# Lower fertility associated with obesity and underweight: the US National Longitudinal Survey of Youth[1–3]

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## ABSTRACT

**Background:** It has been hypothesized that body weight predicts the number of children that a person will have: obese and underweight persons are hypothesized to have fewer children than do their normal-weight counterparts.

**Objective:** We aimed to prospectively examine the association between body weight in young adulthood and achieved fertility in later life.

**Design:** A representative national sample of 12 073 American young adults (aged 17–24 y in 1981) were followed through 2004 (19 survey waves of the National Longitudinal Survey of Youth).

**Results:** Obese young women and men were less likely to have their first child by the age of 47 y than were their normal-weight counterparts [relative risk (RR) = 0.69; 95% CI: 0.61, 0.78 in women; RR = 0.75; 95% CI: 0.66, 0.84 in men). Obesity also predicted a lower probability of having more than one child, particularly for women. These associations were partly explained by a lower probability that obese participants will marry. Underweight men were less likely to have the first, second, third, and fourth child than were normal-weight men (RRs = 0.75–0.88; 95% CIs: 0.61, 0.95). These associations were largely explained by the lower marriage probability of underweight men. Obese women and men and underweight men were less likely to have as many children in adulthood as they had desired as young adults.

**Conclusions:** Obesity may be an important risk factor for lower fertility because of its social and possibly biological effect on reproductive behavior. Further data are needed to assess whether this association holds in more recent cohorts. *Am J Clin Nutr* 2008; 88:886–93.


## INTRODUCTION

The increase in obesity among children and adults is considered to be one of the most important public health concerns in many countries (1, 2), and this ongoing trend may even have an effect on demographic outcomes such as life expectancy (3) and fertility (4). In a recent study of Finns (4), adolescent body weight was found to predict the number of children that a person had as an adult; that is, obese and underweight adolescents were less likely to have children than were their normal-weight counterparts. Because both low and high body weight have been associated with reproductive dysfunctions (5–10) and lower likelihood of marriage (4, 11), the inverse U-shaped association between body weight and fertility is plausible, and it may reflect both biological and social influences.

The association between body weight and number of children has not yet been confirmed in other studies, and, because the participants in the Finnish study were followed only to the ages of 33–39 y, these data may have provided an incomplete reflection of achieved fertility. Furthermore, it is unknown whether the lower fertility associated with extremes of body weight reflects voluntary or involuntary processes. That is, it is possible that underweight and obese persons prefer to have fewer children than do normal-weight persons.

The purpose of the present study was to assess the influence of body weight on fertility in the US National Longitudinal Survey of Youth (NLSY) (12) and to overcome limitations of the previous research noted above. We examined whether body mass index (BMI) at age 17–24 y predicted the likelihood of having the first, second, third, and fourth child by the age of 47 y and, if so, whether this association was mediated by differences in marital status across the life course. We also assessed whether body weight was associated with the number of children desired in young adulthood and, if so, whether body weight predicted the likelihood of attaining this number in adulthood 23 y later.

## SUBJECTS AND METHODS

### Participants

The ongoing NLSY (12–14) has followed, from 1979 onward, a representative sample of Americans born between 1957 and 1964. Follow-up was carried out annually from 1979 to 1994 and biannually since 1994; the most recent data available are from the follow-up in 2004. The original sample ($n$ = 12 686) consisted of 3 subsamples: a representative sample of noninstitutionalized civilian youths ($n$ = 6111); a supplemental sample designed to oversample civilian Hispanic, African American, and economically disadvantaged (other) youths ($n$ = 5295); and a military sample ($n$ = 1280). Because of funding constraints, the number of interviewed members of the military and supplemental samples was limited after 1984 and 1990, respectively.

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Received December 27, 2007.

Accepted for publication June 17, 2008.

We included all participants who provided data on body weight and height in 1981 and data on fertility history in ≥1 follow-up after that, which resulted in 5982 women and 6091 men (total $n = 12\,073$) who were eligible for the study (**Table 1**). Appropriate longitudinal and cross-sectional sampling weights adjusted for sex, race-ethnicity, year of birth, sample type, and location were applied in the statistical analyses to account for differential probabilities of selection into the sample and for attrition. Thus, the sample yielded representative estimates for the US population born between 1957 and 1964. In 2004, 60.4% ($n = 7661$) of the original participants were still participating in the study.

Written informed consent was obtained according to federal law and the policies of the US Office of Management and Budget. The study was approved by institutional review boards of the institutions conducting the surveys.

### Body mass index

In 1981, the then 17–24-y-old participants reported their height in inches and weight in pounds. BMI was calculated from these data (in kg/m$^2$). The BMI scale was positively skewed and was corrected by using a logarithmic transformation:

$$X = \ln(\text{BMI}) \qquad (1)$$

Although the participants have reported their body weight in some of the follow-ups after 1981, we used the earliest possible information on BMI to avoid confounding due to reverse causality between BMI and fertility (*see* Statistical analysis) and to have the largest possible sample size not diminished by missing data on body weight. However, we assessed the stability of BMI in the sample by categorizing BMI in 1981 and 2004 (BMI in 2004 was calculated from data on body weight reported in 2004 and body height reported in 1982) and cross-tabulating these data (**Table 2**).

**TABLE 1**
Descriptive statistics of the sample

| | Women ($n = 5982$) | Men ($n = 6091$) |
|---|---|---|
| Data from 1981 | | |
| Age (y) | 20.7 ± 2.2[1] | 20.6 ± 2.3 |
| Race-ethnicity [*n* (%)] | | |
| African American | 935 (15.6) | 940 (15.4) |
| Hispanic | 1495 (25.0) | 1548 (25.4) |
| Other | 3552 (59.4) | 3603 (59.2) |
| Body-weight group (%)[2] | | |
| Underweight | 11.5 | 3.6 |
| Normal-weight | 74.1 | 72.3 |
| Overweight | 10.7 | 20.1 |
| Obese | 3.8 | 4.0 |
| Children born during the follow-up (*n*) | | |
| First child | 2957 | 3264 |
| Second child | 3003 | 2710 |
| Third child | 1577 | 1341 |
| Fourth child | 596 | 576 |
| Children born outside of marriage (%)[2] | | |
| First child | 32.1 | 34.9 |
| Second child | 18.2 | 17.3 |
| Third child | 23.1 | 23.7 |
| Fourth child | 26.4 | 27.3 |

[1] $\bar{x}$ ± SD (all such values).
[2] Percentages were determined from weighted sample frequencies.

**TABLE 2**
Stability of body-weight categories from the age of 17 to 24 y (in 1981) to the age of 40 to 47 y (in 2004)[1]

| | BMI in 1981 | | | |
|---|---|---|---|---|
| BMI in 2004 | Underweight | Normal-weight | Overweight | Obese |
| | | % | | |
| Underweight | 6.5 | 0.6 | 0.1 | 0.0 |
| Normal-weight | 63.2 | 32.4 | 6.3 | 0.6 |
| Overweight | 23.1 | 43.1 | 29.6 | 12.3 |
| Obese | 7.3 | 23.9 | 64.0 | 87.1 |
| Total | 100 | 100 | 100 | 100 |
| Subjects (*n*) | 494 | 5299 | 1167 | 326 |

[1] BMI, in kg/m$^2$. The percentages show the proportion of participants whose body-weight category in 2004 was the same as or different from that in 1981. BMI: underweight, <18.5; normal-weight, 18.5–24.9; overweight, 25–29.9; obese, >30.

### Fertility

The fertility history of the participants was constructed by using available data from all 19 survey waves between 1981 and 2004. In the final follow-up (ie, 2004), only 4.6% of the participants had ≥5 children, so we restricted our main analyses to the birth of the first, second, third, and fourth biological children (Table 1). In men's fertility data between 1979 and 1998, children have been assigned a "confidence value" indicating the best estimate of whether the child is the participant's biological offspring. When such information was available, we included only the births of children for whom there was at least reasonable confidence that the child was the man's biological offspring. Details of the sample's fertility data may be found in the user's guides for the study (13, 14).

### Desired number of children

In 1982, the participants reported the number of children they desired to have later on as adults (mean ± SD: 2.4 ± 1.4). We created a difference variable by subtracting the number of children desired in 1982 from the number of biological children the participant had in 2004. This variable assessed the difference between desired and achieved fertility; on average, the participants had 0.4 ± 1.8 fewer children in 2004 than they had desired in 1982.

### Covariates

Marital status, urbanicity of residence, and education are potential sociodemographic factors associated with both BMI and fertility (4, 11, 15, 16), and data on these were used as covariates. A time-variant variable of marital status across the life-course was constructed by using data from all available follow-up phases and was coded with 2 categories [0 = not married (ie, single, divorced, separated, or widowed), 1 = married] in each year according to the participant's reports. Missing values were replaced by data from the previous year. In addition, we created a cumulative index of marital status indicating the years of marriage over the study period by summing together dichotomous indicators of marital status in each follow-up phase, which resulted in a scale ranging from 0 to 19 y. This cumulative measure was used as a dependent variable in linear regression models assessing the association between BMI and later marital status.

A time-variant covariate of residence was created in the same way as the marital status variable above—ie, by using data from

all available years and replacing missing values with data from the previous year. Residence was assessed by a dichotomous variable (0 = rural, 1 = urban) in each year. Between 1981 and 1996, residence was defined as urban if the participant lived in a county where the urban population was >50% of the total population. Between 1998 and 2004, residence was defined as urban if the participant was living in an urban area or in an town with a population of >2500.

Education was assessed on the basis of the highest completed grade reported in the latest follow-up phase for each participant. The participants were interviewed on a 20-point scale (0 = no education, 20 = ≥8 y of college; mean = 13.0 ± 2.5 y).

### Statistical analysis

#### *Survival analysis*

Discrete-time survival analysis (17) was used to examine whether and at what age the participants had their first, second, third, and fourth child, and whether BMI predicted these events. In survival analysis, participants are censored either when the event of interest occurs—in this case, when a child is born—or when the study period ends. Here the end of study period was determined by the participant's age when the latest interview took place. The majority of the sample (66%) was censored after the age of 40 y, whereas 16% were censored between the ages of 30 and 39 y and 18% were censored before the age of 30 y.

The participants were 17–24 y old when they reported their weight and height in 1981, and some of them already had children at that time. More specifically, 2667 (22%), 874 (7%), 192 (2%), and 40 (<1%) had the first, second, third, and fourth child, respectively. Having children has been shown to increase body weight in mothers and fathers (18, 19), so retrospective analyses of BMI and fertility would be confounded by reverse causality. To avoid this confounding and to have a completely prospective study design, we excluded from the respective analyses participants who had children in 1981; ie, the birth of the *n*th child was assessed in those participants who did not have the *n*th child in 1981, and no births before the age of 17 y (the age of the youngest participants at baseline) were included in the models. This reduced the number of participants to 4244, 5411, 5873, and 5982 women and 5202, 5828, 6048, and 6091 men in models predicting the birth of the first, second, third, and fourth child, respectively. As a sensitivity analysis, we also fitted the models by including participants who were excluded from the main analyses. These models provided results similar to those presented here, although the former were somewhat attenuated, as might be expected, because of reverse causality between BMI and having children (data available on request).

Age-specific fertility is known to follow a bell-shaped curve, and therefore the effect of time was modeled by linear and quadratic terms of age. The models were fitted separately for women and men. Education level was used as a time-invariant covariate, and marital status and residence were used as time-variant covariates. Because the sample included participants from different subsamples with different racial-ethnic backgrounds, all analyses were adjusted for subsample membership. Furthermore, some of the participants were from the same households, so not all observations were independent. Robust estimator with household clustering instead of maximum likelihood was therefore used to provide appropriate estimates of SEs.

To maximize statistical power, BMI was used as a continuous variable in the analyses. Following a method suggested by Singer and Willett (17), we illustrated the results by calculating the cumulative probabilities of having children by a given age for average members of the underweight (BMI < 18.5), normal-weight (18.5 < BMI < 24.9), overweight (25 < BMI < 29.9), and obese (BMI > 30) groups. We determined the mean value of logged BMI in each weight group and used these values to calculate predictions for average members of each group. In these predictions, other covariates were assigned mean values. In addition, we calculated the relative probabilities of having children in underweight, overweight, and obese participants compared with normal-weight participants.

#### *Regression analysis*

The associations between BMI, the number of children desired in 1982, and the number of children in 2004 were assessed by using Poisson regression analysis. The association between BMI and the difference between desired and achieved fertility was assessed by using linear regression analysis. Age, education, race-ethnicity, and subsample membership were controlled in these analyses. All statistical analyses were conducted with the use of STATA software (version 9.0; StataCorp, College Station, TX).

## RESULTS

The characteristics of the study participants are shown in Table 1, and the cross-tabulation of BMI groups in 1981 and 2004 is shown in Table 2. Most participants moved toward heavier weight groups over the study period, and the obese group was the most stable weight group: 87% of obese young adults were obese 23 y later.

The role of study covariates in predicting fertility is shown in **Table 3**. Being married, having higher educational attainment, and being African American or Hispanic were the strongest predictors of having a child for both women and men.

### Body weight and marital status

First, we assessed the association between BMI and cumulative years of marriage across the life-course after control for age and education. A linear regression analysis indicated a quadratic association between BMI and years of marriage for women [BMI: B = 78.66; SE = 18.61 ($P < 0.001$); $BMI^2$: B = −13.23; SE = 2.94 ($P < 0.001$)] and men [BMI: B = 95.13; SE = 29.57 ($P = 0.001$); $BMI^2$: B = −14.83; SE = 2.67 ($P = 0.002$)]. The predicted years of marriage for underweight, normal-weight, overweight, and obese women was 10.2, 10.3, 9.0, and 6.5 y, respectively. The corresponding values for men were 7.6, 9.1, 9.2, and 8.1 y. In other words, high but not low BMI predicted fewer years of marriage for women, whereas low and high BMI predicted fewer years of marriage in men.

### Body weight and fertility

Survival analysis models of BMI and fertility indicated that there was a curvilinear association between BMI and the probability of having the first, second, third, and fourth child for women and men. The cumulative probabilities (and 95% CIs) of having the first, second, third, and fourth child by a given age are shown in **Figure 1**. The relative probabilities of having children

**TABLE 3**
Predicting the birth of the first, second, third, and fourth child with study covariates in discrete-time survival analysis models

| | First child | Second child | Third child | Fourth child |
|---|---|---|---|---|
| Women ($n$ = 4235–5973) | | | | |
| Age | 1.96 (1.83, 2.10)[1] | 2.37 (2.20, 2.55) | 2.86 (2.56, 3.20) | 3.22 (2.74, 3.80) |
| Age$^2$ | 0.99 (0.99, 0.99) | 0.98 (0.98, 0.99) | 0.98 (0.98, 0.98) | 0.98 (0.98, 0.98) |
| Education | 0.93 (0.91, 0.95) | 0.94 (0.92, 0.96) | 0.92 (0.89, 0.94) | 0.88 (0.84, 0.91) |
| Race-ethnicity | | | | |
| African American | 1.84 (1.64, 2.08) | 2.07 (1.87, 2.31) | 2.44 (2.14, 2.79) | 2.79 (2.29, 3.40) |
| Hispanic | 1.64 (1.43, 1.87) | 1.63 (1.44, 1.84) | 1.98 (1.70, 2.32) | 2.36 (1.86, 2.99) |
| Other | 1.00 | 1.00 | 1.00 | 1.00 |
| Sample | | | | |
| Main | 1.00 | 1.00 | 1.00 | 1.00 |
| Supplementary | 0.84 (0.76, 0.93) | 0.89 (0.82, 0.98) | 0.84 (0.75, 0.95) | 0.83 (0.70, 0.98) |
| Military | 0.19 (0.15, 0.24) | 0.17 (0.14, 0.22) | 0.12 (0.08, 0.18) | 0.01 (0.00, 0.10) |
| Urban residence | 1.05 (0.93, 1.19) | 1.08 (0.96, 1.22) | 0.91 (0.78, 1.07) | 0.73 (0.57, 0.92) |
| Marital status | | | | |
| Married | 7.28 (6.52, 8.13) | 7.68 (6.90, 8.56) | 3.55 (3.08, 4.08) | 2.45 (1.99, 3.01) |
| Other | 1.00 | 1.00 | 1.00 | 1.00 |
| Men ($n$ = 5231–6083) | | | | |
| Age | 2.02 (1.89, 2.16) | 2.28 (2.11, 2.46) | 2.67 (2.40, 2.97) | 3.09 (2.58, 3.71) |
| Age$^2$ | 0.99 (0.99, 0.99) | 0.99 (0.98, 0.99) | 0.98 (0.98, 0.99) | 0.98 (0.98, 0.99) |
| Education | 0.95 (0.93, 0.96) | 0.97 (0.95, 0.99) | 0.96 (0.93, 0.98) | 0.91 (0.87, 0.96) |
| Race-ethnicity | | | | |
| African American | 1.86 (1.61, 2.14) | 2.15 (1.86, 2.49) | 2.55 (2.12, 3.05) | 2.96 (2.25, 3.89) |
| Hispanic | 1.51 (1.29, 1.77) | 1.80 (1.54, 2.11) | 2.46 (2.02, 3.00) | 2.98 (2.21, 4.03) |
| Other | 1.00 | 1.00 | 1.00 | 1.00 |
| Sample | | | | |
| Main | 1.00 | 1.00 | 1.00 | 1.00 |
| Supplementary | 1.06 (0.92, 1.22) | 0.81 (0.70, 0.94) | 0.81 (0.68, 0.96) | 0.97 (0.75, 1.24) |
| Military | 0.30 (0.26, 0.35) | 0.27 (0.22, 0.32) | 0.27 (0.22, 0.32) | 0.42 (0.29, 0.60) |
| Urban residence | 1.02 (0.91, 1.14) | 1.07 (0.94, 1.21) | 1.07 (0.94, 1.21) | 0.95 (0.73, 1.25) |
| Marital status | | | | |
| Married | 9.94 (8.92, 11.08) | 11.10 (9.83, 12.53) | 4.49 (3.83, 5.26) | 3.03 (2.41, 3.82) |
| Other | 1.00 | 1.00 | 1.00 | 1.00 |

Odds ratio; 95% CI in parentheses (all such values). The odds ratios in discrete-time survival analysis refer to changes in the odds of having a child at a given age.

by the age of 47 y for underweight, overweight, and obese participants compared with those for normal-weight participants, predicted by the survival analysis models, are shown in **Table 4**. Compared with normal-weight women, obese women were less likely to have any children, whereas obese men were less likely than normal-weight men to have the first and second child but just as likely to have the third and fourth child. Underweight predicted low fertility in men but not in women.

We then assessed the association between BMI and fertility after adjustment for differences in marital status over the lifecourse (Table 4). In obese women, marital status accounted for 58% of their lower probability of having the first child—ie, from 0.69 to 0.87—and 63%, 32%, and 17% of their lower probability of having the second, third, and fourth child, respectively. After adjustment for marital status, obesity still predicted a lower probability of having the first and third child, but the associations between second and fourth births were not statistically significant. Adjustment for marital status had little effect on the association between men's obesity and fertility, whereas the association between men's underweight and low fertility disappeared almost completely when marital status was taken into account (Table 4).

As an additional analysis, we examined whether BMI moderated the association between marital status and fertility. Assuming that obesity and underweight decrease fertility even when a person is married, we hypothesized that marriage would increase fertility more in normal-weight persons than in obese or underweight persons. This hypothesis was assessed by testing marriage × BMI interaction effects in the survival analysis models. However, these interaction effects were not statistically significant ($P > 0.10$), which suggests that the effect of marriage on fertility did not vary over the BMI distribution.

### Body weight and the number of children desired

Next we examined the association between BMI, fertility desires, and the difference between desired and achieved fertility. In line with results of survival analyses (as described above), a Poisson regression analysis indicated that BMI predicted the number of biological children at the age of 40 to 47 y in a quadratic fashion in women [BMI: B = 5.56; SE = 2.20 ($P$ = 0.012); BMI$^2$: B = −0.92; SE = 0.35 ($P$ = 0.009); $n$ = 3888] and in men [BMI: B = 11.94; SE = 3.95 ($P$ = 0.002); BMI$^2$: B = −1.90; SE = 0.62 ($P$ = 0.002); $n$ = 3591]. The number of children predicted for underweight, normal-weight, overweight, and obese women was 2.06, 2.11, 1.99, and 1.71, respectively; the corresponding values for men were1.63, 1.88, 1.83, and 1.51.

High BMI was associated with a greater number of children desired at the age of 18 to 25 y for women [B = 0.12; SE = 0.05 ($P$ = 0.014); $n$ = 3832] and men [B = 0.17; SE = 0.06 ($P$ =

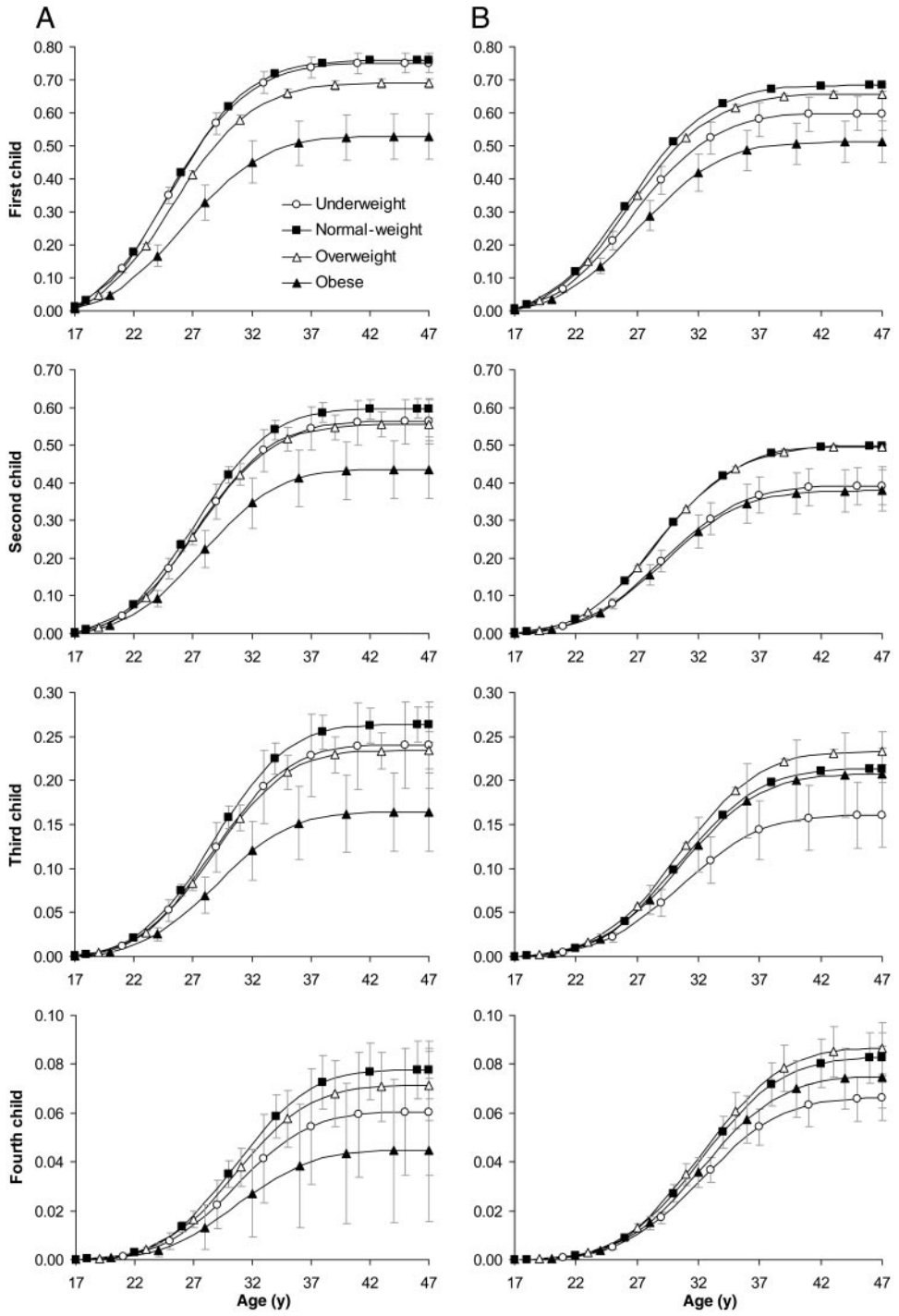


**FIGURE 1.** The predicted cumulative probabilities (and 95% CIs) of having the first, second, third, and fourth child by a given age according to weight group for women (A; $n = 4235–5973$) and men (B; $n = 5231–6083$). The predictions were calculated from discrete-time survival analyses; ie, $P = 1 -$ value of survival function.

0.003); $n = 3504$]. Therefore, when assessing the association between BMI and unachieved fertility, we controlled for the number of children desired. A linear regression analysis with control for age, education, race-ethnicity, subsample membership, and the number of children desired indicated that there was a curvilinear association between BMI and unachieved fertility in women [BMI: B = −9.23; SE = 3.76 ($P = 0.014$); $BMI^2$: B = 1.54; SE = 0.60 ($P = 0.010$)] and men [BMI: B = −17.85; SE = 5.75 ($P = 0.002$); $BMI^2$: B = 2.84; SE = 0.90 ($P = 0.002$)]. Underweight, normal-weight, overweight, and obese women had, on average, 0.21, 0.26, 0.28, and 0.48 fewer children than they had desired, which indicated that high BMI was associated with unachieved fertility. The corresponding values were 0.67, 0.57, 0.70, and 0.97 for men, which indicated that men with high and low body weight were more likely than were normal-weight men to have fewer children than they had desired.

**TABLE 4**
Predicted relative probabilities (and 95% CIs) of having children by the age of 47 y for underweight, overweight, and obese participants compared with normal-weight participants[1]

| | Underweight | Normal-weight | Overweight | Obese |
|---|---|---|---|---|
| Women (n = 4235–5973) | | | | |
| Not adjusted for marital status | | | | |
| First child | 0.99 (0.95, 1.03)[2] | 1.00 | 0.91 (0.89, 0.93) | 0.69 (0.61, 0.78) |
| Second child | 0.94 (0.85, 1.04) | 1.00 | 0.93 (0.88, 0.98) | 0.73 (0.61, 0.86) |
| Third child | 0.91 (0.75, 1.10) | 1.00 | 0.89 (0.82, 0.97) | 0.62 (0.49, 0.79) |
| Fourth child | 0.78 (0.54, 1.11) | 1.00 | 0.91 (0.76, 1.10) | 0.58 (0.35, 0.95) |
| Adjusted for marital status | | | | |
| First child | 0.96 (0.91, 1.00) | 1.00 | 0.97 (0.97, 0.98) | 0.87 (0.79, 0.96) |
| Second child | 0.93 (0.82, 1.04) | 1.00 | 1.00 (0.94, 1.06) | 0.90 (0.76, 1.05) |
| Third child | 0.93 (0.75, 1.13) | 1.00 | 0.94 (0.86, 1.02) | 0.74 (0.58, 0.94) |
| Fourth child | 0.79 (0.55, 1.14) | 1.00 | 0.95 (0.79, 1.14) | 0.65 (0.39, 1.08) |
| Men (*n* = 5231–6083) | | | | |
| Not adjusted for marital status | | | | |
| First child | 0.88 (0.80, 0.95) | 1.00 | 0.96 (0.95, 0.97) | 0.75 (0.66, 0.84) |
| Second child | 0.79 (0.70, 0.89) | 1.00 | 1.00 (0.98, 1.01) | 0.76 (0.66, 0.87) |
| Third child | 0.75 (0.61, 0.93) | 1.00 | 1.09 (1.07, 1.11) | 0.97 (0.78, 1.19) |
| Fourth child | 0.80 (0.70, 0.91) | 1.00 | 1.05 (0.92, 1.19) | 0.90 (0.77, 1.06) |
| Adjusted for marital status | | | | |
| First child | 1.00 (0.93, 1.07) | 1.00 | 0.93 (0.91, 0.94) | 0.78 (0.69, 0.88) |
| Second child | 0.93 (0.82, 1.05) | 1.00 | 0.94 (0.93, 0.96) | 0.78 (0.67, 0.91) |
| Third child | 0.87 (0.70, 1.08) | 1.00 | 1.05 (1.02, 1.09) | 1.00 (0.79, 1.26) |
| Fourth child | 0.91 (0.81, 1.02) | 1.00 | 1.01 (0.89, 1.15) | 0.91 (0.77, 1.07) |

[1] All models were adjusted for race-ethnicity, subsample membership, and urban residence.
[2] Relative probabilities; 95% CI in parentheses (all such values). The relative probabilities and 95% CIs were calculated from the odds ratios obtained from discrete-time survival analysis models.

### Attrition analysis

Finally, we evaluated whether body weight and having children predicted study participation over the follow-up. In a linear regression analysis with control for age, sex, race-ethnicity, and subsample, high BMI predicted longer participation in the study, as indicated by the age at last available interview, so that a 1-unit increase in BMI increased the follow-up period by 0.05 y [untransformed B = 0.05; SE = 0.01 ($P$ = 0.001)]. Likewise, a logistic regression analysis predicting the probability of participating in the most recent follow-up in 2004 indicated that participants with high BMI were more likely to participate [odds ratio = 1.03; SE = 0.01 ($P < 0.001$)] so that a 1-unit increase in BMI increased this probability by 1%. Thus, sample attrition was slightly lower among obese participants. Having ≥1 children in 1981 did not predict the age at the last interview [B = 0.04; SE = 0.14 ($P$ = 0.76)] or the probability of participating in the 2004 follow-up [odds ratio = 0.94; SE = 0.07 ($P$ = 0.39)], which suggests that parenthood status was not related to attrition. These attrition patterns suggested that associations between high BMI and low fertility were unlikely to be biased by selective attrition associated with BMI.

## DISCUSSION

This prospective study from the US NLSY provides evidence for the influence of body weight on achieved fertility. First, after taking into account the effects of education, place of residence, and race-ethnicity, obese young women and men were 31% and 25% less likely than their normal-weight counterparts to have their first child by the age of 47 y. Obesity also predicted lower fertility beyond the first child, particularly in women. In both sexes, obesity in young adulthood was associated with fewer future years of marriage, which explained part of their decreased fertility. Second, underweight men were less likely than normal-weight men to have children, and this difference was due to the former group's lower probability of marriage. Third, no evidence was found to suggest that the fact that obese and underweight persons had fewer children would have reflected their voluntary preference for smaller family size.

Whereas the association between mortality and extremes of body weight was assessed in several studies (20–22), fertility has received far less attention in epidemiologic studies on body weight. With respect to obesity, a meta-analysis (22) of 74 cohort studies concluded that the mortality risk is ≈28% and 20% greater in obese women and men, respectively, than in their normal-weight counterparts. Current evidence thus suggests that obesity may be at least as important a risk factor for lower fertility as it is for greater mortality.

In obese women, marital status was more important in accounting for differences in the first and second births than for those in later births. This finding appeared to reflect the patterns of marriage and fertility: differences in marital status were more important in determining whether a person had children at all but less important in determining the births beyond the first and second child. Obese men also were less likely than were normal-weight men to marry, but that difference did not account the lower fertility of the obese men.

Although marriage accounted for part of the obesity-fertility association, obesity still predicted lower fertility in women and men, even after adjustment for marital status. In addition to measurement imprecision in assessing marital status, this residual effect may reflect physiologic risks of reproductive function

associated with obesity—eg, difficulties in fertilization and pregnancy complications in women (5–7) and lower semen quality and sexual dysfunctions in men (8–10). The lack of biological indicators of fertility in the present sample did not allow us to test this hypothesis. An important area for further research is an exploration of the extent to which the influence of body weight on fertility is mediated by biological factors and by social factors other than marriage.

In contrast to the findings of the Finnish study (4), in the present study, women's underweight was not associated with lower fertility. This finding is in line with studies suggesting that severe underweight related to eating disorders in young women may increase the risk of future pregnancy complications but does not decrease achieved fertility (23–26). The difference between the present study and the Finnish study may be due to the shorter follow-up period in the latter. In the present study, underweight in men was associated with lower achieved fertility, which was also observed in the Finnish study. The present findings suggest that the lower fertility of underweight men may be largely explained by their lower likelihood of marriage compared with normal-weight men.

The results concerning the number of children desired implied that the lower fertility associated with extremes of body weight is unlikely to reflect a voluntary preference for a smaller family size. In fact, obese youth desired larger families than did normal-weight youth, but the former were less likely than the latter to achieve their desired family size in adulthood, even when their higher expectations were taken into account in the analysis. Compared with normal-weight men, underweight men also had fewer children than they had desired.

### Strengths and limitations

The study design had 3 unique strengths. First, this is the largest study to date on body weight and fertility that is based on a representative multiethnic sample. Second, the study models this association up to the age of 47 y and thus allows us to assess the effect of body weight on nearly completed fertility. Third, marital status was modeled as a time-variant covariate, which provided a more accurate method of assessing the role of marital status than did the methods used previously (4).

The main limitation of the study was that data on weight and height were self-reported by the participants. Despite underreporting of weight and overreporting of height by some participants, validity studies suggest that self-reported weight and height are generally accurate (27–29). Considering that persons at the high end of weight distribution may tend to bias their reports toward the mean (30), the use of self-reported data may have diluted the curvilinear BMI-fertility association and thus provided underestimates of its magnitude.

In the present study, 17% to 35% of the children were born to participants who were not married in the year the child was born. These estimates are in agreement with the US national statistics (31), which indicate that the proportion of children born to unmarried women has increased from 18% in 1980 to 34% in 2002. However, our assessment of marital status on the basis of marriage (yes or no) rather than cohabitation may have underestimated the role of social relations in mediating the association between BMI and fertility.

Finally, it must be noted that the role of obesity in fertility patterns may have changed since the time the present cohort members were young adults. Whereas only ≈4% of the participants were obese at the age of 17 to 24 y, the US national statistics (32) indicate that the prevalence of obesity among 20–34-y-olds increased from ≈10% to ≈26% during the period from late 1970s to the early 2000s. On the one hand, with continued increases in the proportion of the population that is obese, the adverse effects of high body weight on fertility may currently be greater than those estimated in the present study. On the other hand, the changing prevalence of obesity may have changed the role of obesity as a determinant of marital status and reproductive behavior. Given that people tend to select partners resembling themselves in body weight (33, 34), it is possible that the increasing prevalence of obesity has decreased the negative effect of obesity on marriage prospects. We are not aware of any studies evaluating these hypotheses, and thus it is currently unclear whether the present findings can be generalized to more recent cohorts.

### Conclusion

We have shown an inverse U-shaped association between BMI and fertility, in which obese men and women and underweight men are less likely than are their normal-weight counterparts to have children by the age of 47 y. Our results provide a basis for further research to determine whether body weight may affect population demographics beyond its effects on mortality. More studies are also needed to evaluate underlying mechanisms and the extent to which the BMI-fertility association is socially and biologically mediated.

The authors' responsibilities were as follows—MJ, ME, and MK: designed the hypothesis and wrote the paper; and MJ: analyzed the data. None of the authors had a personal or financial conflict of interest.

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