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Body mass index during early adulthood and first births: Racial/ethnic and sex differences in the US NLSY79 cohort

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Title page

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Racial/ethnic and sex differences in the US NLSY79 cohort

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Abstract

Growing evidence indicates lifetime fertility is predicted by health conditions during early adulthood such as body mass index ("early BMI"). Less is known if the early BMI to fertility pathway differs by race/ethnicity, a major axis along which disparities in both health and fertility develop. We examined, within each sex, how the deviations of early BMI from healthy range relate to first birth timing and lifetime childlessness in Blacks, Hispanics, and Whites of the US NLSY79 cohort. Obesity was consistently associated with higher childlessness across race/ethnic groups in both sexes, but only in women, its implication in delaying first births manifested after early adulthood. The overall higher childlessness among underweight women was largely driven in Blacks, whereas the lower childlessness among underweight men was detectable in Blacks and Whites. Our findings on the intersectionality of race/ethnicity and sex in the BMI-childlessness pathways encourage more research on the underlying mechanisms.

Keywords: body mass index (BMI), childlessness, first birth, age at first birth, race/ethnicity, Blacks; Hispanics; Whites; United States, NLSY79

Data availability statement

The data that support the findings of this study are available in the NLS Investigator website (https://www.nlsinfo.org/investigator/pages/login) for registered users. Upon publication of this study, the new data on early BMI generated from the present study will be made available on GitHub together with R codes used to generate the data.

Introduction and Background

Childlessness and BMI

Body mass index (BMI; weight [kg] divided by height [m]-squared) is one indicator of adiposity that has received extensive attention in the medical literature in relation to health as well as fertility (Gaskins et al. 2015; Gesink Law, Maclehose, and Longnecker 2007; Ramlau-Hansen et al. 2007). Studies have shown higher chances of experiencing negative birth outcomes or reduced fecundity among underweight (Boutari et al. 2020; Derbyshire 2014) and obese individuals (Cavalcante et al. 2019; Crean and Senior 2019; Kawwass et al. 2016; Luke et al. 2011; Nguyen et al. 2007; Sermondade et al. 2013) for both sexes. Moreover, BMI may shape the social process of partnering, for example by influencing one's self-esteem and self-perceived attractiveness (Kallen and Doughty 1984; La Rocque and Cioe 2011), attractiveness to potential mates (Swami 2006) and chance of being discriminated in education and the labor market (Puhl and Brownell 2001; Spahlholz et al. 2016), all of which can affect individual's standing in mating markets. As yet, studies on the connection between BMI and fertility have typically been concentrated either in the clinical literature of infertility, or in the social sciences literature examining mating dynamics, leaving the association between BMI and completed fertility largely unaddressed from a demographic perspective.

A handful of existing studies conducted in Finland, Sweden, and the United States (US) suggest that BMI measured during early adulthood ("early BMI") influences lifetime fertility (Barclay and Kolk 2020; Jokela et al. 2007; Jokela, Elovainio, and Kivimäki 2008). In general, young adults with BMI higher than the range for healthy weight (18.5-24.9) tend to have fewer children and are more likely to remain childless (i.e., having zero biological children) at the end

of their peak reproductive years. Additionally in men, BMI lower than the healthy range is associated with lower fertility. These patterns appeared independent of socioeconomic status (Barclay and Kolk 2020; Jokela et al. 2007). Understanding the long-term consequences of early BMI for lifetime fertility is intriguing given the important secular trends in contemporary populations – the growing social and health disparities by early BMI (Frederick, Snellman, and Putnam 2014) and the increasing prevalence of delayed first births and childlessness (Kreyenfeld and Konietzka 2017; Ombelet and Goossens 2017).

Less is known about the extent to which the pathway linking early BMI to lifetime childlessness differs by race/ethnicity, a major axis along which disparities develop in terms of both BMI (Clarke et al. 2009; Yang et al. 2021) and childbearing (Darabi and Ortiz 1987; Sweeney and Raley 2014; Torres and Parrado 2022). Although previous work examining the BMI-fertility relationship in the National Longitudinal Study of Youth 1979 (NLSY79) cohort of the US did consider race/ethnicity as a statistical control, heterogeneity by race/ethnicity was not examined in detail (Jokela et al. 2008). This is unfortunate given that the NLSY79 cohort is one of the only data sources that can be used to examine the longitudinal association between early BMI and lifetime fertility by race/ethnicity. With the growing evidence that pathways into childlessness are gendered (Chudnovskaya 2019; Fiori, Rinesi, and Graham 2017; Keizer, Dykstra, and D. Jansen 2008; Saarela and Skirbekk 2020; Trimarchi and Van Bavel 2017), there is a further need to study the intersectionality between race/ethnicity and gender in the BMI-childlessness association.

From early BMI to childlessness: Possible racial/ethnic differences

In this study, we examine whether the association between early BMI and lifetime fertility is consistent across race/ethnicity, and how potential differences between racial/ethnic groups may interact with sex. Despite the well-known racial/ethnic disparities in early BMI (Cali and Caprio 2008) and in childlessness (Boyd 1989; Lundquist, Budig, and Curtis 2009; Stulp et al. 2016) in the US context, they have rarely been studied in a combined framework. The pathways linking early BMI to childlessness may differ by race/ethnicity for several reasons that are not mutually exclusive.

Racial/ethnic differences in BMI

First, the pathways may differ because BMI differs by race/ethnicity. Evidence suggests that racial/ethnic disparities in BMI emerge early in life (Dixon, Peña, and Taveras 2012; Krueger, Coleman-Minahan, and Rooks 2014; Krueger and Reither 2015; Menigoz, Nathan, and Turrell 2016; Rossen and Schoendorf 2012). If the higher childlessness among those who were obese during early adulthood is driven by an ethnic group with particularly high prevalence of obesity, then it would suggest that the racial/ethnic disparity in obesity could exacerbate racial/ethnic disparities in lifetime fertility. On the other hand, if the early BMI to childlessness relationship is relatively consistent across race/ethnicity despite some racial/ethnic differences in BMI, the degree of such consistency might allow us to gauge the physiological or behavioral significance of BMI in mediating lifetime fertility outcomes.

Racial/ethnic differences in preferred BMI

The way BMI is associated with perceived attractiveness may also differ by race/ethnicity and sex (Swami 2006), giving rise to heterogeneous BMI-childlessness relationships if perceived attractiveness influences the chance of partnering and eventually childlessness. Marriage or co-residential union formation, and its timing, have remained one of the strongest predictors of lifetime childlessness in recent US cohorts including the NLSY79 sample cohort (Hayford 2013; Nitsche and Hayford 2020). Previous studies have highlighted marriage (or partnering in general) as a key mediator by which early BMI is associated with lifetime fertility (Barclay and Kolk 2020; Jokela et al. 2007, 2008). As such, to the extent that the probability of union formation or partnering is influenced by early BMI, any racial/ethnic differences in preferred BMI may lead to racial/ethnic heterogeneity in the relationship between early BMI and childlessness.

For instance, it has been hypothesized that ethnic minority women may be 'buffered' from the sociocultural pressure to conform to a slender body type given that the thin body ideal is largely associated with the Western, White-majority culture (Powell and Kahn 1995). In the studies conducted in the US, compared to their White counterparts, Black men reported preferring a heavier body weight for women, and Black women rated slightly heavier body weight to be ideal as well (Flynn and Fitzgibbon 1998), and Black women reported more satisfaction with their body weight despite being heavier than their White counterparts (Chithambo and Huey 2013; Miller et al. 2000). However, ethnic minority women had a similar or even higher tendency to report negative body esteem (Frayon et al. 2021; Grabe and Hyde 2006; Sotiriou and Awad 2020), suggesting that women of non-White groups may nevertheless be susceptible to the slender body ideal. Preferred BMI as perceived by self and others may be

associated with reproductive behaviors and fertility in various ways, and testing them is beyond the scope of this study. To motivate such inquiry, here we ask the first question of whether lifetime fertility among the Black and Hispanic women of the NLSY79 sample would be less affected by deviations from healthy BMI, as implied by the buffering hypothesis.

In men, although body *shape*, such as waist-to-chest ratio, tends to contribute more to perceived bodily attractiveness than does body *size* such as BMI (Tovée et al. 1999), BMI still appears to be a significant factor influencing family formation (Swami 2006). The significance of men's BMI on perceived attractiveness might depend on the context. For example, while Greek women and British women reported to prefer men of healthy BMI range, Greek women preferred slightly thinner male physique presumably because what was perceived as 'healthy body size' differed in Greek society with the highest male obesity rate in Europe (Swami et al. 2007). Unlike in women, there appears no particularly strong preference for slender body size in men. Instead, we might postulate that preference for heavier weight (to the extent that it suggests greater strength) exists. In support, evidence suggests that men tend to underestimate their body weight due to a presumably higher preference for being larger, whereas the opposite is true for women (McCreary 2001, 2002; McCreary and Sasse 2000), and; male undergraduate students in the US aspired for a large, muscular body shape ideal (Mintz and Kashubeck 1999). Such aspirations and related body esteem does not seem to differ by race/ethnicity (Franzoi and Chang 2002; Mintz and Kashubeck 1999). This evidence suggests that there may be no 'buffering' for ethnic minority men from the influence of body size ideals, in contrast to what is hypothesized in women. Nevertheless, racial/ethnic differences in the perception of bodily attractiveness in men, and more generally its possible role in family formation processes, remains less studied for men.

Discrimination and intersectionality

Studies indicate that discrimination based on weight occurs, particularly in terms of obesity. Obese individuals face discrimination in the labor market, in access to and support for education, and in the housing market (Flint et al. 2016; Puhl and Brownell 2001; Spahlholz et al. 2016). Moreover, obesity appears to interact with other individual characteristic such as sex, in affecting the chance of being discriminated across life course. For instance, studies showed that obese women, but not men, faced hiring discrimination (Campos-Vazquez and Gonzalez 2020) and that obese girls, but not boys, were less likely to enroll in college than 'normal' weight peers (Crosnoe 2007). Weight discrimination has also been shown to accumulate with other forms of discrimination, such as race/ethnicity-based discrimination (Pearl et al. 2018). This intersectionality between body weight, sex, and race/ethnicity may place obese or underweight individuals from race/ethnic minority groups in a position of exacerbated disadvantage in the mating market, either in terms of having access to potential partners, union formation processes, or union stability.

Present study

Here, we revisit the NLSY79 data and compare childlessness by early BMI among Hispanics, Blacks, and Whites within each sex. We address one limitation in the NLSY79 data for studying the relationship between early BMI and childlessness, which is that early BMI was measured during young adulthood (average 20 years old) when more than a quarter of the sample were within a year from, or had already finished transitioning to, first parenthood. This condition led the previous study on the same NLSY79 data (Jokela et al. 2008) to exclude early childbearers – here defined as those who already had biological children by 1981, the first year in which BMI

was measured in the NLSY79 sample or those who gave first births before 17 (supplementary material S1). The purpose of excluding early childbearers was to avoid contamination of the early BMI and fertility relationship by the known reverse impact of parenthood on BMI (Corder et al. 2020). However, doing so inevitably overestimates the prevalence of childlessness and potentially distorts racial/ethnic differences in childlessness by early BMI in multiple ways. For example, the median age at first birth is 7 years younger among early childbearers (19 years) than non-early childbearers (26 years), in both sexes, and there are more early childbearers in women (33.5%) than in men (20.3%). Among women whose fertility information at age 40+ was available, the prevalence of childlessness was 15.5, 11.5, and 18.1 % for Blacks, Hispanics, and Whites respectively, but was 27.8, 19.3, 23.3 % after excluding early childbearers. In other words, significantly larger proportions of Black and Hispanic compared with White mothers are excluded using this strategy, introducing bias by race/ethnicity in the sample for the prospective analyses.

Excluding the dynamics of early childbearing also leaves unknown *when* the delayed transition to first parenthood associated with BMI manifests. In light of the previous findings that suggests higher childlessness among those with extreme (either too low or too high) BMI (Barclay and Kolk 2020; Jokela et al. 2007, 2008), one might expect that those who become parents early are more likely to exhibit healthy BMI. But it is also possible that the implications of healthy BMI on accelerating the transition to parenthood depend on age, to the extent that the healthy BMI exerts its effect on lifetime fertility via selection into partner markets, as considered in the above section on racial/ethnic difference in preferred BMI. If so, we would expect that the association between healthy BMI and an accelerated transition to parenthood begins to appear only after young adulthood when women reach typical marriage age. Such temporal dynamics in

the effect of early BMI may further exhibit racial/ethnic differences, if preferred BMI differs by race/ethnicity and in turn affects relational and mating behaviors over the life course.

Thus, incorporating early childbearers into the analysis is crucial to understand the extent to which the influence of early BMI on childlessness differs by race/ethnicity. We achieve this goal by imputing pre-childbearing BMI at a fixed age, so that early childbearers, who are more likely to be non-Whites and were omitted in the previous study on the same NLSY79 data (Jokela et al. 2008), can be incorporated into the analyses. Using the expanded dataset, with 37% more individuals, we seek to answer the following research questions:

- Study 1. Does early BMI predict childlessness at age 40+, and are there racial/ethnic differences?
- Study 2. To what extent does the pace of transition to first birth explain the BMI-childlessness relationship, and are there racial/ethnic differences?

We address these questions separately for each sex. In Study 1, we compare the distribution of predicted probabilities of childlessness for Blacks, Hispanics, and Whites by early BMI. To gauge the significance of racial/ethnic differences in the relationship between early BMI and lifetime childlessness, we conduct probabilistic model comparisons using information criteria and estimate relative quality of predictive accuracy of a model that assumes race/ethnicity interaction in the BMI-childlessness association. In Study 2, we analyze time to first births by early BMI and further by race/ethnicity, and describe patterns based on survival curves and hazard rate differences between early BMI groups. Because the previous study also compared time to first births by early BMI (Jokela et al. 2008), we are also interested in to what extent the

findings regarding the impact of early BMI change once we incorporate early childbearers in the analyses and a larger sample of the NLSY79 cohort more representative in terms of racial/ethnic differences in both timing and quantum of first births.

Data and Methods

NLSY79 cohort

The NLSY79 cohort is a longitudinal project that follows the lives of the birth cohort who were born 1957-1964 in the USA, comprising the younger portion of the baby boom generation (Hogan, Perez, and Bell 2008). The survey started in 1979 when the recruited subjects were aged 14 to 22, and has been repeated annually until 1994 and biennially since, with the most recent survey conducted in 2018. Response rates are high in the sample, e.g., 89% of those from 1979 were followed through 2014 after excluding those deceased (Rothstein, Carr, and Cooksey 2019). Studies found no evidence that attrition has biased the estimated models of employment and wages (MaCurdy, Mroz, and Gritz 1998) or the relationship between educational attainment and labor market outcomes (Aughinbaugh, Pierret, and Rothstein 2017).

Outcome variable: Childlessness

We study childlessness by examining both whether the index person was childless by age 40+ (Study 1), and time to first parenthood, if any (Study 2). We used the "number of children ever born" (NUMKID_XRND) variable to code whether a respondent was childless when her or his last interview was at age 40 or more, and used "age of respondent at first birth" (AGE1B_XRND) variable for those who ever had a child and age at last interview for those who never had a child or were censored before the birth of first child.

Independent variables

Early BMI

We used BMI information available from the 1981, 1982, and 1985 surveys, when the NLSY79 participants was between 16 and 28 years old. In the original 12,686 NLSY79 sample, there were 3,415 individuals (28.2%) whose BMI was only known after their transition to parenthood, and among them 67.1% were females. To be able to incorporate these individuals into our study, we predicted their early BMI before becoming a parent, as follows.

First, we selected 34,995 BMI measurements from 12,599 individuals (6,194 females and 6,405 males). We excluded 87 individuals whose parenthood status at BMI measurement was not known, as this condition would not allow us to predict pre-parenthood BMI. Among the Among the 12,599 individuals, BMI was measured three times for the 83% and two times for the 15%. Second, within each sex, we estimated a model for predicting early BMI. To reflect the data structure of repeated BMI measures within individuals, a mixed-effects model (aka multi-level model) was specified as

$$y_i = \alpha_{j[i]} + X_i\beta + \epsilon_i$$

$$\alpha_j \sim N(\mu_\alpha, \sigma_\alpha^2), \text{for } j = 1, \dots, J,$$

where we have observations of BMI $i = 1, \dots, n$ clustered within individuals $j = 1, \dots, J$, y_i is each observed BMI measurement, and α_j is individual intercepts (random-effects) assumed to follow normal distribution. $X_i\beta$ is a matrix of fixed-effects specific to BMI (age at BMI measurement, whether a BMI was measured at or after the transition to parenthood) and to individual background (age at recruitment to the NLSY79 sample, NLSY79 subsample membership, race/ethnicity, parents highest degree of education, religion during upbringing, residence [urban/rural/farm], with whom a respondent resides). Third, we used the estimated

coefficients, including individual intercepts, to construct for each individual a model to predict BMI at age 16 before transitioning to parenthood, while setting the variables on individual background at each respondent's observed values. Supplementary materials S2 provide more details on the modeling and validation of this method. Lastly, we categorized the predicted BMI values into underweight (<18.5), healthy (18.5-24.9), overweight (25.0-29.9) and obese (>30.0) following the standard clinical classification of BMI. We used the categorical instead of continuous variable to allow for easier interpretations of the interaction between BMI and race/ethnicity.

For the study sample, we excluded individuals who belonged to any race/ethnicity groups other than Blacks, Hispanics, and Whites, and whose reported age at first birth was before 16 because 1) too young a reported age at first birth could indicate reporting error and 2) incorporating them into the analyses would require the predicted BMI to be set at age 10 given that the youngest reported age at first birth was 11. As a result, from the original NLSY79 sample of 12,686 respondents, our study sample size was 10,595 (Figure 1).

[Figure 1 about here]

Race/ethnicity

We used 3 categories of race/ethnicity: Blacks, Hispanics, and Whites, based on the information assessed during the National Opinion Research Center household screening in 1978 when interviewers were instructed to code the race/ethnicity information using a set of guidelines. Respondents were also asked during the first interviews in 1979 to name the racial/ethnic origins with which they identified from a listing of almost 30 categories. Among those who identified

themselves as neither Blacks nor Hispanics, we selected those who identified themselves as of European origin (68%) and created a group of respondents operationally defined as Whites. Within our study sample, there were 2,926 Blacks (27.7%), 1,648 Hispanics (15.6%), and 5,997 Whites (56.7%). The higher representation of Blacks and Hispanics in our sample compared to that at the population-level during early 1980s (Smelser, Wilson, and Mitchell 2001) allows an opportunity to better examine heterogeneity by race/ethnicity.

[Figure 2 about here]

Statistical analysis

We estimate models within each sex, based on the causal diagram where the estimand is the effect of early BMI on childlessness (Figure 3). We model a causal process in which race/ethnicity underlies the variations in both early BMI and childlessness (i.e., a confounder), and behaviors that are hypothesized to be affected in part *by* early BMI – such as union formation, variables directly related to socioeconomic status, or fertility desire – lie on the causal path between early BMI and childlessness (i.e., mediators). As such, only race/ethnicity comprises biasing paths that are necessary and sufficient to be adjusted to estimate the total effect of early BMI on childlessness to avoid over-control bias (Rohrer 2018; Textor et al. 2016) and to examine how the total effect differs by race/ethnicity. In this regard, we do not adjust for mediating variables in the statistical models estimated for the present study. The NLSY79 sample was recruited at ages between 14 and 21 years, and such differences in yearly birth cohort can potentially influence both early BMI and childlessness. As such, yearly birth cohort is another confounder that could potentially compete with race/ethnicity to bias the early BMI to

307 childlessness path. We thus adjust for differences in yearly birth cohort in all models, but do not
308 interpret their estimates.
309 [Figure 3 about here]
310 For Study 1 on early BMI and childlessness, and racial/ethnic differences therein, we
311 modeled within each sex a binary indicator of whether a respondent *i* remained childless at age
312 40+, C_i, using a log link function and as a function of early BMI category (X_1) and race/ethnicity
313 category (X_2):
314
315 $$\text{logit of } (C_i) = \beta_0 + \beta_1 X_1 + \beta_2 X_2$$
316
317 where the inverse logit (or log odds) of β_0, i.e., $e^{\beta_0}/(1 + e^{\beta_0})$, is a predicted proportion of
318 childless individuals in the reference group (who exhibited healthy BMI at age 16 and are
319 Whites), and the inverse logit of $\beta_0 + \beta_1$and $\beta_0 + \beta_2$ are the proportion of childlessness in the
320 non-healthy BMI groups or non-Whites groups, respectively. To be able to fully represent the
321 degree of uncertainty around estimates, we employed Bayesian framework with the Markov
322 chain Monte Carlo (MCMC) algorithm –specifically Hamiltonian Monte Carlo– as simulation
323 technique, and made use of posterior predictive distribution generated from the posterior
324 distribution of estimates. Making use of posterior predictive distribution is recommended over
325 summarizing results based on posterior distribution of estimates, because the former takes into
326 account variance in not only estimates (estimation uncertainty) but also their distribution per se
327 (sampling uncertainty) (McElreath 2020). To obtain the posterior predictive distribution of
328 probability that underweight Hispanic women remain childless, we randomly drew 4,000
329 predicted binary outcomes (childless or not) from the sampling distribution of estimates by

setting early BMI at underweight and race/ethnicity at Hispanics, then took average over the 4,000 draws to get predicted proportion of childlessness, and repeated this process 10,000 times to finally get the distribution of the predicted proportion of childlessness. We interpret the proportion as probability of remaining childless for respective groups. To understand the overall significance of racial/ethnic difference in the BMI to childlessness pathway, we compared models that either allowed or not the interaction between early BMI and race/ethnicity. Specifically, we performed efficient approximate leave-one-out cross-validation (LOO-CV) for Bayesian models using Pareto smoothed importance sampling, to assess if inclusion of the interaction term improves predictive accuracy for a model relative to a simpler model without the interaction term (Vehtari, Gelman, and Gabry 2017).

For Study 2 on the pace of transition to first parenthood by early BMI, and racial/ethnic differences therein, we conducted survival analyses and modeled age at first birth as waiting time until the transition to first parenthood. Using the proportional hazard model, the expected 'hazard' of transitioning to first birth at age t is assumed to be a function of early BMI category (X_1) of respondent i:

$$\lambda(t) = \lambda_0(t)\exp(\beta_1 X_1)$$

where $\lambda_0(t)$ is the baseline hazard rate and represents the probability that the transition to the first parenthood occurs if such transition has not occurred up to age t for the reference group (who exhibited healthy BMI at age 16), and β_1 represents hazard rate of a BMI group relative to the hazard rate for the healthy BMI group. As we test the significance of the interaction between early BMI and race/ethnicity in the Study 1, our focus in the Study 2 is to describe and test

differences in the rate of first births by early BMI categories within each race/ethnicity and sex. As such, models only within each sex and then further within each race/ethnicity group, without testing the interaction between early BMI and race/ethnicity.

We used R version 4.1.0 (R Core Team 2021) for data processing, statistical analysis, and visualization, and the R packages "rstanarm" (Goodrich et al. 2020) for fitting and diagnosing MCMC models and "bayestestR" (Makowski, Ben-Shachar, and Lüdecke 2019) and "bayesplot" (Gabry and Mahr 2017) for processing model outputs. Every model was run for 4 chains each with 5,000 iterations (including a burn-in period of 1,000 iterations which are discarded), examined if the chains converged well by checking split $\hat{R}$ (< 1.1) and trace plots (Muth, Oravecz, and Gabry 2018). More details on modeling results are presented in the supplementary materials 3 and 4.

We report median of posterior samples as point estimate and 90 % credible interval (CI) in brackets as credible interval to express uncertainty around each point estimate. For the credible interval, we compute the lower and higher values comprising the 90% quantile of a given distribution. Both point estimates and credible intervals are calculated based on sampling from the posterior predictive distribution of estimates for the Study 1 and posterior distribution of estimates for the Study 2. Whenever a comparison is made, the 90% indicates the estimated differences expected at 90% chance; if it does not overlap with zero, it means that there is a smaller than 5% chance that the difference is the opposite direction.

Results

Study 1. Does early BMI predict childlessness at age 40+, and are there racial/ethnic differences?

The chance of remaining childless at age 40+ differed by early BMI (Figure 4A for women, Figure 5A for men). In both sexes, being obese during early adulthood was associated with higher probability of childlessness compared to healthy BMI counterparts, by a larger degree in women (0.14 [90% CI: 0.13-0.16]) than in men (0.09 [0.07-0.11]). Being underweight had different implications for men and women: Although being underweight during early adulthood was associated with an average 0.19 [0.17-0.21] *increase* in the probability of childlessness compared to healthy BMI counterparts for men, it was associated with on average 0.03 [0.02-0.04] *decrease* for women. Being overweight during early adulthood was also associated with higher childlessness (in men 0.05 [0.03-0.07], in women 0.02 [0.01-0.04]) relative to healthy BMI counterparts, but the magnitude of the difference relative to the healthy BMI group was not larger than either underweight or obese. Whether the relationship between early BMI and childlessness differs by race/ethnicity was dependent on sex.

Findings for women

Among women, the racial/ethnic differences were most evident for the association between being underweight and childlessness (Figure 4, light grey lines). The lower childlessness associated with the underweight was driven predominantly by the trend in Black women, where the probability of childlessness was lower than that of healthy BMI counterparts by 0.13 [0.12-0.14]. Indeed, underweight Black women are predicted to be least likely to remain childless

(Figure 4B, light grey line). In contrast, being underweight during early adulthood was associated with slightly higher childlessness (Hispanic women: [0.00-0.03]; Figure 4C) or was not distinguishable (White women [-0.02-0.01]; Figure 4D) in the chance of remaining childless compared to healthy BMI counterparts. As indicated by the inclusion of 0 in the 90% CI, certainty around the difference between underweight and healthy BMI group was lower for Hispanic women and White women. This is also shown by some large overlap in the posterior estimates of the probability of childlessness between those for underweight women (Figure 4, light grey lines) and healthy BMI women (Figure 4, turquoise lines) especially in White women.

Racial/ethnic differences are less pronounced among obese or overweight women. Childlessness was consistently higher among the obese across racial/ethnic groups compared to the healthy BMI counterparts. The difference with the healthy BMI group was largest among White women (0.20 [0.18-0.21]), followed by Blacks (0.13 [0.11-0.14] and Hispanics (0.08 [0.07-0.10]). The degree to which being overweight is associated with higher childlessness was consistently small across race/ethnic groups with 0.03 [0.02-0.04] for Hispanic women, 0.05 [0.03-0.06] for White women, and 0.01 [0.00-0.03] for Black women where the overweight and healthy BMI difference in childlessness was the smallest and less certain given the inclusion of zero in the 90% CI.

[Figure 4 about here]

This racial/ethnic contrast, mostly amongst underweight women, likely explains the relatively small support for the model allowing the interaction between race/ethnicity and childlessness, according to model selection. The model with the interaction between race/ethnicity and early BMI received expected log predictive density (ELPD) that is higher by 0.9 (standard error [SE] of the difference = 3.3) compared to the model without the interaction.

Given that the SE of the difference was larger than the difference itself, we interpret this model comparison result as evidence of marginally better accuracy of the model with the interaction term.

Findings for men

Amongst men, the model without the interaction between race/ethnicity and early BMI exhibited *higher* accuracy than the model with the interaction, with the expected log probability density higher by 4.4 (SE = 2.2). The model comparison result contrasts with slightly higher accuracy supported for the model with the interaction term in women, and thus suggests that evidence of racial/ethnic heterogeneity in the BMI-childlessness relationship is clearly weaker in men than in women. This conclusion is supported by the general pattern that deviations from the healthy BMI were all associated with the higher predicted probability of childlessness compared to that of the healthy BMI counterparts in men across race/ethnicity (Figure 5). One visible exception was among underweight Hispanic men, whose probability of remaining childless was lower by 0.03 [0.02-0.05] compared to their healthy BMI counterparts. The opposite is true for underweight Black or White men, where the predicted probability of childlessness jumps up by 0.16 [0.14-0.18] for Blacks and 0.25 [0.23-0.26] for Whites compared to respective racial/ethnic group's healthy BMI counterparts. Because the increase in childlessness is by much larger degree for underweight Black or White men, than for underweight Hispanic men do (0.03), the pattern for underweight Hispanic men is 'masked' if average pattern across race/ethnicity is examined (Figure 5 light grey lines in panel A, compared to panels B, C, and D).

[Figure 5 about here]

Study 2. To what extent does the pace of transition to first birth explain the BMI-childlessness relationship, and is there racial/ethnic difference?

Tracing the impact of early BMI on transition to first births shows that the impact may depend on not only race/ethnicity but also on age in women. We do not find similar evidence for men.

Findings for women

Among women, the average rate of first births at a given age was higher for those who were underweight (Figure 6A, All women) and lower for those who were obese at age 16 (Figure 6C, All women) compared to that of the healthy BMI group (Table 1).

When further examined by race/ethnicity, differences were most pronounced for underweight women. In line with the findings from the Study 1, the faster transition to first births in underweight women compared to the women of healthy BMI was a pattern most visible in Blacks (Figure 6A, Blacks). There was evidence that underweight White women make faster transition to first births but only during early 20s (Figure 6A, Whites). This would explain why lifetime childlessness between underweight White women and White women of healthy BMI was not distinguishable in the Study 1. In Hispanics, nearly 23% of the posterior estimates lied on the negative side, suggesting some degree of uncertainty in the degree to which underweight Hispanic women make faster transition to first births compared to their healthy BMI counterparts (Figure 6A, Hispanics).

In contrast to such racial/ethnic heterogeneity for underweight women, the delayed transition to first parenthood among the obese women compared to the women of healthy BMI was a pattern most consistently and clearly visible across race/ethnicity groups (Figure 6C, Figure 7 bottom).

The relative rate of overweight women becoming a parent was not clearly distinguishable from that of the healthy BMI group (Figure 6B, All women; Table 1), and this observation remained similar across racial/ethnic groups (Figure 7, Overweight). The lower certainty appears to be due to the different impact of being overweight depending on age (faster transition during early adulthood, slower transition in late adulthood), rather than due to similarity with the healthy BMI group. In particular among White women, there was evidence that being overweight 'switches' its direction of impact from positive to negative, such that being overweight accelerates the transition to first parenthood during early adulthood and then delays the transition after 30, compared to the rate of transition for White women of healthy BMI (Figure 6B, Whites). This pattern likely explains why the distribution for differences in posterior estimates distributed 50:50 below and above zero in the overweight White women (Figure 7C middle).

[Figure 6 about here]

[Table 1 about here]

Due to differential impacts of early BMI by age, particularly during early adulthood, we reach a different conclusion about the relationship between early BMI and transition to first births depending on whether early childbearers are included or excluded from the analyses (Table 1). Specifically, after including early childbearers, 1) the relative acceleration in transition to first parenthood among the underweight women becomes stronger and more evident, while 2) the relatively slower transition to first parenthood among the overweight or obese women become weaker, because overweight or obese women are not necessarily delayed in the transition during early adulthood. Supplementary material S5 provides a direct comparison between findings when early childbearers are included versus excluded.

[Figure 7 about here]

Findings for men

Among men, the impact of early BMI on the rate of transition to first births did not depend strongly on age. The faster of transition in the individuals with healthy BMI was visible from early adulthood, most notably compared to underweight (Figure 8A) or obese (Figure 8C) men and less so in overweight men (Figure 8B). This is in contrast to women, where the advantage of a healthy BMI was not apparent during early adulthood. As such, the relationship between early BMI and fertility did not differ significantly depending on whether early childbearers were included or excluded from the analyses (Table 2). In further contrast to women, the pace of transitioning to first parenthood by early BMI was not strongly patterned by race/ethnicity in men. For each of underweight, overweight, and obese groups, the hazard rate difference from the healthy BMI group was similar across race/ethnicity groups (Figure 9). The large uncertainty around the degree of differences for underweight men (as shown by wide distributions in Figure 9) is mostly due to the low number of underweight men in the sample, but the overall negative differences are in line with in average slower transition among the underweight men compared to the healthy BMI counterparts.

[Figure 8 about here]

[Table 2 about here]

[Figure 9 about here]

Discussion

While previous scholarship has established significant differences in first birth rates by BMI in NLSY79 data, ~28% of the sample who had a first child before the assessment of early BMI had to be excluded to avoid confounding by reverse causality by which BMI affects fertility (Jokela et al. 2008). In this study, we revisited the same data and used pre-parental BMI imputed for a larger sample that includes early childbearers, thereby assessing the impact of early BMI on childlessness in a sample more representative in terms of racial/ethnic differences in the timing of first parenthood.

Our results highlight both similarity and heterogeneity by race/ethnicity, and in how they intersect with sex, in the BMI-childlessness relationship. First, we find that overall racial/ethnic heterogeneity may be small, due to the higher childlessness consistently associated with overweight and especially with obesity across race/ethnic groups in both sexes. Within each race/ethnicity, the gap in childlessness between obese and healthy BMI was consistently larger in women. Second, some racial/ethnic heterogeneity is detectable especially in terms of underweight. Amongst women, the average lower childlessness associated with being underweight was driven almost solely by Black women (Figure 4), whereas the exceptional case of lower childlessness among underweight Hispanic men is 'masked' by the much higher childlessness among underweight Black or White men (Figure 5). Third, our new approach of incorporating early childbearers into the analyses offered additional evidence that the slower transition to first births among the overweight or obese women did not manifest until their mid-20s.

First, the higher childlessness among obese individuals across race/ethnicity and sex suggests that there are some shared mechanisms underlying the association between obesity and

fertility. For instance, endocrinological mechanisms by which obesity increases or exacerbates infertility have been widely described in the epidemiological, clinical, genetic, epigenetic, and non-human animal literature (Craig et al. 2017; Pasquali, Patton, and Gambineri 2007; Talmor and Dunphy 2015). To the extent that this body of evidence is generalizable to different sub-groups of population, and that early BMI persists into later BMI (Yang et al. 2021), we can consider higher infertility as one reason why childlessness is higher among obese individuals regardless of sex and race/ethnicity. The physiological impact of obesity on infertility could be larger given that individuals tend to partner with those with similar BMI (Ajslev et al. 2012).

Our findings could also suggest that behavioral mechanisms underlying the obesity-childlessness relationship have become similar across Blacks, Hispanics, and Whites, at least in the US birth cohort representative of the NLSY79 sample. The behavioral mechanisms are likely manifold, rooted in physiological or psychological factors, which interact with mating dynamics as well as structural inequalities such as obesity stigma. One might expect these complex behavioral processes to exhibit heterogeneity by sex and race/ethnicity, depending on how social preference for certain body type differs along those social axes. We find no evidence of heterogeneity by race/ethnicity, and within each race/ethnic group, obesity confers consistently higher childlessness in women than in men. These findings suggest that the socio-behavioral implications of obesity in the family formation process, which tend to be more consequential for women due to the slender body ideal, have in some degree converged across racial/ethnic groups in the US. Alternatively, it could be that different mechanisms are at play across the groups, but combine to produce similar patterns that link obesity to childlessness. It thus remains for future studies to explore the trajectory connecting early adulthood obesity and later life fertility by

553 race/ethnicity within each sex, and examine possible physiological and behavioral pathways in
554 detail.

555 With obesity stigma, in addition to obesity itself, becoming a global health challenge
556 (Brewis, SturtzSreetharan, and Wutich 2018), future studies are needed beyond the racial/ethnic
557 groups examined in this study in the US context, especially in countries at different stages of
558 socioeconomic development where structural factors such as weight-based discrimination might
559 operate differently. Cross-cultural studies have shown that being obese itself might not be
560 stigmatized in some countries (Sohn 2016), for example if large body size is socially valued
561 (Bosire et al. 2020; Cohen et al. 2018, 2019). It is also possible that the importance of BMI on
562 family formation is contingent on the prevalence of extreme BMI types: for example, with
563 increased obesity, the obesity 'penalty' in mating context might become smaller via multiple
564 mechanisms such as increased weight-based assortative mating (Ajslev et al. 2012). Such
565 differences in sociocultural norms or obesity prevalence may partly explain why the relationship
566 between BMI and lifetime fertility differs across countries (Butovskaya et al. 2017; Sear and
567 Marlowe 2009).

568 Second, our key finding regarding racial/ethnic difference was lower childlessness among
569 the underweight women detectable only amongst Black women. The underweight and healthy
570 BMI difference in terms of lifetime childlessness was minimal for Hispanics or White women.
571 As the present study focused on testing the presence of racial/ethnic difference, underlying
572 mechanisms remain to be explored in future research. One mechanism likely in play is the
573 socioeconomic disadvantage experienced by Black young women. It is well known that age at
574 first births is lower among Black women, who, at least between the 1970s to 1990s, were also
575 more likely than other ethnic/racial groups to experience teenage pregnancies that are unwanted,

mistimed, or outside of marriage (Musick et al. 2009; Sweeney and Raley 2014; Ventura and Bachrach 2000). If being underweight is one indicator of socioeconomic disadvantage via increased risk of food insecurity (Burke et al. 2018; Moradi et al. 2019; Myers and Painter 2017), one can expect a stronger association between being underweight and faster transition to first births in a more disadvantaged racial/ethnic group, as we observe among Black women. Within this broader context of socioeconomic disadvantage, behavioral and physiological mechanisms could further play a role.

For instance, it is possible that preferences for slender body type in women have been particularly strong amongst Blacks, a point that is further corroborated by the contrary pattern of higher childlessness among Black obese women. Such preferences, in turn, would underlie higher rates of union formation starting early in life and could be a mechanism which links underweight to lower childlessness among Black women. Under this scenario, the impact of BMI-related selection in the mating context may be even more pronounced in women of non-White ethnicities. Literature suggests that women of non-White groups may be as susceptible to the thin body ideal as Whites (meta-analysis by Grabe and Hyde 2006). Moreover, qualitative evidence considers how slender body type represents a means to social status (Kwan 2009) especially for the women of ethnic minority in the US (Cheney 2011). From this perspective, our finding of lower childlessness among underweight Black women may partly reflect an increasing impact of weight-based mate choice on the lifetime fertility of Black women. For example, the sharp decline in marriage among the early 1960 US birth cohort (to which the NLSY79 sample belongs to) was most pronounced in Black women (Frejka 2017; Raley, Sweeney, and Wondra 2015). This has been explained as the limited overall pool of mates available for Black women, due to many reasons including higher incarceration rate and higher inter-ethnic marriage rate

among Black men, and lower number of Black men with matched educational levels with Black women (Frejka 2017). Within this context, Black women might have experienced higher selection in mate choice, one route in which BMI acted as a criterion.

Another possibility is that the lower childlessness among underweight Black women is due to physiological conditions in favor of fertility (e.g., higher conception rate, better pregnancy outcomes). Given that clinical research has so far given less attention to how reproductive biology by BMI differs by race/ethnicity, it is difficult to assess whether the physiological implications of being underweight differ in Black women. Some studies suggests that eating disorders may be more prevalent among White than Black women, in part due to higher endorsement of slim body type among White women (Gray, Ford, and Kelly 1987; Neff et al. 1997; Powell and Kahn 1995). Further explorations of the intersection of BMI, health and race and its association with reproductive behaviors and processes and may be promising for future research to explore.

Third, the incorporation of early childbearers into the analyses of first births transition allowed us a refined understanding of the findings from the previous study on the same NLSY79 data but without early childbearers (Jokela et al. 2008). With early childbearers in, we find that the hazard ratios for overweight or obese women are closer to 1, i.e., differences compared to the healthy BMI group reduce. This is because obese women appear to be not delayed in their transition to first births, and overweight women even appear to make the transition faster, compared to healthy BMI counterparts at least during early adulthood. As such, the overall delayed first births among obese or overweight individuals are less pronounced than what is implied from the previous study. We also find that the hazard ratios for underweight are further away from 1, i.e., difference compared to the healthy BMI group increases. This is because the

faster rate of first births among the underweight women is evident from early adulthood, allowing the higher chance of underweight women having at least one child to be detected unlike in the previous study.

Our findings thus suggest that the impact of overweight and obese on fertility might be age-dependent, with different mechanisms involved across lifespan. For instance, during adolescence, excess weight and higher body fat are associated with earlier sexual maturity (Barros et al. 2019; Elizondo-Montemayor et al. 2017; Żurawiecka and Wronka 2021) and even with risky sexual behavior (Gordon et al. 2016; Leech and Dias 2012, but see Averett, Corman, and Reichman 2013). This could potentially explain why there seems indistinguishable and even faster rate of transition to first births among obese and overweight women, respectively, in our study sample. However, such adolescence-specific effects might fade when sexual maturation is complete and once the implications of overweight and obese in partnering or infertility become more dominant. Future research is needed to clarify whether and where exactly change points in the impact of early BMI exist, using advanced methods such as piecewise hazard rate, and what mechanisms might explain heterogeneity over time.

To conclude, the present study provides evidence of both similarity and heterogeneity by race/ethnicity, and its intersection with sex, in the early BMI to childlessness association. The patterns described in this study prompt future studies exploring underlying mechanisms of this heterogeneity – possibly involving physiological, psychological, and behavioral mechanisms that interact with social environment where weight-based perception and structural discrimination affect both chance (e.g., partnering) and intention (e.g., fertility expectation or desire) for reproduction. It also remains to be seen to whether our findings from the NLSY79 cohort extends

to more recent US cohorts, and other countries where secular trends of childlessness and BMI are both rapidly changing.

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Figures and Tables

Figure 1. Sample selection procedure.

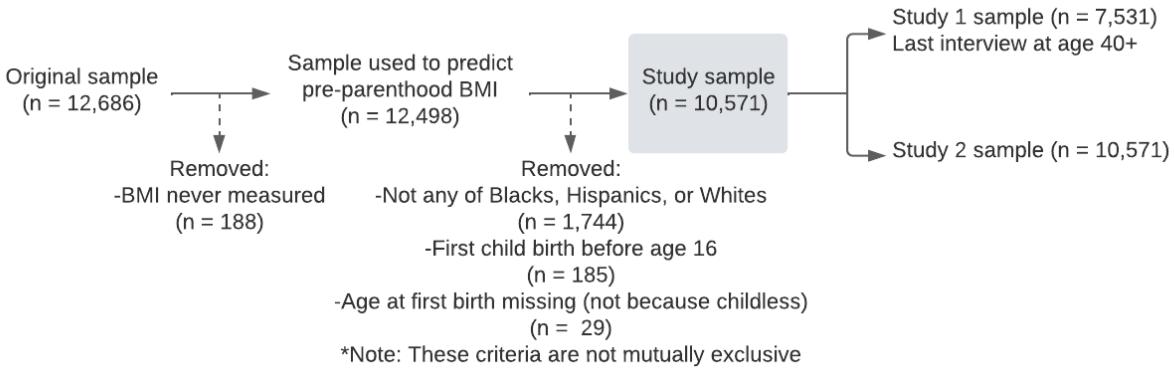

Figure 2. Racial differences in childlessness, age at first birth, and early BMI in the study sample from the US NLSY79 cohort (red = Blacks, green = Hispanics, blue = Whites). Calculations are based on the 10,571 individuals (men = 5,346, women = 5,225) whose BMI was measured during early adulthood. A: Observed proportion of childlessness at the age 40 years or older. When early childbearers are excluded from the sample, average proportion of childless individuals is overestimated and its racial/ethnic distribution changes in especially in women. B: Age at first birth for 7,850 individuals (men = 3,747, women = 4,103) who reported to have at least one biological child. The median age is younger in women (22 vs. 24 in men), and youngest among Blacks (20 in women, 23 in men) and oldest among Whites (23 in women, 25 in men) in both sexes. C: BMI at age 16 before transitioning to parenthood ('early BMI'), as predicted by the method described in the main text. Whereas average early BMI was similar between men (22.5) and women (22.9) in Blacks, it was higher in men for both Hispanics (23.6 vs. 22.4) and Whites (22.8 vs. 21.5).

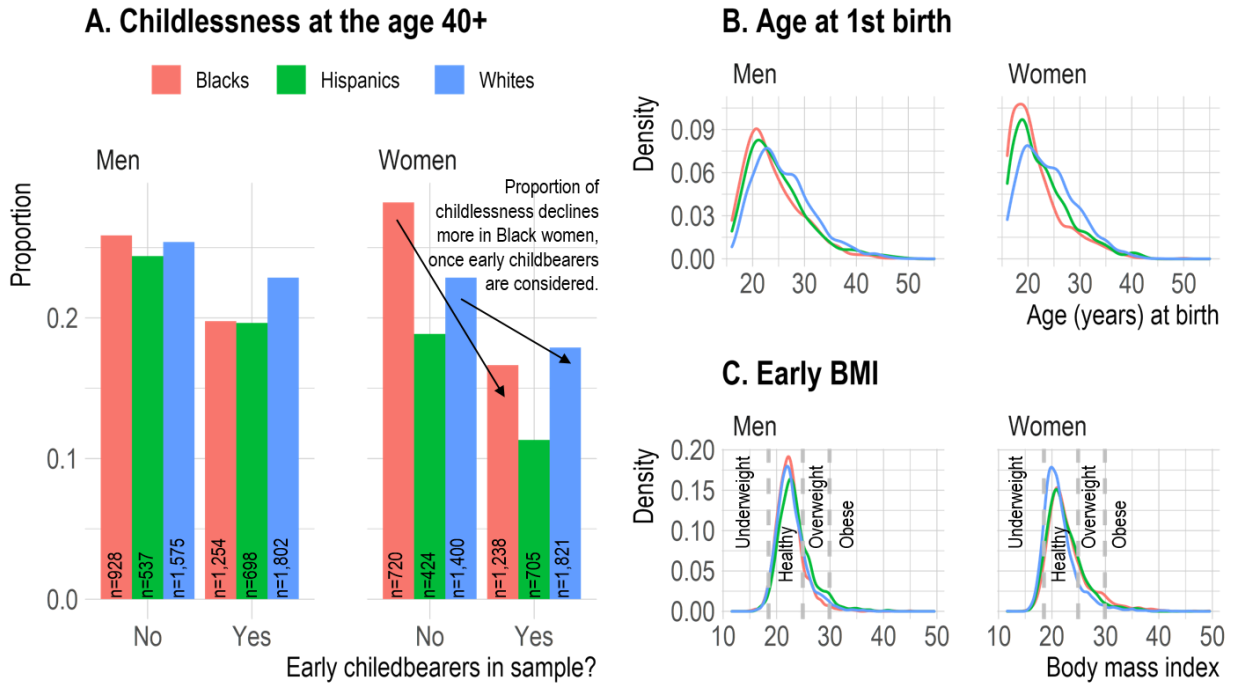

Figure 3. A directed acyclic graph describing the hypothesized causal process linking race/ethnicity, early BMI, and childlessness in the present study.

In our hypothesized causal framework, causal paths (blue lines) exist between exposure (early BMI) and outcome (childlessness), as well as between exposure and mediators. The rest are biasing paths (black lines). Race/ethnicity and birth cohort are confounders, because they affect both exposure and outcome. To estimate the total effect of early BMI on childlessness, adjusting confounders is necessary and sufficient, because adjusting mediators would 'take away' the weight of effects exerted through the causal path between early BMI and mediators. In this study, we are interested in the total effect that combines the direct effect and indirect effects of early BMI, and as such, only race/ethnicity and birth cohort as a potentially competing confounder are adjusted for in the models.

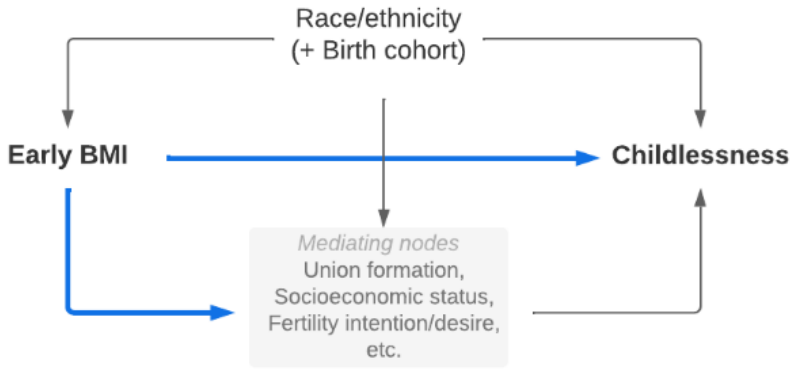

Figure 4. Probability of remaining childless by early BMI (pre-parenthood BMI at age 16), in the NLSY79 cohort women who were last interviewed at age 40+ (n = 3,765).

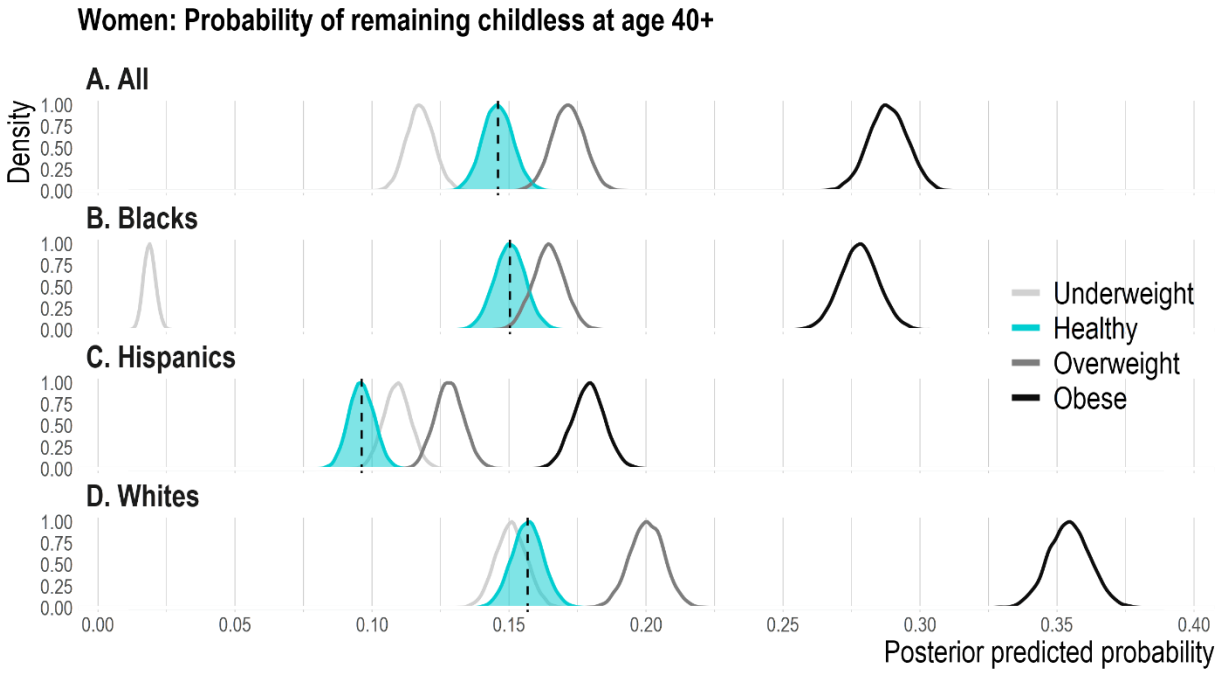

Figure 5. Probability of remaining childless in the NLSY79 cohort men who were last interviewed at age 40+ (n = 3,766).

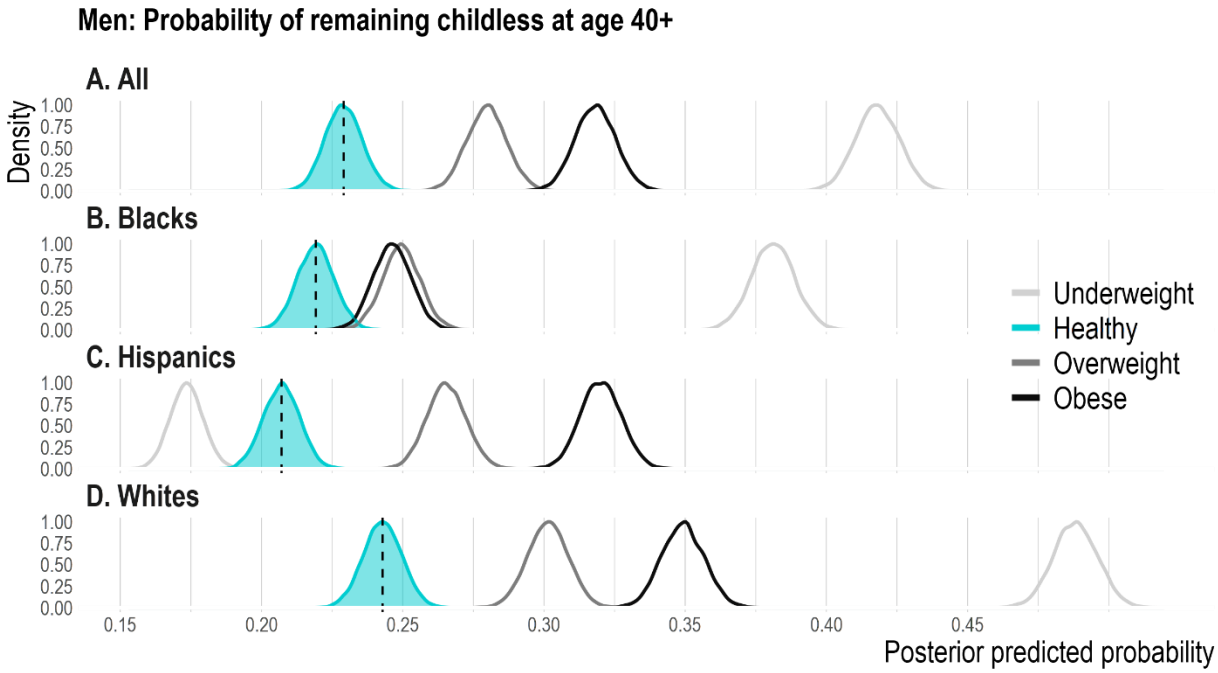

Figure 6. Survival curves for time to first parenthood in the NLSY79 cohort women (n = 5,225).

The survival curves are compared based on pre-parental BMI estimated at age 16. Age at which 50% of women transitioned to first parenthood is indicated by dashed lines.

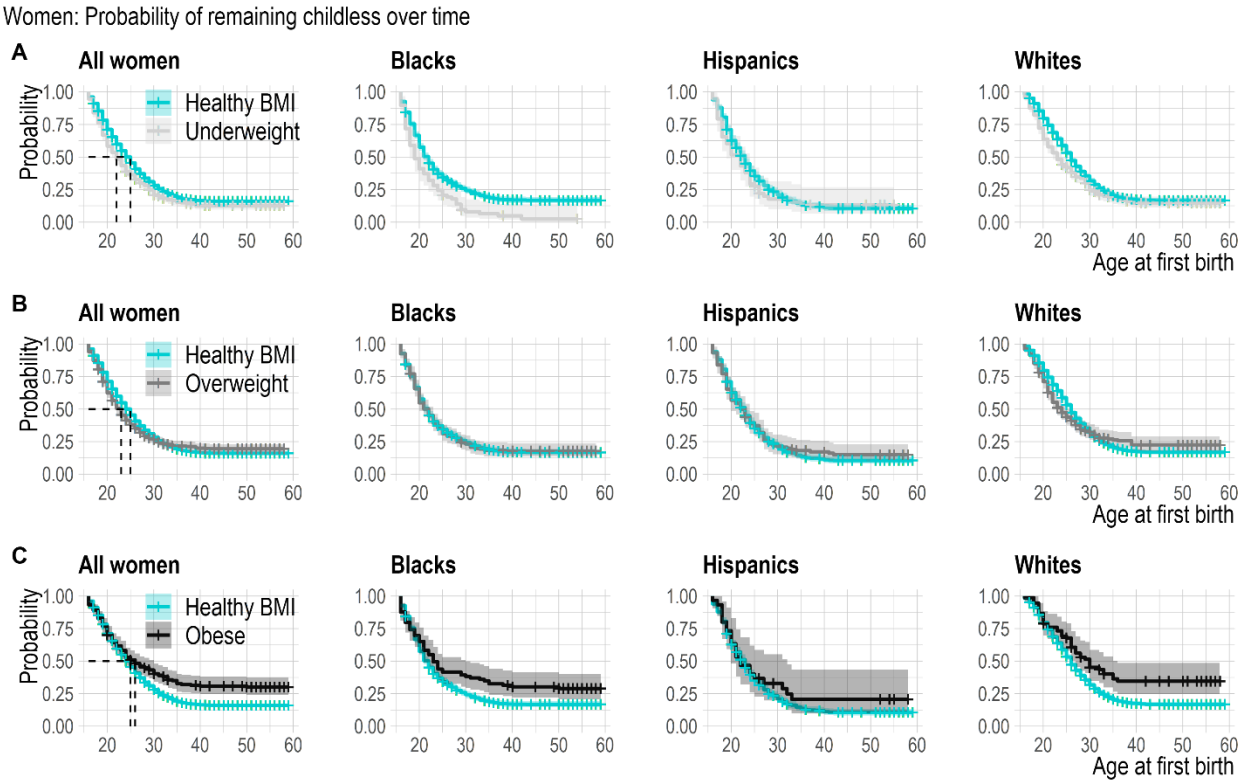

Table 1. Hazard of first births relative to that for healthy BMI in women

Comparing the estimated relative hazards of transitioning to first parenthood by early BMI, depending on whether early childbearers are included (left) or excluded (right) from the analyses. The observed age range is from 16 to 50 years old.

	Sample including early childbearers		**Sample excluding early childbearers**	
Predictors	*Estimates*	*CI*	*Estimates*	*CI*
Underweight	1.26	1.13 – 1.40	1.00	0.86 – 1.17
Overweight	1.08	0.98 – 1.19	0.91	0.80 – 1.04
Obese	0.74	0.62 – 0.88	0.54	0.42 – 0.70
Observations	5225		3494	

Figure 7. Hazard rate of becoming a parent, relative to healthy BMI counterparts, in the NLSY79 cohort women (n = 5,225).

Posterior distributions of estimates were used to calculate hazard rate difference. Negative values indicate lower hazard rate (i.e., slower transition to first births) than healthy BMI. Difference in the distribution of estimates reflects the degree of uncertainty around the estimated hazard rate relative to that of healthy BMI.

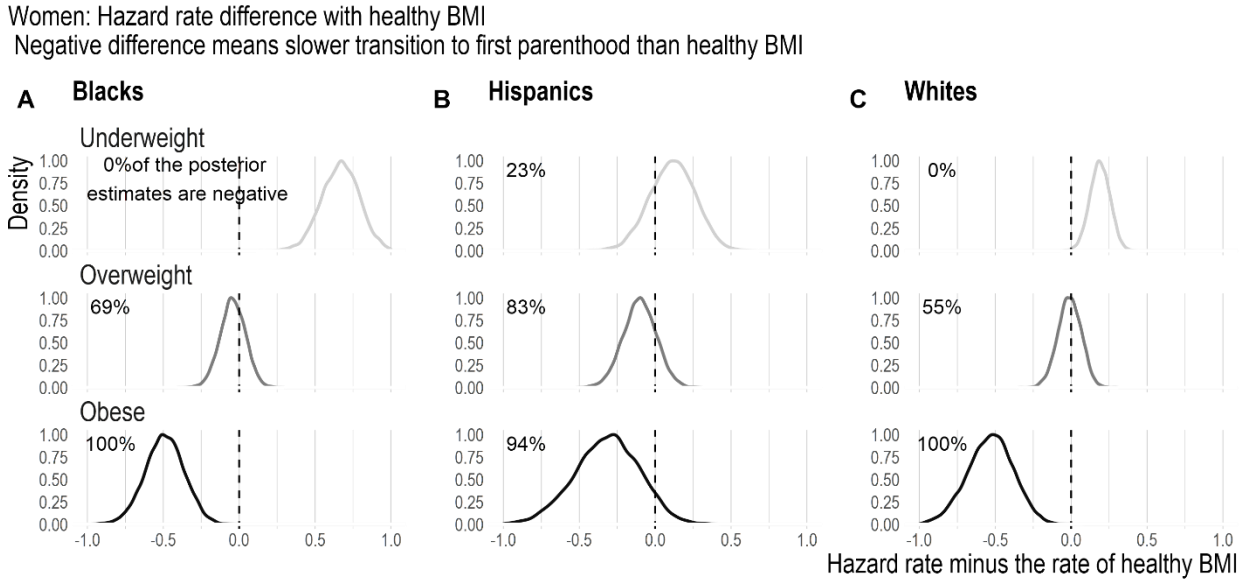

Figure 8. Survival curves for time to first parenthood in the NLSY79 cohort men (n = 5,346).

The survival curves are compared based on pre-parental BMI estimated at age 16. Age at which 50% of men transitioned to first parenthood is indicated by dashed lines.

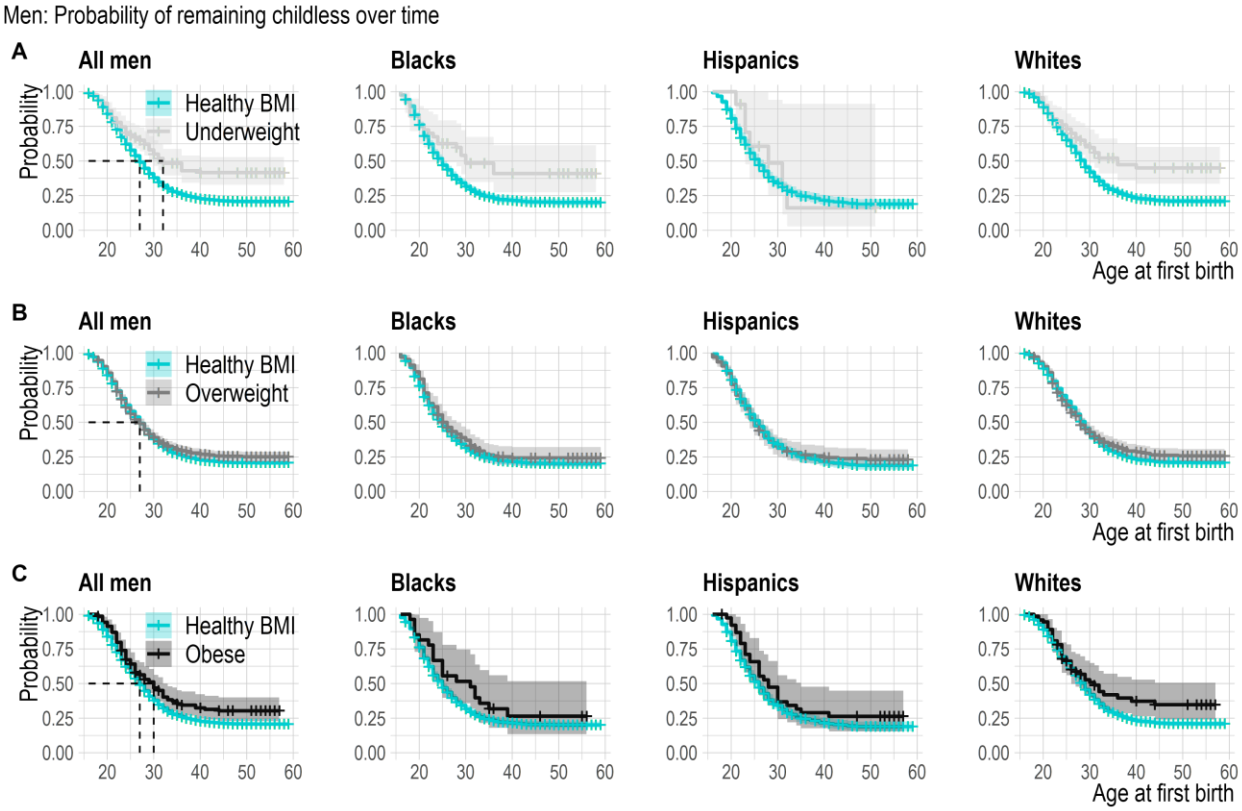

Table 2. Hazard of first births relative to that for healthy BMI in men.

Comparing the estimated relative hazards of transitioning to first parenthood by early BMI, depending on whether early childbearers are included (left) or excluded (right) from the analyses. The observed age range is from 16 to 52 years old.

	Sample including early childbearers		**Sample excluding early childbearers**	
Predictors	*Estimates*	*CI*	*Estimates*	*CI*
Underweight	0.60	0.47 – 0.78	0.59	0.44 – 0.78
Overweight	0.94	0.86 – 1.03	0.89	0.80 – 0.99
Obese	0.77	0.62 – 0.95	0.78	0.61 – 0.99
Observations	5346		4263	

Figure 9. Hazard rate of becoming a parent, relative to healthy BMI counterparts, in the NLSY79 cohort men (n = 5,346).

Posterior distributions of estimates were used to calculate hazard rate difference. Negative values indicate lower hazard rate (i.e., slower transition to first births) than healthy BMI. Difference in the distribution of estimates reflects the degree of uncertainty around the estimated hazard rate relative to that of healthy BMI.