Influence of growth status and placental function on birth weight of infants born to young still-growing teenagers

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ABSTRACT To investigate the determinants of fetal maturation of infants born to adolescent mothers, we studied the obstetric population attended at the Maternity Hospital of Lima, Peru. From this population we selected for inclusion in this study a sample of 412 adolescent mothers ranging in age from 13 to 15 yr. These subjects were selected because the anthropometric measurements of their parents were obtained at the time the adolescents were being attended for delivery. Based on the height measurements of the teenagers' mothers and fathers, the adolescents were classified as either still growing or growth completed depending on whether their height was less or greater than their mothers' height. Infants of young adolescent mothers who had not completed their expected growth in height were significantly lighter in birth weight than those born to adolescent mothers who had completed their growth. Multiple regression and path analyses revealed that the effects of weight gain and placenta weight on birth weight were more effective among the infants born to adolescent mothers who had not completed their growth in height. These data suggest that the reduction in birth weight among immature still-growing adolescents may result from both a decreased net availability of nutrients and/or an inability of the placenta to function adequately for active fetal growth, which results in a retarded fetal growth.

KEY WORDS Adolescent growth, maturity, teenage pregnancy, birth weight, placental function, nutritional status.

Introduction

Previous publications have reported that young adolescent mothers with the same pregnancy weight gain have smaller newborns than older mothers (1–3). In a recent study, we also found that the optimum birth weight, defined as the average population weight, among young teenage mothers (aged 13 to 16 yr) was associated with a pregnancy weight gain of about 16 kg (35 lb), while among older adolescent and adult mothers (aged 17 to 25 yr) it was associated with a pregnancy weight gain of 11 kg (24 lb). The reason for this difference is not clear. It has been postulated that among rapidly growing teenagers the nutritional requirements of pregnancy may be greater than those for adult women, and that this increased requirement competes with the growth needs of the fetus (1, 2). Therefore, among still-growing teenagers, even though they might have the same pregnancy weight gain as adult women, their fetuses might have a lesser access to nutrients than fetuses borne by women who have completed their growth. Hence, the role of growth status as a risk factor in adolescent pregnancy must be defined. With this purpose, we have evaluated the possible differences in the relationship of maternal nutritional factors to weight of infants born to adolescent mothers who have either completed or not completed their expected growth in height.

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Materials and methods

Sample

In 1980 we initiated an anthropometric and nutritional study of adolescent mothers attended at the Maternity Hospital of Lima, Peru. Each year, an average of 35,000 women are attended for delivery at this hospital. The original sample consisted of 1256 participants ranging in age from 12 to 25 yr. Details of the population from which the sample was derived have been provided in an earlier publication (3). In an attempt to understand the role of the adolescent’s relative growth status on prenatal growth, we selected those teenagers whose age at conception was less than 15 yr and whose parents’ anthropometric measurements were obtained at the time the adolescent was attended for delivery. There were a total of 424 teenagers for whom we obtained only the mother’s height and 226 teenagers for whom we obtained information on both mother’s and father’s height.

Measurements

Socioeconomic, obstetric, anthropometric, and biochemical data were obtained from all participants at the time of admission to the program.

Socioeconomic characteristics. Analysis of the socioeconomic information indicated that two-thirds of the gravidas were either single or living in common law union and the remainder were married; about 46% of the sample had elementary education and 36% had between 1 and 5 yr of schooling. About one-half of the gravidas were maids and the rest were unemployed. Less than 3% of the sample smoked, chewed coca leaves, or drank alcohol. All these cases were excluded from the present study. About 95% of the adolescent gravidas were primiparous, and for the present study only these primiparous, nonsmoking subjects were included.

Medical examination. All patients admitted to the Maternity Hospital were given a standard medical examination to determine the health status of the pregnant adolescent. All the information was recorded in the obstetric prenatal record specially designed for this study. The examination included information about previous obstetric history, past medical history, age at menarche, age at first intercourse, and a physical and pelvic examination. At this visit the medical staff also obtained information regarding height of the fundus, weight, blood pressure, position of the fetus, fetal heart rate, and any evidence of maternal edema. The subjects were examined twice, once at the 1st trimester and the second time on the day of delivery.

Biochemical data. The examination included analysis of Hb, hematocrit, serum protein, serum albumin, serum iron, total iron-binding capacity, and percentage transferrin saturation (% transferrin saturation = serum iron / total iron-binding capacity x 100).

Anthropometry. The anthropometric measurements of the mother included evaluations of height (cm), weight (kg), skinfold thickness (mm) at the triceps, subscapula, midaxilla, and calf, and circumference (cm) at the upper arm, calf, and waist. Estimates of upper arm and calf muscle area (mm²) were derived following previously indicated procedures (4–8). In addition, we obtained information on prepregnancy weight and pregnancy weight gain. For this purpose three procedures were used. First, all subjects were asked about their weight before pregnancy. Second, the weight of all subjects admitted at the 1st trimester of pregnancy was measured. Third, prepregnancy weight was estimated retrospectively using the expected weight for height tables of adult Latin American women during pregnancy (9). These reference standards assume an increase of 1.7% in weight during the first 13 wk of pregnancy and a gain of 18.3% spread uniformly over the remaining 27 wk (ie, 0.678%/wk). Therefore, by measuring either weight at the 1st trimester or weight at delivery an estimate of prepregnancy weight for height can be obtained. Comparison of the three estimates of prepregnancy weight gave almost identical values. For this reason, for each individual on whom we had information on either reported prepregnancy weight or weight at 1st trimester of pregnancy, we derived a new average prepregnancy weight that incorporated all three measurements of prepregnancy weight. Hence, the prepregnancy weight and corresponding weight gain used here are based on these corrected values.

Newborns. All newborns were examined by neonatologists and the medical staff. This included standard evaluations for Apgar scores at 1 and 5 min and Dubowitz scores following the protocol of Ballard et al (10). Scores for physical and neuromuscular maturity of newborns were separately recorded. The total Dubowitz scores were then extrapolated to the corresponding gestation age, hereafter referred to as maturation gestation age. As complementary information, gestation age was derived from information about date of last menstrual period of the mother, hereafter referred to as menstruation-based gestation age. All newborns within 6 h of birth were weighed (g) and measured for length (cm), for skinfold thickness (mm) at the triceps, subscapula, and midaxilla, and for circumference (cm) at the head, chest, upper arm, and calf. The skinfolds at the triceps, subscapula, and midaxilla were summed. These measurements were taken by a pediatrician and nurses properly trained by the authors (ARF and JM). Following the procedures of our previous studies (5), a randomly selected 10% of all adolescent mothers and their newborns were remeasured for accuracy. After delivery the placenta was weighed without draining.

Indices of teenager’s growth status

Comparison of the parental and teenager’s height showed that 59% of the teenagers had a height greater than or equal to their parents’ height. On the other hand, with the exception of six teenagers, none equalled their father’s height, and on the average the teenager’s height at delivery equalled only 92% of the father’s height. For operational purposes and for lack of a better indicator of relative growth status, we classified those teenagers whose height was either greater than their mothers’ height or greater than 92% of their fathers’ height as having completed their growth. On the other hand, those whose height was less than the mothers’ height or less than 92% of the fathers’ height were classified as still growing.
Results

General trends

Table 1 compares the birth weight of infants born to adolescent mothers who have either completed their growth or not completed their expected growth. These data show that, at the time of delivery, those teenagers who were shorter than their mothers or had attained less than 92% of their fathers’ height had significantly smaller newborns than their counterparts who were both taller than their mothers and had attained more than 92% of their fathers’ height. Since the results are quite similar when using either the mothers’ or fathers’ height, and because there were more teenagers with information on mother’s height than on father’s height, subsequent analyses are based on mother’s height data rather than the combined father’s and mother’s height.

Multivariate analyses

Multiple regression. The relationship of maternal characteristics to birth weight was evaluated using multiple regression equations in which maternal age, gynecological age, height, prepregnancy weight, pregnancy weight gain, weight at delivery, sum of skinfold thicknesses, maternal upper arm muscle area, Hb, hematocrit, serum albumin, serum protein, serum iron, and percentage serum transferrin were controlled for. Only those independent variables which were significant at the p < 0.05 level were included in the regression equations. Then, the slope (b) of each variable was separately evaluated for significant differences using analysis of covariance.

Because there is an interaction between growth status and age at menarche, to determine the interaction of maternal nutritional status and growth status these factors must be controlled. With this purpose, at each level of growth status (ie, incomplete growth and complete growth) we classified the adolescent mothers into either low gynecological age or high gynecological age categories depending on whether their gynecological age was less than or more than 2 yr. Using the same approach, multiple and stepwise regression analyses were used to determine the predictive relationship of maternal variables and birth weight.

Table 2 presents the multiple regression equations that best predict birth weight among the four groups of adolescent mothers. From these data two points are evident. First, the best prediction of birth weight in all categories includes pregnancy weight gain, gestation age, and placenta weight. Second, at each level of growth status in the adolescent mothers of high gynecological age, the regression coefficients (b1) of pregnancy weight gain are greater than in their counterparts who have not completed their growth.

As shown in Table 3, at the same level of growth status, differences in gynecological maturity are not reflected in concomitant differences in either pregnancy weight gain, gestation length, newborn weight, or placenta weight. On the other hand, differences in maternal growth status are associated with differences in birth weight, so that infants born to mothers who have not completed their growth are significantly (p < 0.05) smaller than their counterparts born to mothers who had completed their growth. Since gynecological age per se does not appear to contribute to differences in birth weight, in subsequent analysis only the ef-
TABLE 2
Comparison of the regression equations for the prediction of birth wt of infants born to young adolescent mothers (13 to 15 yr) who have either completed or not completed their expected growth in ht

<table>
<thead>
<tr>
<th>Regression equation</th>
<th>Incomplete Growth</th>
<th>Complete growth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low gynecological age</td>
<td>High gynecological age</td>
</tr>
<tr>
<td>Constant (a)</td>
<td>-1797.0</td>
<td>-2921.9</td>
</tr>
<tr>
<td>Pregnancy wt gain (b1)</td>
<td>13.4</td>
<td>52.7</td>
</tr>
<tr>
<td>Gestation length (b2)</td>
<td>112.4</td>
<td>122.4</td>
</tr>
<tr>
<td>Placenta wt (b3)</td>
<td>0.4</td>
<td>1.1</td>
</tr>
<tr>
<td>SE (g)</td>
<td>247.9</td>
<td>323.7</td>
</tr>
<tr>
<td>n</td>
<td>43</td>
<td>76</td>
</tr>
</tbody>
</table>

TABLE 3
Comparison of pregnancy wt gain, gestation length, placenta wt, and infant birth wt of young adolescent mothers (13 to 15 yr) who have either completed or not completed their expected growth in ht

<table>
<thead>
<tr>
<th>Variable</th>
<th>Incomplete growth</th>
<th>Complete growth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low gynecological age</td>
<td>High gynecological age</td>
</tr>
<tr>
<td>n</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Age at conception (yr)</td>
<td>60</td>
<td>14.2</td>
</tr>
<tr>
<td>Pregnancy wt gain (kg)</td>
<td>56</td>
<td>9.4</td>
</tr>
<tr>
<td>Gestation length (wk)</td>
<td>54</td>
<td>39.4</td>
</tr>
<tr>
<td>Placenta wt (g)</td>
<td>52</td>
<td>561.4</td>
</tr>
<tr>
<td>Newborn wt (g)</td>
<td>60</td>
<td>3001.9</td>
</tr>
</tbody>
</table>

Effects on birth weight of pregnancy weight gain, gestation length, and placenta weight are evaluated.

Path analysis. To explore the interdependence and direct influence of the three best predictors (pregnancy weight gain, gestation length, and placenta weight) of birth weight, a path analysis was carried out. Following the protocol of Li (11), all the variables were first standardized whereby each individual value is expressed as the difference between the age-specific means divided by the SD (ie, if an individual value for birth weight is 2900 and the age-specific mean and SD are 3200 ± 400, the corresponding standardized value would be 0.75). Thereafter, one-way causal models were tested by calculating path coefficients after performing consecutive and exclusive multiple regression analyses.

Figure 1 summarizes the direct and indirect path coefficients of each variable on weight of infants born to young adolescent mothers who either completed or had not completed their growth. Since the placenta is the medium whereby the maternal factors influence prenatal growth (12, 13), the path assumes that the indirect effects of pregnancy weight gain and gestation length occur through their influence on the placenta, with the placenta in turn directly affecting the newborn weight. These data show that, as expected, the effects of pregnancy weight gain on birth weight are greater among the adolescents who completed their growth than in their counterparts that are still growing (this information was also evident from the original regression analysis). The net direct effects of the independent variables on birth weight can be calculated by multiplying the path coefficient by the SD of birth weight. For example, for the adolescents who had not completed their expected growth, assuming a SD of birth weight of 400 g, an increase of 1.5 kg above the mean pregnancy weight gain of the group would result in an additional gain of only 81 g (0.203 x 400) in birth weight. In contrast, among the adolescents who had completed their growth, an increase of 1.5 kg in pregnancy weight gain would cause an increase of 122 g (0.306 x 400) in birth weight. Not evident from the regression analysis is that the contribution of gestation length to birth weight is similar in both groups of adolescents. Furthermore, not evident from the
regression analysis is the finding that placenta weight exerts a strong indirect and direct effect on birth weight of the adolescents who completed their growth, while among the still-growing adolescents placenta weight is less effective in modifying birth weight.

Discussion

The present findings demonstrate that among young adolescents (15 yr or less), prenatal growth is affected by differences in maternal growth status so that those adolescents who have not completed their expected growth have smaller newborns than their counterparts who have completed their growth. As inferred from both multiple regression and path analyses, the contribution of pregnancy weight gain toward newborn weight is less in the adolescents who are expected to continue growing than in their counterparts who have completed growth. For example, among the adolescents who have completed growth, the proportion of newborn weight to pregnancy weight gain (newborn weight/pregnancy weight gain × 100) equals about 33.05 and 31.08% for adolescents who have not completed their growth.

At present, we do not know by what physiological mechanisms adolescent mothers who are still expected to grow have smaller newborns than their counterparts who have completed their growth. There are, however, several possible explanations.

First, the reduction in birth weight might be due to a competition for nutrients between the adolescent mother and her fetus. Previous investigations on twins, siblings, parent-offspring similarities in growth, and comparative population studies (14–18) have demonstrated that the influence of genetic factors on growth in height is greater during adolescence than during childhood. Furthermore, adolescent growth is characterized by an extreme variability in the age of growth spurt onset and height growth termination. Although about 95% of the mature height is attained by menarche, a great deal of variability occurs in the amount of growth afterward. For example, the US Fels longitudinal study showed that the increase in height after menarche of those in
the 10th percentile of height for age was about 4.3 cm. On the other hand, the increase in the height of those in the 90th percentile for the same study was about 10.6 cm (19). In terms of nutritional requirements, one would expect from these data that a pregnant teenager who had not completed her growth would have higher nutritional needs than the young woman who had attained her mature height. Furthermore, in view of the prevalent influence of the genetic component during adolescence one would expect that pregnant teenagers who have not yet reached their adult statures would be actively growing. Thus, unless she had a higher nutritional intake, the pregnant teenager who had not completed her growth would end up competing for nutrients with her own fetus. Applying this concept to the present study, it is likely that the adolescents who were both physically (growth completed) and gynecologically (gynecological age > 2 yr) mature at the time of pregnancy would be spared the taxing influence of pregnancy and so produce larger babies. On the other hand, those individuals who were both physically (growth not yet complete) and gynecologically (gynecological age < 2 yr) immature at the time of pregnancy would attempt to fulfill their genetic growth potential even at the expense of their own fetuses' growth, resulting in low birth weight.

Second, the reduction in birth weight might reflect an inability of the immature mother to deliver nutrients to the fetus. The path analysis (fig 1) indicates that the contribution of the placenta weight on newborn weight is enhanced among the adolescents who have completed their growth as compared to those who are still growing. Since placental size does not limit fetal growth, the greater contribution of placenta weight to newborn weight indicates that the reduction in birth weight among immature adolescents is related to differences in placental function rather than in placental size per se. Because uteroplacental blood flow is the main regulator of placental growth (12, 13), it is quite possible that among the adolescents who had not completed their expected growth the uteroplacental blood flow was reduced. Although we do not know the actual mechanism, how this occurs can be inferred from animal experimental studies. In vitro studies of the placenta have demonstrated that while malnutrition does not interfere with the rate of α-amino isobutyric acid uptake, it does reduce the rate of placentental blood flow (20). Furthermore, measurements of cardiac output and organ blood flow using radioactive microspheres in anesthetized control and food-restricted rats have demonstrated that malnourished mothers do not expand cardiac output and placentental blood to the same extent as well-fed mothers (21). Applying these findings to the present study would imply that the fetal growth-retarding effects of the maternal-fetal competition for nutrients in the immature still-growing adolescent occur through a deficiency of placentental function. These findings together would suggest that the reduction in birth weight among immature still-growing adolescents may result from both a decreased net availability of nutrients and a deficient placentental function that causes a reduction of fetal growth.

However, because US teenagers lack a postpregnancy growth (2) and since the low birth weight is more a function of prematurity than of prenatal growth retardation, the present findings cannot be extrapolated to all populations.

References