Early Childhood Determinants of Age at Menarche in Rural Guatemala

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ABSTRACT The influence of early childhood determinants on age at menarche was investigated in a sample of Guatemalan women who participated as children in a nutrition intervention study conducted from 1969 to 1977. Age at menarche was retrospectively estimated in 1991 and 1992. Mean age at menarche was 13.7 (±1.3) years. Data on linear growth, diarrhea and respiratory illnesses, and energy intake from supplementation as well as home sources were available between birth and 7 years of age. Socioeconomic status (SES) data were collected in 1975. Four hundred and ninety-seven women who had reached menarche by 1992 were grouped into three categories of stunting based on their height-for-age z-scores (none, >-2.0; moderate, -2.0 SD to -3.0 SD; severe, <-3.0 SD relative to National Center for Health Statistics reference data) at 3 years of age. About 78% of the sample was moderately or severely stunted at 3 years of age. The group that was severely stunted in childhood reached menarche at 14.1 ± 1.4 years, significantly later than those who were moderately stunted (13.7 ± 1.2 years) or not stunted (13.5 ± 1.3 years). Using multiple linear regression methods, stunting was a significant predictor of age at menarche. Average energy intake (kcal/d) from home diet was associated with earlier menarche independent of preschool growth status. Percent time ill with diarrhea was positively associated with age at menarche. When the effects of diet, supplement, percent time ill with diarrhea and respiratory illnesses, and SES were taken into account, the independent influence of stunting on age at menarche persisted and remained significant.

Poverty and poor nutrition in infancy and early childhood cause growth stunting and later maturation. Socioeconomic status (SES) influences the timing of menarche, with better-off females attaining menarche earlier than those of lower social class (Attallah et al., 1983; Bielicki et al., 1986; Charzewska et al., 1976; Chowdhury et al., 1977; Eveleth and Tanner, 1991; Rona et al., 1986; Tan-Boom et al., 1983; Uche and Okorafor, 1979). Reported differences between social classes range from few months to about 2 years.

The majority of studies of the determinants of menarche have been cross-sectional in design, with characteristics assessed at adolescence or later. Prospective studies of the effects of early childhood factors are limited because of the high cost and logistic problems of collecting data over a long time span. Available information is mostly limited to retrospective investigations of children who were severely malnourished in early childhood and/or adopted and relocated to a developed country.

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A history of severe undernutrition was associated with a delay of 1.2 years in menarche among Barbadian girls who were hospitalized during their first year of life compared with nonhospitalized girls (Galler et al., 1985). Girls diagnosed with kwashiorkor as young children experienced menarche 1.3 years later than girls without a history of malnutrition, while those with a history of marasmus were 0.3 years later (Galler et al., 1987).

In a study from Hyderabad, India, Satyanarayana and Naidu (1979) reported that girls with a severe height deficit (<-4.0 SD using Harvard reference data) at 5 years of age attained menarche at a significantly later age than those who were short (-3.0 to -4.0 SD), mild to moderately stunted (-2.0 to -3.0 SD) or not stunted (>-2.0 SD) at this age. The mean ages at menarche were, respectively, 15.2, 14.9, 14.1, and 13.7 years for these groups.

Improvements in the quality of life during early childhood were associated wtih earlier menarche and accelerated pubertal growth among Indian and Bangladeshi girls adopted during childhood (between 3 and 72 months) by Swedish families compared to Swedish and affluent Indian girls (Adolfsson and Westphal, 1981; Proos et al., 1991a,b). Mean age at menarche for the adopted girls was 11.6 years, or about 3 years earlier than rural Indian females and about 2 years earlier than urban Indian females. The value was, in fact, lower than 13.0 years observed for Swedish girls (Hagman et al., 1986).

In summary, severe undernutrition and poor standards of living are associated with later age at menarche in several societies. Little is known, however, about the causal mechanisms by which these conditions in early childhood delay menarche.

The purpose of this study was to examine the association of growth stunting in early childhood with age at menarche while considering various nutritional, health, and environmental determinants. The overall hypothesis tested was that linear growth stunting summarizes the impact of health, nutritional, and environmental factors during early childhood, and is the most important nutritional status predictor in early childhood of age at menarche.

MATERIALS AND METHODS

Study population

Data for this study are drawn from a longitudinal, community-based nutritional intervention study carried out between 1969 and 1977 in rural Guatemala and from data collected on the same study subjects in 1991 and 1992. The intervention study was conducted by the Institute of Nutrition of Central America and Panama (INCAP) and was designed to test the hypothesis that improved nutrition in early childhood results in accelerated physical growth and mental development. The study populations were of Spanish and Amerindian heritage and Spanish speaking. Four villages (two large and two small) were randomly selected to receive either a high energy, high protein dietary supplement called Atole (163 kcal and 11.5 g per cup of supplement or 180 ml), or a low energy, no protein supplement called Fresco (59 kcal per 180 ml). Both supplements contained equal amounts of vitamins and minerals. Consumption was ad libitum and was measured carefully on a daily basis, but only for children between birth and 7 years of age. In addition, preventive and curative health services were offered in all four villages. More details about the intervention study are given elsewhere (Martorell et al., 1995a).

Reproductive histories of 976 women residents of the study villages born between 1962 and 1977 were collected in two cross-sectional rounds in 1991 and 1992. For the current analysis, subjects who were former participants of the longitudinal intervention study (excluding 144 in-migrants) and for whom there were growth, skeletal maturation, diet, and health data from birth to 3 years of age are presented. The final sample consisted of 497 subjects who had data on preschool height and had reached menarche.

Age at menarche

Menarche data were collected retrospectively in two rounds in 1991 and 1992 (Khan et al., 1995). During each round, age at onset of menarche was recalled by the year and month of occurrence. Comparison of the reported ages at menarche for subjects giving year and month in both rounds ($n = 213$) revealed inconsistencies in menarche of more than 1 month in 117 subjects. When differences were between 1 and 24 months, values were averaged and included in the analyses ($n = 115$); two cases with differences of more than 24 months were excluded. If age at menarche was missing or not recalled in one of the two rounds, the nonmissing value was selected ($n = 223$). When a month was not reported in either round but the reported year was the same, menarche was assumed to have occurred in mid-year and an additional 6 months was added to the reported age (n = 21). For cases for which years differed by 1 or 2 but months were not given, the 1991 record for that year was selected and an additional 6 months was added to the reported year (n = 40). Twenty-six subjects failed to report any information on menarche or could not remember the date of the event in either round.

Determinants of menarche

Diet and infection are major determinants of poor growth during childhood in less developed countries. Data on supplement intake, diet, anthropometry, morbidity, and skeletal maturation were routinely collected for children less than 7 years of age. Data on SES of the household were collected in 1975.

Supine length was measured every 3 months on each subject for the first 2 years, at 6-month intervals between 2 and 4 years, and at 12-month intervals between 4 and 7 years of age. Lengths were measured to the nearest 0.1 cm; statures were not measured. To make these data comparable to international stature (standing height) reference data (National Center for Health Statistics, NCHS, Hamill et al., 1977), 1.0 cm was added to supine lengths after 2 years of age. The term "height-for-age" is used because of its familiarity and conventionality.

Height-for-age is a summary measure of dietary and disease experience. Descriptive analyses indicated that height-for-age z-scores declined significantly throughout the first 2 years but remained virtually constant after about 30 months (Martorell et al., 1995b). Height-for-age z-scores at later ages are thus satisfactory as measures of achieved status. For this analysis, height-for-age z-score at 3 years of age (HAZAT3) was used if available (N = 324), or when not available, it was calculated using regression estimates with data taken at 30, 42, 48, or 60 months of age.

Morbidity data on diarrhea and respiratory illnesses were obtained every 2 weeks by maternal or caretaker recall. For these analyses, percent time ill with each illness was calculated as the number of days ill over the total days at risk from 3 months to 3 years of age. Only children for whom at least 360 days of data were available during this time were included. Regression parameter estimates of the impact of morbidity on age at menarche were calculated as per 100 days ill.

Data on dietary supplement intake were gathered daily from birth to 7 years of age. A 24-hour dietary recall was conducted to gather information on home intakes every 3 months between 15 and 36 months of age and at 42, 48, 60, 72, and 84 months of age. Supplementation intake data were analyzed as average caloric intake from supplement ingested, regardless of type of supplement (Atole or Fresco). Average daily intake of energy (kcal/d) from dietary supplement was estimated for children with at least 360 days of information from birth to 3 years of age. Average daily energy (kcal/d) from home diet was calculated for subjects with information on at least 360 days between 15 and 36 months of age. Regression parameter estimates of the impact of energy intake on age at menarche, either from supplement or home diet, were calculated as per 100 kcal/d.

Maturation status at 3 years of age (NOCSAT3) was defined as the number of ossification centers of the left hand and wrist assessed from radiographs. Data on skeletal maturation were gathered every 6 months for the first 48 months of age and yearly thereafter until 7 years of age. Raw data on the number of centers at each age were transformed into internal z-scores, dividing the differences between each subject's raw value and the mean value for the whole sample at each age interval by their respective standard deviations for that age interval. Descriptive analysis indicated the number of centers increased slowly from birth to 18 months and then increased rapidly between 24 and 48 months until the maximum 28 centers were ossified. Maturation status at age 3 years (NOCSAT3) was derived from the internal z-scores of number of ossification centers at 36 months, and when not available (N = 25), values at ages between 24 and 48 months were used, based on regression estimates.

A summary variable for SES was created using principal components analysis based on household characteristics and an inventory of household possessions in 1975; as such, the score measured accumulated wealth within the household. The SES score was normalized with a mean of 0 and a SD of 1 (Rivera et al., 1995).

In summary, early childhood determinants included height-for-age z-score, percent time ill with diarrhea and respiratory infections, average daily energy (kcal) from supplementation, average daily energy (kcal) consumed from home diet, and an indicator of skeletal maturity.

Statistical analysis

The analysis was carried out in two steps: first, the association between growth retardation and menarche was examined; next, this association was examined while simultaneously considering other independent variables. For the first step, a one-way analysis of variance (ANOVA) was performed to compare mean age at menarche among three groups of subjects who had preschool height measurements (n = 497). The subjects were categorized into three groups based on height deficit at age 3 years. Subjects who were above −2.0 SD of the NCHS reference for height-for-age were considered "not stunted." Subjects who had height values between −2.0 and −3.0 SD or below −3.0 SD were considered as having moderate and severe growth stunting, respectively.

To estimate the magnitiude of the effect of growth stunting on menarche, multiple regression analysis was used with the subset of data with complete information on all variables of interest (n = 250). In the first model, growth stunting (HAZAT3) as a continuous variable was entered in an OLS regression with age at menarche as the dependent variable. The influence of growth stunting on age at menarche, controlling for diet, morbidity, and SES variables, was then tested in several multiple linear regression models. $P \leq 0.05$ was considered statistically significant. Data analyses were completed using SAS-PC software of version 6.04 (SAS institute, 1990).

TABLE 1. Mean ages at menarche (years) according to childhood growth stunting

Degree of stunting[1]	Sample size	Age at menarche Mean	SD	ANOVA difference*
Severely stunted	188	14.1	1.4	A
Moderately stunted	199	13.7	1.2	B
Not stunted	110	13.5	1.3	B
All groups	497	13.7	1.3	

[1]Height-for-age z-scores (HAZ) were used; severe if HAZ <= −3.00; moderate if −2.00 >= HAZ < −3.00; and not stunted if HAZ > −2.00.
*The groups with dissimilar letters were significantly different at $P < 0.05$.

TABLE 2. Descriptive statistics for predictor variables for subjects with complete information (N = 250)

Variable	Mean	SD	Range
Age at menarche (years)	13.8	1.3	11.0–17.5
Supplement (kcal/day)	76.1	78.0	1.7–357.3
Home diet (kcal/day)	365.5	141.6	53.0–1017.4
Diarrhea[1]	3.9	4.5	0.0–33.0
Respiratory[1]	17.9	15.6	0.0–65.6
SES in 1975	−0.1	0.9	−1.5–2.7
NOCSAT3[2]	20.0	4.2	2.0–27.0
Height-for-age at 3	−2.8	1.1	−7.0–−0.6

[1]Percent time ill with diarrhea or respiratory illness.
[2]Number of ossification centers at age 3 years.

RESULTS

Effect of childhood nutritional status

The overall mean age at menarche was 13.7 ± 1.3 years (n = 497; Table 1). Age at menarche differed by stunting category with values of 14.1 ± 1.4, 13.7 ± 1.2, and 13.5 ± 1.3 years for severe, moderate, and not stunted subjects, respectively. A post hoc Duncan multiple range test showed that the severely stunted group had significantly later menarche compared to the moderately stunted and nonstunted groups (ANOVA, $P < 0.003$).

Effects of other early childhood determinants

To investigate the effects of early childhood determinants, in addition to childhood growth stunting, on menarche, only the 250 subjects who had complete information on all variables were included. Summary statistics of these variables are presented in Table 2. The mean age at menarche for this sample was 13.8 ± 1.3 years which is nearly identical to the mean from the larger sample (Table 1). The percentages of severely, moderately, and not-stunted preschool children were 38, 40, and 22%, respectively, identical to the percentages found in the larger sample. Mean energy intake from supplementation was 76 kcal/d with a range of 2–350 kcal/d, while energy from home diet was 365 kcal/d with a wide range of 53–1,017 kcal/d.

Table 3 shows the bivariate associations of HAZAT3 and age at menarche with energy intake from supplement and home diet, morbidity, maturation, and SES. Height at 3 years of age (HAZAT3) was correlated with energy intake from supplement and home diet as well as SES. The association of HAZAT3 and skeletal maturation status at age 3 years (NOCSAT3) was also significant ($r = 0.61, P < 0.0001$). For age at menarche, the correlations with both HAZAT3 and energy consumed from home diet were significant and negative. None of the other vari-

TABLE 3. Correlations (r) of height-for-age at 3 years (HAZAT3) and age at menarche with supplement, home diet, morbidity, maturity, and SES

Variable	Height-for-age at 3 years (HAZAT3)	Age at menarche
Supplement (kcal/day)	0.32***	−0.01
Home diet (kcal/day)	0.21***	−0.19**
Diarrhea[1]	0.002	0.11
Respiratory[1]	0.09	0.03
NOCSAT3[2]	0.61***	0.09
SES in 1975	0.20***	0.02
HAZAT3		−0.15*

[1]Percent time ill with diarrhea or respiratory illness.
[2]Number of ossification centers at age 3 years and expressed as z-scores created internally.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$ (N = 250).

ables were significantly correlated with age at menarche.

The results of three separate regression models with age at menarche as the dependent variable and the independent variables of interest are presented in Table 4. In model 1, growth stunting at age 3 years (HAZAT3) was significantly associated with age at menarche. As hypothesized, girls who were growth stunted during early childhood reached menarche at a later age. A decrease of 1 SD in childhood height-for-age resulted in a 0.17 year (about 2 months) increase in age at menarche.

Including diet and illness histories in model 2, the effect of HAZAT3 was only slightly attenuated. Energy (kcal/d) consumed from home diet during early childhood was inversely associated with age at menarche; girls with higher energy intake reached menarche significantly earlier than girls with lower energy intake. The percent time ill with diarrhea during early childhood approaches statistical significance ($P < 10$) as an independent predictor of age at menarche.

Model 3 included all variables used in model 2 plus SES. The inclusion of SES in the model increased the significance of the independent effect of HAZAT3 on age at menarche to a magnitude approximately the same as that found in model 1. On the other hand, the effect of SES on age at menarche was not significant. The magnitude and significance of the other variables included in model 3 were nearly indentical as in model 2. In sum, the regression coefficients for HAZAT3 were negative and of nearly the same magnitude in all three models.

DISCUSSION

This study demonstrates that linear growth retardation during early childhood is associated with later age at menarche. In the bivariate analysis, subjects who were severely stunted (height-for-age z-score ≤ −3 SD) at 3 years of age reached menarche at a significantly later age than their moderately (−2 to −3 SD) or not stunted (> −2 SD) counterparts; the mean ages at menarche for the three groups were 14.1, 13.7, and 13.5 years, respectively. Multivariate models, in which childhood illness, dietary energy intakes from supplement and at home, and SES were controlled confirmed the hypothesis that growth stunting at age 3 years has a significant, independent effect on age at menarche (Table 4).

The coefficient of determination of the regression model was low ($R^2 = .05$), but the fit of the models was highly significant. One possible explanation for the low R^2 values might be errors in the estimation of age at menarche (recall bias). The independent variables, on the other hand, were collected prospectively or by direct observation (SES) and were measured more reliably. Low R^2 values are observed frequently in epidemiological studies where dependent variables are commonly of a complex, multi-causal nature; an example is an analysis of cancer rates as a function of diet and lifestyle variables. In these studies, as in the present study, the usual aim is to identify key determinants and to infer causality. In contrast, when the aim of the study or analysis is prediction of a variable, e.g., percentage body fat, from a set of independent variables, the validity of the exercise depends on obtaining high R^2 values. Such a use was noted in this paper, namely the prediction of height-for-age z-score at 3 years from values at 30, 42, 48, and 60 months; the R^2 for the regressions were all above 80%.

Average energy (kcal/d) consumed from home diet was a significant predictor of age at menarche even with growth stunting at age 3 and the other covariates in the models. This finding was unexpected in that it was assumed that the majority of the effect of dietary intake during early childhood on menarche would be captured by the height-for-age variable. The only other studies linking energy intake and age at menarche are from industrialized countries and intakes were recorded just 1–3 years prior to menar-

TABLE 4. Regression analysis of the early childhood factors on age at menarche

Variable	Model 1 B ± SE	Model 2 B ± SE	Model 3 B ± SE
HAZAT3	−0.17 ± 0.07**	−0.14 ± 0.08*	−0.16 ± 0.08**
Supplement (100 kcal/d)		0.06 ± 0.11	0.07 ± 0.11
Home diet (100 kcal/d)		−0.16 ± 0.06***	−0.16 ± 0.06***
% Diarrhea		0.04 ± 0.02*	0.04 ± 0.02*
% Respiratory		0.00 ± 0.01	0.00 ± 0.01
SES in 1975			0.08 ± 0.09
Constant	13.46 ± 0.21	13.93 ± 0.36	13.88 ± 0.37
Adjusted R^2	0.02	0.05	0.05
df	(249)	(245)	(244)

*P = 0.10, **$P < 0.05$; ***$P < 0.01$.

che (Kissinger and Sanchez, 1987; Maclure et al., 1991; Meyer et al., 1990). When analyses were done, excluding average intake from home diet from model 2, the magnitude of the effect of growth stunting on mean age at menarche was increased (i.e., from b= −0.14 to b= −0.21).

One possible biological explanation of the importance of energy intake from the home diet in the present study is that it affects body composition (e.g., relative body fat, lean body mass, and skinfold thicknesses) in adolescence, which, in turn, may be related to endocrinological responses which affect age at menarche. However, this explanation appears unlikely because energy from the supplements, which was measured more precisely than home diets, was not related to menarche (Table 4, models 3 and 4). Indeed, extensive analyses of exposure to the supplements failed to identify an impact on menarche (Khan et al., 1995). Alternately, the home diet measured throughout the preschool years may serve as a proxy for diet quantity and/or quality during later growth periods, including adolescence.

Controlling for SES in the complete model (model 3) did not significantly alter the magnitude or significance of the parameter estimate for stunting at age 3 years on age at menarche. One explanation for the lack of association between SES and age at menarche may be that nutritional status (HAZAT3) is closely related to SES (Table 3) and that any effect of SES on menarche is fully mediated through nutritional status. Other possible explanations are that variation in SES is insufficient or the factors used to estimate SES in the sample are inadequate to capture its effect on age at menarche.

The number of ossification centers at 3 years of age (NOCSAT3) was not included in either model 2 or 3 because of its high collinearity with height-for-age z-score at 3 years of age.

In conclusion, the prevalence of linear growth stunting in early childhood (at age 3 years) was high in this population and was due to deficient diets, high rates of illness, and poor SES. Women who were severely growth stunted during early childhood (i.e., z-score values 3 SD or more below the reference mean) reached menarche about 6 months later than those who were less stunted (i.e., z-score values above −3 SD). Therefore, growth stunting at 3 years of age is a good summary of nutritional insults and socioeconomic conditions present during early childhood, and affects age at menarche. Future research should attempt to determine if early childhood undernutrition affects age at menarche by altering the pattern of adolescent growth and/or body composition.

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