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Racial Disparities and the Effect of Maternal Sexually Transmitted Infections on Adverse Birth Outcomes

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Abstract

Background: Understanding the role of sexually transmitted infections (STIs) in disparities associated with adverse birth outcomes (ABOs) may help target specific interventions for black and white women. Despite extensive research existing on racial disparities in ABOs and STIs, a gap in the literature exists regarding disparities in the association between STIs and ABOs.

Methods: This cross-sectional study utilizes data from Missouri vital statistics (n=173,624), years 2010-2012. Exposure was classified as: primary-HIV, either alone or with another STI (gonorrhea, syphilis, hepatitis B, or hepatitis C); secondary-aforementioned STIs without HIV; and unexposed. Outcomes of interest included preterm birth (birth prior to 37 weeks of gestation), low birth weight (less than 2500g), and small for gestational age (weight below the 10th percentile for gestational age). Bivariate and multivariate logistic regression models estimated risk of STIs on ABOs and adjusted for potential confounders (age, education, marital status, prenatal care, health insurance, previous preterm birth, smoking, BMI, weight gain, and chronic health conditions), by calculating odds ratios and 95% confidence intervals for primary and secondary exposures on all three outcomes.

Results: HIV exposure was significantly associated with a 72% increased odds (OR: 1.72, 95% CI: 1.03-2.89) for having a low birth weight birth and a 71% increased odds (OR: 1.71, 95% CI: 1.03-2.84) for being small for gestational age, when compared with women without an STI, after adjusting for potential confounding variables. Race was not an effect modifier.

Conclusions: Reducing one's risk of having an STI before pregnancy provides an important primary prevention tactic that can reduce risks for ABOs should one become pregnant.

Keywords: Adverse Birth Outcomes; Sexually Transmitted Infections; Maternal and Child health; Racial Disparities

Abbreviations: ABOs: Adverse Birth Outcomes; CDC: Centers for Disease Control and Prevention; PTB: Preterm Birth; LBW: Low Birth Weight.

Introduction

Racial disparities among adverse birth outcomes (ABOs) have become a growing public health concern. The Centers for Disease Control and Prevention (CDC) reported that the overall preterm birth (PTB) rate in 2010 in the United States was 12.0%, with blacks having a 60% higher rate than Whites (17.1% vs. 10.8%, respectively) [1]. ABOs were associated with several other adverse health outcomes, with PTB associated with breathing problems, feeding difficulties, cerebral palsy, developmental delay, vision problems, and hearing impairment [2]. PTB and low birth weight (LBW) were also largely associated with infant mortality. In the United States, the cause-specific infant mortality rate for combined PTB and LBW in 2013 was 106.9 per 100,000 infants. Racial disparities were evident in these mortality rates as well, with cause-specific infant mortality rates of 80.5 per 100,000 infants and 254.1 per 100,000 infants in whites and blacks, respectively [3].

Understanding factors that are associated with disparities in ABOs is key to developing interventions to combat them. One factor that may help explain these disparities is maternal sexually transmitted infections (STIs), as there is growing literature associating STIs and ABOs. In a large population-based cohort study of Canadian women, for example, HIV was found to be a significant risk factor for PTB (aOR 1.76), SGA (aOR 1.43), and LBW (aOR 1.90) [4]. Another study utilized a retrospective cohort design and serological HIV testing for women delivering in an inner-city hospital in Atlanta found HIV to be a significant risk factor for both LBW (aOR 2.11) and PTB (aOR 1.83) [5]. A large population based study in Puerto Rico also found a significant increase in risk for PTB (aOR 2.13) for mothers with HIV [6]. The relationship between HIV and ABO's has been consistently reported in the literature.

What is less conclusive in the literature is the relationship between other STIs and ABOs is less conclusive. Insignificant associations have been shown for the risk of PTB and LBW in a state-wide sample of mothers in Louisiana with chlamydia, gonorrhea, and syphilis [7]. In contrast, a study of women attending inner city STI clinics found significant associations between gonorrhea and PTB [8], consistent with findings from the previously mentioned investigation in Puerto Rico where women with gonorrhea were found to have a 49% increased odds of PTB in reference to women without gonorrhea [6]. The study of inner city STI clinics also found a significant association between chlamydia and PTB in contrast with Waight, et al. (2013) in Louisiana. In another recent study conducted in California, syphilis was found to be significantly associated with an increased odds of PTB, while chlamydia and gonorrhea were not significantly associated with increased odds of PTB [9]. Other studies have reported mixed results in the impact of Hepatitis B Virus and Heptatitis C Virus on birth outcomes, and they excluded other STIs from their exposure categories [10-12]. These inconsistent findings highlight the need for additional population-based research on the relationship between maternal STIs and ABOs.

Research has shown that rates of STIs vary across racial groups. Incidence rates of chlamydia, syphilis, and HIV were about eight times greater among non-Hispanic blacks than non-Hispanic whites and the incidence rate of gonorrhea was over 18 times greater among non-Hispanic blacks than non-Hispanic whites in 2010 [13]. In Missouri, this study's setting, STI disparities were especially evident, with HIV rates of 786.9 per 100,000 residents and 120.9 per 100,000 residents in blacks and whites, respectively [14]. A review indicated that segregation was a social determinant of these disparities in STI rates between racial groups [15]. A biological determinant of STI disparities may be the higher prevalence of bacterial vaginosis (BV) in non-Hispanic blacks, which increases the risk of acquiring STIs. A study of women aged 14-49 from the National Health and Nutrition Examination Survey found that BV prevalence was over twice as high in non-Hispanic Blacks (51.4%) than non-Hispanic Whites (23.2%) [16].

Despite extensive research existing on the racial disparities among both ABOs and STIs, there is a gap in the literature regarding disparities in the association between STIs and ABOs. Understanding the role of STIs in disparities associated with ABOs may help practitioners target specific interventions for black and white women. To further understand how STIs during pregnancy affect ABOs across racial groups in Missouri, a cross-sectional study utilizing birth certificate data comprising all singleton live births in the state between 2010 and 2012 was performed. Missouri was an ideal setting for this

study, as Missouri ranks among the highest in the country regarding STI rates for women [17], and Missouri birth certificate data includes an expanded list of STIs that includes Hepatitis B and C. The purpose of this study was to estimate the overall effects of maternal STIs on ABOs (PTB, LBW, SGA), and to evaluate race as an effect modifier in this relationship.

Materials and Methods

Study Design

The investigation was a cross-sectional study that utilized data from Missouri vital statistics. Maternal characteristics, along with data on ABOs, are collected on birth certificates. The standardized birth certificate format was adopted by Missouri in 2010, thus the study sampled all live births from 2010-2012 in the state. All outcome, exposure, and covariate data for subjects in the study was acquired from the standardized birth certificate.

Population

In total, there were 220,214 live births in Missouri from 2010-2012. The study cohort included all singleton live births during this period (n=212,440). Singleton births with improbable birth weight or gestational age were removed (n=212,367). As the investigation compares disparities between blacks and whites, the sample was restricted to mothers who identify as non-Hispanic Black and non-Hispanic White (n=192,783) on the birth certificates. Births that were missing information on gestational age, birth weights, or any other analytic variable were excluded from the analysis. The prenatal care variable was identified as being potentially problematic due to missing data (10.1% of subjects missing prenatal care data). To remedy this, an "unknown" category was included in the prenatal care variable in the analyses. After this, 19,159 (9.9%) subjects were missing data for one or more other predictors or outcomes and were excluded from the analyses. This lead to a final analytic sample of 173,624 singleton live births. Chi-square tests were performed to compare covariate frequencies in subjects with missing data and subjects included in the analysis.

Exposure

Mothers were classified into three exposure categories (primary, secondary, or unexposed). Missouri birth certificate data contained information on the following STI's: HIV, Hepatitis B and C, Gonorrhea, and Syphilis. The primary exposure level was defined as any woman with HIV, as the literature indicates that HIV has had the strongest effect on the adverse birth outcomes of interest in the study, compared to other STIs [4-6,18]. Mothers who had HIV alone or HIV and another STI were categorized into the primary exposure group. The secondary exposure group included any women infected with Hepatitis B, Hepatitis C, gonorrhea, or syphilis, which were chosen both based on their availability on birth certificates and indication from the CDC that they may complicate pregnancy [19]. Mothers not infected with an STI were categorized into the unexposed (reference) group. The exposure level information was determined by a "Yes" answer to any of the respective STIs on the child's birth certificate, which indicated if the woman had the respective STI at any point during her pregnancy.

Outcomes

Three outcomes of interest were investigated: PTB, defined as any birth prior to 37 weeks of gestation; LBW, defined as any birth with a birth weight less than 2500g regardless of gestational age; and SGA, defined as an infant born below the 10th percentile of birth weight for their gestational age; data to identify all three outcomes were readily available on Missouri birth certificates. PTB was calculated from the gestational age variable; LBW was calculated from the birth weight variable; and SGA was calculated in reference to gestational age cutoffs by race as seen in Alexander [20].

Effect Modifier

Since prior literature suggests racial disparities existed for both birth outcome rates and STI rates [13,21,22], effect modification by race was assessed, and analyses were stratified if effect modification was present.

Covariates

Demographic and maternal health factors present on Missouri Birth Certificates that were investigated as potential confounders were mother's age (less than 19, 20-34, 35 and older), mother's education (less than high school, high school diploma/GED, some college, 4 year college degree or higher), mother's marital status (yes, no), Kotelchuck Index (level of prenatal care; adequate or inadequate), health insurance type (Medicaid, private, other), previous preterm birth (yes, no), cigarette exposure during pregnancy (yes=any exposure during first, second or third trimester; no=no exposure), pre-pregnancy BMI (underweight, normal weight, overweight, obese), appropriate weight gain during pregnancy

(adequate vs. inadequate as defined by Kotch) [23], history of hypertension (yes=pre-pregnancy hypertension, gestational hypertension, preeclampsia or eclampsia; no=no exposure), and history of diabetes (yes=pre-pregnancy or gestational diabetes; no=no exposure). The study protocol was deemed to be exempt from approval by the Institutional Review Board at Saint Louis University. All analyses were conducted in SAS v9.4 (SAS Institute, Cary, NC).

Data Analysis

To first determine if any significant associations between exposure and all other variables (covariates and outcomes) exist, chi-square tests were performed. Logistic regression models were built to estimate the effect of STIs on PTB, LBW, and SGA before and after controlling for potential confounders. A preliminary analysis including the interaction term of race and the STI exposure variable in full models for PTB, LBW and SGA were built to investigate effect modification. If the interaction term was significant at p<.20, stratified models were presented. Crude and adjusted logistic regression models estimated risk of STIs on ABOs by calculating odds ratios (ORs) and 95% confidence intervals (CIs).

Results

Characteristics of the study population by exposure group is displayed in Table 1. Overall, 1,660 (1.0%) subjects had any STI other than HIV and 110 (0.6%) had HIV. For the outcomes, 13,107 (7.5%) individuals had a PTB, 10,410 (6.0%) had a LBW, and 16,445 (9.5%) had births that were SGA. Demographically, 84% of the sample was white, 80% of the sample was between the ages of 20 and 34, 59% were married, and 85% had at least a high school degree or GED. Nearly half (48%) of the sample was overweight or obese. Insurance coverage was about an even split, with 45% of the sample having Medicaid while 51% had private insurance. Table 1 also displays that there are significant differences between exposure groups for all covariates included in our analysis except for diabetes (chi-square *P*=0.2131). All other covariates were significant at the *P*<.05 level, with most having a chi-square *P*<.0001.

Covariates n(%)	Other STI (n=1,660)	HIV (n=110)	Unexposed (n=171,854)	Total (n=173,624)	*P*-value*
PTB	193 (11.6)	18 (16.4)	12,896 (7.5)	13,107 (7.6)	<.0001
LBW	188 (11.3)	20 (18.2)	10,202 (5.9)	10,410 (6.0)	<.0001
SGA	228 (13.7)	19 (17.3)	16,191 (9.4)	16,445 (9.5)	<.0001
Age					
<20	265 (16.0)	6 (5.5)	16,512 (9.6)	16,783 (9.7)	<.0001
20-34	1,271 (76.6)	91 (82.7)	138,265 (80.5)	139,627 (80.4)	
>34	124 (7.5)	13 (11.8)	17,077 (9.9)	17,214 (9.9)	
Race					
White	1,018 (61.3)	35 (31.8)	144,936 (84.3)	145,989 (84.1)	<.0001
Black	642 (38.7)	75 (68.2)	26,918 (15.7)	27,635 (15.9)	
Education					
Less than HS	604 (36.4)	44 (40.0)	25,120 (14.6)	25,768 (14.8)	<.0001
HS or GED	576 (34.7)	43 (39.1)	41,894 (24.4)	42,513 (24.5)	
Some college	408 (24.6)	15 (13.6)	56,178 (32.7)	56,601 (32.6)	
College or more	72 (4.3)	8 (7.3)	48,662 (28.3)	48,742 (28.1)	
Previous PTB	123 (7.4)	15 (13.6)	5,447 (3.2)	5,585 (3.2)	<.0001
Insurance					
Medicaid	1,375 (82.8)	85 (77.3)	76,422 (44.5)	77,882 (44.9)	<.0001
Private	225 (13.6)	21 (19.1)	87,919 (51.2)	88,165 (50.8)	
Other	60 (3.6)	4 (3.6)	7,513 (4.4)	7,577 (4.4)	
Marriage Status					
Married	427 (25.7)	32 (39.1)	102,922 (59.9)	103,381 (59.5)	<.0001
Not married	1,233 (74.3)	78 (70.9)	68,932 (40.1)	70,243 (40.5)	
Prenatal Care					
Adequate	861 (51.9)	67 (60.9)	122,499 (71.3)	123,427 (71.1)	<.0001

Inadequate	617 (37.2)	34 (30.9)	33,726 (19.6)	34,377 (19.8)	
Unknown	182 (11.0)	9 (8.2)	15,629 (9.1)	15,820 (9.1)	
Weight Gain					
Appropriate	372 (22.4)	18 (16.4)	42,074 (24.5)	42,464 (24.5)	0.021
Inappropriate	1,288 (77.6)	92 (83.6)	129,780 (75.5)	131,160 (75.5)	
Smoking	786 (47.4)	31 (28.2)	33,751 (19.6)	34,568 (19.9)	<.0001
Hypertension	155 (9.3)	13 (11.8)	12,231 (7.1)	12,399 (7.1)	0.0004
Diabetes	109 (6.6)	7 (6.4)	9,605 (5.6)	9,721 (5.6)	0.2131
Pre-pregnancy BMI					
Underweight	113 (6.8)	5 (4.6)	7,873 (4.6)	7,991 (4.6)	<.0001
Normal	786 (47.4)	30 (27.3)	81,556 (47.5)	82,372 (47.4)	
Overweight	358 (21.6)	29 (26.4)	40,638 (23.7)	41,025 (23.6)	
Obese	403 (24.3)	46 (41.8)	41,787 (24.3)	42,236 (24.3)	

Table 1: Prevalence of outcomes and covariates among all live births, Missouri 2010-2012, by sexually transmitted infection (STI) status (n=173,624).
P-values from chi-square test of independence.

After testing for effect modification by race, it was determined that race was not an effect modifier for any of the three outcomes of interest at the *P*<.20 level. *P*-values for the STI and race interaction term for each outcome were: PTB, *P*=.370; LBW, *P*=.726; and SGA, *P*=.948. Following this determination, race was included as a confounder in the final, overall logistic regression models. Table 2 displays the results of the crude and adjusted logistic regression models for STI exposures on the three outcomes while controlling for key covariates.

Covariate	Crude OR (95% CI)	Adjusted OR (95% CI)
Pre-term birth (PTB)		
Unexposed	1.00 (referent)	1.00 (referent)
Maternal STI	1.62 (1.39-1.89)	1.13 (0.97-1.33)
Maternal HIV Infection	2.41 (1.46-4.00)	1.42 (0.83-2.43)
Low birth weight (LBW)		
Unexposed	1.00 (referent)	1.00 (referent)
Maternal STI	2.02 (1.76-2.36)	1.14 (0.97-1.33)
Maternal HIV Infection	3.52 (2.17-5.72)	1.72 (1.03-2.89)
Small for gestational age (SGA)		
Unexposed	1.00 (referent)	1.00 (referent)
Maternal STI	1.53 (1.33-1.76)	1.03 (0.89-1.19)
Maternal HIV Infection	2.01 (1.22-3.29)	1.71 (1.03-2.84)

Table 2: Crude and adjusted* odds ratios (ORs) and 95% confidence intervals (CIs) for STI exposures on ABOs.

Adjusted for age, race, education, previous PTB, medical insurance, marriage status, adequacy of prenatal care, weight gain during pregnancy, smoking during pregnancy, hypertension, diabetes, and BMI.

Before adjusting for potential confounding, both STI and HIV infection were significant risk factors for PTB, LBW, and SGA (OR range: 1.53-3.52). After adjusting for the covariates identified as potential confounders, any STI infection was found to not be related to ABOs. However, HIV infection was significantly associated with over a 70% increased odds of LBW (aOR and 95% CI: 1.72, 1.03-2.89) and SGA (aOR and 95% CI: 1.71, 1.03-2.84).

Discussion

Results showed that only HIV is associated with increased odds of LBW and SGA; however, race was shown to not be an effect modifier in the relationship between STIs and ABOs. It was hypothesized that race may be an effect modifier in this relationship since STIs have been shown to be a risk factor for ABOs [4-6,9] and there is a large racial disparity in ABO rates [1]. Since race is not an effect modifier in this relationship, there is some other reason for the large disparity in ABO outcome rates between whites and blacks. Despite the absence of effect modification, race was shown to be an important confounding factor in the relationship between HIV and other STI exposure for all three outcomes. This finding, that race is not an effect modifier in the relationships studied, was not previously characterized, making it an important finding even though it is a null result.

The present investigation is one of only a dearth of population-based studies that have investigated both HIV and other STIs (including Hepatitis B and C) while adjusting for relevant sociodemographic characteristics important in a US population. Having HIV was significantly associated with a 72% increased odds for having a LBW birth and a 71% increased odds for being SGA, when compared with women without an STI, in our study. These findings are consistent with earlier studies [4,5]. The current study did not find a significant relationship between HIV and PTB, however, in contrast with MacDonald, et al. (2015) [4] and Ellis, et al. (2002) [5]. We speculate that these differences may be due to our sample size, as the adjusted odds showed a 42% increased risk for PTB in comparison to women with an STI. With a larger sample, the span of the confidence interval of 0.83-2.43 would likely shrink and become significant.

Our second exposure of any STI was initially found to be significantly associated with ABO's in the crude analysis; however these relationships were no longer significant after adjusting for demographic, prenatal care, and other chronic conditions. This insignificant finding is consistent with Waight, et al. discussed earlier [7]. However, this finding is in contrast with Eick, et al (2017) who reported a significant association between syphilis and PTB (aOR 1.45) [6], and both Eick, et al. (2017) and Baer, et al (2018) who reported gonorrhea (aOR 1.49) as a significant risk for PTB [6,9]. We again speculate this may be due to sample size. For example, Eick, et al. (2017) [6] combined eight years of data for their study, thus increasing the power of their sample to detect a significant association while this study only used three years of data. Because this study is trending in the direction of the previous literature, we suspect that a larger sample size would have resulted in significant findings for all of the outcomes explored. In addition, our inclusion of Hepatitis B virus (HBV) and Hepatitis C virus (HCV) to the list of STI's included in our analytic modeling is important and notable. HBV and HCV are other STIS that have been shown to be related to PTB and LBW, are treatable, and yet often excluded from population-based studies looking at STIs and ABOs [24,25].

This study has several limitations. The cross-sectional nature of the study (all information collected at one point in time from birth certificates) prohibits any temporality or incidence calculations, thus limiting conclusions to association rather than causation. Further, our data set did not include chlamydia. Additionally, there are potential issues from residual and unmeasured confounding, such as education, income, neighborhood exposures, or when a person contracted the STI. Smoking was self-reported and because of social desirability associated with not smoking during pregnancy, there is some suspicion this may be an underreported variable. Another limitation is the low exposure rates in the sample, especially for HIV, which may reflect low screening rates rather than low rates of infection. Expanding this study to include both live births and fetal deaths may also capture a larger sample of STI exposed pregnancies that may not have resulted in a live birth. Future studies that prospectively follow pregnant women would be one way to address some of the limitations outlined here.

Despite these limitations, the study has much strength. First, this study was the first of its kind to investigate race as an effect modifier in the relationship between STIs and ABOs, and discovered that race was not an effect modifier in the relationships analyzed. The study had a representative sample of all singleton live births in Missouri between 2010 and 2012, and was able to discern a significant association between HIV and two outcomes (LBW and SGA), in spite of the very low exposure rate in the sample (0.6%). This suggests that HIV likely was an important risk factor for LBW and SGA. The large sample size limited the likelihood of selection bias and provides external validity to pregnant women in the state of Missouri. Additionally, the low rates of missing data (only 9.9% of the sample excluded from final analysis) and large number of key covariates present on birth certificates allowed for the controlling of important potential confounders. Finally, chi-square tests comparing frequencies for covariates in subjects missing data and

subjects that were included in the analysis showed no significant differences, indicating that the study is robust against selection bias.

In conclusion, this representative cross-sectional sample of singleton live births in Missouri from 2010-2012 showed that HIV was a significant risk factor for LBW and SGA, and, that race was not an effect modifier in this relationship. These results have implications for public health practitioners as well as clinicians. To reduce the glaring racial disparity among ABO rates, one intervention may be implementing programs aimed at lowering STI rates in the black population. For clinicians, this study underscores the importance of caring for women with HIV. Clinicians must inform and educate pregnant women with HIV and other STIs about their risks for poor birth outcomes. Primary prevention strategies must also be used by clinicians to prevent HIV before pregnancy, increasing the chances that should a woman become pregnant, she would enter pregnancy with less risk. Early and regular prenatal care provides important opportunities for STI screening among pregnant women, which means access to affordable, quality of prenatal care is essential to combating the deleterious outcomes associated with an STI exposed pregnancy.

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Summary

A study of birth certificate records in Missouri found that pregnant women with HIV had increased risk of having a low birth weight or small for gestational age birth.

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