Pregnancy and Lactation Hinder Growth and Nutritional Status of Adolescent Girls in Rural Bangladesh[1,2]

Jee H. Rah,[3] Parul Christian,[3]* Abu Ahmed Shamim,[4] Ummeh T. Arju,[4] Alain B. Labrique,[3] and Mahbubur Rashid[4]

[3]Center for Human Nutrition, Department of International Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD 21205 and [4]JiVitA Maternal and Child Research Project, 5400 Rangpur, Bangladesh

Abstract

Adolescent pregnancy is associated with adverse birth outcomes. Less is known about its influence on maternal growth and nutritional status. We determined how pregnancy and lactation during adolescence affects postmenarcheal linear and ponderal growth and body composition of 12–19 y olds in rural Bangladesh. In a prospective cohort study, anthropometric measurements were taken among primigravidae ($n = 229$) in the early first trimester of pregnancy and at 6 mo postpartum. Randomly selected never-pregnant adolescents ($n = 458$) of the same age and time since menarche were measured within 1 wk of these assessments. Annual changes in anthropometric measurements were compared between the 2 groups adjusting for confounders using mixed effects regression models. The mean ± SD age and age at menarche of adolescents were 16.3 ± 1.6 y and 12.7 ± 1.2 y, respectively. Unlike pregnant girls who did not grow in height (−0.09 ± 0.08 cm/y), never-pregnant girls increased in stature by 0.35 ± 0.05 cm/y. The adjusted mean difference between the 2 groups was 0.43 ± 0.1cm ($P < 0.001$). Similarly, whereas never-pregnant girls gained BMI, mid-upper arm circumference, and percent body fat, pregnant girls declined in every measurement by 6 mo postpartum, resulting in adjusted mean ± SD differences in annual changes of 0.62 ± 0.11 kg/m^2, 0.89 ± 0.12 cm, and 1.54 ± 0.25%, respectively (all $P < 0.001$). Differences in changes in all anthropometric measurements except height were greater among adolescents whose first pregnancy occurred <24 mo vs. ≥24 mo since menarche (BMI, −1.40 ± 0.18 vs. −0.60 ± 0.11 kg/m^2; all interaction terms, $P < 0.05$). Pregnancy and lactation during adolescence ceased linear growth and resulted in weight loss and depletion of fat and lean body mass of young girls.   J. Nutr. 138: 1505–1511, 2008.

Introduction

Adolescent pregnancy is a worldwide concern, particularly in areas of poverty and social disadvantage. Nearly two-thirds of women in Sub-Saharan Africa and a few countries in South Asia have their first child before the age of 20 y (1). Pregnancy during adolescence has been shown to increase the risk of adverse birth outcomes, poor fetal growth, and infant and maternal health and mortality (2,3). Less is known about how pregnancy and lactation affect the growth and nutritional status of adolescent mothers.

Adolescence is a period of rapid growth and development. Approximately 50% of adult body weight and 15% of final adult height is attained during adolescence, along with changes in body shape and composition (4,5). In South Asian countries such as Bangladesh, chronic undernutrition can delay physical maturation and extend the adolescent growth period beyond 20 y of age, coinciding with the age of first pregnancy (6). Adolescents in developing countries exhibit poor growth and nutritional status. A study of rural Bangladeshi teenagers showed that stunting (height-for-age <3rd percentile of the National Center for Health Statistics/WHO reference) and thinness (BMI-for-age <5th percentile of the National Center for Health Statistics/WHO reference) were prevalent among 48 and 60% of adolescent girls, respectively (7). Other than genetic factors, adverse environmental conditions such as low socioeconomic status (SES)[5] and poor quality diets explain the suboptimal growth of teenagers (8,9).

Conventionally, it has been accepted that growth is largely completed by the time adolescents become pregnant, because preg-

[5] Abbreviations used: MUAC, mid-upper arm circumference; SES, socioeconomic status; SSF, subscapular skinfold; TEM, technical error of measurement; TSF, triceps skinfold.

THE JOURNAL OF NUTRITION

nancy occurs subsequent to menarche, when growth rate drops to a nadir (10,11). However, in a recent study conducted in Camden, NJ, it was demonstrated that a large proportion of pregnant adolescents were still growing (using knee height changes as a measure of growth), reflecting continuing growth in stature during and following pregnancy (12). This finding may be particularly relevant to malnourished adolescent girls in developing countries whose physical growth period is extended and in whom pregnancy occurs at an early age.

A large proportion of adolescents in developing countries enters pregnancy with a poor nutritional status and is likely to have suboptimal dietary intake during pregnancy and lactation (13–15). Consequently, in severely undernourished pregnant adolescents, maternal nutritional depletion and impaired growth of the fetus are likely to occur concurrently due to the competition for nutrients between the growing teenage mother and the fetus (13).

The present study was conducted to investigate how pregnancy and lactation during adolescence affects postmenarcheal linear and ponderal growth and body composition of young mothers in rural Bangladesh. We hypothesized that annual changes in stature, weight, BMI, lean body mass, and body fat measures among adolescents who experienced a pregnancy and were followed through 6 mo postpartum would be lower compared with annual changes in these anthropometric indicators among never-pregnant adolescents.

Materials and Methods

Study area and subjects. We conducted a prospective cohort study in which 2 groups of adolescent girls, 12–19 y old primigravidae and never-pregnant subjects at the same age and time since menarche, were concurrently followed for 1 y. The study was carried out in rural northwestern Bangladesh where a double-masked, randomized, controlled trial was ongoing (2001–2007) to examine the impact of vitamin A and β-carotene supplementation on maternal and infant mortality (the JiVitA-1 trial). The trial area comprised 596 community units, called sectors, in the districts of Gaibandha and Rangpur, encompassing an area of 580 km^2 with an estimated total population of 650,000. This densely populated (~1000 per km^2) study area is typical of rural Bangladesh with respect to SES, health service utilization, childhood malnutrition and morbidity, and reproductive health.

In July 2004, 96 contiguous sectors in the western part of the JiVitA study area were purposively selected for conducting the present study. A census was conducted at the outset in these selected sectors to enumerate all married and unmarried female adolescents. Data on date of birth, month and year of menarche, marital status, and pregnancy history were collected from all enumerated adolescents. The self-reported month and year of birth, checked against the home records of date of birth or the parents, was used to calculate age. Data on menarcheal status was gathered by asking the adolescent the year and month of the onset of menstruation. Help from the mother or other female relatives living the household was sought. Those who were pregnant at the time of census, had a previous pregnancy, were premenarcheal, or were <12 y or >19 y of age were excluded. A total of 2605 married nulligravid ($n = 385$) and unmarried ($n = 2220$) adolescents aged 12–19 y who had reached menarche were identified. Subsequently, over a period of 1 y, newly married women who were enumerated and registered into the study by the JiVitA project were added to the list of enumerated adolescents if they met the above criteria ($n = 419$).

Married adolescents were visited once every 5 wk to identify incident pregnancies as part of the JiVitA project. At each of these visits, women who reported being amenstrual in the past 30 d were administered an human chorionic gonadotropin-based urine test to detect pregnancy. As part of the present study, each time an adolescent was ascertained as being pregnant for the first time, 2 never-pregnant adolescents were randomly selected from the pool of enumerated adolescents matched on age (in years) and time since menarche (in months).
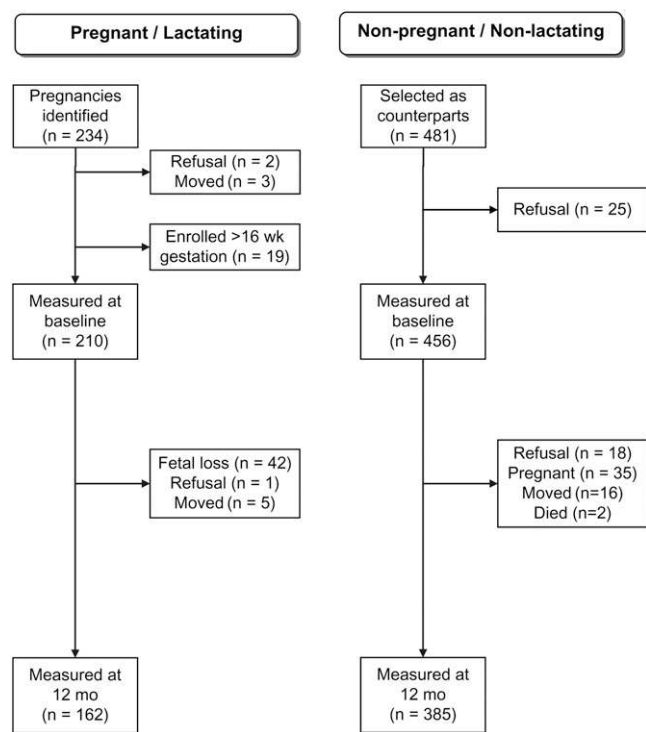
Data collection. After obtaining consent, within 1 ± 1.5 wk of pregnancy ascertainment, pregnant adolescents were interviewed at home regarding their dietary intake and physical activity in the preceding week, morbidity in the previous month, and smoking and alcohol consumption. An FFQ was used to determine intake of 30 different food items. Physical activity was assessed by self-reported performance of 15 common work activities. Height, weight, mid-upper arm circumference (MUAC) and triceps (TSF) and subscapular (SSF) skinfolds were measured during this visit. Interviews and measurements were conducted at a mean $\pm$ SD gestational age of 10.1 ± 2.8 wk and at 6 mo postpartum for pregnant adolescents. Never-pregnant adolescents were also assessed within a week of the assessments conducted in their pregnant counterparts. Information on SES was collected at the first interview for both groups. At 3 and 6 mo postpartum, women were asked whether they were breast-feeding and were asked to recall the number of times had they breast-fed in the past 24 h. Timing of initiation of breast-feeding at birth and feeding of prelacteals was determined soon after birth during a home visit to conduct newborn anthropometry.

All interviews and measurements were conducted by 8 trained and standardized female interviewers/anthropometrists. Anthropometric measurements were taken following standard procedures as described by Gibson (16). Height was measured to the nearest 0.1 cm using a Harpenden stadiometer (Shorr Productions) and weight was measured to the nearest 0.1 kg using a digital scale (UNICEF scale S0141015, by SECA). Both TSF and SSF were measured to the nearest 0.2 mm using Holtain calipers and MUAC to the nearest 0.1 cm using a nonstretch locally manufactured insertion tape (JiVitA) based on the Ross nonelastic tape design (Ross Laboratories). All measurements except weight were taken 3 times and the median value for each measurement was used for this analysis.

Standardization for each anthropometric measurement was conducted every 3 mo throughout the data collection period. During the sessions, each female interviewer measured a group of 10–15 women twice with an hour between measurements. Both intra- and interobserver reliability were assessed by calculating the technical error of measurement (TEM) as described by Ulijaszek and Kerr (17). The mean $\pm$ SD intra- and interobserver TEM of 8 female interviewers were 0.21 ± 0.03 and 0.25 ± 0.07 cm, respectively, for height; 0.17 ± 0.02 and 0.20 ± 0.05 cm, respectively, for MUAC; 0.52 ± 0.10 and 0.69 ± 0.14 mm, respectively, for TSF; 0.5 ± 0.09 and 0.5 ± 0.08 mm, respectively, for SSF. Throughout the study, the TEM for all of the measurements were maintained at approximately one-half the acceptable intra- and interobserver TEM values suggested by Shorr (18): 0.69 and 0.95 cm for height; 0.35 and 0.43 cm for MUAC; 0.8 and 1.89 mm for TSF; and 1.83 and 1.53 mm for SSF.

Sample size. The sample size was calculated using $\alpha = 0.05$ and $\beta = 0.2$ to detect a difference of ≥ 0.5 cm in annual height increments between pregnant and never-pregnant adolescents using a SD of an annual change in height of 1.5 cm (19). A 1:2 ratio of pregnant to never-pregnant adolescents was used to ensure at least 1 never-pregnant comparison in case 1 of the 2 were to become pregnant during the course of the study. This sample size was increased by 35% to account for pregnancy loss and loss to follow-up, resulting in a required (rounded) sample of 220 pregnant and 440 never-pregnant adolescents. A total of 229 pregnant and 456 never-pregnant adolescents was enrolled into the study between April and November 2005.

Although the 5-weekly pregnancy surveillance allowed us to identify most pregnancies early in gestation, some women who later became pregnant were not met at each round and were identified later in gestation. A total of 19 pregnant girls enrolled after 16 wk of gestation were excluded because their baseline (early pregnancy) anthropometric measurements were less likely to be proxy for prepregnancy measurements (**Fig. 1**). Adolescents who had a fetal loss ($n = 42$) were excluded from the analysis to keep the exposure of pregnancy and lactation approximately constant within the pregnant adolescent group (Fig. 1). Baseline characteristics did not differ between adolescents who had live births compared with those with fetal loss (data not shown). The present analysis uses the baseline and 12-mo follow-up interview and anthropometric data to address the primary research question of whether the

FIGURE 1 Study participation and follow-up among pregnant and never-pregnant adolescents.

annual changes in anthropometric measurements differed by pregnancy status.

Calculations and statistical analyses. BMI was defined as weight (kg)/height^2 (m). Upper-arm-muscle and fat areas were derived from MUAC and TSF as described by Frisancho (20) and percent body fat from TSF and SSF [$1.33 \times (SSF + TSF) - 0.013 \cdot (SSF + TSF)^2 - 2.5$] as described by Slaughter et al. (21). Stunting and underweight were defined as height-for-age and weight-for-age Z-scores < −2, respectively, using the CDC (22) reference standards in EpiInfo 2005 software.

Descriptive statistics were employed to examine the distributions of the full range of variables. We created categorical variables using meaningful cutoffs: dietary intake variables were classified into 6 food groups and dichotomized using a cut-off of ≤3 times/wk; work activities were classified into strenuous vs. light-to-moderate groups and dichotomized using a cutoff of ≤2 activities/d; morbidity variables were grouped and dichotomized into no morbidity and any day of illness in the past 30 d. A wealth index was developed based on dwelling characteristics, land ownership, productive and durable assets, and human capital (S. Gunnsteinsson, A. B. Labrique, K. P. West, Jr, P. Christian, S. Mehra, A. A. Shamim, M. Rashid, J. Katz, and R. Klemm, unpublished data) using principal components analysis (23).

We compared baseline characteristics between pregnant and never-pregnant adolescents using *t* tests and chi-square tests. The Kruskal-Wallis test was used to compare the means of continuous variables that were not normally distributed, such as TSF and SSF. Changes in height, weight, BMI, and body composition indicators were annualized after being divided by the actual number of days between measurements and tested for significance using paired *t* tests. Mean annual changes in anthropometric measurements were compared by pregnancy status using *t* tests.

Changes in anthropometric measurements over time were compared by pregnancy status adjusting for confounders using linear mixed effects regression models (24,25). The mixed effects regression models permit accounting for the within-subject correlation of repeated measurements (in this case, only 2) in estimation of the regression parameters and their standard errors (24). Dependent variables included anthropometric measures; separate models were constructed for height, weight, BMI,

MUAC, TSF, SSF, upper-arm muscle and fat area, and percent body fat. Independent variables included time of measurement in years since the baseline visit, pregnancy status, interaction between time and pregnancy status, age, time since menarche, and other potential confounders. Confounding factors included characteristics that differed by pregnancy status at the baseline and 12-mo follow-up and were associated with annual change in each anthropometric measurement in the bivariate analyses ($P < 0.05$). The robust estimates of standard errors were computed. The interaction between time of measurement and pregnancy status reflected the difference in annual change in anthropometric measures between pregnant and never-pregnant adolescents.

We hypothesized that differences in changes in anthropometric measures between pregnant and never-pregnant adolescents would be larger among adolescents with a shorter time since menarche. Thus, a 3-way interaction between time of measurement, pregnancy status, and time since menarche as a dichotomous variable was tested at the 5% significance level. If there was a significant interaction, linear mixed effects regression models were fitted separately for adolescents with time since menarche <24 mo and ≥24 mo. We also tested a 3-way interaction between time of measurement, pregnancy status, and age as a dichotomous variable. All analyses were performed using STATA version 9.0 (Stata Corp). Results are presented as means ± SD and percentages and the significance level throughout was $P < 0.05$.

This study was carried out between July 2004 and December 2006. The study was approved by the Bangladesh Medical Research Council, Dhaka, Bangladesh and the Committee on Human Research at the Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD.

Results

Of 210 pregnant adolescents who were enrolled within 16 wk of gestation, 162 (77%) who had live births completed the baseline and 12-mo assessments (Fig. 1). Among a total of 456 never-pregnant adolescents who were enrolled, 385 (83%) were measured at the 12-mo follow-up visit (Fig. 1). The girls who did not complete the study due to migration, pregnancy, and refusal were comparable to those who remained in the study by baseline characteristics (data not shown).

The age at the time of entry into the study and age at menarche of the subjects in both groups combined was 16.3 ± 1.6 y and 12.7 ± 1.2 y, respectively. All pregnant and only 9.4% of never-pregnant adolescents were married at the time of interview (**Table 1**). The height and weight at baseline did not differ between pregnant and never-pregnant adolescents (Table 1). However, TSF, upper-arm fat area, and percent body fat were higher ($P < 0.05$) and BMI and upper-arm muscle area were lower ($P < 0.05$) among pregnant compared with never-pregnant adolescents at baseline (Table 1).

Frequency of food intake did not differ between pregnant and never-pregnant adolescents, but a higher proportion of pregnant than never-pregnant adolescents reported experiencing morbidity symptoms such as painful urination or lower abdominal pain and symptoms of anemia in the month preceding the interview (Table 1). A greater proportion of never-pregnant adolescents came from households with higher SES compared with pregnant adolescents.

All pregnant adolescents (100%) who had live births and whose infants were alive reported breast-feeding at 3 and 6 mo postpartum. Among adolescents who were breast-feeding and reported the frequency of breast-feeding in the past 24 h, the median frequency was 12 at both 3 and 6 mo postpartum.

Pregnant adolescents did not gain height during the follow-up period, whereas never-pregnant adolescents increased their stature by 0.35 ± 0.85 cm/y ($P < 0.001$) (**Table 2**). Whereas

TABLE 1 Characteristics of pregnant and never-pregnant adolescents at baseline[1–3]

	Pregnant adolescents	Nonpregnant adolescents
n	162	385
Chronological age, y	16.4 ± 1.6	16.3 ± 1.6
Age at menarche, y	12.8 ± 1.2	12.7 ± 1.2
Time since menarche, mo	43.4 ± 20	43.0 ± 20
Married, %	100	9.4
Baseline anthropometry		
Height, cm	149.1 ± 5.4	149.4 ± 5.1
Weight, kg	43.1 ± 5.0	42.4 ± 5.5
MUAC, cm	23.4 ± 1.7	23.2 ± 2.0
TSF, mm	10.8 ± 3.0	11.6 ± 3.5[†]
SSF, mm	9.5 ± 2.3	9.8 ± 2.7
BMI, kg/m[2]	19.3 ± 1.7	19.0 ± 2.0[‡]
Upper-arm-muscle area, cm[2]	32.2 ± 4.2	30.7 ± 4.5[‡]
Upper-arm-fat area, cm[2]	11.8 ± 3.8	12.6 ± 4.5[‡]
Body fat, %	18.8 ± 3.6	19.6 ± 0.2[‡]
Stunting,[4] %	58.0	47.0*
Underweight,[4] %	40.1	40.1
Past 7-d dietary intake,[5] %		
Meat and eggs	26.7	22.0
Fish	54.1	52.9
Milk and dairy	34.6	28.2
Dark green leafy vegetables	8.0	9.1
Fruits	35.8	34.7
Nuts and beans	5.6	5.7
Past 30-d morbidity,[6] %		
Diarrhea or dysentery	8.7	15.6*
Productive cough or rapid breathing	24.4	24.8
Painful urination or lower abdominal pain	50.3	31.5*
Nausea or vomiting	74.5	18.8*
Hepatitis[7]	12.0	6.3*
Anemia[8]	50.9	21.0*
Past 7-d history of activities,[9] %		
Carried heavy objects (>20 kg)	14.8	13.3
Husked, ground, or pounded grain	14.2	11.4
Chopped loads of firewood	12.4	6.2*
Carried a child a long distance	4.9	12.2*
Swept the floor or courtyard	98.2	93.0*
Walked >1 h	7.4	16.9*
Washed dishes	96.9	83.1*
Cared for a child	20.4	34.6*
Cooked	96.9	66.8*
SES indicators		
Literate	74.1	87.3*
Currently enrolled in school	0	52.0
Highest grade completed (>5th grade)	65.4	83.6*
Income earning job	25.9	14.3*
Religion		
Muslim	90.7	84.4
Hindu	8.0	15.1*
Other	1.3	0.5
Wealth index (>0.5)[10]	38.3	57.1*

[1] Values are means ± SD or percent. Symbols indicate different from pregnant adolescents, $P < 0.05$: [†] Kruskal-Wallis test; [‡] *t* test; * chi-square test.
[2] Missing values existed for: weight (n = 1), BMI (n = 1), TSF (n = 1), SSF (n = 1), upper-arm muscle area (n = 1), upper-arm-fat area (n = 1), % body fat (n = 2), underweight (n = 1), meat and eggs (n = 4), fish (n = 21), milk and dairy (n = 2), fruits (n = 42), diarrhea and dysentery (n = 2), productive cough (n = 16), painful urination (n = 19), nausea and vomiting (n = 20), hepatitis (n = 7), anemia (n = 1), swept floor (n = 2), washed dishes (n = 1), cared for a child (n = 1).

1508   Rah et al.

pregnant adolescents had lower weight, BMI, MUAC, upper-arm-muscle area, and percent body fat by 6 mo postpartum, never-pregnant adolescents gained in each ponderal dimension over a 1-y period (all $P < 0.001$) (Table 2). Similarly, annual losses in TSF and upper-arm fat area were greater and gain in SSF was lower among pregnant than never-pregnant adolescents (all $P < 0.01$). All differences remained significant after adjusting for age, time since menarche, wealth index at baseline, and morbidity, dietary intake, and physical activity at baseline and at the 12-mo follow-up (**Table 3**). Annual changes in height and weight were lower among pregnant than never-pregnant adolescents by 0.43 ± 0.10 cm and 1.70 ± 0.25 kg, respectively, after adjusting for confounders (both $P < 0.001$) (Table 3).

There was a significant interaction between pregnancy status and time since menarche in their association with changes in all anthropometric measurements except height (**Fig. 2**). Differences in annual changes in anthropometric measurements were larger among adolescents whose first pregnancy occurred <24 mo since menarche compared with those who had their first pregnancy ≥24 mo after the onset of menarche, adjusting for confounders (weight, −3.35 ± 0.42 vs. −1.60 ± 0.26 kg/y; BMI, −1.40 ± 0.18 vs. −0.60 ± 0.11 kg/(m[2]·y); upper-arm muscle area, −2.53 ± 0.65 vs. −1.52 ± 0.27 cm[2]/y; percent body fat, −3.24 ± 0.42 vs. −1.24 ± 0.25 %/y; P-values for all interaction terms < 0.05) (Fig. 2). No interactions between pregnancy status and time since menarche were observed in their association with annual change in height (0.54 ± 0.25 vs. 0.40 ± 0.10 cm/y; P-value for interaction, 0.29). The difference between pregnant and never-pregnant adolescents in changes in anthropometric measurements did not differ by chronological age (data not shown).

Discussion

Few studies from developing countries have examined the consequences of premature childbearing on the growth and nutritional status of the young mother. In this prospective cohort study of 12- to 19-y-old girls in a poor undernourished rural population in Bangladesh, we assessed how pregnancy and lactation during adolescence affect linear and ponderal growth and body composition of young mothers. Pregnancy and lactation during adolescence appeared to halt linear growth and resulted in weight loss and depletion of fat and lean body mass of young girls at the end of a year of follow-up. This phenomenon was more prominent among adolescents who became pregnant at an earlier gynecological age.

Pregnant girls did not gain in stature between early pregnancy and 6 mo postpartum compared with the annual growth rate of 0.35 ± 0.85 cm/y observed among never-pregnant adolescents

TABLE 1 Continued

[3] Mean ± SD gestational age for pregnant adolescents at baseline measurement was 10.1 ± 2.8 wk.
[4] Stunting and underweight were defined as height-for-age and weight-for-age Z-scores < −2, respectively.
[5] Percentage of adolescents who consumed each food group more than 3 times in the past 7 d.
[6] Percentage of adolescents who had each morbidity symptom at least once in the past 30 d.
[7] Percentage of adolescents who had at least 1 clinical symptom of hepatitis, including ash-colored/gray stool, tea-colored urine, liver pain, jaundice, and yellow eyes.
[8] Percentage of adolescents who had at least 3 clinical symptoms of anemia in the past 30 d.
[9] Percentage of adolescents performing each work activity at least once in the past 7 d.
[10] Adolescents with wealth index score >0.5 are from higher SES.

TABLE 2 Annual changes in anthropometric measurements in adolescent girls by pregnancy status[1]

		Height	Weight	MUAC	BMI	TSF	SSF	Arm muscle area	Arm fat area	Body fat
		cm	*kg*	*cm*	*kg/m²*	*mm*		*cm²*		*%*
Pregnant,	Baseline	149.1 ± 5.4	43.1 ± 5.0	23.4 ± 1.7	19.3 ± 1.7	10.8 ± 3.0	9.5 ± 2.3	32.2 ± 4.2	11.8 ± 3.8	18.8 ± 3.6
n = 162	1-y follow-up	149.0 ± 5.4	42.3 ± 5.1[†]	22.8 ± 1.8[†]	19.0 ± 1.8[†]	9.2 ± 2.7[†]	9.5 ± 2.6	31.7 ± 4.1[†]	10.0 ± 3.5[†]	17.6 ± 3.8[†]
	(Δ)[2]	−0.04 ± 0.69	−0.76 ± 2.51	−0.58 ± 1.11	−0.34 ± 1.12	−1.46 ± 2.09	0.04 ± 1.78	−0.39 ± 2.64	−1.73 ± 2.59	−1.18 ± 2.50
Nonpregnant,	Baseline	149.4 ± 5.1	42.4 ± 5.4	23.2 ± 2.0	19.0 ± 2.0	11.6 ± 3.5	9.8 ± 2.7	30.7 ± 4.5	12.6 ± 4.5	19.6 ± 4.2
n = 385	1-y follow-up	149.7 ± 5.0[†]	43.2 ± 5.6[†]	23.5 ± 2.0[†]	19.3 ± 2.0[†]	11.4 ± 3.4	10.4 ± 3.0[†]	31.7 ± 4.5[†]	12.5 ± 4.5	19.8 ± 4.1[†]
	(Δ)[2]	0.35 ± 0.85	0.91 ± 2.43	0.30 ± 1.00	0.31 ± 1.05	−0.20 ± 2.26	0.59 ± 1.92	1.13 ± 2.33	−0.03 ± 2.76	0.26 ± 2.49
Between group difference in Δ, *P*-value		<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001	<0.001

[1] Values are means ± SD or percent. [†] Different from baseline, $P < 0.05$ (paired t test).

[2] Changes in anthropometric measurements were annualized, after being divided by the actual number of days between measurements.

of the same age and time since menarche. Although peak height velocity occurs prior to menarche, linear growth continues during late adolescence and most girls grow 5–7.6 cm after the onset of menarche (4,26). In developing countries, chronic undernutrition can delay puberty and extend the adolescent growth period beyond 20 y of age (6). Using cross-sectional data on height of rural Bangladeshi primigravidae enrolled in the JiVitA trial, we found that height increased by 0.35 ± 0.02 cm/y from 12 to 21 y, when it plateaued and remained constant (data not shown). In the present study, never-pregnant adolescents of the same age and time since menarche grew ~0.3 cm during a 1-y period. Assuming a constant height increment of 0.3 cm/y until 21 y of age, we estimate that the apparent cessation of linear growth due to early pregnancy may result in an overall decrement ranging from 0.6 to 2.7 cm in attained height among 12- to 19-y-old girls in rural Bangladesh.

Pregnant girls declined in weight and BMI by 0.76 ± 2.51 kg and 0.34 ± 1.12 kg/m², respectively, by 6 mo postpartum. Body weight and BMI of pregnant adolescents reflect maternal energy stores and, thus, weight loss indicates the depletion of maternal energy reserves to meet the demands of pregnancy and lactation (27). Pregnant adolescents lost MUAC and upper-arm muscle area, which reflects the depletion of lean body mass. On the other hand, never-pregnant adolescents in this rural population continued to gain weight, BMI, percent body fat, and upper-arm muscle area over the 1-y period.

Differences in annual changes in ponderal and body composition measures between pregnant and never-pregnant adolescents were greater among those with a shorter time since menarche.

The annual growth rate in ponderal and body composition measures of never-pregnant adolescents was higher among those who were enrolled within a shorter time since menarche (all $P < 0.05$). Adolescents who had their first pregnancy <24 mo since menarche appeared to have similar reductions in weight and BMI and slightly higher reductions in percent body fat by 6 mo postpartum compared with those whose first pregnancy occurred ≥24 mo after the onset of menarche. This implies that the depletion of maternal fat stores during pregnancy and early stages of lactation was more profound among adolescents who became pregnant at an earlier gynecological age.

Our results indicating the cessation of linear growth among pregnant adolescents correspond with the recent findings from a study conducted in Mexico City in which pregnant adolescents <18 y of age did not grow in height between the 2nd trimester of pregnancy and 1 mo postpartum, whereas never-pregnant adolescents matched for age, menarche age, BMI, and SES grew 0.94 ± 0.3 cm during the 5-mo period (28). There is evidence from developed countries suggesting that teenagers continue to grow in height during pregnancy (29). In a study from Camden, NJ, Scholl et al. (12,29) noted a tendency for most gravidas to measure slightly less in stature during pregnancy as a result of vertebral compression or weight gain. However, using knee height measurement, which is less susceptible to the shrinkage effects of pregnancy, the study found a significant increase in knee height, reflecting gains in stature among pregnant adolescents (12). In the present study, we measured maternal height not during pregnancy but at 6 mo postpartum when a rebound in maternal height is expected (10). Thus, change in height between
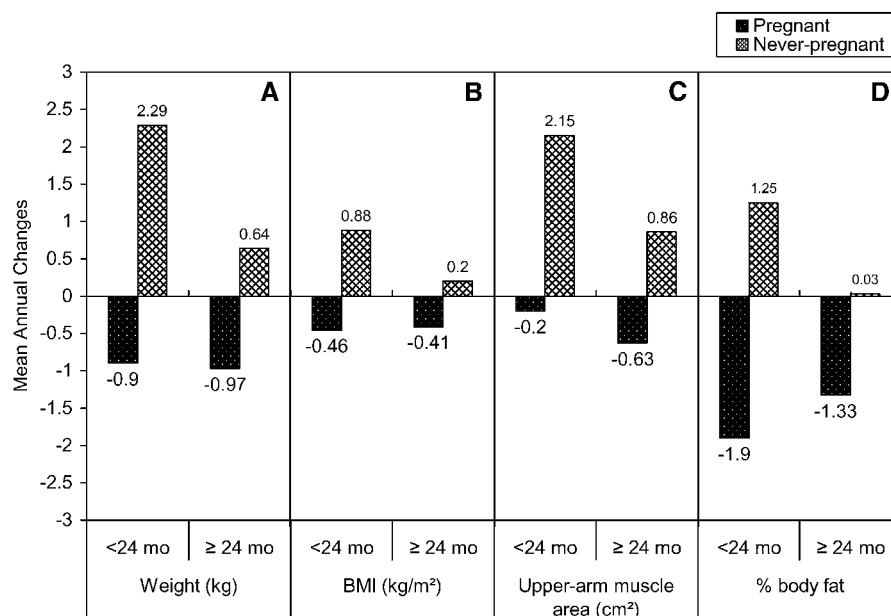
TABLE 3 Adjusted differences in annual changes in anthropometric measurements between pregnant and never-pregnant adolescents[1]

	Pregnant		Never pregnant		Differences[2]	
	Adjusted β-coefficient (SE)	*P*-value	Adjusted β-coefficient (SE)	*P*-value	Adjusted β-coefficient (SE)	*P*-value
Height, *cm*	−0.09 (0.08)	0.26	0.35 (0.05)	<0.001	−0.43 (0.10)	<0.001
Weight, *kg*	−0.94 (0.20)	<0.001	0.97 (0.13)	<0.001	−1.70 (0.25)	<0.001
BMI, *kg/m²*	−0.41 (0.09)	<0.001	0.34 (0.06)	<0.001	−0.62 (0.11)	<0.001
MUAC, *cm*	−0.76 (0.10)	<0.001	0.30 (0.06)	<0.001	−0.89 (0.12)	<0.001
TSF, *mm*	−1.69 (0.17)	<0.001	−0.18 (0.12)	0.13	−1.48 (0.22)	<0.001
SSF, *mm*	−0.03 (0.14)	0.84	0.64 (0.10)	<0.001	−0.45 (0.19)	0.02
Upper-arm muscle area, *cm²*	−0.61 (0.24)	0.01	1.08 (0.13)	<0.001	−1.16 (0.30)	<0.001
Upper-arm fat area, *cm²*	−2.13 (0.24)	<0.001	0.01 (0.14)	0.96	−1.96 (0.32)	<0.001
Body fat, *%*	−1.41 (0.20)	<0.001	0.30 (0.13)	0.02	−1.54 (0.25)	<0.001

[1] Values are mean adjusted β-coefficients (SE) and associated *P*-values.

[2] Differences in annual changes in anthropometric measurements were calculated between pregnant vs. nonpregnant adolescents using the linear mixed effects regression models adjusting for confounders.

FIGURE 2 Adjusted annual changes in weight (*A*), BMI (*B*), upper-arm muscle area (*C*), and percent body fat (*D*) by time since menarche (<24 mo vs. ≥24 mo) and pregnancy status. Annual changes were adjusted for confounders using the linear mixed effects regression models. *P*-values for all interaction terms were <0.05. There were 40 and 163 pregnant girls with time since menarche <24 mo and ≥24 mo, respectively, and 77 and 315 never-pregnant girls with time since menarche <24 mo and ≥24 mo, respectively.

early pregnancy and 6 mo postpartum in the present study is less likely to be affected by the downward bias in maternal height.

Weight loss and depletion of fat and lean body mass observed among pregnant girls in our study contrast with findings from studies in well-nourished populations in which adolescent pregnancy has been associated with large gestational weight gains (15.2 ± 0.6 kg) and postpartum weight retention (6.9 ± 0.5 kg) and continuing accrual of fat mass during late pregnancy through 4–6 wk postpartum (30,31). Adolescents appeared to continue to synthesize maternal tissues instead of mobilizing maternal nutrient stores for fetal nutritional demands in late pregnancy and early postpartum (30,31). Maternal prepregnancy nutritional status affects nutrient partitioning (13). In a well-nourished population and overnourished rapidly growing adolescent ewes, a preferential compartmentalization of nutrients to promote rapid maternal growth at the expense of the fetus has been observed (31–33) compared with both maternal weight loss and poor fetal growth that may occur among severely undernourished adolescents (13).

We assumed the burden of lactation to be similar among pregnant women, because all pregnant adolescents who had live births and whose infants were alive were breast-feeding at 3 and 6 mo postpartum. The rate of exclusive breast-feeding was low at 8% at 3 mo and 4% at 6 mo, but this can be explained by the universal practice of prelacteal feeding (>90%) in this population. Breast-feeding, however, was predominant through 6 mo of age as determined by its median frequency of 12 as determined by the 3- and 6-mo visits.

It is essential to consider why pregnancy and lactation during adolescence ceased linear growth of these young girls. One explanation may be the inadequate nutrient supply to support maternal growth due to the competition for nutrients between the teenage mother and the fetus (13). In severely undernourished adolescents, competition for nutrients between the mother and her conceptus is likely to result in both cessation of maternal linear growth and poor fetal growth. Another potential mechanism, which remains to be examined, is the acceleration of epiphyseal closure due to the increased estrogen concentration during pregnancy (34). Epiphyseal closure is normally induced by an increased secretion of estrogen during puberty (34). In pregnant adolescents, further elevation of estrogen level during pregnancy may accelerate the termination of bone growth by stimulating epiphyseal closure (34).

A few limitations to this study need to be considered. Although the present study had a 1-y follow-up period, which was longer than that of comparable studies, pregnant adolescents were measured up to 6 mo postpartum when the burden of lactation was still high. Therefore, this study was unable to determine whether a catch-up growth in stature, weight, fat mass, and muscle mass occurred after 6 mo postpartum when the frequency and intensity of lactation decrease. We did not assess energy, protein, and other macro and micronutrient intakes and our estimates or the level of physical work activity were based on the frequency of a few work activities. Information on the duration and intensity of physical activity was not collected. Future studies of energy intake and expenditure among pregnant adolescents in these malnourished settings would enhance our understanding of energy balance in this population.

In conclusion, childbearing during adolescence may hamper postmenarcheal linear and ponderal growth of young girls, a potential window of opportunity for catch-up growth in an undernourished population. The cessation of linear growth in adolescents due to an early pregnancy may result in an overall loss ranging between 0.6 and 2.7 cm in attained height in rural Bangladeshi women, which may contribute to stunting and increased obstetric risk. The depletion of maternal fat stores and lean body mass during pregnancy and an early stage of lactation, especially among adolescents who become pregnant at an earlier gynecological age, may exacerbate the outcome of future pregnancies and increase the risk of maternal morbidity and mortality. Similar studies with a longer follow-up period need to be conducted in other developing country settings, where marriage and pregnancy occur early and where undernutrition is common, to better understand the risk of early pregnancy to the young mother.

Acknowledgments

for computer programming and data management support and Andre Hackman for technical assistance.

Literature Cited

1. Bongaarts J, Cohen B. Adolescent reproductive behavior in the developing world. Introduction and Review. Stud Fam Plann. 1998;29:99–105.
2. Malamitsi-Puchner A, Boutsikou T. Adolescent pregnancy and perinatal outcome. Pediatr Endocrinol Rev. 2006;3 Suppl 1:170–1.
3. Stewart CP, Katz J, Khatry SK, LeClerq SC, Shrestha SR, West KP, Christian P. Preterm delivery but not intrauterine growth retardation is associated with young maternal age among primiparae in rural Nepal. Matern Child Nutr. 2007;3:174–85.
4. Spear BA. Adolescent growth and development. J Am Diet Assoc. 2002;102:S23–9.
5. Rogol AD, Clark PA, Roemmich JN. Growth and pubertal development in children and adolescents: effects of diet and physical activity. Am J Clin Nutr. 2000;72:S521–8.
6. Riley AP, Huffman SL, Chowdhury AKM. Age at menarche and postmenarcheal growth in rural Bangladeshi females. Ann Hum Biol. 1989;16:347–59.
7. Shahabuddin AK, Talukder K, Talukder MK, Hassan M, Seal A, Rahman Q, Mannan A, Tomkins A, Costello A. Adolescent nutrition in a rural community in Bangladesh. Indian J Pediatr. 2000;67:93–8.
8. Qamra SR, Mehta S, Deodhar SD. Physical growth in school girls: relationship to socioeconomic status and dietary intake-II. Indian Pediatr. 1990;27:1051–65.
9. Qamra SR, Mehta S, Deodhar SD. A mixed-longitudinal study on the pattern of pubertal growth: relationship to socioeconomic status and caloric intake-IV. Indian Pediatr. 1991;28:147–56.
10. Scholl TO, Hediger ML. A review of the epidemiology of nutrition and adolescent pregnancy: maternal growth during pregnancy and its effect on the fetus. J Am Coll Nutr. 1993;12:101–7.
11. Garn SM, La Velle M, Pesick SD, Ridella SA. Are pregnant teenagers still in rapid growth? Am J Dis Child. 1984;138:32–4.
12. Scholl TO, Hediger ML, Ances IG. Maternal growth during pregnancy and decreased infant birth weight. Am J Clin Nutr. 1990;51:790–3.
13. King JC. The risk of maternal nutritional depletion and poor outcomes increases in early and closely spaced pregnancies. J Nutr. 2003;133:S1732–6.
14. Pathak P, Singh P, Kapil U, Raghuvanshi RS. Prevalence of iron, vitamin A, and iodine deficiencies amongst adolescent pregnant mothers. Indian J Pediatr. 2003;70:299–301.
15. Chaturvedi S, Kapil U, Gnanasekaran N, Sachdev HPS, Pandey RM, Bhanti T. Nutrient intake amongst adolescent girls belonging to poor socioeconomic group of rural area of Rajasthan. Indian Pediatr. 1996;33:197–201.
16. Gibson RS. Principles of nutritional assessment. New York: Oxford University Press; 1990.
17. Ulijaszek SJ, Kerr DA. Anthropometric measurement error and the assessment of nutritional status. Br J Nutr. 1999;82:165–77.
18. Shorr I. How to weigh and measure children. New York: United Nations Department of Technical Cooperation and Development and Statistical Office; 1986.
19. Fleming AF, Briggs ND, Rossiter CE. Growth during pregnancy in Nigerian teenage primigravidae. Br J Obstet Gynaecol. 1985;92:S32–9.
20. Frisancho AR. Anthropometric standards for the assessment of growth and nutritional status. Ann Arbor (MI): University of Michigan Press; 1990.
21. Slaughter MH, Lohman TG, Boileau RA, Horswill CA, Stillman RJ, Van Loan MD, Bemben DA. Skinfold equations for estimation of body fatness in children and youth. Hum Biol. 1988;60:709–23.
22. CDC. CDC growth charts for the United States: methods and development. Vital and health statistics series 11, no. 246. Hyattsville (MD): National Center for Health Statistics; 2002.
23. Vyas S, Kumaranayake L. Constructing socio-economic status indices: how to use principal components analysis. Health Policy Plan. 2006;21:459–68.
24. Finucane MM, Samet JH, Horton NJ. Translational methods in biostatistics: linear mixed effect regression models of alcohol consumption and HIV disease progression over time. Epidemiol Perspect Innov. 2007;4:8.
25. Brown T, Wang Z, Chu H, Palella FJ, Kingsley L, Witt MD, Dobs AS. Longitudinal anthropometric changes in HIV-infected and HIV-uninfected men. J Acquir Immune Defic Syndr. 2006;43:356–62.
26. Roche AF, Davila GH. Late adolescent growth in stature. Pediatrics. 1972;50:874–80.
27. Adair LS, Popkin BM. Prolonged lactation contributes to depletion of maternal energy reserves in Filipino women. J Nutr. 1992;122:1643–55.
28. Casanueva E, Rosello-Soberon ME, Da-Regil LM, Arguelles M, Cespedes MI. Adolescents with adequate birth weight newborns diminish energy expenditure and cease growth. J Nutr. 2006;136:2498–501.
29. Scholl TO, Hediger ML, Cronk CE, Schall JI. Maternal growth during pregnancy and lactation. Horm Res. 1993;39 Suppl 3:59–67.
30. Hediger ML, Scholl TO, Schall JI. Implications of the Camden study of adolescent pregnancy: interactions among maternal growth, nutritional status, and body composition. Ann N Y Acad Sci. 1997;817:281–91.
31. Scholl TO, Hediger ML, Schall JI, Khoo CS, Fischer RL. Maternal growth during pregnancy and the competition for nutrients. Am J Clin Nutr. 1994;60:183–8.
32. Wallace JM, Da Silva P, Aitken RP, Cruickshank MA. Maternal endocrine status in relation to pregnancy outcome in rapidly growing adolescent sheep. J Endocrinol. 1997;155:359–68.
33. Wallace JM, Aitken RP, Cheyne MA. Nutrient partitioning and fetal growth in rapidly growing adolescent ewes. J Reprod Fertil. 1996;107:183–90.
34. Vander A, Sherman J, Luciano D. Human physiology: the mechanisms of body function. 7th ed. Boston: WCB McGraw-Hill; 1998.

THE JOURNAL OF NUTRITION

JN