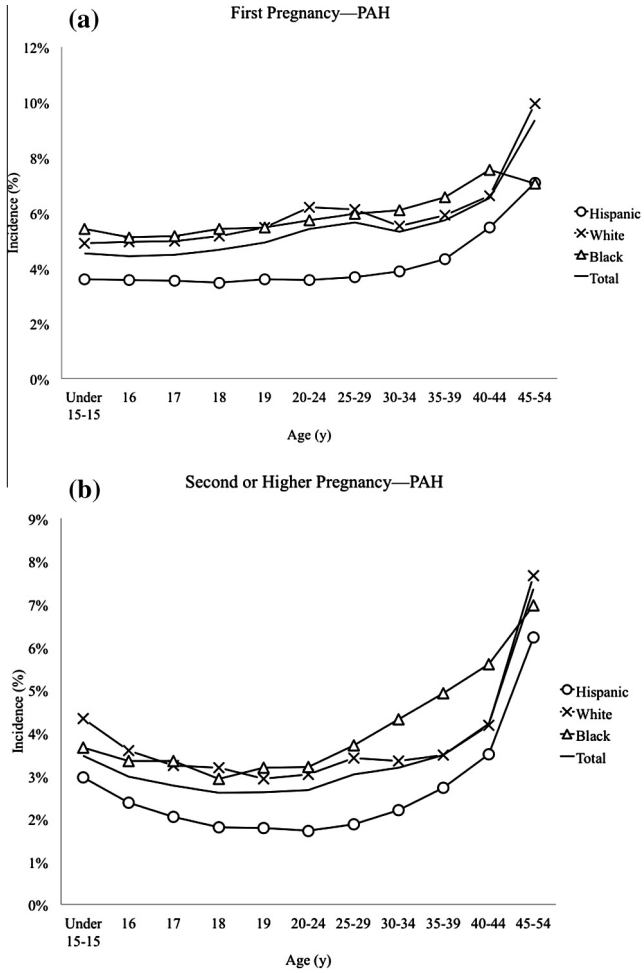

Fig. 1. Relation between the incidence of pregnancy associated hypertension and maternal age for different racial groups in the United States. (a) First pregnancy. Incidence of pregnancy associated hypertension was significantly greater for the non-Hispanic black or non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .02$) except age 45–54 years, which had low birth numbers. (b) Second or higher pregnancy. Incidence of pregnancy associated hypertension was significantly greater for the non-Hispanic black or non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .02$) except age $\leqslant$15 years and 45–54 years, which had low birth numbers.

blood pressure >95 mmHg) is associated with a 3-fold increase in frequency of fetal death.[3] Gestational hypertension and preeclampsia have similar risk factors and are precursors of insulin resistance and future cardiovascular disease [15,17–21].

There are conflicting reports about the risk of preeclampsia in adolescents compared with young adults, in part because most studies lack information about parity or lack sufficient statistical power [1,2,9]. A systematic review of controlled studies about the effect of age concluded that women aged >40 years have a 2-fold increased risk of developing preeclampsia, and no adverse risk was noted in pregnancies in adolescents [2]. Two studies reported similar age-related incidence trends in preeclampsia in adolescence as reported in the present study. A randomized cohort study that used the National Hospital Discharge Survey in the United States reported a decreasing risk of preeclampsia with increased age in adolescence, but the results were not stratified by parity [1]. A randomized cohort study using data from the State of Florida Bureau of Vital Statistics noted an age-related decreasing trend in the incidence pattern in adolescence for preeclampsia [9]. Although the study also did not stratify by parity, parity was considered in a logistic regression model to adjust the incidence results [9]. Neither study excluded women with preexisting hypertension [1,9].

The rank order of risk among racial groups for PAH and eclampsia in the present study is consistent with the prevalence of hypertension in the general population among these racial groups [27–32]. There are few studies available that have compared racial groups for the incidence of hypertensive disorders of pregnancy [33,34]. G1 Hispanic women have an increased risk of preeclampsia and a decreased risk of gestational hypertension than white women [34]. In a study of the New York State hospital discharge database, total hypertensive disorders of pregnancy were more frequent in blacks than Hispanic or white women; black and Hispanic women were at a greater risk

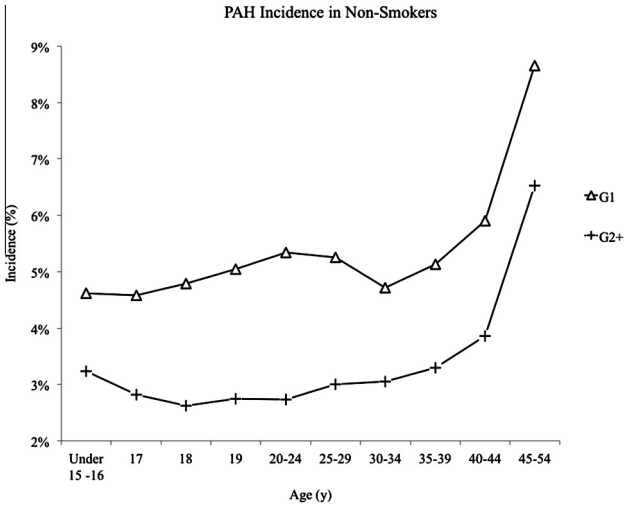

Fig. 2. Relation between the incidence of pregnancy associated hypertension and maternal age for different parity in nonsmoking women in the United States. The incidence of PAH significantly decreased in the G2+ group for ages 15–18 in nonsmoking women ($P \leqslant .0003$).

Table 3
Incidence of eclampsia in adolescents in the United States.[a]

Race	Age (y)	G1			G2+		
		No. subjects with eclampsia	Total No. subjects in age group	Incidence of eclampsia (%)	No. subjects with eclampsia	Total No. subjects in age group	Incidence of eclampsia (%)
Total							
	⩽15	1740	314285	0.55	79	19415	0.41
	16	2128	486260	0.44	217	67897	0.32
	17	3094	787145	0.39	498	196384	0.25
	18	4280	1085436	0.39	967	437472	0.22
	19	4982	1291295	0.39	1647	783026	0.21
	$P \leqslant$ [b]			.0001			.0001
Hispanic							
	⩽15	488	115512	0.42	26	7533	0.35
	16	585	177288	0.33	62	25851	0.24
	17	745	261096	0.29	107	70407	0.15
	18	1002	325165	0.31	227	142216	0.16
	19	1042	358846	0.29	349	233078	0.15
	$P \leqslant$ [b]			NS			.04
Non-Hispanic white							
	⩽15	439	86333	0.51	12	4046	0.30
	16	713	173197	0.41	61	18001	0.34
	17	1230	332614	0.37	178	62945	0.28
	18	2001	514316	0.39	367	165101	0.22
	19	2578	665283	0.39	687	332600	0.21
	$P \leqslant$ [b]			NS			.04
Non-Hispanic black							
	⩽15	813	112440	0.72	41	7836	0.52
	16	830	135775	0.61	94	24045	0.39
	17	1119	193435	0.58	213	63032	0.34
	18	1277	245955	0.52	373	130155	0.29
	19	1362	267166	0.51	611	217348	0.28
	$P \leqslant$ [b]			.03			.03

[a] Data reported as number or % subjects. Data from years 1997–2009. *Abbreviations:* G1, first pregnancy; G2+, second or higher pregnancy.
[b] Change in incidence with age. NS, not significant ($P > .05$).

of developing preeclampsia than white women [33]. The lower prevalence of hypertension in the general population in Hispanic people, despite the higher level of risk factors compared with other racial groups, has been previously noted [34]. Risk factors such as higher body mass index, older age, and glucose intolerance may increase the risk of developing hypertension in people within varied socioeconomic and racial backgrounds. Thus, the

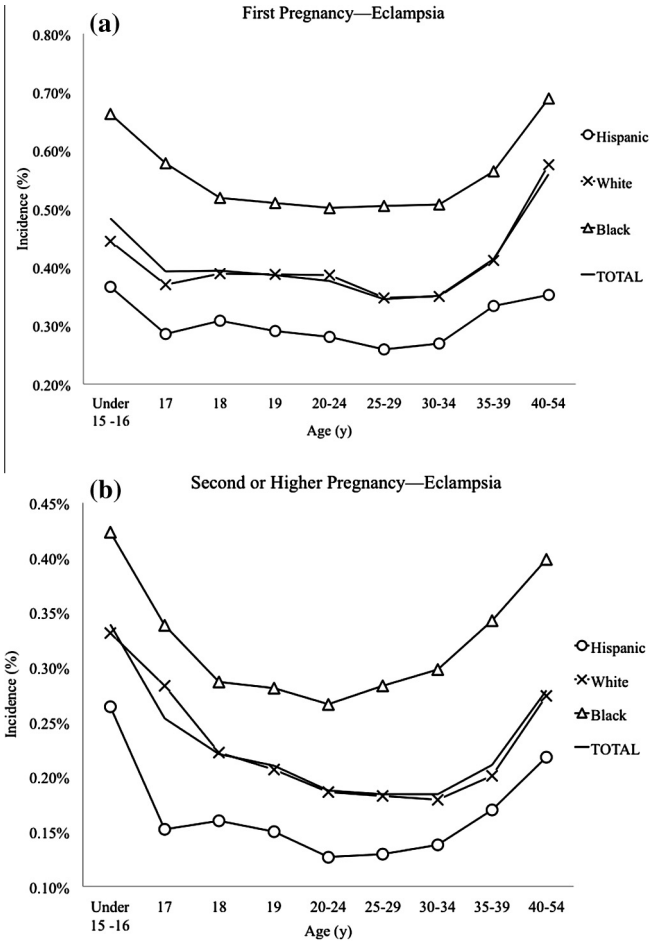

Fig. 3. Relation between the incidence of eclampsia and maternal age for different racial groups in the United States. (a) First pregnancy. Incidence of eclampsia was significantly greater for the non-Hispanic black than non-Hispanic white and for non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .006$). (b) Second or higher pregnancy. Incidence of eclampsia was significantly greater for the non-Hispanic black than non-Hispanic white and for non-Hispanic white than Hispanic groups for all age groups ($P \leqslant .002$) except age $\leqslant$16 years, which had low birth numbers.

overall lower prevalence of hypertension in the Hispanic population may be secondary to genetic, lifestyle, or cultural factors [34].

Tobacco use during pregnancy is associated with a decrease in the risk of developing hypertensive disorders of pregnancy [35]. However, G2+ women who denied tobacco use had a similar decrease in the incidence of PAH in late adolescence (Fig. 2). Therefore, tobacco use is unlikely to have influenced the results in the larger PAH cohort utilized in the present investigation.

Puberty is characterized by physiological increase in insulin resistance. Following puberty a reduction in insulin resistance proceeds into late adolescence [10,11]. The postpubertal reduction in insulin resistance may have caused the decreased incidence of PAH and eclampsia noted in the present study. The increased incidence of PAH and eclampsia in the G1 and G2+ groups in the fifth decade may be associated with the increased incidence of insulin resistance and metabolic syndrome with increased age. The postpubertal decrease in insulin resistance has been documented in several studies [10,11], and this decrease in insulin resistance may continue into the third decade of life [36]. Prepregnancy metabolic syndrome and insulin resistance in early pregnancy are associated with the risk of developing preeclampsia in the future [12,13,15,16]. Specific components of prepregnancy metabolic syndrome are risk factors for developing preeclampsia [16]. Insulin resistance in early pregnancy in nonoverweight primigravidas has a high predictive value for the risk of developing preeclampsia [13]. The future risk of developing the metabolic syndrome and adverse cardiovascular outcomes has been demonstrated in women with a history of preeclampsia [19–21,37,38].

There are 2 distinct categories of risk factors for developing preeclampsia: (1) couple-related risk factors; and (2) factors associated with maternal metabolic syndrome/insulin resistance [39]. Preeclampsia may be considered in 2 stages: (1) poor placental perfusion may cause the elaboration of factors that interact with maternal constitutional factors, resulting in the clinical manifestations of preeclampsia; and (2) maternal constitutional factors may additionally increase the risk of developing abnormal

placentation and decreased placental perfusion [40]. Biochemical changes associated with PAH and other implantation disorders may be recognized in the first trimester prior to vascular remodeling of the failed placenta [40]. The factors released by the poorly perfused placenta may be an appropriate response to overcome reduced nutrient delivery. This appropriate response in conjunction with maternal constitutional factors such as abnormal insulin resistance may result in PAH.

Limitations of the present study include the absence of stratified data about socioeconomic factors, educational level, marital status, and use of prenatal care. The young age of many study subjects precluded the assessment of highest educational level and marital status. Prenatal care, as determined by the graduated index algorithm, was not incorporated because the necessary variables were not available. However, the increased risk of poor obstetric outcomes in adolescent pregnancy may be independent of social status, educational level, and dependent on biologic immaturity [8]. In addition, several potential confounding variables which could have biased the data were not evaluated including prepregnancy high body mass index, multifetal gestation, thrombophilia, connective tissue disease, poor outcome in a previous pregnancy, chronic renal disease, and partner related factors; however, these variables may have little importance in younger women who were the primary focus of this investigation.

Physiologic changes in insulin resistance may be a possible explanation for the observations of the present study. Increased insulin resistance, according to the 2-stage model, may cause abnormal placentation, maternal predisposition to the effects of systemically released factors by the poorly perfused placenta, and PAH [40]. Future studies that directly measure insulin resistance before and early in pregnancy may be useful to identify further risk factors for PAH.

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Conflict of interest statement

The authors have declared that no competing interests exist.

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