

Weight as a bio-social filter of the proximate determinants of fertility

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Abstract

A woman's weight is located theoretically at the intersection between internal biological factors that influence her fertility and external social, economic and cultural factors that also influence her fertility. Integrating research from multiple disciplines, we use a proximate determinants framework to develop and test hypotheses about these pathways. Data on fertility histories come from nulliparous women sampled in the National Longitudinal Survey of Youth 1979-1997 and Longitudinal Study of Adolescent Health 1994/5-2000/1. Findings suggest that obese women in both cohorts experience later and fewer pregnancies and births. At the same time, obesity is associated with a reduced likelihood of abortion in the younger and more recent birth cohort, while in the older and less contemporary birth cohort, overweight increases the likelihood of miscarriage or stillbirth. We discuss the implications of these findings for future trends in U.S. fertility during an era when the prevalence of obesity is on the rise.

Introduction

The U.S. prevalence of overweight and obesity has grown markedly during the last two and a half decades (Ogden et al. 2007, Troiano & Flegal 1998), and both are now classified as critical medical and social problems. Overweight, and obesity in particular, are tied to a wide range of outcomes including serious health and reproductive risks for women and their children. Obese women are at a greater risk than leaner women of gestational diabetes, polycystic ovarian syndrome, and infertility (see Siega-Rits and Larain 2006 for review), while their babies face a greater risk of birth defects (Watkins et al. *Pediatrics* 2003, Anderson et al. *Epidemiology* 2005) and of being macrosomic, or large for gestational age (Seibre et al. 2001 *Int. Journ of Obesity*).

Research relating unhealthy weight with poor reproductive outcomes ranges from studies of infertility, unsuccessful infertility treatment, and poor pregnancy outcomes following infertility treatment (Norian et. al. 2005, Styne-Gross et al. 2005, Beliver et al. 2004) and high-risk labor and delivery (e.g. Beaten, Bukusi and Lambe 2001, Seibre et al. 2001). The majority of these studies have been conducted among clinical samples and local populations and there is a clear lack of population-based research on the relationship between overweight and fertility. Therefore, the generalizability of the findings above to the total U.S. population is questionable. This is particularly concerning with respect to specific subpopulations who may be at greater risk for both obesity and poor reproductive outcomes (e.g. young women, women of low socioeconomic status and minorities).

We use the clinical findings just cited along with theory and substantive findings from the biomedical and social sciences to motivate specific questions about the distribution, disparities, and magnitude of the relationship between women's weight and reproductive outcomes. Using data from two nationally representative samples of women who participated in the National

Longitudinal Survey of Youth (NLSY) and the National Longitudinal Survey of Adolescent Health (Add Health) we investigate whether weight is related to the timing of first pregnancy and birth, first pregnancy outcomes, and women's total number of pregnancies and births. These datasets contain information on two cohorts of women; the first is older and less recent while the second is younger and more recent. Using both sources of data allow us to confirm whether associations between weight and individual-level reproductive outcomes are similar across two samples, among women who belong to two unique birth cohorts, and among women who are overweight and report first births at slightly different stages of the life course. Our findings have important implications for the U.S. fertility rate given the high prevalence of overweight and obesity in this low fertility context.

Background

Obesity and Proximate Determinants of Fertility

At a population level, Bongaarts' (1978) proximate determinants of fertility provides a framework for linking distal social factors to the total fertility rate through biological characteristics (e.g. infecundity, amonherra, reproductive potential) and individual behaviors (e.g. contraceptive use and breastfeeding). This framework has recently been extended to low fertility settings (Morgan and Taylor 2006) and individual-level fertility outcomes (Quisnelle-Vallee and Morgan 2003). We use the proximate determinants framework here to organize our thinking about the pathways through which obesity might lead to higher or lower fertility. From this framework, a woman's body mass is located theoretically at the intersection between the external social, economic and cultural factors influencing her fertility and the internal biological factors influencing her fertility.

Obesity and Fertility Exposure

Obesity likely influences fertility through access to sexual partnership and mate selection, key factors for fertility exposure (Stover 1998). A recent literature review concludes that overweight and obese women have a harder time finding sexual, romantic and marriage partners than their leaner counterparts (Sobal 2006). Obesity is associated with dating less frequently and at later ages (Pearce, Boergers, and Prinstein 2002), the likelihood of never marrying and of marrying later (Fu and Goldman 1996 JMF). It is also associated with social isolation (Strauss and Pollack, 2003), stigmatization (Dejong, 1980; Puhl & Brownell, 2003), and discrimination (see Sobal 2006 for a review), and it is tied to women being rated as unattractive and unfeminine (Benjamin and Kamin-Shaaltiel, 2004).

Obesity and Fecundity

Obesity is also associated with three aspects of fecundity-- the duration of a women's fertile period, infertility and spontaneous intrauterine mortality.¹ Obese women are at a greater risk of polycystic ovarian disease (PCOD) and other menstrual irregularities that lead to a failure to ovulate (@@), which eliminates women's fertile period each month. Ovulation failure and other endocrinological problems also increase obese women's risk for infertility (@@). Furthermore, obese women are less responsive to fertility treatment (@@) and are less likely to have assisted reproductive technology lead to pregnancy and implantation (@@). Obese women who do become pregnant as a result of infertility treatment are also at a greater risk of miscarriage (@@). The relationship between obesity and spontaneous intrauterine mortality among women who conceive naturally is inconsistent. The majority of studies find higher risk of

¹ To our knowledge, research has not investigated whether obesity is related to frequency of sexual intercourse within a relationship and lactational infecundity the other two natural fertility factors identified by Bongaarts (1978).

spontaneous abortion (@@), but limited research contradicts these findings (@@). Similarly, obesity and the risk of stillbirth are linked in most (@@) but not all studies (@@).

Obesity and Deliberate Fertility Control

Obesity is also associated with contraceptive failure, an important aspect of deliberate fertility control. Estimates from case control studies (Holt et al. 2005) and retrospective cohort analysis (Holt et al. 2002) suggest that obese women are more likely than normal weight women to report pregnancy due to failure of oral contraceptives. Obesity may also predict contraceptive non-use, based on research linking obesity to poor body image and poor body image to contraceptive self-efficacy (Wingood et al. 2002; Eisenberg et al 2005). Body image and weight are related and not synonymous, but the findings suggest that obese women may be less likely to use contraception due to an inability (or perceived inability) to negotiate its use. To our knowledge, research has not explored whether obese women are more likely to seek induced abortion, another indicator of deliberate fertility control, but evidence about obesity and both fertility exposure and contraceptive use can be used to propose both positive and negative associations. Obese women who want children may be less likely to seek induced abortion if they recognize their limited access to romantic partners and their increased risk of infertility. Conversely, given that obese women are at a greater risk of unintended pregnancy due to contraceptive failure, they may be more likely to seek induced abortion.

The Current Study

The evidence suggesting that obesity is linked to fertility exposure, natural fertility control and deliberate fertility control lead us to develop competing hypotheses about how overweight and obesity are related to individual-level fertility outcomes. We focus on

consequences of being medical classified as both overweight and obese to discern whether both weight classifications above what is medically classified as a normal weight predict reproductive events or whether only the heaviest women are at risk of weight influencing reproductive outcomes.

The literature linking obesity to fertility exposure and natural fertility control suggest that compared to leaner women:

H01a: obese women have later ages at first pregnancy and birth and

H02a: obese women have fewer total pregnancies and births.

However, obese women are at greater risk of contraceptive failure and contraceptive non-use, which leads to opposing hypotheses. These associations could be so prominent that compared to learner women:

H01b: obese women actually have earlier ages at first pregnancy and birth and

H02b: obese women have more total pregnancies and births.

A final scenario is that associations between obesity and fertility exposure, natural fertility control, and deliberate fertility control counteract each other, which would lead us to expect that:

H01c: weight is not associated with women's ages at first pregnancy and birth

H02c: nor is weight associated with women's total number of pregnancies and births.

The connections between obesity and the proximate determinants of fertility also lead us to speculate on associations between obesity and first pregnancy outcomes. The literature on obesity and spontaneous intrauterine mortality leads us to speculate that compared to learner women:

H03a: obese women are at greater risk of a first pregnancy ending in stillbirth or miscarriage versus a live birth.

Conversely, if the association between obesity and spontaneous intrauterine mortality is specific to women who undergo infertility treatment, then:

H03b: obesity will not be linked to the risk of first pregnancy ending in stillbirth or miscarriage.

There is no clear evidence as to whether obesity may also be associated with the likelihood that a woman's first pregnancy will end in induced abortion versus live birth.

Associations between obesity and both contraceptive failure and non-use suggest that:

H04a: obese women are more likely than normal weight women to have first pregnancies end in induced abortion.

However, if obese women desire children, realize that finding suitable sexual partners may be difficult, and recognize their risk of infertility, then:

H04a: obese women may be less likely than normal weight women to have first pregnancies end in induced abortion.

Our study is the first of which we are aware to take what we know about how obesity is related to the proximate determinants of fertility and use it as a springboard to explore whether weight is associated with important individual-level reproductive events in the U.S.

Methods

We use data from two nationally representative longitudinal datasets, the NLSY and Add Health to investigate how overweight and obesity early in a woman's reproductive years are

related to age at first pregnancy, first pregnancy outcomes (live birth versus abortion, miscarriage, or stillbirth), and women's total number of pregnancies and births. We predict how weight during the early reproductive years influences these individual-level fertility outcomes to ensure that our indicators of weight occur before first pregnancy. In addition, women tend to gain, not lose weight during their reproductive years. Weight trajectories generally increase with age until middle age and then begin to decline (@@) and few obese women lose weight and are able to maintain a normal weight (@@). Thus, obesity early in the reproductive life course places women at great risk of having weight influence the individual-level fertility outcomes investigated by this study.

Data and Sample

The National Longitudinal Survey of Labor Market Experience, Youth Survey 1979-2000 (NLSY79) is our first source of data. From this nationally representative sample we identify 3698 women who were observed both in 1981 when body weight was first assessed and in 1998 at the close of the study period. We exclude 77 women with missing data on weight or the fertility outcomes. Then, in order to reduce the potential for a reverse-causal bias whereby pregnancy or childbirth leads to higher subsequent weight, we exclude 1247 women (34%) whose first pregnancy occurred prior to 1981. This leaves an analytical sample of 2374 women. Due to the over-sample of selected minority subpopulations, contrasts can be made among women of African American, European American and Hispanic ethnic origin.²

The NLSY is an excellent data source for estimating how adolescent and early adult weight are related to fertility outcomes throughout women's reproductive lives. We can predict

² Although we do have data from women of other racial and ethnic backgrounds, small sample sizes limit our ability to conduct within group analyses for these respondents.

how weight relatively early in women's reproductive life span is related to pregnancies and births among those who delay a first pregnancy until the later teen years and adulthood. As other scholars have noted, the NLSY is arguably the best source of U.S. data for studying women's fertility histories throughout their reproductive years (Quisnelle-Vallee and Morgan 2003). The one group that is lost from analyses is women who experience a first pregnancy before 1981, when the sample ranged in age from 16 to 24 (i.e., the 34% of the women noted above). To capture associations between weight and earlier births, we turn to data from the National Longitudinal Study of Adolescent Health.

The National Longitudinal Study of Adolescent Health (Add Health) is a school-based, longitudinal, nationally representative survey of 20,745 adolescents who were in grades 7 through 12 in one of 34 public, private, and parochial schools in 1994 (Bearman, Jones, & Udry, 1997). Respondents were selected from a stratified random sample of students in schools. Three waves of in-home survey data were collected in 1995, 1996 and 2000-2001 and provide researchers with a wealth of information about adolescents' health and well-being. For comparability to the NLSY, we restrict our sample to the 8,887 African American, European American and Hispanic female respondents who participated in Wave I and Wave III data collection efforts who were nulliparous and not pregnant at Wave I. Our sample is further constrained to young women with no missing data on weight or fertility outcomes. The final samples vary for each analysis, depending on the necessary restrictions of each different outcome (see the descriptive statistics in the Appendix for more detail). Sample restrictions in our least restrictive analysis (number of pregnancies) lead to a final analytic sample of 4,072 female respondents who are between the ages of 11 and 21 at Wave I and 18 and 27 at Wave III.

Add Health is an excellent complementary data source for estimating how adolescent weight is related to fertility outcomes earlier in women's reproductive lives. It contains an in-depth fertility history and respondents' height and weight, which can be used to construct Centers for Disease Control and Prevention (CDC) age- and sex-specific BMI scores for children and teens (Centers for Disease Control and Prevention, 2000). Excellent data about background characteristics associated with weight and reproductive outcomes are also available, which allows us to better ensure that any observed associations between weight and individual-level fertility outcomes do not result from joint factors influencing them both.

Individual-level Fertility Outcomes

In the NLSY79, data on the fertility outcomes are abstracted from the full reproductive histories compiled for each woman in the study by the Ohio State University Center for Human Resource Research. We use the data on the cumulative number of pregnancies and of live births in 1998. In addition we use the data on the pregnancy outcome (live birth=1, miscarriage, spontaneous abortion and stillbirth=2, induced abortion=3) for the first pregnancy among all women in the analytical sample. Lastly, we calculate the survival time in years from 1981 until the year of their first pregnancy for all women in the analytical sample. As noted earlier, to avoid reverse-causality bias this sample does not include women whose first birth occurred prior to 1981.

In Add Health, all female respondents are asked to complete a pregnancy and birth history during Wave III data collection efforts in 2000-2001. Unfortunately, these data were collected within the context of women's history of romantic and sexual relationships, which led some pregnancies and births to be excluded from this fertility history. We used the Add Health household roster to complete the fertility history by obtaining the date of birth for other

biological children born to female Add Health respondents. When the fertility history is supplemented with these data, estimates of adjusted age-specific birth rates for women between the ages of 15 and 23 are comparable to national estimates of first birth rates (see Schoen, Landale, and Daniels, forthcoming, for a full discussion of this issue). We supplement the pregnancy data from the Wave III fertility history using data from the household roster and responses to questions during Wave I and II about whether young women had ever been pregnant, the date(s) of pregnancies, and the outcome(s) of pregnancies. This data is used to create outcome variables parallel to the NLSY79 variables described above.

Weight Classification Prior to First Pregnancy

We compute BMI scores from Add Health and NLSY indicators of respondent-reported height and weight at the beginning of the study period for each respective study. For the NLSY79, the BMI classification is based on assessments of weight and height in 1981. As noted earlier, fewer than 2% of the sample had missing data on these variables. Because the NLSY79 sample involves women above the age of 18, we are able to apply the universal CDC standards for BMI categorization of adults where underweight is < 18.5 , normal is ≥ 18.5 and < 25 , overweight is ≥ 25 and < 30 , and obese is 30 or higher.

Because the majority of Add Health sample members (roughly 99%) are under age 20 when weight is assessed at Wave I, BMI is constructed differently for this sample. We use guidelines from the Centers for Disease Control and Prevention (CDC) for assessing age- and sex-specific BMI scores for children and teens (Centers for Disease Control and Prevention, 2000) and then classify young women with respect to the categories for underweight (BMI below the 5th percentile of age- and sex-specific scores), normal weight (BMI in the 5th through and 84th

percentile), at risk of overweight (BMI above the 85th percentile and below the 95th percentile), and overweight (BMI above the 95th percentile).

The weight classifications used across datasets are different, but they best reflect age-appropriate, medically accurate BMI classifications. Discrepancies in age-appropriate terminology for referring to individuals with BMI scores above the normal range also exist. Among children and adolescents, individuals are classified as at-risk of overweight and overweight. The comparable classifications for adults are overweight and obese. For simplicity, we will consistently use the terminology for adults to ease comparisons of results across datasets.

Control Variables

We control for precursors of both BMI and fertility whose omission might otherwise lead to an over or under estimation of the association between weight status and fertility. They include socioeconomic status, personality characteristics, and indicators of the pace of social and biological development. Socioeconomic status is indicated by the *educational attainment of respondents' mothers* and the *household poverty status* at the beginning of the analytical period (Wave I for Add Health and 1981 for NLSY).

Controls for personality and developmental characteristics include indicators that have been associated with overweight and the timing of fertility in previous studies. The Rosenberg Scale of *self esteem* scale is assessed in 1980 in the NLSY and in Wave I of the Add Health. Cognitive ability is measured in the Add Health at Wave I using a standardized score on the *Peabody Picture Vocabulary Test* (PPVT) and in 1979 in the NLSY using standardized scores on the *Armed Forces Qualification Test* (AFQT). (This control is not included in the models presented here, but will be included in the final analyses.) We also control for academic performance using GPA in core courses (Mathematics, English, Science, and Social Studies)

from the Add Health at Wave I and educational attainment in 1981 from the NLSY. These indicators of academic performance control for both aspects of the individual's own socioeconomic status (and potential socioeconomic status) as well as cognitive development. Two indicators of biological and social development are included—women's age at menarche and age of sexual initiation. In addition, in the Add Health we are able to control for self-reported health at Wave I. Data on health status is not available from the NLSY.

We construct indicators for race and ethnic identity to be parallel for the NLSY and Add Health samples. Respondents are classified as either “White” (reference), “Black” or “Hispanic.” The controls for race and ethnicity serve a double purpose; they allow us to adjust our findings for the oversample of minority subpopulations in both datasets and they are also important indicators of unmeasured social factors that influence both childbearing and weight. We also include region and urbanicity in models for Add Health sample members to adjust for the Add Health sampling frame. As we proceed with analyses, we will develop more parallel models across datasets that include a more similar and wider array of control variables.

Analytic Plan

We estimate relationships between weight and five fertility outcomes using estimation techniques appropriate for each dependent variable of interest. The first two outcomes, age at first pregnancy and age at first birth, are modeled using hazards regression models. The third outcome, first pregnancy outcome, is modeled using multinomial regression. The final two outcomes, the cumulative measures of total pregnancies and total births, are modeled using ordered logistic regression.

Hazards regression models

The relationship between weight status and the timing of first pregnancy is assessed using two types of hazards regression models-- Cox proportional hazards model and the discrete-time

hazards model. Both of these models allow us to test whether there is a statistically significant difference in the hazard of first pregnancy by weight status. The Cox model makes no distributional assumptions about the distribution of the survival times from the weight assessment at the beginning of the analytical period to the year in which either a woman becomes pregnant or she is censored from the analysis. It does, however, make the assumption that the differences between groups that are specified by covariates are proportional over time (or, in this case, age) (Powers and Xie 2000). In other words, with respect to the covariate of main interest in this study, the proportionality assumption means that the ratio of the hazard of pregnancy by weight status is constant with increasing age. The discrete-time model allows us to test this assumption of proportionality using age interactions with the main covariate of interest for the outcome of the status competition. In findings to be completed prior to the final presentation, we will examine whether the analyses violate the proportionality assumption. Non-unique survival times, i.e. ties, are handled in the Cox model using the Breslow method (Breslow 1974).

Multinomial logistic regression model

The multinomial regression model allows us to assess the relationship between weight status and the risk of the three possible first birth outcomes –miscarriage or stillbirth, induced abortion, or live birth. It is a favorite estimation technique for contrasting the odds of non-ordered, categorical outcomes because: it generalizes to the binomial logit model when there are two categories and thus provides more clearly interpretable results; it models grouped data equivalently to the log-linear model; and there is widely available statistical software for estimation (Powers and Xie, 2000).

Ordinal logistic regression model.

The ordered logistic regression (OLR) model allows us to assess the relationship between weight status and the number of pregnancies and births in two respective models. Like the multinomial logistic model, the ordinal logistic model is an extension of the simple logistic model. It is a favorite estimation technique for modeling ordered, categorical outcomes (Powers and Xie, 2000). The OLR model fits a simultaneous regression model across the categories of the dependent variable in which it iteratively compares pooled categories with the remaining categories. For example in the case of a birth outcome with a range of 0 to five possible births, the model simultaneously compares the likelihood of all of the following: 1) 0 versus 1-5 births; 2) 0-1 versus 2-5 births; 3) 0-2 versus 3-5 births; 0-3 versus 4-5 births; and 0-4 versus 5 births. These simultaneous regressions are made under the assumption that the coefficients vary in a proportional way across these different intervals. In analyses underway, we test this proportionality assumption in the analyses of birth and pregnancy for both the Add Health and NLSY samples. We suspect that greater understanding of the role of weight in fertility will be developed by considering the likelihood of any pregnancies or births separately from the likelihood of progression to second and higher order reproductive outcomes.

Sample weights

Guidelines for analyzing the NLSY and the Add Health data suggest that researchers either weight statistical models and correct for design effect or that they control for variables associated with the Add Health sampling design (Chantala 2006; CHRR 2006). We take the latter approach and present unweighted estimates from NLSY and Add Health models of reproductive outcomes. Supplementary analyses (available upon request) of both the Add Health and NLSY datasets show that estimates of relationships between weight and reproductive

outcomes obtained when models that are weighted and corrected for design effects produce substantively and statistically similar results to those presented here.

Findings

Hazard of first pregnancy and birth

Our analyses of the hazards to first pregnancy and birth allow us to estimate whether weight is related to earlier or later first reproductive events due to biological or social consequences of weight. Estimates in Table 1 indicate that obesity, but not overweight is associated with age at first pregnancy. In both cohorts, obese women are roughly 25% less likely to experience pregnancy in a given year than their normal weight counterparts. Obese women in both cohorts are also less likely to experience a first birth in a given year. In the younger cohort, the risk of a first birth among obese women compared to normal weight women is 25% lower. In the older cohort, the risk of a first birth is 30% lower among obese (versus normal weight) women.

First pregnancy outcome

Our multinomial analyses of *the outcome of first pregnancy* allows us to assess whether obesity is related to a woman's ability to carry a pregnancy to term (live birth versus miscarriage or stillbirth) and her desire to carry a pregnancy to term (live birth versus abortion). Estimates in Table 2 suggest that weight is related to both outcomes, but not consistently across the two cohorts. Among the younger cohort, obese young women are almost 51% $[(1 - \exp(-.709)) * 100]$ less likely to end a first pregnancy with induced abortion. This association is not evident among the older, less recent cohort of women. Estimates in this older cohort do suggest that overweight, not obese, women have a 65% $[(\exp(.504) * 100)]$ higher likelihood of experiencing

a miscarriage or stillbirth versus a live birth. We intend to conduct further analyses to explore both the cohort differences in associations between weight and first pregnancy outcomes and the reason why overweight, but not obesity is associated with miscarriage or stillbirth among the NLSY cohort. We suspect that discrepancies among the two cohorts result in part from younger mothers' exclusion from analyses using the NLSY. Research suggesting that obesity is tied to a decreased likelihood of pregnancy, lead us to hypothesize that overweight, but not obese women in the NLSY are at greater risk of miscarriage or still birth because they are more likely to experience a pregnancy than their overweight counterparts.

Number of pregnancies and births

In the NLSY, we use the two outcomes the *total number of pregnancies* and *total number of births* as reasonable estimates of women's total lifetime fertility history (Morgan). In the Add Health, these variables represent earlier births and foreshadow young women's expected total number of births. We assess the relationship between these cumulative measures of fertility and weight using ordinal logistic regression. Estimates in Table 3 suggest that weight is similarly related to total lifetime fertility history and number of births earlier in the life course. Among the younger and older cohorts, obese women report fewer pregnancies and births, which suggests that the lifetime fertility rate among obese women is lower than the fertility rate of their leaner counterparts. Young obese women in the Add Health are 29% $[(1 - \exp(-.343)) * 100]$ less likely to report an additional birth compared with their normal weight peers. Similarly, in the NLSY obese women are 40% $[(1 - \exp(-.503)) * 100]$ less likely to report an additional birth.

Current analyses presented in Table 3 may place heavy weight upon failure to conceive and bring a pregnancy to term. The findings on first birth outcomes for obese women reported in Table 1 and Table 2 suggest that this type of non-proportionality in the association between

obesity and reproductive outcomes may be likely, since obese women had a lower hazard to first birth and were more likely to experience stillbirth and spontaneous abortion. We are currently conducting analyses which test the OLR assumption of proportionality that will be presented in the final paper. These analyses compare the likelihood of progression to second and higher order reproductive outcomes separately from first birth.

Discussion

The findings from this study allow us to develop a cohesive understanding of the role of unhealthy and socially unacceptable weight in the reproductive outcomes of U.S. women at different points along their life course.

First, our findings support the role of a biological pathway which unhealthy weight reduces fertility by increasing stillbirth and spontaneous abortion. At the same time, our findings fail to support some of the social and psychological pathways through which we had hypothesized that socially unacceptable levels of weight might increase fertility. We found no evidence that overweight and obesity operate to increase the risk of unwanted pregnancy and childbearing. Nor did we find evidence that heavier than normal weight is associated with higher fertility. In fact, unwanted fertility—as indicated by the likelihood of abortion—was lower among the younger women sampled in the Add Health dataset. Additionally, pregnancy and childbearing was delayed among obese adolescents and adult women. While it may be that overweight and obese women experience stigma and suffer lower self-confidence and sense of control (Dejong, 1980; Puhl & Brownell, 2003; Wingood et al. 2002; Eisenberg et al 2005), our findings suggest that these experiences do not translate into poorer quality communication with reproductive partners and associated contraceptive failure and contraceptive non-use.

Although, our findings allow us to exclude the hypothesis that obesity places women at risk for higher fertility through unwanted or mistimed pregnancies, we can not conclude that it is infecundity alone which leads to lower fertility. The later and lower levels of fertility among obese women is consistent both with the biological hypotheses that highlight the role of weight-related infecundity, as well as with the social and psychological hypotheses that highlight the role of weight-related stigma and social selection in partnering. In fact, the biological pathway leading to lower fecundity, may be enhanced by social factors and interpersonal interactions. For example, we hypothesized that young obese women who desire children are actually aware of the fact that it may be difficult for them to find suitable sexual partners as they grow older. The lower likelihood of termination of first pregnancies we observe in the Add Health sample, may thus reflect strategies to reach desired fertility in the context of lower fecundity and partnership success.

In future analyses, we will estimate associations between weight and reproductive outcomes among women of the same age in the NLSY and Add health studies. This will allow us to better assess the similarity of these relationships across cohorts. We also plan to estimate whether these relationship vary among women in different social groups (e.g. different racial/ethnic groups, women from different socioeconomic backgrounds, etc.) to begin exploring social explanations for relationships between weight and reproductive events. A final goal of this study is to explore why obesity and overweight are differentially related to first pregnancy outcomes among women from different birth cohorts. As noted previously, we suspect that this finding may result from the different age compositions of the two samples and the exclusion of younger women from analyses using the NLSY, but we have not confirmed this hypothesis.

This study has found a consistent association between obesity and both delayed and diminished reproduction. The consistency of the findings across the different age-groups and periods of study suggest that obesity is an important factor in the current and future fertility of US women.

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Table 1: Estimates Predicting Relationships between Weight and Women's Time to First Pregnancy and Birth

Variable	Time to First Pregnancy			Time to First Birth		
	AddHealth (N=4072)		NLSY (N=2056)	AddHealth (N=4068)		NLSY (N=2056)
	hazard ratio	SE	hazard ratio	SE	hazard ratio	SE
<i>Weight Category (reference: normal weight)</i>						
underweight	1.080	0.210	0.875	0.072	1.094	0.254
overweight	0.892	0.071	0.905	0.082	0.839	0.083
obese	0.751	** 0.076	0.756 *	0.106	0.747 *	0.092
<i>Age (at first time point: w1 or 1981)</i>	0.922	** 0.022	0.915	*** 0.016	0.983	0.029
<i>Race/Ethnicity (ref.: non-Hispanic white)</i>						
non-Hispanic black	1.252	** 0.094	0.997	0.063	1.073	0.100
Hispanic	0.907	0.084	1.308	*** 0.099	0.750 *	0.088
<i>Mother's Educational Attainment</i>	0.940	*** 0.012	0.974	** 0.009	0.912	0.015
<i>Age at First Intercourse</i>	0.855	*** 0.016	0.970	* 0.012	0.857	0.019
<i>Age at Menarche</i>	0.928	*** 0.019	1.013	0.016	0.917	0.022
<i>Self-esteem</i>	1.008	0.008	0.984	* 0.006	1.001	0.010
<i>Poverty</i>	1.124	0.165	1.140	* 0.074	1.069	0.187
<i>Grade Point Average (NLSY: attainment in years)</i>	0.677	*** 0.028	1.000	0.022	0.638	0.032
<i>PPVT Score (NLSY: not available)</i>	0.993	** 0.002			0.987	0.003
<i>Region (to be added to NLSY; ref.: West)</i>						
Midwest	0.952	0.082			1.227	0.137
South	0.975	0.079			1.350	** 0.141
Northeast	0.682	*** 0.073			0.725	* 0.105
<i>Urbanicity (to be added to NLSY; ref.: rural)</i>						
urban	0.907	0.067			0.940	0.085
suburban	0.833	* 0.062			0.849	0.077
<i>High Self-reported General Health (NLSY: not available)</i>	0.907	0.056			0.816	** 0.061
<i>Marital Status (collinear in AddHealth; ref.: married)</i>						
never married			0.808	* 0.069		
divorced/separated			1.156	0.139		
					0.746	*** 0.067
					0.987	0.124

***p<=.001 **p<=.010 *p<=.050

Sources: The National Longitudinal Study of Adolescent Health & The National Longitudinal Study of Adolescent Health

Table 2: Estimates Predicting Relationships between Women's Weight and First Pregnancy Outcomes

Variable	AddHealth (N=4072)				NLSY (N=2056)			
	Miscarriage/Stillbirth vs. Live Birth		Abortion vs. Live Birth		Miscarriage/Stillbirth vs. Live Birth		Abortion vs. Live Birth	
	coefficient	SE	coefficient	SE	coefficient	SE	coefficient	SE
<i>Weight Category (reference: normal weight)</i>								
underweight	0.251	0.528	-0.169	0.594	0.217	0.233	-0.211	0.270
overweight	0.157	0.221	-0.461	0.252	0.504 *	0.231	0.224	0.280
obese	0.318	0.261	-0.709 *	0.342	-0.317	0.446	0.396	0.385
<i>Age (at first time point: w1 or 1981)</i>								
<i>Race/Ethnicity (ref.: non-Hispanic white)</i>								
non-Hispanic black	0.346	0.208	0.657 **	0.212	0.180	0.180	-0.314	0.207
Hispanic	0.332	0.245	0.344	0.251	-0.360	0.247	0.174	0.232
<i>Mother's Educational Attainment</i>								
Age at First Intercourse	0.001	0.041	0.168 ***	0.040	0.010	0.030	0.140 ***	0.032
Age at Menarche	-0.034	0.062	0.020	0.070				
Age at Menarche	0.061	0.055	0.092	0.059	-0.021	0.048	-0.007	0.051
Self-esteem	-0.003	0.023	0.040	0.023	0.018	0.019	-0.017	0.020
Poverty	-0.218	0.420	-0.308	0.487	0.147	0.190	0.131	0.208
<i>Grade Point Average (NLSY: attainment in years)</i>								
Grade Point Average (NLSY: attainment in years)	0.042	0.114	0.360 **	0.118	-0.129 *	0.062	-0.026	0.073
<i>PPVT Score (NLSY: not available)</i>								
PPVT Score (NLSY: not available)	0.007	0.007	0.030 ***	0.007	0.172	0.256	0.797 *	0.345
<i>Region (to be added to NLSY; ref.: West)</i>								
Midwest	-0.551 *	0.241	-0.872 ***	0.226				
South	-0.553 *	0.227	-1.154 ***	0.227				
Northeast	0.112	0.299	0.235	0.271				
<i>Urbanicity (to be added to NLSY; ref.: rural)</i>								
urban	-0.289	0.210	0.208	0.224				
suburban	-0.056	0.206	0.342	0.217				
<i>High Self-reported General Health (NLSY: not available)</i>								
High Self-reported General Health (NLSY: not available)	0.271	0.173	0.309	0.180				
<i>Marital Status (collinear in AddHealth; ref.: married)</i>								
never married					0.126	0.355	0.407	0.441
divorced/separated					-0.214	0.351	-0.865	0.475

***p<=.001 **p<=.010 *p<=.050

Sources: The National Longitudinal Study of Adolescent Health & The National Longitudinal Study of Adolescent Health

Table 3: Estimates Predicting Relationships between Women's Weight and Their Number of Pregnancies and Births

Variable	Number of Pregnancies		Number of Births	
	AddHealth (N=4072) coefficient	SE	AddHealth (N=4068) coefficient	SE
<i>Weight Category (reference: normal weight)</i>				
underweight	0.218	0.244	-0.184	0.128
overweight	-0.139	0.098	-0.097	0.139
obese	-0.343	**	-0.503	*
<i>Age (at first time point: w1 or 1981)</i>	0.331	***	-0.085	**
<i>Race/Ethnicity (ref.: non-Hispanic white)</i>				
non-Hispanic black	0.266	**	-0.169	0.097
Hispanic	-0.132	0.113	0.161	0.122
<i>Mother's Educational Attainment</i>	-0.091	***	-0.037	*
<i>Age at First Intercourse</i>	-0.270	***	0.005	0.020
<i>Age at Menarche</i>	-0.079	**	-0.010	0.025
<i>Self-esteem</i>	0.015	0.010	0.017	0.010
<i>Poverty</i>	0.243	0.194	0.224	*
<i>Grade Point Average (NLSY: attainment in years)</i>	-0.501	***	0.017	0.034
<i>PPVT Score (NLSY: not available)</i>	-0.008	**	0.003	0.003
<i>Region (to be added to NLSY; ref.: West)</i>				
Midwest	-0.092	0.105	0.254	*
South	-0.012	0.099	0.367	**
Northeast	-0.482	***	-0.414	*
<i>Urbanicity (to be added to NLSY; ref.: rural)</i>				
urban	-0.039	0.091	-0.054	0.106
suburban	-0.192	*	-0.199	0.105
<i>High Self-reported Health (NLSY: not available)</i>	-0.139	0.077	-0.249	**
<i>Marital Status (collinear in AddHealth; ref.: married)</i>				
never married			-0.011	0.130
divorced/separated			0.160	0.191
			-0.264	*
			-0.037	0.1954

***p<=.001 **p<=.010 *p<=.050

Sources: The National Longitudinal Study of Adolescent Health & The National Longitudinal Study of Adolescent Health