Body Mass Index in Adolescence and Number of Children in Adulthood

Markus Jokela, Mika Kivimäki,*† Marko Elovainio,*†‡ Jorma Viikari,§ Olli T. Raitakari,¶ and Liisa Keltikangas-Järvinen**

Background: Body weight is associated with reproduction and related behaviors, but it is unknown whether it has significance for fertility differences in the general population. We examined whether adolescent body mass index (BMI; kg/m^2) predicted the number of children in adulthood 21 years later.

Methods: The participants were 1298 Finnish women and men (ages 12, 15, and 18 years at baseline) followed in a prospective population-based cohort study (the Cardiovascular Risk in Young Finns) from year 1980 to 2001.

Results: There was an inverted J-shaped association between BMI and the number of children, such that underweight adolescents had 10–16% fewer children in adulthood, overweight adolescents 4–8% fewer, and obese adolescents 32–38% fewer than individuals with normal adolescent weight. This association was similar in women and men, and independent of age, education, urbanicity of residence, and timing of menarche (in women). Adolescents with low or high BMI were less likely to have lived with a partner in adulthood, which partly accounted for their decreased number of children. The influence of adolescent BMI was independent of adulthood BMI in women but not in men. Age at menarche also predicted the number of children, such that women with early or late menarche had more children than those with average age at menarche.

Conclusion: Underweight and especially obesity may have a negative impact on fertility in the general population. The increasing prevalence of obesity in children and adolescents may represent a concern for future reproductive health.

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From the *Department of Psychology, University of Helsinki, Finland; †International Centre for Health and Society, Department of Epidemiology and Public Health, University College London, UK; ‡National Research and Development Centre for Welfare and Health, Helsinki, Finland; §Department of Medicine, Turku University, Turku, Finland; and ¶Department of Clinical Physiology, Turku University Central Hospital, Turku, Finland.

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Correspondence: Liisa Keltikangas-Järvinen, Department of Psychology, University of Helsinki, P. O. Box 9, FIN-00014, University of Helsinki, Finland. E-mail: liisa.keltikangas-jarvinen@helsinki.fi

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Obesity is associated with elevated levels of morbidity[1] and mortality,[2] and is considered to be one of the most important threats to public health.[3] Obesity increases the risk of sexual and reproductive dysfunction in women[4–6] and men,[7–9] and obese people are less likely to get married than people of normal weight.[10] Very low body mass index (BMI) has also been associated with dysfunctions of reproductive physiology in both men and women.[4,8]

Despite the global increase in the prevalence of obesity,[3] very little is known about the potential influence of body weight on fertility (ie, the number of children) in the general population. In a sample of parents of college students, Ellis and Haman[11] found that higher BMI was associated with greater number of children, particularly in women. However, this study was not prospective and, therefore, the direction of causality between weight and number of children could not be determined definitely. The number of births (ie, parity) is associated with increased risk of obesity in women,[12] and parenthood may also increase the risk of obesity in men.[13] Moreover, the sample of Ellis and Haman[11] included only individuals with one or more children, and potential confounding factors, such as age or level of education, were not controlled for.

Prospective epidemiologic studies with representative samples are needed to evaluate the influence of body weight on fertility in the general population. In the present study we examined whether adolescent BMI predicted the number of children in adulthood 21 years later in a population-based sample of Finnish women and men participating in a prospective study.[14,15] We hypothesized that there is an inverted U-shaped association between BMI and number of children, because both low and high BMI have been associated with risks for reproduction. We also assessed whether the influence of adolescent BMI was mediated by adulthood body weight or the likelihood of attaining a mate. In women, high adolescent BMI has been shown to be related to younger menarche[16] which, in turn, may predict earlier initiation of sexual behavior.[17] We therefore examined whether age at menarche was involved in the association between BMI and fertility. Skinfold thickness was used as an additional indicator of body fatness.

METHODS

Participants

The participants were 1298 women (n = 715) and men (n = 583) participating in the on-going population-based study of Cardiovascular Risk in Young Finns.[14,15] In this

study, a randomly selected sample of 3596 Finnish healthy children and adolescents from 6 birth cohorts (age 3, 6, 9, 12, 15, and 18 years at baseline) have been followed since 1980, focusing on the development of cardiovascular risk factors. Complete details of the study are provided elsewhere.[14,15] For the present study, we included 3 of the oldest cohorts, who were adolescents (ie, 12, 15 and 18 years of age) at baseline and thus were adults (ie, 33, 36 and 39 years of age) in the most recent follow-up phase in the year 2001. Of 1790 study participants in these 3 cohorts at baseline, 1298 had data on the present study variables (Table 1). The participants gave written informed consent, and the study was approved by local ethics committees. There was no linear or nonlinear association between adolescent BMI and the likelihood of having participated in the follow-up, indicating that adolescent BMI was not related to study attrition.

Measures in Adolescence

Adolescent height and weight were measured at the baseline when the participants were 12, 15, and 18 years of age. Measurements were taken in a medical examination with a Seca weight scale and anthropometer. BMI was calculated as weight in kilograms/(height in meters)^2.

Measures of skinfold thickness were obtained at the baseline examination by Harpenden calipers (Holtain and Bull-British Indicators instruments) to 0.2-mm readings. The combined thickness of 3 skinfold measurements (subscapular, triceps, and biceps) was used in the analysis. The partial correlation (controlling for age and sex) between BMI and skinfold thickness was r = 0.69.

The women participants reported the age at menarche at the baseline and in 2 subsequent follow-up phases 3 and 6 years later. For those who had reported the age at menarche in more than one follow-up, data from the earliest possible follow-up phase were used.

TABLE 1. Descriptive Statistics* of the Sample (n = 1298)

Sex; no. (%)	
Women	715 (55.1)
Men	583 (44.9)
Age in 2001	35.8 ± 2.4
Education	14.3 ± 3.3
Residence	2.5 ± 1.0
Has lived with a partner; no. (%)	1130 (87.0)
Adolescent body mass index (kg/m^2)	19.7 ± 2.9
Adulthood body mass index (kg/m^2)	25.4 ± 4.2
Adolescent skinfold thickness (mm)	29.7 ± 13.4
Adulthood waist circumference (cm)	85.9 ± 12.3
Age at menarche in women	13.2 ± 1.2
Number of children	
Mean ± SD	1.6 ± 1.3
No. (%)	
0	335 (25.8)
1	244 (18.8)
2	439 (33.8)
3	205 (15.8)
4	52 (4.0)
5+	23 (1.8)

*Mean ± SD, unless otherwise indicated.

Measures in Adulthood

Body mass index was calculated as above based on adulthood height and weight as measured in a medical examination in the follow-up phase in 2001. Waist circumference was measured midway between iliac crest and lowest rib as the average of 2 measurements with an accuracy of 0.1 cm. The partial correlation (controlling for age and gender) between adulthood BMI and waist circumference was r = 0.91.

The number of the participant's children and their years of birth were reported by the participants in the follow-up phase when the participants were 33, 36, and 39 years of age. For partnership history we created a dichotomous variable that indicated whether or not the participant had ever lived together with a partner (ie, had either been married or cohabiting). Data for this variable were taken from all the 6 follow-up phases, in which the participants have reported their current marital status and changes in marital status between the follow-ups.

Education level was measured by the completed years of education in adulthood reported by the participants. Place of residence in adulthood was reported by the participants on a four-point scale (1 = city; 2 = suburban area; 3 = rural area; 4 = remote rural area), and was used as a continuous covariate in the analyses.

Statistical Analysis

Poisson Regression

The association between BMI and the number of children was assessed with Poisson regression,[18] for which the equation with n independent variables is

$$\mu_i = \exp(\beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} + \ldots + \beta_n x_{in})$$

The Poisson model provided an adequate fit for the present data (Pearson goodness-of-fit $\chi^2 = 1310.65$, $df = 1291$), and there was no significant overdispersion in the dependent variable (mean = 1.6, variance = 1.7). The Poisson regression was fitted with adolescent BMI and its square as the independent variables; adulthood number of children was the dependent variable; and covariates were age, sex, years of education and place of residence. The associations of skinfold thickness and of age at menarche with the number of children were also assessed with Poisson regression, with the same covariates as mentioned above. The parameter estimates were expressed as incidence-risk ratios (IRRs).

Survival Analysis

In order to determine the temporal patterning of the fertility differences, discrete-time survival analysis[19] was used to examine whether and when the participants had their first, second, and third child, and whether adolescent BMI predicted these events. The birth of the fourth or any subsequent child was not considered since only 6% of the participants had a fourth child and only 2% had 5 or more children (Table 1). Survival analysis takes into account the phenom-

enon of censoring, ie, the fact that not all participants have their children within the study period, and some participants do not have children at all. In survival analysis a participant is censored either when the event of interest occurs (ie, a child is born) or when the study period ends (ie, at the age of 33, 36, or 39, depending on whether the participant belonged to the youngest, middle or oldest birth cohort). The survival analyses thus allowed us to determine the population estimates for the probabilities of having the first, second, and third child by the age 39. Age, sex, education and place of residence were entered as covariates. The age-specific fertility is known to follow a bell-shaped curve, so the effect of time was modeled as a nonlinear function. The parameter estimates were expressed as odds ratios of logit hazard function for one unit increase in the independent variable.

Adolescent BMI Categorization for Illustration

The results were illustrated by categorizing participants into groups of underweight, normal weight, overweight, and obese on the basis of their adolescent BMI. For the overweight and obese groups we used the international cut-off values provided by Cole et al[20] Based on these cut-offs, 7% of adolescent females and 9% of males were categorized as overweight and 0.9% of females and 1.4% of males were categorized as obese. In the absence of established cut-off values for adolescent underweight, we categorized BMI in the lowest 5% as underweight. This categorizing was carried out within sex and birth cohort groups, and provided the respective BMI cut-off values of 14.8, 16.6, and 17.6 for females 12, 15, and 18 years of age, and the corresponding values of 15.2, 17.0, and 18.3 for males. Next we determined the median adolescent BMI values within the underweight, normal weight, overweight and obese groups, (16.2, 19.4, 25.1, and 30.4, respectively). The corresponding values were 15.7, 19.5, 25.1, and 29.9 for females, and 16.4, 19.2, 25.1, and 30.5 for males. These values were used as prototypical or average cases of the 4 adolescent BMI groups in illustrations of the model-predicted estimates. In these calculations other covariates were assigned their mean values.

Logistic Regression

The association between adolescent BMI and partnership history was assessed with logistic regression analysis. Adolescent BMI and its square were the independent variables; adulthood number of children was the dependent variable; and the covariates were age, sex, years of education and place of residence.

Regression Diagnostics

BMI was positively skewed, so we examined whether a transformation of BMI affected the results using an inverse transformation (ie, X=1/BMI) which corrected the skewness. The results were similar and, in terms of confidence intervals of the estimates, even slightly more precise when the transformed BMI was used (data not shown). However, the original scale provided more convenient regression coefficients, so the analyses were fitted with the original BMI scale. Outlier diagnostics indicated that extreme observations did not substantially influence the results.

With one exception (see below), the findings were similar for women and men, with no statistical evidence of differences by sex. However, because of the sex-specific nature of fertility, we also present the results separately for women and men. In some analyses the confidence intervals of the relevant parameter estimates included 1.00 in separate analyses for women and men, but not when sexes were combined. In the absence of statistical interaction by sex, we will interpret these results based on the estimates for women and men combined.

RESULTS

There was a nonlinear association between adolescent BMI and the number of children in adulthood (Table 2, Model 1), indicating that underweight and obese individuals had fewer children than those with normal weight (Fig. 1A). The predicted number of children of average members (ie, median values) of adolescent underweight, normal weight, overweight, and obese groups were, respectively, 1.49, 1.78, 1.64, and 1.10 in women and 1.32, 1.46, 1.40, and 0.99 in men. In other words, average underweight, overweight, and obese women had, respectively, 16%, 8%, and 38% fewer children than women with normal weight. The corresponding percentages in men were 10%, 4%, and 32%. Individuals with BMI about 2 units above the mean (ie, around the cut-off point of the 75th percentile) were estimated to have 2–3% more children than individuals with mean BMI. Note that women had more children than men because women begin to have children at a younger age.[21,22]

The survival analyses indicated that there was a nonlinear association between adolescent BMI and the probability of having the first, second or third child at a given age (Table 3; Fig. 2). There were no linear or nonlinear interaction effects between BMI and time, indicating that the strength of the association between BMI and the probability of having children did not increase or decrease with time. Figure 2 shows the cumulative probability (ie, $P = 1 -$ value of survival function) of having children plotted against age in women and men. Table 4 shows in numerical form the likelihoods of having the first, second, and third child by the age of 39 in women and men.

The partial correlation (controlling for age and sex) between Year-0 adolescent and Year-21 adulthood BMI was r=0.54. We tested whether adulthood BMI mediated the association between adolescent BMI and the number of children by entering both adolescent and adulthood BMI into the same model. The results differed by sex (sex × adulthood BMI interaction effect: linear IRR=1.28, 95% confidence interval [95% CI] = 1.02–1.60; quadratic IRR = 0.996, 95% CI = 0.992–0.999). When both BMIs were in the model, only adolescent BMI was important in women, while only adulthood BMI was important in men (Table 2, Model 3). In other words, the influence of adolescent BMI was mediated by adulthood BMI in men but not in women. This result was the same when waist circumference rather than BMI was used as an indicator of adulthood body fatness (data not shown).

Next we assessed whether adolescent BMI predicted the likelihood of having ever lived with a partner (ie,

TABLE 2. Association of Adolescent BMI With Number of Children in Adulthood: Three Adjusted* Poisson Regression Models (n = 1298)

	Model 1 IRR (95% CI)	Model 2 IRR (95% CI)	Model 3 IRR (95% CI)
All (n = 1298)			
BMI	1.28 (1.10–1.48)	1.18 (1.02–1.36)	1.24 (1.07–1.45)
BMI^2	0.994 (0.990–0.998)	0.996 (0.993–0.999)	0.995 (0.991–0.998)
Partnership		5.01 (3.92–6.40)	
Adult BMI			1.04 (0.95–1.14)
Adult BMI^2			0.999 (0.998–1.001)
Women (n = 715)			
BMI	1.32 (1.07–1.63)	1.19 (0.97–1.46)	1.30 (1.04–1.63)
BMI^2	0.993 (0.988–0.998)	0.996 (0.991–1.001)	0.994 (0.989–0.999)
Partnership		4.72 (3.35–6.64)	
Adult BMI			0.92 (0.87–1.08)
Adult BMI^2			1.000 (0.999–1.002)
Men (n = 583)			
BMI	1.24 (1.00–1.54)	1.16 (0.93–1.43)	1.16 (0.93–1.44)
BMI^2	0.995 (0.990–0.999)	0.996 (0.992–1.001)	0.996 (0.991–1.001)
Partnership		5.30 (3.74–7.52)	
Adult BMI			1.28 (1.05–1.55)
Adult BMI^2			0.996 (0.993–0.999)

*All models were adjusted for age, education, residence, and sex.

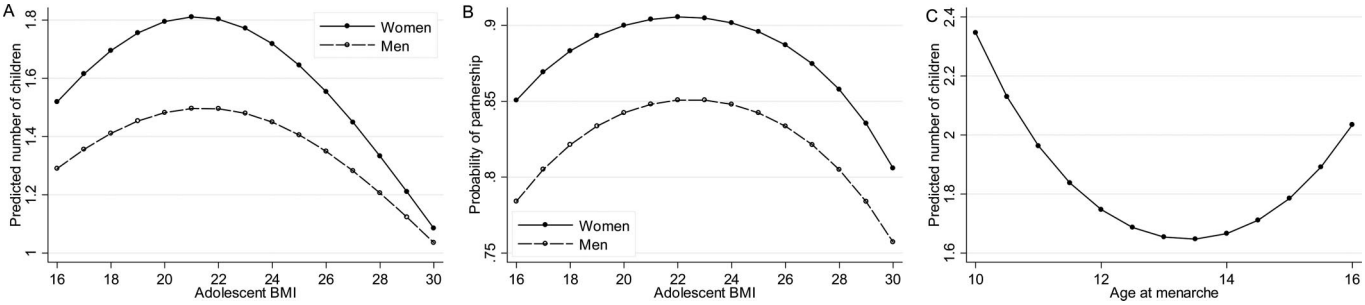

FIGURE 1. (A) and (B) show the predicted number of children and the likelihood of having ever lived with a partner, respectively, plotted against adolescent BMI. In (C), the predicted number of children is plotted against the age at menarche (in women). In (A) and (B), the range of the x-axis is approximately from the median value of the underweight group to the median value of the obese group. The range of the x-axis in (C) is approximately ± 2 standard deviations.

having been married or cohabiting), and the degree to which this accounted for the association between BMI and the number of children. Adolescent BMI predicted in a nonlinear fashion the likelihood of having ever lived with a partner in women (linear OR = 1.83, 95% CI = 1.03–3.26; quadratic OR = 0.986, 95% CI = 0.973–0.999) and men (linear OR = 1.62, 95% CI = 0.996–2.64; quadratic OR = 0.989, 95% CI = 0.978–0.999; Fig. 1B) with the respective probability estimates of 0.84, 0.90, 0.90, and 0.81 in women and 0.79, 0.84, 0.84, and 0.74 in men for average members of the 4 BMI groups. Adolescent BMI was still associated with number of children even after controlling for partnership history (Table 2, Model 2).

When the Poisson regression models were fit including only participants who had ever lived together with a partner, the evidence of the BMI-fertility association was weaker, all (n = 1130): linear IRR = 1.14, 95% CI = 0.99–1.33; quadratic IRR = 0.997, 95% CI = 0.993–1.000; Women (n = 640): linear IRR = 1.15, 95% CI = 0.94–1.42; quadratic IRR = 0.997, 95% CI = 0.992–1.001; Men (n = 490): linear IRR = 1.13, 95% CI = 0.91–1.40; quadratic IRR = 0.997, 95% CI = 0.992–1.002], although not absent. In this subsample, the predicted number of children in the 4 adolescent BMI groups was 1.77, 1.93, 1.82 and 1.46 in women, and 1.63, 1.69, 1.56, and 1.21 in men. In other words, underweight, overweight and obese women had approximately 8%, 5%, and 24% fewer children, respectively, than women with normal adolescent weight. In men the corresponding percentages were 4%, 8%, and 29%, respectively.

TABLE 3. Association of Adolescent BMI With the Birth of the First, Second, and Third Child: Three Survival Analysis Models (n = 1298)

	First Child OR (95% CI)	Second Child OR (95% CI)	Third Child OR (95% CI)
All			
Time	3.24 (2.81–3.75)	4.71 (3.78–5.87)	5.46 (3.58–8.33)
Time^2	0.98 (0.98–0.98)	0.98 (0.97–0.98)	0.97 (0.97–0.98)
BMI	1.37 (1.10–1.70)	1.36 (1.05–1.76)	2.21 (1.31–3.75)
BMI^2	0.993 (0.988–0.998)	0.993 (0.987–0.998)	0.982 (0.970–0.994)
Women			
Time	2.97 (2.48–3.56)	4.76 (3.60–6.30)	4.58 (2.80–7.48)
Time^2	0.98 (0.98–0.98)	0.98 (0.97–0.98)	0.98 (0.97–0.99)
BMI	1.49 (1.08–2.06)	1.46 (0.998–2.13)	2.78 (1.33–5.79)
BMI^2	0.991 (0.983–0.998)	0.991 (0.982–1.000)	0.976 (0.959–0.994)
Men			
Time	4.05 (3.12–5.24)	5.30 (3.62–7.77)	8.70 (3.78–20.05)
Time^2	0.98 (0.97–0.98)	0.97 (0.97–0.98)	0.97 (0.96–0.98)
BMI	1.36 (1.01–1.83)	1.32 (0.92–1.89)	1.72 (0.82–3.62)
BMI^2	0.993 (0.987–1.000)	0.994 (0.986–1.002)	0.988 (0.971–1.005)

All models were adjusted for age, education, residence, and sex.

Among women the partial correlation (controlling for age) between BMI and age at menarche was $r=-0.27$, indicating that heavier women had earlier menarche. There was a nonlinear association between age at menarche and the number of children, such that women with early or late menarche had more children than those in between (linear IRR=0.44; CI=0.25–0.76; quadratic IRR = 1.031; 95% CI = 1.010–1.053; Fig. 1C). Controlling for age at menarche did not change the association between adolescent BMI and fertility (data not shown).

The association between adolescent skinfold thickness and adulthood number of children was in the same direction (all: linear IRR = 1.13, 95% CI = 0.98–1.31; quadratic IRR = 0.988, 95% CI = 0.973–1.004; women: linear IRR = 1.13, 95% CI = 0.92–1.39; quadratic IRR = 0.986, 95% CI = 0.960–1.013; men: linear IRR = 1.16, 95% CI = 0.92–1.48; quadratic IRR = 0.987, 95% CI = 0.963–1.012) as with BMI, although the confidence intervals of the parameter estimates included 1.00.

DISCUSSION

Our findings suggest that underweight and obesity are associated with reduced reproduction in the general population. The participants of the present study were still in their reproductive years, so their current fertility may not have accurately represented their completed fertility. However, the survival analyses suggested that this may not have been a major limitation, because the reproductive differentials between BMI groups became stronger over time. Moreover, less than 5% of babies in Finland are born to women over 39 years of age,[21] the corresponding percentage being somewhat higher for men.[22] It is therefore unlikely that the observed fertility differences related to body weight would represent only transitory effects, although this should be confirmed with further follow-up of this cohort.

In women, underweight is known to cause menstrual dysfunction,[23] and obesity has been associated with decreased fecundability and increased risk of pregnancy complications.[4–6] Obesity is also a risk factor for polycystic ovary syndrome, which causes infertility.[24] In men, low (<20 kg/m^2) and high (>25 kg/m^2) adulthood BMI have been associated with decreased semen quality,[8] and obesity has been shown to increase the risk of erectile dysfunction.[9] These physiological correlates of body weight may contribute to the lower fertility in underweight and obese people.

The lowered fertility of underweight and obese individuals was partly accounted by their decreased likelihood of attaining a partner. We were not able to examine whether other factors related to mating (eg, the number of partners, the stability of relationships) might also be involved. The role of other psychologic and social variables should also be considered. Taller young women have been found to have lower maternal tendencies and decreased preference for having children.[25] Whether a similar phenomenon is related to BMI is unclear[26]; the possible correlation between childbearing motivation and body weight merits further research. The extremes of body weight may also be related to psychiatric disorders.[27,28]

Research suggests that there is assortative mating (ie, nonrandom mating that results in similarity between spouses) for BMI in general,[29] and obesity in particular.[30] This may act to cumulate the influence of BMI on fertility in underweight and obese couples. Assortative mating also implies that the BMI-fertility association observed in one sex might be partly accounted by their spouse's BMI, for which we did not have data. This possibility has received little attention in fertility

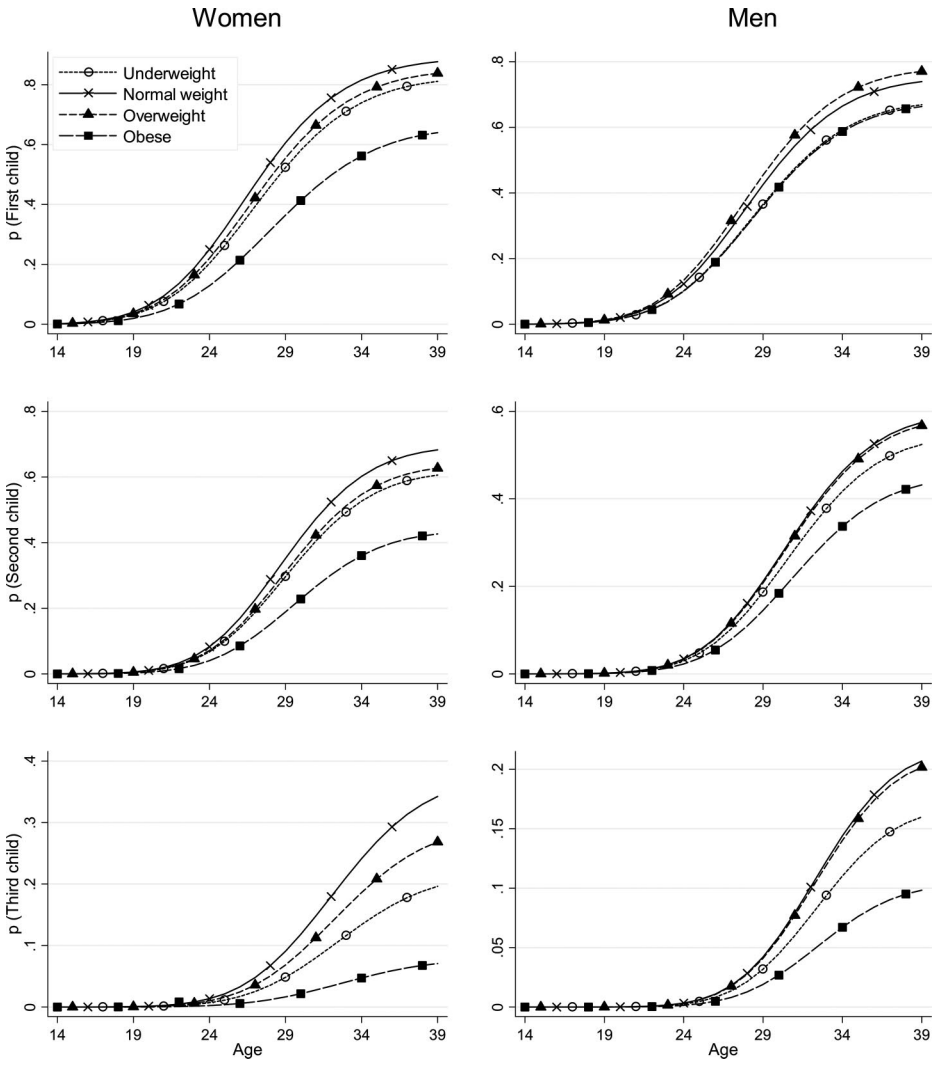

FIGURE 2. The cumulative probability (P = 1 – value of survivor function) of having the first, second, and third child plotted against age in women and men by body weight groups. Note the different scales of y-axis in each of the panels.

TABLE 4. The Predicted Probability of Having the First, Second, and Third Child by the Age of 39 (ie, P = 1 − Value of Survivor Function at 39) by BMI Group

	Underweight	Normal Weight	Overweight	Obese	Total
All	0.50	0.57	0.55	0.39	0.50
First child	0.75	0.81	0.80	0.65	0.75
Second child	0.57	0.63	0.60	0.42	0.55
Third child	0.19	0.28	0.25	0.09	0.20
Women	0.54	0.63	0.58	0.38	0.53
First child	0.81	0.88	0.84	0.64	0.79
Second child	0.61	0.68	0.63	0.43	0.59
Third child	0.20	0.34	0.27	0.07	0.22
Men	0.45	0.51	0.51	0.40	0.47
First child	0.67	0.74	0.77	0.66	0.71
Second child	0.52	0.57	0.57	0.43	0.52
Third child	0.16	0.21	0.20	0.10	0.17

research. Sallmen et al[7] found that men's overweight decreased the likelihood of conception independently of their wives' BMI. Further data on couples are needed to investigate the combined influence of partners' body weight on the number of their children.

Adolescent BMI predicted fertility independently of adulthood BMI in women but not in men. This difference may be because in women the association between adulthood BMI and fertility is probably confounded by the influence of pregnancies on adulthood BMI.[12] It may also be that women's adolescent body weight has independent long-term implications for reproductive potential. This would parallel the finding that adolescent overweight may increase adulthood mortality independently of adulthood body weight.[2] Indeed, adolescent overweight has been implicated as a risk factor for adulthood polycystic ovary syndrome[31] and menstrual disturbances.[31–33] Women with a history of eating disorders and severe underweight appear to suffer no major long-term consequences of infertility,[34–36] although they are more likely to have obstetric complications.[27,34–36] More detailed information on possible biologic and social mechanisms would help to evaluate the relative importance of adolescent and adulthood body weight on fertility.

The ongoing increases in obesity[3] may represent a concern for future reproductive health.[37] In Finnish adolescents the prevalence of obesity increased 2- to 3-fold from 1977 to 1999.[38] Hence, the lowered fertility associated with high BMI may affect an increasing proportion of the population in generations younger than the present study participants, who were adolescents in the early 1980s.

Adolescent BMI is a highly heritable trait with an estimated heritability of about 85% in Finnish adolescent twins.[39] Heritable traits associated with reproductive differentials have the potential to evolve via the mechanism of natural selection.[40] The present results suggest that stabilizing selection may be acting on BMI, since the low and high ends of BMI are selected against in terms of reproductive success. On the other hand, individuals with a BMI about one-half standard deviations above the mean were estimated to have 2–3% more children than those with mean BMI. If this pattern were to hold over generations, genetic factors might be shifting the mean BMI of the population about 2 units upwards in future generations, albeit only at a modest rate. Reproductive differentials are unlikely to account for any substantial portion of the recent obesity epidemic.

The higher fertility of women with early or late menarche was an unexpected finding. Early-maturing girls tend to enter sexual and marital relations earlier,[17] which might contribute to their greater number of children in adulthood. On the other hand, a study conducted in China[41] found that late rather than early menarche was associated with increased fertility. The pattern of the present finding suggests that timing of menarche may be under disruptive natural selection where individuals in the extreme ends of a trait have a reproductive advantage over the others. It would be of interest to investigate further the developmental pathways linking early and late menarche to increased adulthood fertility.

REFERENCES

1. Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes Relat Metab Disord*. 1997;2: 507–526.
2. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard growth study of 1922 to 1935. *N Engl J Med*. 1992;327:1350–1355.
3. World Health Organization. *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation.* Geneva, Switzerland: WHO; 2000.
4. Zaadstra BM, Seidell JC, Van Noord PAH, et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *BMJ*. 1993;306:484–487.
5. Norman RJ, Clark AM. Obesity and reproductive disorders: a review. *Reprod Fertil Dev*. 1998;10:55–63.
6. Siega-Riz AM, Siega-Riz AM, Laraia B. The implications of maternal overweight and obesity on the course of pregnancy and birth outcomes. *Matern Child Health J*. 2006;10(Suppl):153–156.
7. Sallmen M, Sandler DP, Hoppin JA, et al. Reduced fertility among overweight and obese men. *Epidemiology*. 2006;17:520–523.
8. Jensen TK, Andersson AM, Jorgensen N, et al. Body mass index in relation to semen quality and reproductive hormones among 1,558 Danish men. *Fertil Steril*. 2004;82:863–870.
9. Bacon CG, Mittleman MA, Kawachi I, et al. A prospective study of risk factors for erectile dysfunction. *J Urol*. 2006;176:217–221.
10. Gortmaker SL, Must A, Perrin JM, et al. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med*. 1993;329:1008–1012.
11. Ellis L, Haman D. Population increases in obesity appear to be partly due to genetics. *J Biosoc Sci*. 2004;36:547–559.
12. Smith DE, Lewis CE, Caveny JL, et al. Longitudinal changes in adiposity associated with pregnancy. The CARDIA Study. Coronary Artery Risk Development in Young Adults Study. *JAMA*. 1994;271:1747–1751.
13. Weng HH, Bastian LA, Taylor DH, et al. Number of children associated with obesity in middle-aged women and men: results from the Health and Retirement Study. *J Womens Health*. 2000;13:85–91.
14. Åkerblom HK, Uhari M, Pesonen E, et al. Cardiovascular Risk in Young Finns. *Ann Med*. 1991;23:35–40.
15. Raitakari OT, Juonala M, Kähönen M, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study. *JAMA*. 2003;290:2277–2283.
16. Biro FM, Khoury P, Morrison JA. Influence of obesity on timing of puberty. *Int J Androl*. 2006;29:272–277.
17. Udry RJ, Cliquet RL. A cross-cultural examination of the relationship between ages at menarche, marriage, and first birth. *Demography*. 1982;19:53–63.
18. Long JS, Freese J. *Regression Models for Categorical Dependent Variables Using Stata, Revised Edition.* College Station, TX: Stata Press; 2003.
19. Singer JB, Willett JB. *Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence.* Oxford, Oxford University Press; 2003.
20. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;320:1240–1246.
21. Gissler M. Hedelmällisyys Suomessa ennen, nyt ja tulevaisuudessa [Fertility in Finland in the past, now, and in the future] *Yhteiskuntapolitiikka*. 2003;68:553–567.
22. Nikander T. *Suomalaismiehen perheellistyminen* [The family formation in Finnish men] (Statistics Finland, Helsinki, 1995; in Finnish).
23. Frisch RE. Body fat, menarche, fitness and fertility. *Hum Reprod*. 1987;2:521–533.
24. Pasquali R, Gambineri A, Pagotto U. The impact of obesity on reproduction in women with polycystic ovary syndrome. *BJOG*. 2006;113: 1148–1159.
25. Deady DK, Smith MJL. Height in women predicts maternal tendencies and career orientation. *Pers Ind Diff*. 2006;40:17–25.
26. Ball K, Crawford D, Kenardy J. Longitudinal relationships among overweight, life satisfaction, and aspirations in young women. *Obes Res*. 2004;12:1019–1030.

27. Finfgeld DL. Anorexia nervosa: analysis of long-term outcomes and clinical implications. *Arch Psychiatr Nurs*. 2002;16:176–186.
28. Simon GE, Von Korff M, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. *Arch Gen Psychiatry*. 2006;63:824–830.
29. Silventoinen K, Kaprio J, Lahelma E, Viken RJ, Rose RJ. Assortative mating by body height and BMI: Finnish twins and their spouses. *Am J Hum Biol*. 2003;15:620–627.
30. Hebebrand J, Wulftange H, Goerg T, et al. Epidemic obesity: are genetic factors involved via increased rates of assortative mating? *Int J Obes Relat Metab Disord*. 2000;24:345–353.
31. Laitinen J, Taponen S, Martikainen H, et al. Body size from birth to adulthood as a predictor of self-reported polycystic ovary syndrome symptoms. *Int J Obes Relat Metab Disord*. 2003;27:710–715.
32. Rich-Edwards JW, Goldman MB, Willett WC, et al. Adolescent body mass index and infertility caused by ovulatory disorder. *Am J Obstet Gynecol*. 1994;171:171–177.
33. Lake JK, Power C, Cole TJ. Women's reproductive health: the role of body mass index in early and adult life. *Int J Obes Relat Metab Disord*. 1997;21:432–438.
34. Crow SJ, Thuras P, Keel PK, Mitchell JE. Long-term menstrual and reproductive function in patients with bulimia nervosa. *Am J Psychiatry*. 2002;159:1048–1050.
35. Bulik CM, Sullivan PF, Fear JL, et al. Fertility and reproduction in women with anorexia nervosa: a controlled study. *J Clin Psychiatry*. 1999;60:130–135.
36. Wentz E, Gillberg IC, Gillberg C, Rastam M. Fertility and history of sexual abuse at 10-year follow-up of adolescent-onset anorexia nervosa. *Int J Eat Disord*. 2005;37:294–298.
37. Skakkebaek NE, Jorgensen N, Main KM, et al. Is human fecundity declining? *Int J Androl*. 2006;29:2–11.
38. Kautiainen S, Rimpelä A, Vikat A, Virtanen SM. Secular trends in overweight and obesity among Finnish adolescents in 1977–1999. *Int J Obes Relat Metab Disord*. 2002;26:544–552.
39. Pietiläinen KH, Kaprio J, Rissanen A, et al. Distribution and heritability of BMI in Finnish adolescents aged 16y and 17y: a study of 4884 twins and 2509 singletons. *Int J Obes Relat Metab Disord*. 1999;23:107–115.
40. Darwin C. *On the Origin of Species.* Cambridge: Harvard University Press; 2005/1859.
41. McKibben SL, Poston Jr., DL. The influence of age at menarche on the fertility of Chinese women. *Soc Biol.* 2003;50:222–237.