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Reproductive Health Disparities in the USA: Self-Reported Race/Ethnicity Predicts Age of Menarche and Live Birth Ratios, but Not Infertility

Talia N. Shirazi [1] · Asher Y. Rosinger [1,2]

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Abstract

Self-identified race/ethnicity and socioeconomic status (SES) contribute to disparities in several health domains, although research on their effects on women's reproductive function has largely focused on links between SES and age of menarche. Here, we assessed whether race/ethnicity, SES, and downstream correlates of SES such as food security and health-insurance security are associated with age of menarche, infertility, and live birth ratios (ratios of recognized pregnancies resulting in live births) in the USA. We used cross-sectional data from 1694 women aged 12–18 years for menarche (2007–2016), 974 women aged 23–45 for infertility (2013–2016), and 1714 women aged 23–45 for live birth ratios (2007–2016) from the National Health and Nutrition Examination Survey. We estimated multiple linear and logistic regressions with survey weights to test these associations. When controlling for lifestyle (activity levels, smoking, alcohol consumption) and physiological factors (diabetes, weight status), non-Hispanic (NH) black and Hispanic girls reported a significantly lower age of menarche by about 4.3 (standard error [SE] = 0.08, $p < 0.001$), and 3.2 months (SE = 0.09, $p < 0.001$), respectively, relative to NH white girls. NH black women reported live birth ratios 9% (SE = 0.02, $p < 0.001$) lower than NH white women. Women with unstable health insurance reported live birth ratios 6% (SE = 0.02, $p = 0.02$) lower than women with stable health insurance. Race/ethnicity, SES, and its downstream correlates were not associated with infertility. One hypothesized explanation for observed disparities in age of menarche and live birth ratios is the embodiment of discrimination faced by NH black women within the USA. Our findings also underscore the importance of health insurance access for favorable reproductive health outcomes. Future work should elucidate the role of embodied discrimination and other downstream correlates of SES in modulating women's reproductive health outcomes to inform strategies to mitigate health disparities.

Keywords Socioeconomic status · Race · Ethnicity · Menarche · Live birth ratios · Health insurance

For decades, studies have suggested the role of social and environmental factors in significantly modulating the population patterns of health and disease [1]. The accumulation of such studies led to the creation of the World Health Organization (WHO) Commission on Social Determinants of Health in 2005, tasked with studying the effect of socioeconomic status (SES) and its correlates on country indices of morbidity and mortality on a global scale [2]. Results of this commission mirrored prior findings in that within and across countries, structural determinants of individuals' environments, experiences, and lifestyles play powerful roles in shaping health outcomes [3]. Subsequent WHO initiatives have confirmed significant effects of social factors on mortality [4]. Thus, while research on the proximate, causal physiological factors of disease is necessary to reduce health disparities, research on upstream social and environmental factors that modulate risk factors of poor health within and between populations is crucial as well.

Within the USA, correlates of social and economic vulnerability predict health outcomes on a wide range of physiological and psychological phenotypes. SES and neighborhood-level measures of SES negatively predict coronary heart

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✉ Asher Y. Rosinger
axr579@psu.edu

[1] Department of Anthropology, Pennsylvania State University, Carpenter Building, University Park, PA 16802, USA

[2] Department of Biobehavioral Health, Pennsylvania State University, 219 Biobehavioral Health Building, University Park, PA 16802, USA

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disease [5], depressive symptoms [6], diabetes [6], obesity [6], and allostatic load [7], among other disease states. In countries with historical de facto and de jure segregation and racism, self-identified race/ethnicity is not independently associated with SES. In the USA, self-identified non-Hispanic (NH) black and Hispanic individuals trail behind NH white individuals in both income and education [8, 9]. Racial and ethnic minorities also report higher levels of self-reported discrimination or prejudice as compared with NH white individuals [10–12], and individuals with darker skin within minority groups report higher levels of discrimination [13]. Higher levels of discrimination, in turn, predict more unfavorable health outcomes [14]. Thus, lower SES and higher levels of discrimination may explain why racial and ethnic minorities in the USA bear the disproportionate burden of coronary heart disease [15], early mortality [16], obesity [17], and allostatic load more generally [18], among others.

Disparities in reproductive health outcomes have also been noted along the lines of both SES and race/ethnicity. While some studies suggest an inverse association between SES and age of menarche [19–21], others suggest a direct relationship [22, 23], such that age of menarche is lowest among adolescents with parents of low SES. As research has demonstrated that earlier age of menarche is associated with higher cancer risk [24] as well as negative psychosocial outcomes in young adulthood and beyond [22, 25], SES disparities in age of menarche may contribute to disparities in health in adulthood.

There is also an inverse association between SES and pregnancy outcomes such as stillbirths and low birth weight. Stillbirths [26] and low birth weight [27] are disproportionately observed among women of low SES. Patterns across similar outcomes are observed along self-identified racial and ethnic categories within the USA, such that women self-identifying with racial and ethnic minority groups experience menarche at a younger age [28, 29], and higher rates of stillbirths and miscarriages [30]. SES and race/ethnicity may also interact in modulating certain pregnancy-related risk factors; for example, whereas high SES may reduce the preeclampsia risk among white women, high SES does not attenuate preeclampsia risk among black women, who are overall at greater risk [31].

Many questions remain regarding the associations between self-identified race/ethnicity, SES, SES's downstream correlates, and women's reproductive health and function. As most prior studies have focused on age of menarche and outcomes related to infant and maternal health, other metrics of reproductive function remain understudied. For example, it is unknown whether minority groups and women of lower SES experience higher rates of infertility, which is in part modulated by hypothalamic-pituitary-gonadal axis function. While some work suggests infertility may be predicted by education but not income [32], studies of infertility in the USA typically rely on samples obtained at infertility clinics, which are comprised largely of women of high SES due to treatment costs [32]. As a result, the majority of women with impaired fertility do not obtain infertility services [33], and accurate surveillance of women experiencing infertility at various SES levels is obscured.

Additionally, although the total fertility rate and probability of stillbirths differ as a function of SES and race/ethnicity, it is unknown whether live birth ratios, or the ratios of clinically recognized pregnancies resulting in live births [34], exhibit a similar association with these predictors. Further, studies looking at self-identified race/ethnicity and variables related to SES have rarely looked at these factors in conjunction, and instead look at either race/ethnicity or SES-related variables in isolation.

Because of the conflation between race/ethnicity and SES in the USA, and because of work suggesting that race/ethnicity and SES may independently and uniquely lead to health disparities [35], it is important that studies on health disparities examine such factors concurrently. These limitations of prior work highlight the extent to which the effect of the social determinants of women's reproductive health outcomes requires further study.

Therefore, we aim to fill these gaps by evaluating how self-identified race/ethnicity, SES, and downstream correlates of SES are associated with age of menarche, prevalence of infertility, and live birth ratios using nationally representative data collected in the USA between 2007 and 2016 from the National Health and Nutrition Examination Survey (NHANES). Based on the extensive body of work suggesting disparities across SES and racial/ethnic groups, we hypothesized that self-reported racial/ethnic minority group identity and low SES indicators would be associated with a lower age of menarche, a higher prevalence of infertility, and lower live birth ratios.

Method

Data Source

Conducted by the Centers for Disease Control and Prevention's (CDC) National Center for Health Statistics (NCHS), NHANES uses a stratified, cluster sampling design that assigns participants' sample weights based on demographic variables, allowing for the calculation of representative population-level estimates of effects. Full descriptions of the study design have been described in detail elsewhere [36]. Data have been collected continuously since 1999 and are publicly released in 2-year cycles. The present study employs data from women aged between 12 and 45 (inclusive) collected between 2007 and 2016. NHANES examinations include laboratory tests, comprehensive physical examinations, and either researcher-administered or computer-administered

surveys. Between 2007 and 2016, unweighted response rates varied between 64 and 86% for interviews, and between 61 and 83% for medical examinations.

NHANES is approved by the NCHS research ethics review board. Children aged 7–17 years provided assent and parents provided consent for children under 18, and adults provided consent for themselves. All NHANES data used in this analysis were publicly available for download without identifiers via the NCHS website [36].

Measures

Reproductive Health Outcomes

The reproductive health questionnaire was administered to all women participating in NHANES 12 and older. This questionnaire requires women to self-report age of menarche ("How old were you when you had your first menstrual period?"), number of pregnancies ("How many times have you been pregnant?"), infertility ("Have you ever attempted to become pregnant over a period of at least a year without becoming pregnant?"), and number of live births ("How many of your deliveries resulted in a live birth?"). Live birth ratios were calculated as the number of reported pregnancies resulting in a live birth [34]. While age of menarche and live birth ratios were available for all women, questions about infertility were first added during the 2013–2014 survey cycle; thus, analyses on infertility are restricted to women participating in the 2013–2014 and 2015–2016 cycles. Across all survey cycles, women also reported whether they were currently pregnant, breastfeeding, or using any form of hormonal contraceptives.

Race/Ethnicity

NHANES contains survey measures on race and ethnicity which are made publicly available, and participants are categorized as non-Hispanic (NH) white, NH black, Hispanic (including Mexican American and other Hispanic), and other. Participants who identified as other race, including multiracial and NH Asian, were included in analyses but not shown in regressions separately here due to small sample sizes and the heterogeneity comprising this category. It is important to note that race/ethnicity, operationalized in this way, does not classify individuals into biologically meaningful or culturally homogenous groups. Rather, these socially based categories are based on physical phenotypes, and it is the differential treatment of individuals in these categories that consequently modulate exposures, risks, and constraints within race-conscious societies [37]. Therefore, we consider race/ethnicity defined in this way to be a proxy for the differential exposures, risks, and constraints experienced across socially and phenotypically based categories.

SES Measures

The two markers of SES used in this analysis were federal income-to-poverty ratio and education completion. Federal income-to-poverty ratio (FIPR) is an index of family income to poverty, with poverty guidelines enumerated by the US Department of Health and Human Services. FIPR was categorized as ≤ 130%, 131–350%, and > 350% for analyses following published guidelines [38]. Education was assessed through a self-report question, "What is the highest grade or level of school you have completed or the highest degree you received?" Participants were categorized into the following four categories: some high school, high school graduate or high school diploma equivalent, some college or associates degree, and college graduate or above. For women 18 and under, education completion of the household reference was used in place of participant education and categorized as described above.

Downstream Correlates of SES

Food [39, 40] and health-insurance insecurity [41, 42] are associated with SES and race/ethnicity, and may predict objective markers of health. Adult food security over the past year was assessed in all participants using a standard food security questionnaire, which includes questions such as "In the last 12 months, did you ever eat less than you felt you should because there wasn't enough money for food?" Food security questionnaire responses were used to divide participants into high, marginal, low, and very low food security groups based on previously established guidelines [43].

We used questionnaire data on current health insurance status as well as consistency of health insurance over the past 12 months to divide participants into two groups. Participants were either classified as health-insurance secure if they currently had health insurance and had health insurance over the last 12 months. They were classified as health-insurance insecure if they did not have health insurance or had a lapse in insurance over the previous year.

Finally, we examined foreign-born status of participants and household references. Foreign-born participants often have greater health disparities across a wide range of outcomes including those related to reproductive health [44], may experience more discrimination, and may have trouble navigating health care settings [45]. Thus, participants were categorized as being born in the USA, being born outside of the USA and living in the USA for less than 10 years, or being born outside of the USA and living in the USA for more than 10 years due to the healthy immigrant paradox [46].

Covariates

Many behavioral and physiological factors may modulate hormone production and reproductive function. Body mass index

Table 1 Sample size and descriptive characteristics for US adolescents and women, National Health and Nutrition Examination Survey (NHANES)[a,b,c]

	Age of menarche	Infertility	Live birth ratios
Sample *n*	1694	974	1714
Age range (inclusive)	12–18	23–45	23–45
Mean age (SE)	14.97 (0.06)	34.39 (0.32)	36.00 (0.20)
Ethnicity (%)			
White, non-Hispanic	61.45 (2.08)	67.67 (2.42)	68.01 (1.68)
Black, non-Hispanic	14.47 (1.37)	11.01 (1.55)	13.44 (1.12)
Hispanic	16.67 (1.61)	12.72 (1.76)	12.43 (1.04)
Other	7.40 (0.65)	8.60 (0.86)	6.13 (0.58)
FIPR (%)			
≤ 130%	28.55 (1.80)	18.14 (2.13)	20.15 (1.44)
131–350%	37.38 (1.92)	37.02 (1.86)	38.73 (1.56)
> 350%	34.07 (2.28)	44.84 (2.41)	41.12 (1.92)
Highest level of education* (%)			
Less than high school	19.15 (1.29)	5.67 (0.82)	10.06 (0.81)
High school	19.74 (1.56)	14.77 (1.64)	16.00 (1.13)
Some college	31.25 (1.80)	37.94 (2.31)	40.50 (1.51)
College degree or higher	29.86 (1.93)	40.62 (2.47)	33.44 (1.64)
Adult food security category (%)			
High	70.02 (1.77)	72.01 (2.17)	70.63 (1.44)
Marginal	10.35 (1.00)	10.58 (1.37)	10.82 (1.00)
Low	12.70 (1.16)	11.11 (1.27)	11.96 (1.01)
Very low	6.93 (0.77)	6.29 (0.77)	6.59 (0.57)
Health-insurance security (%)			
Secure	93.75 (0.91)	88.18 (1.32)	89.26 (0.96)
Insecure	6.24 (0.91)	11.82 (1.32)	10.74 (0.96)
Time in the USA (%)			
Born in the USA	95.42 (0. 65)	87.74 (1.38)	87.61 (1.06)
Foreign born, in the USA < 10 years	2.59 (0.47)	2.78 (0.57)	2.28 (0.42)
Foreign born, in the USA ≥ 10 years	2.00 (0.36)	9.49 (1.24)	10.11 (0.90)
BMI category (%)			
Underweight	0.79 (0.24)	1.10 (0.41)	1.12 (0.33)
Normal weight	60.00 (1.70)	36.67 (2.40)	34.32 (1.42)
Overweight	18.90 (1.24)	22.28 (1.41)	27.05 (1.27)
Obesity	20.31 (1.24)	39.36 (1.76)	37.51 (1.43)
Smoking (%)^			
Never	76.24 (1.40)	63.20 (2.23)	57.76 (1.60)
Past	N/A	16.62 (1.78)	18.67 (1.21)
Current (some days)	N/A	5.20 (0.77)	4.73 (0.54)
Current (every day)	N/A	14.98 (1.39)	18.83 (1.28)
Diabetes (%)			
No	99.42 (0.26)	94.64 (0.79)	95.62 (0.54)
Yes	0.58 (0.26)	5.36 (0.79)	4.38 (0.54)
Mean drinks/week (SE)	N/A	0.41 (0.04)	0.36 (0.03)
Physical activity			
Sedentary (< 150 min/week)	88.55 (1.14)	79.29 (1.70)	79.97 (1.26)
Exercising (≥ 150 min/week)	11.45 (1.14)	20.71 (1.70)	20.04 (1.26)

*For age of menarche subsample, highest level of education for household reference person

† For age of menarche subsample, BMI-for-age *z* score

^For age of menarche subsample, smoking collapsed into never and ever

[a] Unweighted sample size

[b] Weighted means

[c] Without missing covariate data

FIPR: federal income poverty ratio. Numbers in brackets represent standard errors. Other race/Hispanic origin included in analyses but not shown

(BMI) exhibits a U-shaped relationship with fertility, such that very low and high BMIs are associated with decreased fertility [47]. BMI may also negatively predict age of menarche [48]. As such, controlling for the effects of BMI is necessary to isolate the link between the predictors of interest here and reproductive outcomes. Some work suggests that reproductive physiology is influenced by smoking [49], alcohol consumption [50], age [51], physical activity [52], and diabetes [53].

Measures of the aforementioned variables obtained via self-report or medical examination were included as covariates in all models.

Height and weight were measured by an examiner, and BMI was calculated as weight in kilograms divided by height in meters squared. BMI *z* scores were calculated for participants age 12 to 18 using the *zanthro* function [54] and were categorized following CDC guidelines as underweight (BMI *z* score ≤ 1.645), normal weight (BMI *z* score greater than or equal to − 1.645 but less than 1.04), overweight (BMI *z* score greater than or equal to 1.04 and but less than 1.645), and obesity (BMI *z* score at or above 1.645). Standard BMI categories [underweight (< 18.5 kg/m^2), normal weight (18.5–24.9), overweight (25–29.9), and obesity (≥ 30)] were created for all adult participants. For all participants, BMI was analyzed as a categorical variable. Smoking measures were obtained via self-report; specifically, here we used responses to "Have you smoked at least 100 cigarettes in your lifetime?" and "Do you now smoke cigarettes every day, some days, or not at all?" to classify participants into never, past, and current smoker groups. For alcohol consumption, participants were first categorized as either never, former, or current drinkers [55]. For current drinkers, an average number of drinks per day was calculated. Diabetes was evaluated using responses to the question, "Has a doctor ever told you that you have diabetes?" as well as using lab-measured blood glycohemoglobin (HBa1c) levels. Any adult who reported that a doctor diagnosis of diabetes or had HBa1c ≥ 6.5% was classified as having diabetes [56]. Physical activity was scored following previous studies [57]; briefly, women reporting at least 150 min per week of moderate intensity aerobic activity were classified as "exercising," meeting national physical activity guidelines [58], while women reporting less than 150 min per week of moderate intensity aerobic activity were classified as sedentary. Similar dichotomizations of physical activity have been used in previous work assessing the link between physical activity and ovarian function [59].

In summary, the covariates included in regressions indexed the following: age, highest level of education achieved, food security category, health-insurance security, time in the USA, BMI category, smoking and alcohol consumption (with the exception of age of menarche analyses, which did not include alcohol consumption), diabetes, and physical activity. Descriptive information for these covariates can be found in Table 1.

Statistical Analyses and Analytic Samples

Inclusion criteria for the reproductive health outcomes are displayed in Fig. 1. For age of menarche, the sample was restricted to women between 12 and 18 (inclusive). Rationale for excluding women over 18 for these analyses was based on previous demonstrations of (albeit, limited) SES fluidity across adolescence and adulthood [57]; by focusing on a sample of adolescent women whose current SES would more likely reflect their SES at age of menarche, limitations of changing SES across time are somewhat circumvented. Limiting our sample to women between 12 and 18 further allowed us to best capture other exposures and conditions present around the time of menarche. Household reference education completion was used as a predictor rather than participant education completion for this sample only. To assess the association between race/ethnicity, SES, other predictors and age of menarche, we estimated multiple linear regressions adjusting for covariates between 2007 and 2016.

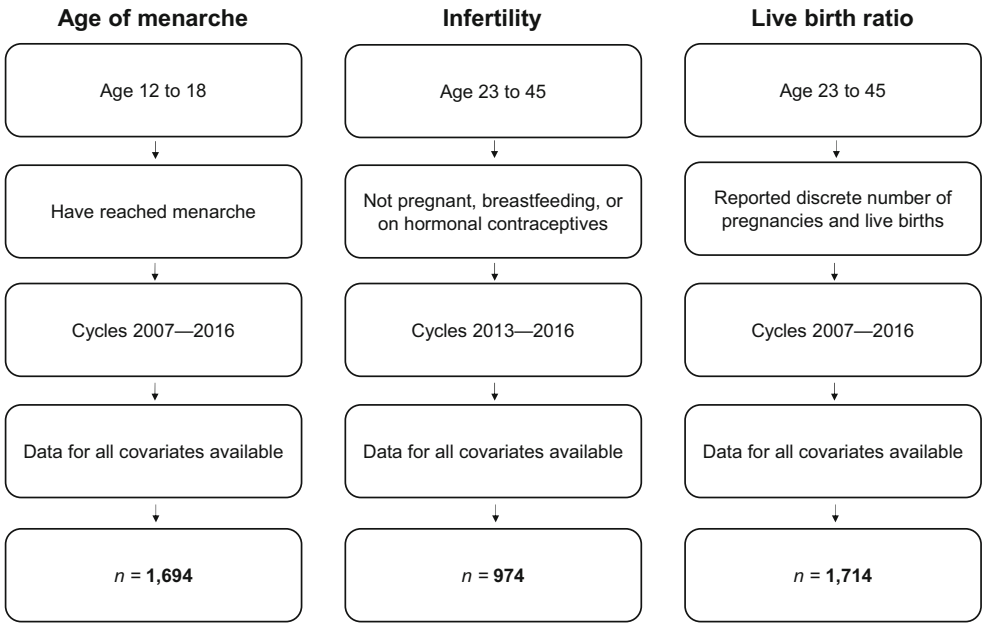

Fig. 1 Information on inclusion criteria and sample sizes of study

For analyses on infertility, women who reported being pregnant, breastfeeding, or on hormonal contraceptives were excluded. We also excluded women below 23 and above 45 years. The upper limit of 45 was chosen to exclude any women who were likely to be menopausal or peri-menopausal. A lower limit of 23 was chosen as the majority of participants pursuing college education would have likely completed their degree by this age. Since the infertility variable was dichotomous, we estimated multiple logistic regression models to assess the association between race/ethnicity, SES, and the other SES proxies on the odds of infertility. When exponentiated, these provide the odds ratios. Because infertility questions were added in the 2013–14 cycle, infertility analyses draw on data from the 2013–14 and 2015–16 cycles only.

For analyses on live birth ratios, we restricted our sample to women between 23 and 45 for the reasons cited above. In the 2015–2016 survey cycle, women were able to select "greater than 10" for number of live births or pregnancies, rendering us unable to calculate accurate live birth ratios for such women. Thus, analyses on live birth ratios are further restricted to women who reported a discrete number of both pregnancies and live births. We analyzed live birth ratios with multiple linear regressions between 2007 and 2016. However, as the outcome is a continuous ratio that includes zeros and ones, we also estimated fractional outcome regressions [60] as sensitivity analyses. While ratio dependent variables are often analyzed effectively using linear models [61], it is possible for linear regressions to predict values below zero or below one, which are not possible given the nature of the data.

As some prior work has exhibited significant interactions between SES and race/ethnicity [62, 63], we estimated models including both main effects of our markers of SES and race/ethnicity as well as their interactions. However, because we observed no clear or consistent significant interactions between SES and race/ethnicity in predicting our outcomes of interest, we present models including the main, but not interactive, effects of SES and race/ethnicity.

Statistical significance was set at $p < 0.05$. We aggregated all survey cycles to maximize sample size to test these relationships. We used the mobile examination center (MEC) weights to adjust for the complex survey design, non-response, oversampling, non-coverage, and day of the week to have nationally representative estimates. All estimates are weighted, except for sample sizes. All analyses were conducted in Stata (version 16.1 Stata Corp, College Station, TX), and done in accordance with NCHS analytical guidelines [38]. Variation inflation factors (VIF) and tolerance values were examined for all regressions to assess multicollinearity. All tolerance values were between 0.70 and 0.77, and all VIFs were between 1.30 and 1.42, well below the general limit of 10 as indicative of multicollinearity. To examine the predicted values of our three outcomes by race/ethnicity, we used post-estimation marginal standardization for regressions adjusting for the distribution of other covariates included in the models including SES to illustrate the practical significance of the results [64].

Results

Sample Demographics

Sample sizes and demographic information on the samples for each outcome of interest can be found in Table 1. Briefly, across samples, the majority of women self-identified as NH white (61.45 to 68.01%), had a FIPR > 350% (34.07 to 44.84%), had completed some college (31.25 to 40.50%), and had high food security (70.02 to 72.01%) were classified as health-insurance secure (88.18 to 93.75%), and were born in the USA (87.61 to 95.42%).

Age of Menarche

Data from girls between the ages of 12 and 18 years who had previously experienced menarche were included in a linear regression predicting age of menarche (Table 2; Fig. 2). Relative to NH white girls, NH black girls (beta = − 0.36, SE = 0.08, $p < 0.001$) and Hispanic girls (beta = − 0.27, SE = 0.09, $p = 0.005$) reported younger age of menarche. As age of menarche was measured in years, a beta of − 0.36 corresponds to a difference of 4.3 months, while a beta of − 0.27 corresponds to a difference of 3.2 months. FIPR did not predict age of menarche. Household reference education exhibited a positive, but not significant, relationship with age of menarche, such that girls from households with the reference person's highest education level being less than high school had the lowest age of menarche (beta = − 0.28, SE = 0.14, $p = 0.05$), and girls from households with the reference person's highest education level being college or greater had the highest age of menarche. Food security, health-insurance security, and time in the USA did not predict age of menarche.

Infertility

Data from women between the ages of 23 and 45 who were not currently pregnant, lactating, or on a hormonal contraceptive were included in a logistic regression predicting infertility over the last 12 months (Table 3; Fig. 3). Race/ethnicity and FIPR did not predict infertility. Relative to women who had completed college, women who did not complete high school had a lower odds of infertility (OR = 0.18, 95% CI = 0.06, 0.55, $p < 0.01$). Food security, health-insurance security, and time in the USA did not predict infertility.

Table 2 Multiple linear regression predicting age at menarche among USA women between 12 and 18, National Health and Nutrition Examination Survey (NHANES)

Independent variable	Age at menarche coefficient (SE)
Age	0.22 (0.02)**
Ethnicity	
White, non-Hispanic	REF
Black, non-Hispanic	− 0.36 (0.08)**
Hispanic	− 0.27 (0.09)**
FIPR	
≤ 130%	0.15 (0.13)
131–350%	0.02 (0.11)
> 350%	REF
HH REF highest level of education	
Less than high school	− 0.28 (0.15)
High school	− 0.15 (0.11)
Some college	− 0.01 (0.09)
College degree or higher	REF
Adult food security category	
High	REF
Marginal	− 0.17 (0.10)
Low	− 0.17 (0.12)
Very low	− 0.04 (0.11)
Health-insurance security	
Secure	REF
Insecure	− 0.08 (0.17)
Time in the USA	
Born in the USA	REF
Foreign born, in the USA < 10 years	0.13 (0.14)
Foreign born, in the USA ≥ 10 years	− 0.38 (0.32)
BMI category	
Underweight	0.83 (0.36)*
Normal weight	REF
Overweight	− 0.31 (0.11)**
Obesity	− 0.38 (0.10)**
Smoking	
Never	REF
Ever	0.23 (0.10)*
Diabetes	
No	REF
Yes	0.43 (0.21)*
Physical activity	
Sedentary (< 150 min/week)	REF
Exercising (≥ 150 min/week)	− 0.002 (0.13)
n	1694
r^2	0.15

FIPR Federal income poverty ratio. *HH REF* Household reference

*$p < 0.05$, ** $p < 0.01$

Because age significantly modulated infertility, we conducted a follow-up analysis to determine whether there were differences in age of first birth as a function of race/ethnicity and markers of SES. A subset of women ($n = 548$) also reported information on age at first live birth. We estimated a multiple linear regression predicting age of first live birth for

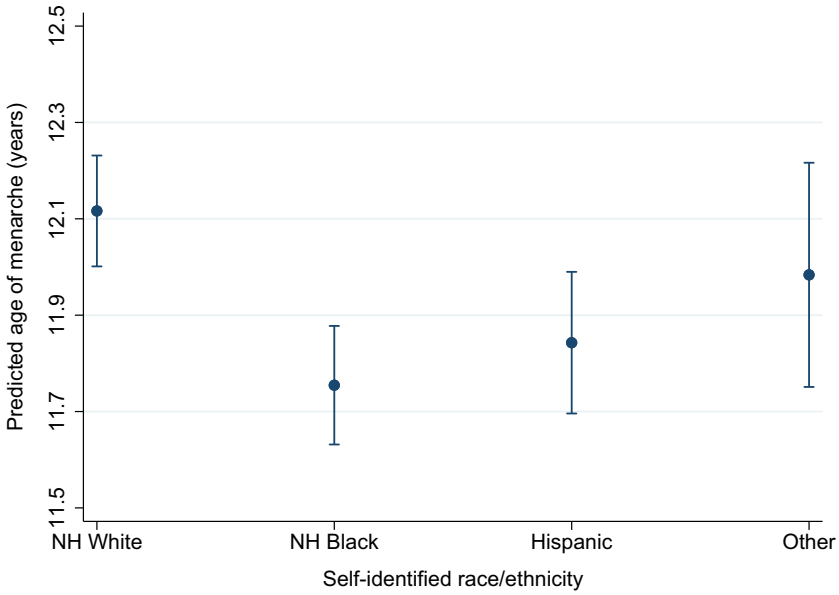

Fig. 2 Predicted age (and 95% CIs) of menarche among US women between 12 and 18 (n = 1694), National Health and Nutrition Examination Survey. Notes: Figure generated using marginal standardization adjusted for age, FIPR, household reference education, health-insurance security, time in the USA, BMI, smoking, diabetes, alcohol use, and physical activity

women on whom infertility data were available, when controlling for the same covariates included in the logistic regression predicting infertility. In this subset, relative to NH white women, NH black women (beta = −2.11 years, SE = 0.48, $p < 0.001$) and Hispanic women (beta = − 2.13 years, SE = 0.60, $p = 0.001$) reported younger ages of first live birth (full results not shown). This translated into predicted ages of first birth of 24.4 years for NH white women, 22.3 for NH black women, and 22.3 for Hispanic women (Fig. 4). Women who had completed less than high school (beta = − 4.99, SE = 1.00, $p < 0.001$), high school (beta = − 4.66, SE = 0.75, $p < 0.001$), and some college (beta = −3.27, SE = 0.65, $p = 0.001$) reported an age at first live birth significantly lower than women who had graduated college (full results not shown). Food security, health-insurance security, and time in the USA did not predict age of first birth.

Live Birth Ratios

Data from women between the ages of 23 and 45 were included in a linear regression predicting live birth ratios (Table 4; Fig. 5). Live birth ratios were lower in NH black women (beta = 9% lower, SE = 0.02, $p < 0.001$), but not Hispanic women (beta = 3% lower, SE = 0.02, $p = 0.154$) relative to NH white women. NH white and Hispanic women reported 81 and 78 live births per 100 reported pregnancies, respectively, while NH black women reported 72 live births per 100 reported pregnancies. FIPR, education, and food security were not associated with live birth ratios. Women who were categorized as health insurance insecure reported live birth ratios 6% lower relative to women categorized as health insurance secure (SE = 0.02, $p = 0.02$). Time in the USA did not predict live birth ratios. Results from a sensitivity analysis using a fractional outcome regression is displayed in Electronic Supplementary Material Table 1; importantly, the directionality and statistical significance of results were consistent with the linear regression results.

Discussion

The present study utilized data from a nationally representative sample of USA adolescent and adult women of reproductive age to assess whether age of menarche, infertility, and live birth ratios were associated with self-identified race/ethnicity, SES, and variables indexing downstream correlates of SES. Race/ethnicity was associated with a lower age of menarche and lower live birth ratios when controlling for SES and other variables, while SES and its downstream correlates were not consistently associated with these outcomes. Specifically, age of menarche was 4.3 and 3.2 months earlier in NH black and Hispanic girls, respectively, relative to NH white girls. Live birth ratios were lower in NH black women relative to NH white woman such that per 100 reported pregnancies, NH black women reported 9 fewer live births than did NH white women. Earlier age of menarche may predict increased risk of certain cancers [24] and psychosocial challenges in adulthood [22], making early age of menarche an unfavorable health outcome. As such, self-reported racial and ethnic minorities in the present study reported less favorable health outcomes as compared with NH white women across two of the three outcomes examined. Thus, our results replicate prior work and expand the range of women's reproductive health-related phenotypes that exhibit differences across race/ethnicity within the USA.

Table 3 Multiple logistic regression predicting infertility among US women between 23 and 45, National Health and Nutrition Examination Survey (NHANES)

Independent variable	Infertility OR (95% CI)
Age	1.09 (1.06, 1.13)**
Ethnicity	
White, non-Hispanic	1 (REF)
Black, non-Hispanic	0.73 (0.34, 1.55)
Hispanic	0.79 (0.36, 1.74)
FIPR	
≤ 130%	0.91 (0.39, 2.09)
131–350%	0.81 (0.48, 1.36)
> 350%	1 (REF)
Highest level of education	
Less than high school	0.18 (0.06, 0.55)**
High school	1.08 (0.48, 2.42)
Some college	0.84 (0.50, 1.40)
College degree or higher	1 (REF)
Adult food security category	
High	1 (REF)
Marginal	1.06 (0.48, 2.34)
Low	0.75 (0.28, 1.96)
Very low	1.30 (0.63, 2.67)
Health-insurance security	
Secure	1 (REF)
Insecure	0.83 (0.43, 1.60)
Time in the USA	
Born in the USA	1 (REF)
Foreign born, in the USA < 10 years	0.73 (034, 1.55)
Foreign born, in the USA ≥ 10 years	1.68 (0.77, 3.68)
BMI category	
Underweight	2.08 (0.31, 14.23)
Normal weight	1 (REF)
Overweight	1.04 (0.44, 2.46)
Obesity	2.00 (0.94, 4.28)
Smoking	
Never	1 (REF)
Past	0.72 (0.35, 1.47)
Current (some days)	1.11 (0.21, 6.64)
Current (every day)	1.04 (0.47, 2.31)
Diabetes	
No	1 (REF)
Yes	1.54 (0.61, 3.94)
Mean drinks/week	0.97 (0.79, 1.20)
Physical activity	
Sedentary (< 150 min/week)	1 (REF)
Exercising (≥ 150 min/week)	1.46 (0.87, 2.47)
n	974

FIPR Federal income poverty ratio

*$p < 0.05$, **$p < 0.01$

That the disparities in these outcomes persisted even when controlling for other variables previously shown to also modulate reproductive and general health, such as SES [35] suggests that self-identified race/ethnicity uniquely captures information not captured by any other variables in our models. To explain this finding, we look to the ecosocial theory of disease distribution, which posits that racial disparities in health outcomes may be in part explained by institutional variables such as economic deprivation and exposure to environmental toxins, as well as by interpersonal variables such as self-reported discrimination [65]. Embodiment specifically refers to the processes by which lived social and psychological influences become physioanatomically incorporated to influence biological processes [66], and a robust body of work supports links between subjective levels of experienced racism and a wide range of behavioral, psychological, and physical health outcomes [14]. Though work on the effect of interpersonal discrimination on women's reproductive function specifically is comparatively sparse, some studies have found links between self-reports of discrimination and perceptions of pubertal timing [61], and a recent systematic review of the limited existing literature on discrimination and pregnancy in NH black women found that discrimination may negatively influence biomarkers of maternal neuroendocrine, cardiovascular, and immune health [67]. It is reasonable to hypothesize that just as embodiment may contribute to observed racial and ethnic disparities in pregnancy-related outcomes, it may too contribute to racial/ethnic disparities in age of menarche and live birth ratios.

It is important to note that differences in live birth ratios were noted when comparing NH black and NH white women, but not when comparing Hispanic and NH white women. Some potential explanations for the observation that live birth ratios were not lower in both minority groups are that embodied discrimination in US minorities may manifest differently, or that discrimination is experienced at different scales. In fact, NH black adolescents [10] and adults [12] report higher levels of perceived discrimination than do their Hispanic counterparts. While we found that NH black women had live birth ratios that were 9% lower than those of NH white women, future research is needed to determine whether differential perceived discrimination among NH black and Hispanic women contribute to differences in reproductive outcomes.

Health-insurance security also was associated with lower live birth ratios, such that health-insurance insecure women had birth ratios that were approximately 6% lower. Health-insurance insecurity may be a downstream correlate of SES [68], and it may exert its effects on live birth ratios because lapses in healthcare access may limit women's access to preconception and prenatal care, both of which promote favorable pregnancy and birth outcomes [69]. Our results here underscore the importance of improving access to preconception and prenatal care

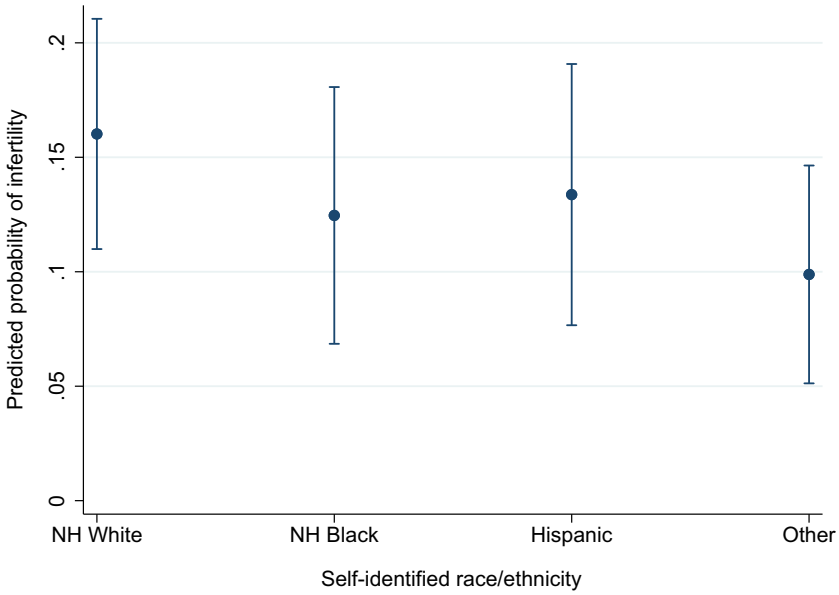

Fig. 3 Predicted probability (and 95% CIs) of infertility among US women between 23 and 45 (n = 974), National Health and Nutrition Examination Survey. Notes: Figure generated using marginal standardization adjusted for age, FIPR, household reference education, health-insurance security, time in the USA, BMI, smoking, diabetes, alcohol use, and physical activity

for women of reproductive age, which has been facilitated by the Affordable Care Act and Medicaid expansion [70]; as such, future efforts should continue to ensure accessible and affordable care for women of all race/ethnicity and SES groups.

Self-reported race/ethnicity, SES, and downstream correlates of SES were not associated with infertility in the present study. While we did not observe a stepwise relationship between SES and infertility, women who did not complete high school did have a significantly lower odds of infertility as compared with women who completed college. Relative to other analyses, those for infertility were based on a smaller sample, making it possible that these analyses were underpowered to detect any present, but small, effects, or that our pattern of results would not replicate in larger samples. Follow-up analyses here corroborate previously reported patterns in which racial/ethnic minorities and women with less education begin having children earlier than white women or women with more education [71, 72]. It is possible that by beginning their reproductive careers sooner, women of racial minorities and women with less education circumvent the effects of age-related declines in fertility [51]. Future work with larger samples and with women actively trying to conceive are necessary to better elucidate relationships between infertility, race/ethnicity, and SES.

The pathways by which racism and discrimination may act to modulate women's reproductive function are likely many. Here, we highlight one institution-level pathway and one interindividual-level pathway that may be theoretically likely to modulate reproductive outcomes. Minorities experience a

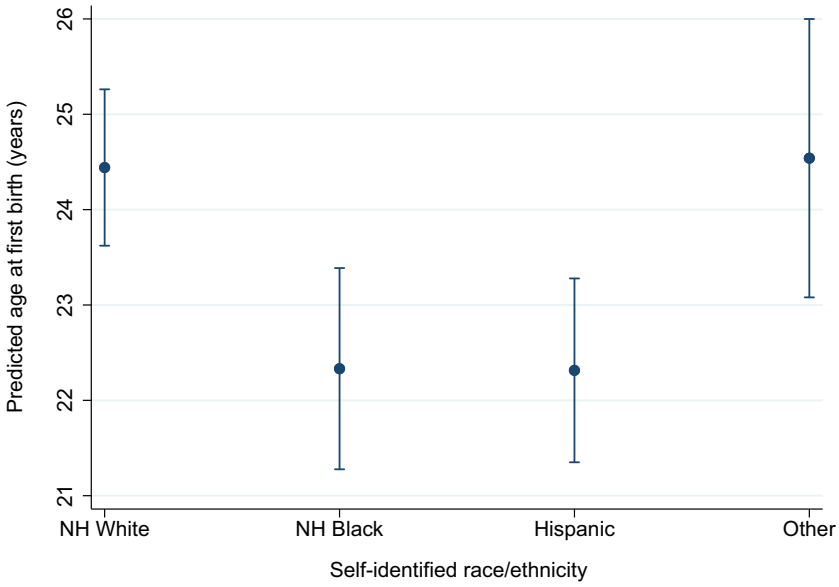

Fig. 4 Predicted age of first live birth (and 95% CIs) among US women between 23 and 45 (n = 548), National Health and Nutrition Examination Survey. Notes: Figure generated using marginal standardization adjusted for age, FIPR, household reference education, health-insurance security, time in the USA, BMI, smoking, diabetes, alcohol use, and physical activity

Table 4 Multiple linear regression predicting live birth ratios among US women between 23 and 45, National Health and Nutrition Examination Survey (NHANES)

Independent variable	Live birth ratios coefficient (SE)
Age	− 0.001 (0.001)
Ethnicity	
White, non-Hispanic	1 (REF)
Black, non-Hispanic	− 0.09 (0.02)**
Hispanic	− 0.03 (0.02)
FIPR	
≤ 130%	0.02 (0.02)
131–350%	0.01 (0.02)
> 350%	1 (REF)
Highest level of education	
Less than high school	0.02 (0.03)
High school	− 0.02 (0.03)
Some college	0.01 (0.02)
College degree or higher	1 (REF)
Adult food security category	
High	1 (REF)
Marginal	− 0.03 (0.03)
Low	< 0.001 (0.02)
Very low	− 0.004 (0.03)
Health-insurance security	
Secure	1 (REF)
Insecure	− 0.06 (0.02)*
Time in the USA	
Born in the USA	1 (REF)
Foreign born, in the USA < 10 years	− 0.06 (0.05)
Foreign born, in the USA ≥ 10 years	− 0.002 (0.02)
BMI category	
Underweight	−0.10 (0.07)
Normal weight	1 (REF)
Overweight	− 0.3 (0.02)
Obesity	− 0.02 (0.02)
Smoking	
Never	1 (REF)
Past	− 0.06 (0.02)*
Current (some days)	− 0.04 (0.03)
Current (every day)	− 0.05 (0.02)*
Diabetes	
No	1 (REF)
Yes	− 0.09 (0.04)*
Mean drinks/week	< 0.001 (0.01)
Physical activity	
Sedentary (< 150 min/week)	1 (REF)
Exercising (≥ 150 min/week)	0.02 (0.02)
n	1714
r^2	0.05

FIPR federal income poverty ratio

*$p < 0.05$, **$p < 0.01$

disproportionally high burden of environmental toxin exposure, such as exposure to endocrine disrupting chemicals (EDCs) [73]. Certain subtypes of EDCs are associated with earlier age of menarche [73], markers of infertility [74], and miscarriages [75]. EDCs exert deleterious effects by changing hormone regulation and action, as well as by inducing oxidative stress, inflammation, and gene expression [76], and it is possible that through influencing these physiological processes, EDCs modulate racial differences in reproductive axis function. The effects of EDCs on racial differences in reproductive function as well as the effects of other institution-level variables more generally require further investigation [65].

Interindividual differences in physiological and psychological stress may also modulate reproductive outcomes [77]. For example, lower self-reported anxiety and distress is associated with the likelihood of conception within a menstrual cycle [78] and successful outcomes in in vitro fertilization [79]. Biomarkers of stress may also predict early pregnancy loss [80] and unfavorable outcomes in in vitro fertilization [79]. One pathway by which psychological stress influences reproductive function is through influencing estradiol [81]: psychological stress dampens the hypothalamic-pituitary-gonadal (HPG) axis, likely through cortisol's negative effect on gonadotropin production [82]. This in turn lowers estradiol production, and lower estradiol is associated with decreased probabilities of conception [83]. Therefore, previously well-established links between discrimination and elevated psychological stress may affect reproductive outcomes. We hypothesize that discrimination-induced stress may decrease or otherwise disrupt HPG function, and that this decreased production of HPG axis outputs such as estradiol and progesterone may be acting as the proximate cause of the differences in age of menarche and live birth ratios observed in the present study (see Fig. 6).

Strengths and Limitations

Our study has several limitations. All outcome variables of interest and the majority of predictors were ascertained via self-report, which is prone to recall error and reporting bias. Although we believe embodiment and differential lived experiences best explain the observed racial disparities in age of menarche and live birth ratios, measures of self-reported racism and discrimination are not administered as part of NHANES; thus, we are unable to empirically point to embodiment as the explanation for our results. To test this hypothesis, future studies should collect detailed data on physical and mental health, reproductive outcomes, and demographic factors in concert with measures of institutionalized and personally mediated discrimination [37]. Finally, longitudinal studies should be employed to evaluate how interindividual differences and intra-individual changes are linked to discrimination, physical health, access to care, and reproductive function

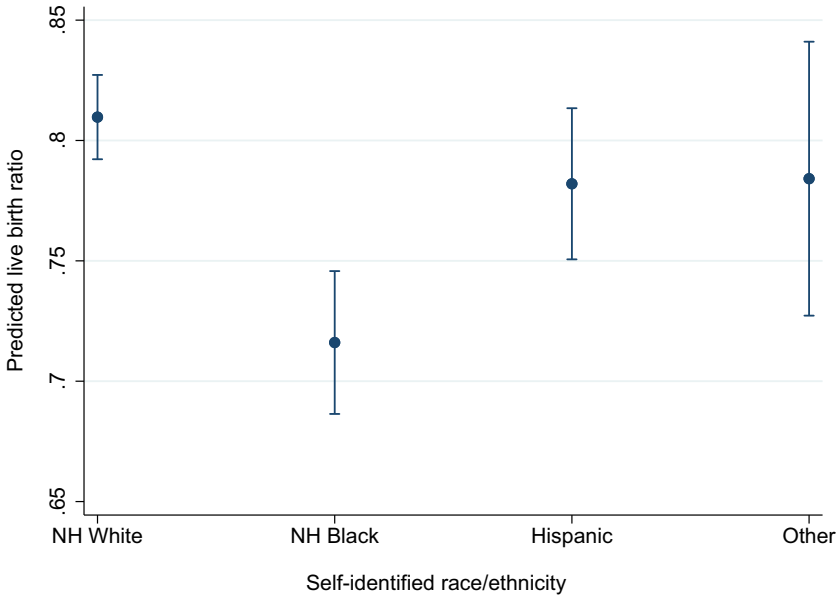

Fig. 5 Predicted live birth ratios (and 95% CIs) among US women between 23 and 45 ($n = 1714$), National Health and Nutrition Examination Survey. Notes: Figure generated using marginal standardization adjusted for age, FIPR, household reference education, health-insurance security, time in the USA, BMI, smoking, diabetes, alcohol use, and physical activity

across time. Despite these limitations, this study uses the most recent 10 years of NHANES data to estimate associations among variables related to lifestyle, SES, race/ethnicity, general health, and reproductive function with nationally representative estimates and thus contributes to our understanding of reproductive health disparities, as well as to our understanding of the unique effects (or lack thereof) of SES and race/ethnicity on such disparities.

Conclusion

In conclusion, our data suggest disparities across self-identified race/ethnicity categories in the USA in age of menarche and live birth ratios, and differences in live birth ratios as a function of health insurance security. These associations are apparent when statistically controlling for SES, downstream correlates of SES, and physical states associated with alterations reproductive function. We theorize that just as discrimination gives rise to disparities in general physical and mental health, so too may it give rise to disparities in women's reproductive function, although empirical data are needed to evaluate this working hypothesis. Future work should identify the specific biological pathways by which discrimination affects reproductive function, and be used to create evidence-based programs to mitigate reproductive health disparities across self-identified racial and ethnic categories.

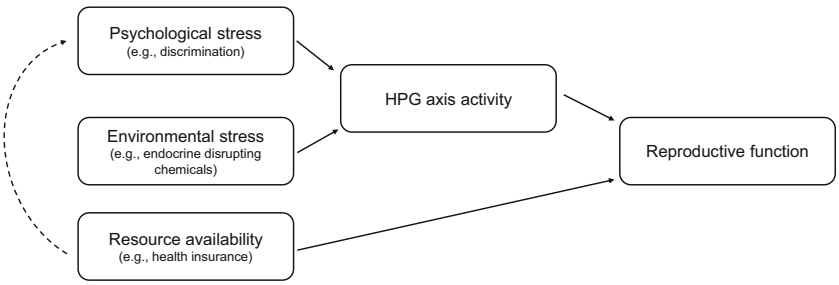

Fig. 6 Hypothesized relationships linking psychological distress to hypothalamic-pituitary-ovarian axis activity and concomitantly, reproductive function. Notes: dashed line indicates indirect effect

Compliance with Ethical Standards

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (NCHS Research Ethics Review Board) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflicts of Interest The authors declare that they have no conflict of interest.

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