

## Anemia in Pregnancy and Race in the United States: Blacks at Risk

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**Objectives:** This study's objectives were to determine the national prevalence of anemia in pregnancy (AIP) in the United States, compare racial differences in the prevalence, compare the AIP risk factor profiles between non-Hispanic whites and non-Hispanic blacks, and to analyze the associations between AIP and some maternal and fetal/neonatal complications between whites and blacks.

**Methods:** The data used were from the United States natality data files (1995 through 2000), which included 23,654,695 live births. All mothers diagnosed with AIP, defined as hemoglobin-concentration <10 g/dl, were included. The cohorts were analyzed in two groups. The "whole group" (WG) comprised all women in the data set who had anemia status reported. The "low-risk group" (LRG) comprised women with low-risk factors for AIP. Race was determined by mothers' skin colors and racial self-identifications. Logistic regression was used to explore associations between race and AIP while controlling for other covariates. **Results:** The national prevalence of AIP among the general population was 21.55/1,000 among the WG and 11.51/1,000 among the LRG. Among the WG, the prevalence of AIP was two times higher among non-Hispanic blacks (35.38/1,000) than among non-Hispanic whites (18.02/1,000). Among the LRG, the prevalence was 1.94 times higher among non-Hispanic blacks (20.44/1,000) than among non-Hispanic whites (10.63/1,000). The other risk factor profiles for AIP were similar among the races. Many serious maternal and fetal/neonatal complications occurred more frequently among anemic patients when compared with non-anemic patients. **Conclusions:** The results of this study showed that black race was significantly associated with higher risk of AIP. The other risk factor profiles of AIP were significantly similar between whites and blacks. This study also confirmed that AIP was significantly associated with some serious maternal and fetal/neonatal complications. The findings of this study indicate that race is an important risk factor of AIP.

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Anemia in pregnancy (AIP) is a condition with effects that may be deleterious to mothers and fetuses. Indeed, AIP is a known risk factor for many maternal and fetal complications. Maternal complications include premature labor, poor weight gain, and dysfunctional labor.<sup>1-6</sup> Fetal or neonatal complications include prematurity, low birth weight, poor Apgar score, fetal distress, and neonatal distress, requiring prolonged resuscitation and causing neonatal anemia due to poor reserve.<sup>1,2,4-8</sup> Infants with anemia have higher prevalence of failure to thrive, poorer intellectual developmental milestones, and higher rates of morbidities and neonatal mortalities than infants without anemia.<sup>1,2,7-9</sup> Moreover, babies whose mothers had AIP during their first trimester *in utero* experienced higher rates of cardiovascular mor-

bilities and mortalities in their adult lives than babies whose mothers did not have AIP.<sup>10</sup>

A search of the Ovid MEDLINE 1966 to 2004 database yielded 399 articles, using "anemia and prevalence" as the keywords. Only two of these articles reported prevalence of anemia among women in the United States. Feroli et al reported a 30% prevalence of anemia (hematocrit <35%) among preadolescent African American females in local schools.<sup>11</sup> Ania reported anemia prevalence to be 12.4% in Minnesota.<sup>12</sup> No racial comparisons in the prevalence of anemia were performed in either of these studies. The same database search yielded six articles using "prevalence of anemia in pregnancy" (PAIP) as the keywords.<sup>13-19</sup> None of these six studies were conducted in the United States. Thus, the statistics on the national and racial PAIP were limited, and most of the existing anemia studies were locally conducted and focused on specific populations such as individuals with low income.<sup>1,11,12,20</sup>

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Race is recognized as an important risk factor for infant anemia and in screening for anemia in infants.<sup>21-23</sup> Race is, however, not currently recognized as a risk factor of AIP.<sup>1,20,23,24</sup> Understanding racial differences in the PAIP may help in identifying factors that may be responsible for differences in racial prevalence of many pregnancy-related complications<sup>1,3-6,10,25</sup> and thus help in planning prevention strategies.

This study had four objectives. The first was to determine the national and racial PAIP. The second, and the primary objective, was to compare the PAIP between whites and blacks. The third was to compare the risk factors for AIP between blacks and whites. The fourth objective was to identify associations between AIP and selected maternal and fetal/neonatal complications in blacks and whites.

## Methods

### Data Description

Data for this cross-sectional cohort study were derived from the US natality data files from 1995 to 2000.<sup>26</sup> These data were collected by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention under a cooperative agreement with all 50 states and the District of Columbia. These data files are produced every year. They include statistical data from births that are provided to the NCHS by the individual states under the Vital Statistics Cooperative Program.<sup>3,27</sup> The data conform to uniform coding specifications and have passed rigorous statistical quality checks.<sup>3,27</sup>

The studied variables were obtained from the aforementioned database. AIP was defined as hemoglobin concentration less than 10 g/dL.<sup>26</sup> The diagnosis was made from routine blood tests done during the first prenatal visit and was recorded on the US national standard birth certificates with the use of a check-box format.<sup>26,28-32</sup>

### Other Variables

Tables 1 and 2 depict other variables reported in the data and analyzed in this study. Race was determined by mothers' self-identifications. The reported racial groups included whites, blacks, American Indians, Asian Indians, Chinese, Japanese, Hawaiian, Filipi-

Table 1

### AIP Risk Factors Reported in the National Center for Health Statistics 1995–2000 Natality Data Set

- Sociodemographic factors (age, race, level of formal education, marital status, areas and cities of residence)
- Obstetrical factors (gravidity, parity, history of previous preterm or small-for-gestational-age deliveries, plurality of pregnancy—multiple or singleton)
- Behavioral factors (smoking or tobacco usage, alcohol usage, utilization of prenatal care services)
- Medical conditions (diabetes, renal or cardio-respiratory diseases, chronic hypertension—blood pressure  $\geq 140/90$  or usage of antihypertensive medication prior to 20 weeks' completed gestation)

AIP—anemia in pregnancy

nos and Vietnamese.<sup>26</sup> The remaining minority races, including Pacific Islanders, were classified as "others" in the NHCS data set. Races were digitally coded as "01" for whites, "02" for blacks, and so on. Code "99" was used for those whose racial status was not reported. Race was further subcategorized as either Hispanic or non-Hispanic. Of the 23,654,695 live births documented in the data, 98.6% (23,326,064) had their racial and anemia status reported. We recoded the racial groups into non-Hispanics and Hispanics.

Table 2

### Maternal and Fetal Complications Reported

Maternal Complications	Fetal and Neonatal Complications
Premature rupture of membrane (rupture of membranes $>12$ hours before labor's onset)	Low birth weight
Premature labor (labor's onset before 37 completed weeks of gestation)	Prematurity
Premature delivery (delivery before 37 completed weeks of gestation)	Neonatal morbidities (low Apgar scores, birth asphyxia, meconium aspiration, respiratory distress, hyaline membrane syndrome and neonatal anemia).
Uterine bleeding	
Hydramnios	
Pregnancy-induced hypertension (PIH) (Blood pressure [BP] of $\geq 140/90$ mmHg after 20 completed weeks of gestation or increase in systolic BP of $\geq 30$ mmHg or diastolic BP $\geq 15$ mmHg above the preconception baseline BP)	Congenital anomalies (cardiorespiratory, central nervous system, renal agenesis and other genitourinary anomalies, omphalocele, gastroschisis and other gastrointestinal anomalies, digital and chromosomal anomalies)
Preeclampsia (PIH plus proteinuria)	
Eclampsia (preeclampsia plus convulsion)	
Seizure without PIH	
Weight gain	
Placenta previa	
Placenta abruption	
Anesthetic complications	

### Statistical Analysis

The determination of the national and racial PAIP was performed by analyses of the cohort as a “whole group” (WG), which included all 23,326,064 live births who had racial and anemia status reported. Non-Hispanic white race (henceforth referred to as whites) was used as the referent group for all racial comparisons.

The differences in PAIP between non-Hispanic blacks (henceforth referred to as blacks) and whites, as analyzed in the WG, were further explored by comparing the racial PAIP in the low-risk group (LRG) that was computed out of the WG. The inclusion criteria into the LRG are listed in Table 3. A total of 475,518 (2%) cases out of the 23,326,064 live births that constituted the WG met these criteria. These criteria were based on the known risk factors for AIP<sup>1,33-35</sup> and the factors that had statistically significant associations with AIP in our analyses. Though there is currently no standard definition of an “AIP low-risk pregnancy,” it is plausible that women who met the aforementioned criteria were less likely to have AIP than those who did not meet these criteria.

The analyzed AIP risk factors in whites and blacks were chosen based on the already known AIP risk factors<sup>1,33-35</sup> and those factors that showed statistically significant associations with AIP in our chi-square and regression analyses. Selected maternal and fetal/neonatal complications were analyzed to determine the associations between these complications and AIP in blacks and whites.

The statistical analyses were performed with SPSS software (version 10.0). Unadjusted odds ratios (ORs), with 95% confidence intervals (CIs), were derived from chi-square analyses and were used to estimate the relative risk (RR) since this was a cohort study.<sup>33</sup> Adjusted ORs were derived from logistic regression. Statistically significant associations were those with 95% CI without 1 and *P* value less than .05.

The choice of risk factors for inclusion in the logistic regression analyses was based on the results of bivariable analyses or the already-documented known risk factors for AIP.<sup>1,33-35</sup> Since the data used in this study were from a very large data set, we realized that a small difference in the PAIP might be significantly magnified. We therefore chose unadjusted RR >1.25 or <0.75 as a criterion for inclusion of risk factors (variables) in the logistic regression analysis if such variables had not been documented as known risk factors for AIP.<sup>1,3,33-35</sup>

For the purpose of this study, early prenatal care was considered as prenatal registration within the first 2 months of pregnancy. This is because the developing fetus is more sensitive to deleterious conditions, like the effects of anemia, during embryogenesis and organogenesis that mostly occur during the early weeks of gestation.<sup>10,36-38</sup>

Table 3

### Inclusion Criteria of the Low-risk Group

- Ages between 20 and 29 years
- Attained  $\geq$  16 grades of former education
- No smoking or tobacco use
- Free from any medical conditions like hemoglobinopathy, chronic hypertension, diabetes, or renal or cardiovascular disease
- No previous preterm delivery
- Married
- Primigravida with singleton pregnancy
- No alcohol use
- Free from obstetrical complications like placenta abruption, placenta previa, bleeding, rhesus incompatibility, pregnancy-induced hypertension, and eclampsia
- No previous history of small-for-gestational-age deliveries
- Commencement of prenatal care and screening for anemia in pregnancy within the first 2 months of gestation

### Results

A total of 502,728 (21.55/1,000) cases out of the studied 23,326,064 live births had AIP. The WG population distributions were 60.9% whites, 15.1% blacks, and .98% American Indians. PAIP was two times higher in blacks (35.38/1,000; RR=2) than in whites (18.02/1,000).

### Prevalence

The PAIP dropped across all races in the LRG when compared with the corresponding prevalence in the WG. The PAIP was 10.6/1,000 in whites and 20.4/1,000 in blacks in the LRG. PAIP in American Indians (12.4/1,000) dropped to about the same level as the PAIP in whites in the LRG, with unadjusted RR of 1.17, in contrast to the unadjusted RR of 2.8 resulting from the PAIP of 50.5/1,000 in American Indians in the WG when compared with the PAIP of 18.0/1,000 in whites in the same WG. The racial difference in the prevalence of AIP was, however, still maintained between blacks (20.4/1,000) and whites (10.6/1,000) with unadjusted RR of 1.94 in the LRG.

### Sociodemographic Factors

The PAIP was inversely proportional to the mothers’ ages until the age of 35, when the prevalence leveled off. Teenage mothers had the highest prevalence in all races, while mothers between ages 35 and 39 had the lowest prevalence. The higher the mother’s attained level of formal education, the lower the observed PAIP. Increased parity, unmarried status, multiple pregnancy, and nonmetropolitan residence were all associated with higher PAIP. The observed PAIP increased directly proportionally to the delay in registration for prenatal care. The risk factors for AIP were similar in whites and blacks, though lack of formal education had stronger statistical significance in blacks (unadjusted RR=1.95)

when compared to in whites (unadjusted RR=1.30). The PAIP was consistently about two times higher in blacks than in whites in almost every group.

#### Parity

There was significant increase in the PAIP with each additional increase in parity, irrespective of the race. PAIP was 16.7/1,000 in primiparous as compared to 21.8/1,000 in pentaparous (para five) whites (RR=1.31, 95% CI=1.27–1.36,  $P<.000001$ ). The same trend was observed in blacks with PAIP of 31.8/1,000 in primiparous and 38.1/1,000 in pentaparous blacks (RR=1.21, 95% CI=1.16–1.25,  $P<.000001$ ), though there was less dramatic change in the PAIP in blacks in comparison with whites from para four and above.

#### Pregnancy Complications

Women with previous history of preterm or premature deliveries had a 2.4 times higher risk of developing AIP among whites (17.7/1,000 versus 41.2/1,000, 95% CI=2.33–2.44,  $P<.00001$ ) and a 2.8 times higher risk among blacks (34.5/1,000 versus 91.1/1,000, 95% CI=2.72–2.89,  $P<.00001$ ) when compared with women without such history.

#### Other Medical Problems

Hemoglobinopathies and renal, cardiac, and lung diseases had strong statistically significant associations with AIP in the two racial groups (Table 4). Tobacco usage during pregnancy was associated with a 1.35 times higher risk of AIP in both races, while alcohol usage was associated with a 1.59 times higher risk. A higher percentage of whites used tobacco, while a higher percentage of blacks used alcohol during pregnancy. The risk factors for AIP were once again similar between the two racial groups, though hemoglobinopathy had stronger association with AIP in whites (unadjusted RR=11.3) than in blacks (unadjusted RR=4.8).

Uterine bleeding, or other forms of excessive bleeding,

followed by rhesus incompatibility, placenta previa, and then placenta abruption were strongly associated with AIP in that descending order. The risk factors for AIP were similar in whites and blacks, though there was a little stronger association between eclampsia and AIP in whites (unadjusted RR=2.2) when compared to in blacks (unadjusted RR=1.7). Note the close to two times higher PAIP in blacks as compared to whites in almost every compared risk factor.

The majority of blacks delayed prenatal care registration. Hemoglobinopathies, lung diseases, and rhesus incompatibility had the strongest adjusted statistically significant associations with AIP in blacks, while renal diseases, rhesus incompatibility, and alcohol use had the strongest statistically significant associations with AIP in whites in logistic regression analyses. The regression analysis showed that black race was a significantly associated risk factor for AIP with adjusted  $P<.001$ .

Table 4

Maternal Medical Comorbidities and Tobacco/Alcohol Use in Association With Prevalence of AIP Among Non-Hispanic Whites and Blacks

Mothers' Comorbidity	Non-Hispanic Whites		Non-Hispanic Blacks	
	Prevalence Cases/1,000 (n)	Unadjusted RR (95% CI)	Prevalence Cases/1,000 (n)	Unadjusted RR (95% CI)
Cardiac disease				
Absent	17.77 (250,857)	1.00 (referent)	35.06 (122,908)	1.00 (referent)
Present	55.80 (5,146)	3.27 (3.18–3.36)	111.3 (1,652)	3.45 (3.27–3.63)
Lung disease				
Absent	17.55 (246,722)	1.00 (referent)	34.48 (119,828)	1.00 (referent)
Present	63.75 (9,281)	3.81 (3.73–3.90)	105.3 (4,732)	3.29 (3.20–3.40)
Diabetes				
Absent	17.79 (246,084)	1.00 (referent)	35.25 (121,014)	1.00 (referent)
Present	26.68 (9,919)	1.51 (1.48–1.55)	40.43 (3,546)	1.15 (1.11–1.19)
Hemoglobinopathy				
Absent	17.96 (255,126)	1.00 (referent)	35.04 (122,960)	1.00 (referent)
Present	171.36 (877)	11.3 (10.5–12.2)	148.5 (1,600)	4.80 (4.55–5.07)
Chronic hypertension				
Absent	17.97 (253,499)	1.00 (referent)	35.23 (122,464)	1.00 (referent)
Present	25.27 (2,504)	1.42 (1.36–1.48)	47.23 (2,096)	1.36 (1.30–1.42)
Renal disease				
Absent	17.83 (251,146)	1.00 (referent)	35.14 (122,066)	1.00 (referent)
Present	82.11 (3,660)	4.93 (4.76–5.10)	162.9 (1,287)	5.34 (5.03–5.67)
Tobacco use				
No	17.09 (175,920)	1.00 (referent)	34.44 (99,136)	1.00 (referent)
Yes	23.42 (47,037)	1.38 (1.37–1.39)	45.91 (14,301)	1.35 (1.33–1.37)
Alcohol use				
No	18.74 (239,977)	1.00 (referent)	36.40 (117,404)	1.00 (referent)
Yes	27.53 (4,286)	1.48 (1.44–1.53)	60.01 (3,144)	1.69 (1.63–1.75)

AIP—anemia in pregnancy, RR—relative risk, CI—confidence interval, n—number of total cases of patients with the corresponding condition.  $P<.00001$  for all comparisons.

Table 5

## Associations Between Few Maternal and Fetal/Neonatal Complications and Anemia in Pregnancy Among Non-Hispanic Whites and Blacks

Maternal Characteristics	Non-Hispanic White Mothers		Non-Hispanic Black Mothers	
	Non-anemic Cases/1,000 [# of cases] (Referent)	Anemic Cases/1,000 [# of cases] (RR: 95% CI)	Non-Anemic Cases/1,000 [# of cases] (Referent)	Anemic Cases/1,000 [# of cases] (RR: 95% CI)
Tocolysis performed	23.15 [322,636]	42.93 [10,979] (1.89: 1.86–1.93)	20.89 [70,867]	39.12 [4,868] (1.91: 1.85–1.97)
Precipitous labor < 3 hours	20.75 [288,676]	28.65 [7,311] (1.39: 1.36–1.43)	20.76 [70,362]	35.97 [4,468] (1.76: 1.71–1.82)
Dysfunctional labor	29.17 [405,899]	49.45 [12,618] (1.73: 1.70–1.76)	24.65 [83,553]	42.06 [5,225] (1.74: 1.69–1.79)
Anesthetic complication	0.68 [8,958]	2.27 [524] (3.32: 3.04–3.63)	0.52 [1,643]	1.39 [156] (2.68: 2.27–3.16)
Maternal seizure	0.29 [4,059]	0.56 [142] (1.91: 1.61–2.26)	0.52 [1,770]	0.86 [107] (1.65: 1.36–2.01)
Incompetent cervix	2.63 [36,744]	5.44 [1,393] (2.07: 1.96–2.19)	4.50 [15,272]	7.02 [875] (1.57: 1.46–1.68)
Maternal herpes	10.33 [135,774]	23.64 [5,482] (2.32: 2.26–2.38)	10.09 [32,021]	23.52 [2,647] (2.36: 2.27–2.46)
Hydramnios	12.15 [169,522]	22.88 [5,858] (1.90: 1.85–1.96)	15.44 [52,435]	28.38 [3,535] (1.86: 1.80–1.93)
<b>Fetal/Neonatal Characteristics</b>				
Fetal distress	39.47 [517,166]	59.25 [13,692] (1.53: 1.51–1.56)	51.37 [162,604]	72.93 [8,185] (1.45: 1.42–1.49)
Neonatal anemia	0.99 [13,721]	4.16 [1,056] (4.20: 3.94–4.47)	1.28 [4,294]	7.00 [863] (5.50: 5.11–5.92)
Meconium aspiration syndrome	1.97 [27,131]	3.51 [892] (1.79: 1.67–1.91)	2.91 [9,783]	5.56 [686] (1.91: 1.77–2.07)
Fetal alcohol syndrome	0.04 [560]	0.09 [23] (2.26: 1.49–3.43)	0.11 [349]	0.21 [25] (1.99: 1.33–2.99)*
Assisted ventilation >30 minutes	9.28 [125,844]	18.10 [4,556] (1.97: 1.91–2.03)	11.34 [35,718]	19.01 [2,258] (1.69: 1.62–1.76)
Cardiac anomalies	1.32 [18,169]	2.21 [554] (1.67: 1.54–1.82)	1.04 [3,435]	2.29 [278] (2.22: 1.96–2.50)
Omphalocele or gastroschisis	0.29 [3,998]	0.39 [98] (1.34: 1.10–1.64)+	0.33 [1,107]	0.69 [84] (2.08: 1.66–2.59)
Genital anomalies	0.93 [12,715]	1.95 [489] (2.11: 1.93–2.31)	0.54 [1,774]	0.94 [114] (1.76: 1.45–2.12)
Musculoskeletal anomalies	1.95 [26,738]	3.58 [897] (1.84: 1.72–1.97)	2.33 [7,737]	7.78 [943] (3.35: 3.13–3.59)

RR=relative risk, CI=confidence interval,  $P<.00001$  except \*  $P=.0001$  and +  $P=.004$ .

AIP was significantly associated with higher incidence of many maternal and fetal/neonatal complications in blacks and whites, with unadjusted RR ranging between 1.5 and 5.5 (Table 5). The notable maternal complications that were overrepresented among anemic mothers included hydramnios, anesthetic complications, maternal seizure, dysfunctional labor, incompetent cervix, herpes, and requirement of tocolysis. Neonatal anemia, fetal distress, meconium aspiration syndrome, fetal alcohol syndrome, requirement

of assisted ventilation for more than 30 minutes, and systemic organ congenital anomalies were some of the serious complications that were overrepresented among anemic mothers' offspring in comparison to the offspring of the mothers without AIP (Table 5).

## Discussion

This study established that, using the hemoglobin concentration of 10 g/dl as the cutoff point for definition of AIP, the prevalence of AIP was two times higher among blacks (35/1,000 in WG, RR=2.0 and 20/1,000 in LRG, RR=1.9) than among whites (18/1,000 in WG and 10/1,000 in LRG), irrespective of presence or absence of other AIP risks factors, with the national prevalence of AIP being 22/1,000 between 1995 and 2000 in the United States. It also showed that the risk factors for AIP were similar in whites and blacks. The study further confirmed strong associations between AIP and many maternal and fetal/neonatal complications in whites and blacks.

This study also showed that black race, maternal age, marital status, parity, socio-economic factors as measured by the level of education attained, use of alcohol and/or tobacco during pregnancy, presence of conditions like hemoglobinopathy, renal disease, cardiac disease, and lung disease were all significant risk factors associated with AIP. Many of these risk factors for AIP identified in this study have been documented and recognized in management and prevention of AIP.<sup>1,33–35</sup> Black race, however, has not been officially recognized as a significant risk factor for AIP in the United States. Moreover, this would be the first nationally conducted study on AIP to determine the national and racial PAIP and the racial differences in the AIP prevalence rates in the United States.

### Limitations

The strengths of this study include the fact that we studied large, nationally collected and accepted natality data.<sup>3,26</sup> The detailed information provided in the data gave us opportunity to compare the PAIP in whites and blacks, both in the presence and absence of all recognized risk factors of AIP.<sup>1,3,33-35</sup> We were also able to confirm the serious complications that AIP may be associated with both in the mothers and their offspring (Table 5).<sup>1,3-6,10,25</sup>

Despite those strengths, the study had limitations. One important limitation of this study was the fact that the hemoglobin concentration cutoff point of 10 g/dl, used in defining AIP in the studied data,<sup>26</sup> might be associated with significant underreporting of the actual prevalence of AIP. The current Centers for Disease Control and Prevention recommended cutoff points for definition of AIP are 11.0 g/dl for the first and third trimester and 10.5 g/dl for the second trimester of pregnancy.<sup>1,2,33,34,39,40</sup> This may partly explain the lower prevalence of AIP observed in this study when compared with the reports of other local and international studies on AIP.<sup>1,4,13-19,23</sup> This limitation, however, would not significantly affect the observed racial differences in PAIP between whites and blacks.

Other limitations include arguments about the racial differences in the prevalence of risk factors for AIP as possible explanations for the observed racial differences in prevalence of AIP. The notable risk factors that may have racial predilection include socioeconomic and demographic factors (age, level of education, income level), nutritional (especially iron deficiency-related anemia), hemoglobinopathies (like sickle cell disease), behavioral (smoking, alcohol usage, parity, utilization of prenatal care), maternal conditions, and obstetrical history.<sup>1,3,33-35</sup> Though the income levels of the participants were not reported in the studied data, it is plausible to roughly deduce these economic strata from the attained former educational levels of the participants since strong correlations have been established between the attained levels of former education and income.<sup>41-43</sup> Persistence of the racial difference in the prevalence of AIP between whites and blacks after controlling for all these risk factors both in the LRG and in the logistic regression analyses implied that black race is a factor on its own, with strong association with AIP. Indeed, iron deficiency, the globally recognized most common cause of anemia,<sup>4,44,46-48</sup> has been reported not to be more common in blacks when compared to whites in the study of Lazebnik et al, who reported 7.6 ng/ml higher average level of ferritin (the marker of iron storage level) in pregnant black women than in pregnant white women.<sup>49</sup> Thus, the possibility of higher level of iron deficiency anemia of any cause in blacks could not explain this observed twofold higher prevalence of AIP in blacks than in whites.

The timing of AIP diagnosis may be another limitation, in that timing may affect the result of the quantitative hemoglobin measurement as a result of hemodilution that occurs with pregnancy.<sup>2,33,34,39,40</sup> This could not, however, explain the persistence of the racial differences in the PAIP after controlling for this factor in the regression analysis and in the LRG, where one of the inclusion criteria was registration for prenatal care and screening for AIP during the first 2 months of gestation (Table 3).

### Conclusions

The Centers for Disease Control and Prevention's (CDC) recommendations for screening for anemia among childbearing-age women state that "Starting in adolescence, screen all nonpregnant women for anemia every 5–10 years throughout their childbearing years during routine health examinations. Annually screen for anemia in women having risk factors for iron deficiency (eg, extensive menstrual or other blood loss, low iron intake, or a previous diagnosis of iron-deficiency anemia)."<sup>51</sup> This guideline and other existing national clinical guidelines do not recognize black race as a risk factor for AIP that may warrant screening for anemia more frequently than once every 5–10 years in asymptomatic blacks of childbearing age to prevent AIP in this at-risk group.<sup>20,23,24</sup>

Many women complete their childbearing within 5 to 10 consecutive years of their lives. Performing anemia screening only once every 5–10 years may lead to failure to diagnose and treat asymptomatic anemia among childbearing-age blacks before they become pregnant. More-frequent anemia screening and treatment of any diagnosed anemia in these at-risk blacks of childbearing age group may lead to significant reduction in the prevalence of AIP in this racial group and may result in significant reduction in the prevalence of the serious maternal and fetal/neonatal complications associated with AIP.

We recommend that black race should be recognized as a risk factor of AIP that warrants annual or biennial anemia screening among childbearing-age black women. Health care providers who take care of black childbearing-age women may also help reduce the prevalence of AIP in this racial group by counseling them to have a balanced diet and take supplemental iron as part of preparation for conception.

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## REFERENCES

1. Recommendations to prevent and control iron deficiency in the United States. *Morb Mortal Wkly Rep* 1998;47(RR-3)April 3:1-36.
2. Taylor DJ, Mallen C, McDougall N, Lind T. Effect of iron supplementation on serum ferritin levels during and after pregnancy. *Br J Obstet Gynecol* 1982;89:1011-7.
3. Ananth CV, Demissie K, Smulian JC, Vintzileos AM. Placenta previa in singleton and twin births in the United States, 1989 through 1998: a comparison of risk factor profiles and associated conditions. *Am J Obstet Gynecol* 2003;188(1):275-81.
4. Klebanoff MA, Shiono PH, Selby JV, Trachtenberg AI, Graubard BI. Anemia and spontaneous preterm birth. *Am J Obstet Gynecol* 1991;164(1 Pt 1):59-63.
5. Lieberman E, Ryan KJ, Monson RR, Schoenbaum SC. Association of maternal hematocrit with premature labor. *Am J Obstet Gynecol* 1988;159(1):107-14. Comments in: *Am J Obstet Gynecol* 1989;161 (6 part 1):1750-1 and *Am J Obstet Gynecol* 1990;162(1):293-5.
6. Scholl TO, Hediger ML, Fischer RL, Shearer JW. Anemia versus iron deficiency: increased risk of preterm delivery in a prospective study. *Am J Clin Nutr* 1992;55:985-8.
7. Pritchard JA, Cunningham FG, Pritchard SA, Mason RA. On reducing the frequency of severe abruptio placentae. *Am J Obstet Gynecol* 1991;165(5 Pt 1):1345-51. Comment in: *Am J Obstet Gynecol* 1993;168(5):1641-2.
8. Denmead DT, Ariagno RL, Carson SH, Benirschke KB. Placental pathology is not predictive for sudden infant death syndrome (SIDS). *Am J Perinatol* 1987;4(4):308-12.
9. Wu AC, Lesperance L, Bernstein H. Screening for iron deficiency. *Pediatrics in Review* 2002;23(5):171-8.
10. Kadyrov M, Kosanke G, Kingdom J, et al. Increased fetoplacental angiogenesis during first trimester in anaemic women. *Lancet* 1998;352: 1747-9.
11. Feroli K, Hobson S. Defining anemia in a preadolescent African American population. *J Pediatr Health Care* 1995;9(5):199-204.
12. Ania BJ, Suman VJ, Fairbanks VF, Melton LJ III. Prevalence of anemia in medical practice: community versus referral patients. *Mayo Clin Proc* 1994;69(8):730-5. Comments in: *Mayo Clin Proc* 1994;69(8): 808-9.
13. Xiong X, Buekens P, Fraser WD, Guo Z. Anemia during pregnancy in a Chinese population. *Int J Gynaecol Obstet* 2003;83(2):159-64.
14. Mardones F, Rioseco A, Ocqueteau M, et al. [Anemia in pregnant women from the community of Puento Alto, Chile]. [Spanish] *Revista Medica de Chile* 2003;131(5):520-5.
15. Sharma JB, Soni D, Murthy NS, Malhotra M. Effect of dietary habits on prevalence of anemia in pregnant women of Delhi. *J Obstet Gynaecol Res* 2003;29(2):73-8.
16. Marti-Carvajal A, Pena-Marti G, Comunian G, Munoz S. Prevalence of anemia during pregnancy: results of Valencia (Venezuela) anemia during pregnancy study. *Archivos Latinoamericanos de Nutricion* 2002;52(1):5-11.
17. Nurdia DS, Sumarni S, Suyoko Hakim M, Winkvist A. Impact of intestinal helminth infection on anemia and iron status during pregnancy: a community-based study in Indonesia. *Southeast Asian J Trop Med Public Health* 2001;32(1):14-22.
18. Massawe SN, Urassa EN, Nystrom L, Lindmark G. Effectiveness of primary level antenatal care in decreasing anemia at term in Tanzania. *Acta Obstet Gynecol Scand* 1999;78(7):573-9.
19. Becerra C, Gonzales GF, Villena A, de la Cruz D, Florian A. [Prevalence of anemia in pregnancy, Pucallpa Regional Hospital, Peru]. [Spanish] *Pan American Journal of Public Health* 1998;3(5):285-92.
20. Guide to clinical preventive services, second edition. (1996) Section one, Screening; Part C. Metabolic, nutritional, and environmental disorders. 1. No recommendation for screening asymptomatic non-pregnant women. 2. The prevalence of AIP among pregnant women not known. <http://hstat.nlm.nih.gov/hquest/db/local.gcps.cps/screen/browse/s/62501/cmd/hf/action/gettext?ihr=ch22>.
21. American Academy of Family Physicians. Summary of policy recommendations for periodic health examination. In: American Academy of Family Physicians reference manual, 2000. [www.aafp.org/exam/](http://www.aafp.org/exam/)
22. US Preventive Services Task Force. Screening for iron deficiency anemia, including iron prophylaxis. In: Guide to clinical preventive services: report of the US Preventive Services Task Force, second edition. Baltimore: Williams & Wilkins, 1996:231-46.
23. Guide to clinical preventive services, second edition. (1996) Section one, Screening; Part C. Metabolic, nutritional, and environmental disorders. 1. No recommendation for screening asymptomatic child bearing-aged women. 2. Black kids to screen. <http://hstat.nlm.nih.gov/hquest/db/local.gcps.cps/screen/browse/s/62705/cmd/hf/action/gettext?ihr=ch22cnt>.
24. US Preventive Services Task Force, 1996. No recommendation on screening for AIP among asymptomatic non-pregnant women. [www.aepublic.org/clinic/uspsft/uspsiron.htm](http://www.aepublic.org/clinic/uspsft/uspsiron.htm).
25. Gambling L. Effect of iron deficiency on placental cytokine expression and fetal growth in the pregnant rat. *Biol Reprod* 2002;66(2):516-23.
26. Natality data sets, 1995 to 2000. Washington, DC: National Center for Health Statistics, Centers for Disease Control and Prevention, United States Department of Health and Human Services, 2000.
27. MacDorman MF, Atkinson JO. Infant mortality statistics from the linked birth/infant death data set—1995 period data. Monthly vital statistics report; vol 46 (2 suppl). Hyattsville, Md: National Center for Health Statistics, 1998.
28. Buescher PA, Taylor KP, Davis MH, Bowling JM. The quality of the new birth certificate: a validation study in North Carolina. *Am J Public Health* 1993;83:1163-5.
29. Dobie SA, Baldwin LM, Rosenblatt RA, Fordyce MA, Andrilla CH, Hart LG. How well do birth certificates describe the pregnancies they report? The Washington state experience with low-risk pregnancies. *Matern Child Health J* 1998;2:145-54.
30. Taffel SM, Ventura SJ, Gay GA. Revised US certificate of birth—new opportunities for research on birth outcome. *Birth* 1989;16:188-93.
31. Taffel S, Johnson D, Heuser R. A method of imputing length of gestation on birth certificates. *Vital Health Stat* 2 1982;93:1-11.
32. Zhang J, Yu FK. What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *JAMA* 1998;280: 1690-1.
33. Current trends. CDC criteria for anemia in children and childbearing-aged women. *Morb Mortal Wkly Rep CDC* 1989;38(June 9):400-4.
34. Sjostedt JE, Manner P, Nummi S, Ekenved G. Oral iron prophylaxis during pregnancy: a comparative study on different dosage regimens. *Acta Obstet Gynecol Scand Suppl* 1977;60:3-9.
35. Perry GS, Yip R, Zyrkowski C. Nutritional risk factors among low-income pregnant US women: the Centers for Disease Control and Prevention (CDC) Pregnancy Nutrition Surveillance System, 1979 through 1993. *Semin Perinatol* 1995;19(3):211-21.
36. NSAID danger around conception. [www.merckmedicus.com/pps/us/hcp/frame\\_micromedex.jsp?pg=http%3A%2F%2Fmerck.micro-medex.com%2Findex.asp%3Fpage%3Dnewsarchive%26news\\_id%3D5482%26news%3DMDM](http://www.merckmedicus.com/pps/us/hcp/frame_micromedex.jsp?pg=http%3A%2F%2Fmerck.micro-medex.com%2Findex.asp%3Fpage%3Dnewsarchive%26news_id%3D5482%26news%3DMDM).
37. Cunniff C, Frias JL, Kaye C, et al. Pediatrics. Available on MerckMedicus February 1, 2001. Maternal phenylketonuria. [www.merckmedicus.com/pps/us/hcp/journalarticle.jsp?newsid=146279&journal=Pediatrics](http://www.merckmedicus.com/pps/us/hcp/journalarticle.jsp?newsid=146279&journal=Pediatrics).
38. Correa A, Botto L, Liu Y, Mulinare J, Erickson, JD. Pediatrics. Do multivitamin supplements attenuate the risk for diabetes-associated birth defects? Available on MerckMedicus May 30, 2003, Originally published: May 1, 2003. [www.merckmedicus.com/pps/us/hcp/journalarticle.jsp?newsid=185244&journal=Pediatrics](http://www.merckmedicus.com/pps/us/hcp/journalarticle.jsp?newsid=185244&journal=Pediatrics).
39. Svanberg B, Arvidsson B, Norrby A, Rybo G, Solvell L. Absorption of supplemental iron during pregnancy: a longitudinal study with repeated bone-marrow studies and absorption measurements. *Acta Obstet Gynecol Scand Suppl* 1975;48:87-108.
40. Puolakka J, Janne O, Pakarinen A, Jarvinen A, Vihko R. Serum ferritin as a measure of iron stores during and after normal pregnancy with and without iron supplements. *Acta Obstet Gynecol Scand Suppl* 1980;95:43-51.
41. Mead H, Witkowsky K, Gault B, Hartmann H. The influence of income, education, and work status on women's well-being. *Womens Health Issues* 2001;11(3):160-72.

42. Robles BJ. Latinas in the Academy: profiling leadership, promoting future scholars. LBJ School of Public Affairs and Center for Mexican American Studies, University of Texas at Austin. [www.utexas.edu/lbj/faculty/robles/research/pdf/latinas.pdf](http://www.utexas.edu/lbj/faculty/robles/research/pdf/latinas.pdf).
43. Ostrove JM, Feldman P, Adler NE. Relations among socioeconomic status indicators and health for African Americans and whites. *Journal of Health Psychology* 1999;4(4):451-63.
44. Singh K, Fong YF, Arulkumaran S. Department of Obstetrics and Gynaecology, National University Hospital, Singapore, Singapore. Anaemia in pregnancy—a cross-sectional study in Singapore. *Eur J Clin Nutr* 1998;52(1):65-70.
45. Herrmann RP. Iron deficiency. Current trends and fads. *Aust Fam Physician* 1994;23(8):1457-61.
46. Watson DS, Tozer RA. Anaemia in Yirrkala. *Med J Aust* 1986;144 suppl: S13-S15.
47. Dallman PR, Siimes MA, Stekel A. Iron deficiency in infancy and childhood. *Am J Clin Nutr* 1980;33:86-118.
48. Earl R, Wotecki CE, eds. *Iron deficiency anemia: recommended guidelines for the prevention, detection, and management among US children and women of childbearing age*. Washington, DC: National Academy Press, 1993.
49. Lazebnik N, Kuhnert BR, Kuhnert PM. The effect of race on serum ferritin during parturition. *J Am Coll Nutr* 1989;8(6):591-6.