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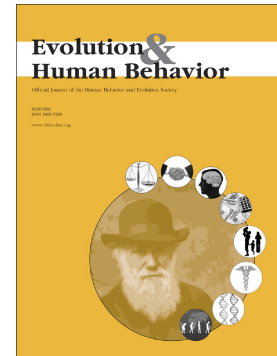
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Menarcheal timing is accelerated by favorable nutrition but unrelated to developmental cues of mortality or familial instability in Cebu, Philippines

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Abstract

Understanding the determinants of pubertal timing, particularly menarche in girls, is an important area of investigation owing to the many health, psychosocial, and demographic outcomes related to reproductive maturation. Traditional explanations emphasized the role of favorable nutrition in maturational acceleration. More recently, work has documented early maturity in relation to markers of familial and environmental instability (e.g. paternal absence), which are hypothesized to serve as cues triggering adaptive adjustment of life history scheduling. While these studies hint at an ability of human females to accelerate maturity in stressful environments, most have focused on populations characterized by energetic excess. The present study investigates the role of developmental nutrition alongside cues of environmental risk and instability (maternal absence, paternal absence, and sibling death) as predictors of menarcheal age in a well-characterized birth cohort born in 1983 in metropolitan Cebu, the Philippines. In this sample, which was marked by a near-absence of childhood overweight and obesity, we find that menarcheal age is not predicted by cues of risk and instability measured at birth, during childhood and early adolescence, but that infancy weight gain and measures of favorable childhood nutrition are strong predictors of maturational acceleration. These findings contrast with studies of populations in which psychosocial stress and instability co-occur with excess weight. The present findings suggest that infancy and childhood nutrition may exert greater influence on age at menarche than psychosocial cues in environments characterized by marginal nutrition, and that puberty is often delayed, rather than accelerated, in the context of stressful environments.

1. Introduction

There is extensive variation in the timing of puberty across human populations, raising questions about the causes of these differences (Eveleth & Tanner, 1990). To date, most studies investigating the determinants of human maturational tempo have focused on female age at menarche, or first menses, which provides an objective, directly comparable marker. This work has demonstrated a roughly four to five year acceleration in age at menarche in northern European countries with good historical records, from a mean of approximately 17 years of age in the mid 1800s to a recent mean of 12.5 in wealthy nations (Konner, 2010; Marshall & Tanner, 1969; Parent, Teilmann, Juul, Skakkebaek,Toppari, & Bourguignon, 2003). A historical acceleration in age at menarche has similarly been documented in many lower and middle-income countries, and earlier menarcheal age is also characteristic of individuals of higher socioeconomic status within populations (Parent et al., 2003). Traditionally, these findings have been interpreted as a reflection of improvements in nutrition and control of common early life infections, which led to a more rapid pace of growth and an earlier onset of puberty and reproductive maturation (Eveleth & Tanner, 1990; Kuzawa & Bragg, 2012; Parent et al., 2003).

In contrast to this nutrition-focused work, a largely separate line of investigation within the psychological and anthropological literatures has borrowed principles from evolutionary theory to posit a link between earlier maturity and various forms of psychosocial adversity (Ellis & Boyce, 2008; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999). Following the proliferation of research on paternal absence and menarche, Draper and Harpending (1982) originally proposed that daughters raised without their fathers present might learn to view paternal investment as non-essential to child rearing—thus leading to an earlier age at first sex

and subsequent earlier age at first reproduction. Belsky, Steinberg, and Draper (1991) expanded this model to include age at menarche.

More recent adaptations of these ideas borrow from life history theory (LHT), a branch of evolutionary theory developed to explain the extensive variation in life cycle timing and reproductive strategies across mammalian taxa (Stearns, 1992). Within this framework, the timing of reproductive maturity is considered a key developmental decision point, since it is the transition at which an organism's priorities shift from growth to reproduction (Charnov, 1991; Hill & Kaplan, 1999). Because high extrinsic (unavoidable) mortality increases the risk of dying before reproducing and leads to discounting of future fitness, prominent LHT models assume that mortality is a key determinant of variation in the optimal age of maturity across species (e.g. Charnov, 1991; Promislow & Harvey, 1990). All else equal, higher extrinsic mortality is expected to favor a "faster" life history strategy characterized by earlier age at maturity and a faster pace of reproduction, with consequent trade-offs in somatic maintenance and a shortened lifespan.

During the past few decades, these principles have been used to help explain plasticity-derived variation in maturational tempo in humans (Chisholm, 1993; Ellis, 2004). Specifically, organisms experiencing cues reflecting high local mortality rates, or other related dimensions of environmental instability, are predicted to respond by accelerating maturity and adopting a fast life history strategy. Support for this hypothesis comes from studies showing that early maturity is predicted by developmental experiences that are consistent with ecological harshness (e.g. low socioeconomic status, impoverished surroundings, and trauma) or environmental unpredictability (e.g. stochastic parental investment or paternal absence during childhood) (Anderson, 2015; Belsky, Schlomer, & Ellis, 2012; Boyce & Ellis, 2005; Chisholm, Quinlivan, Petersen, & Coall,

2005; Coall & Chisholm, 2003; Ellis, 2004; Moffitt, Caspi, Belsky & Silva, 1992; Quinlan,

2003; Wilson & Daly, 1997). As a particularly salient cue of harshness, experience of a

heightened mortality risk—such as through the death of a parent or sibling—have also been

shown to predict accelerated maturation and earlier onset of reproduction (Chisholm, 1993; Ellis,

Figueredo, Brumbach, & Schlomer, 2009). These findings have been interpreted as evidence that

individuals can selectively adopt a "faster" life history when local conditions signal an uncertain

or predictably harsh future (Ellis et al., 1999; Wilson & Daly, 1997; Ellis, 2004; Belsky,

Schlomer, & Ellis, 2012).

Although many studies generally support the expectations of this LHT-inspired model, it

is notable that the majority of studies investigating links between psychosocial stress,

environmental risk, and pubertal timing have focused on populations living in high income, low-

fertility countries in which the maturation-accelerating effect of over-nutrition and overweight

are common (Anderson, 2015; Sohn, 2017). In these higher income populations, lower

socioeconomic status, social stress, and environmental instability tend to co-occur with a high-

calorie environment, and greater risk of overweight and obesity, such as seen in the United States

and parts of Western Europe (see Moffitt et al., 1992; Ellis et al., 1999). From the perspective of

human evolution, the co-occurrence of caloric abundance alongside stress related to ecological

harshness is likely a recent phenomenon unique to the rise of wealthy countries with pronounced

income inequality; such environments, in which high psychosocial stressors may occur in the

absence of energetic growth constraint, have the potential to inflate the apparent importance of

developmental stressors as accelerators of maturational timing (Kuzawa & Bragg, 2012). It is

plausible that certain indicators of environmental unpredictability, such as paternal absence or

instability, did co-occur with sufficient nutrition in the evolutionary past (Ellis et al., 2009;

Marlowe, 2003; but see Quinlan, 2007). However, although relatively few, studies in populations faced with less abundant nutrition generally find that psychosocial stress and familial instability are not predictive of earlier maturity for females (Anderson, 2015; Sheppard, Snopkowski, & Sear, 2014; Sohn, 2017), raising doubts about the applicability of this model beyond contemporary high income societies.

Building upon this work, here we examine experiences of early life psychosocial stressors as predictors of maturational tempo in a population in which cues of harshness and unpredictability do not co-occur with nutritional abundance and excess weight, as is more common in many higher income countries. Data come from the Cebu Longitudinal Health and Nutrition Survey (CLHNS), a birth cohort study located in Metropolitan Cebu, the Philippines, that has followed a large group from birth into middle adulthood. In this cohort, stunting was present in 69% of the rural and 60% of the urban children by two years of age (Adair & Guilkey, 1997), compared to a recent high (in 2002) of only 3.9% in the United States (UNICEF, 2013). In the year that the study began (1983), gross domestic product (GDP) per capita in the Philippines was $645, compared to $15,531 in the United States (World Bank, 1983), and overweight and obesity were nearly absent at Cebu during adolescence (Kuzawa, Adair, Avila, Cadungog, & Le, 2003). Among men in Cebu, early life cues of mortality and familial instability including maternal absence, paternal instability, and sibling death did not predict maturational tempo, which instead was strongly accelerated in relation to favorable developmental nutrition (Gettler, McDade, Bragg, Feranil, & Kuzawa, 2015). Building on this work among Cebu males, and prior work on the role of growth patterns as predictors of pubertal timing in Cebu women (Adair, 2001), here we evaluate the role of familial instability and cues of mortality as predictors of menarcheal age against a backdrop of minimal overweight during childhood and adolescence.

2. Methods

2.1 Study Design

The Cebu Longitudinal Health and Nutrition Survey (CLHNS) is a population-based longitudinal survey ongoing since 1983 (Adair et al., 2011). A total of 3,327 pregnant women who gave birth in 1983-1984 in 33 randomly selected urban and rural *barangays* (smallest administrative villages) were recruited and 3080 of these women had singleton newborns. Information on mother and child were collected by interviews with mothers conducted immediately after birth and every two months during the infant's first two years of life (Adair et al., 2011). The analyses reported here primarily use data collected at the child's birth, at 12 and 24 months of age, at 8.5 years, and at 11.5 years. In addition, participants reported whether they had experienced menses, and if so at what age, during interviews conducted at 11.5, 14.5, 19.5 and 22 years of age. Additionally, we use data on child anthropometrics, childhood nutrition, and the social and economic characteristics of the household. We focus on 807 daughters who had all necessary variables. There were no significant differences between the analysis sub-sample and the full sample in terms of household assets or income, parental age or education, birth order or educational levels (all $p>0.1$). The data reported here were collected under conditions of written informed consent with Institutional Review Board oversight by the University of North Carolina, Chapel Hill.

2.2 *Data Collection*

Anthropometric data were collected using standard techniques (Lohman, Roche, & Martorell, 1988). Birth weight was recorded immediately after birth by birth attendants (see Adair et al., 2011). Gestational age was calculated from the mother's last menstrual period; if pregnancy complications occurred or if infant birth weight was less than 2.2 kg, trained nurses

obtained Ballard scores. Birth weight was adjusted for gestational age at birth to capture variation in fetal growth rate, rather than differences in birth size due to variation in gestational duration. Birth weight and weight at the 2 year study visit were used to calculate infancy growth velocity during the first 2 years of life. A food frequency questionnaire survey for children was carried out in 1991 and caloric intake was estimated from these data using the Philippines Food Composition Tables developed by the Food and Nutrition Research Institute (FNRI, 1990). Body mass index (BMI) at age 8.5 years was calculated as weight (kg)/height (m^2). Age at menarche for daughters was obtained from self-reported month and year of first menstrual period assessed during periodic surveys during adolescence and early adulthood. The mother's age at menarche was obtained by recall during the baseline survey in 1983.

Following analyses in Cebu males (Gettler et al., 2015), girls were classified as experiencing "paternal instability" if their father was deceased or absent up to age 11.5 years, if their mother was unmarried during their infancy or childhood (measured from birth to age 11.5), or if their mother remarried during their childhood. This paternal instability variable includes data collected from mothers at the time of the participants' birth and when they were 1, 2, 8.5, and 11.5 years of age. Maternal absence was classified as girls who lived in a separate household than their mothers during the surveys at ages 8.5 or 11.5. Sibling death was assessed based upon maternal reports of her children's births and deaths from 1983 until 1994 when subjects were 11.5 years old.

2.3 Statistical analysis

All analyses were conducted using Stata Version 13.1 (Stata Corp, College Park, TX). Descriptive statistics were reported for the full analytical sample and then divided into early and late maturers (median split on age), with *p*-values (2-sided t-tests and Chi-square) reported for

differences by maturational tempo. After examining the bivariate relationships between

menarcheal age and each predictor, a series of regression models were used to evaluate the

independent and combined effects of psychosocial factors and developmental nutrition. We

considered relationships before and after adjustment for the mother's age at menarche, which we

interpret as a rough indicator of genetic influences on maturational tempo (Adair, 2001).

Variance inflation factors for all predictor variables were below 2, suggesting multicollinearity

was not a concern. For graphical illustration of adjusted relationships between menarcheal age

and each key predictor variable, a multiple regression model was run including all predictors

from the full model (Table 2, Model 4), minus the key predictor of interest (e.g. childhood

energy intake). Residuals were calculated and then re-centered on the mean menarcheal age

before being plotted against the predictor.

3. Results

As expected, early maturers grew faster from birth to 24 months, and had higher caloric

intake and BMI in childhood. However, early and late maturers did not differ in the proportion of

individuals who experienced familial instability (Table 1).

In bivariate models predicting menarcheal age, early weight velocity, childhood caloric

intake, and BMI were all found to be significant independent predictors of accelerated

menarcheal timing (Table 2, Model 1). In contrast, all of the markers of early life adversity were

highly non-significant as predictors of menarcheal age (Table 2, Model 2). When both nutrition

and adversity variables were considered together, this had no appreciable effect on the nutrition

coefficients but further attenuated the adversity coefficients (Table 2, Model 3). Although the

mother's menarcheal age was a significant positive predictor of her daughter's menarcheal age,

adjustment for this did not appreciably alter any of the coefficients linking nutritional or

psychosocial factors to daughter's menarcheal age (Figure 1 A-C; Table 2, Model 4).

4. Discussion

In this cohort of women living in metropolitan Cebu, Philippines, we find that measures of early life adversity or familial instability—as reflected in paternal absence, maternal absence or sibling death—did not predict maturational tempo. Instead, measures reflecting favorable infancy and childhood nutrition and growth were strong predictors of maturational acceleration. These findings run counter to the expectations of psychosocial models of maturational acceleration derived from life history theory, and suggest that in contexts like Cebu nutrition is likely the primary determinant of pubertal timing, with more favorable nutrition leading to earlier menarche.

The majority of studies reporting that paternal absence predicts earlier menarche have been conducted in low fertility, high-income nations (see review by Webster, Graber, Gesselman, Crosier, & Schember, 2014). Results more consistent with the present findings have been reported in the few populations studied in which the nutritional environment during childhood was more comparable to that experienced during early childhood by the Cebu cohort studied here. For instance, Sheppard and colleagues (2014) found no relationship between paternal absence and age at menarche in a cohort of girls living in Malaysia where nutritional environments more closely resemble that of the Cebu study population. Another recent analysis of 11,138 randomly selected Indonesian women found no relationship between paternal absence and recalled age at menarche; higher socioeconomic status and greater resource abundance, did, however, predict earlier menarche (Sohn, 2017). Although not reporting information on nutritional status, a study of diverse racial and socioeconomic groups in and around Cape Town, South Africa, found no effect of father absence on age at menarche in girls, although it did

predict earlier sexual debut and earlier first pregnancy (Anderson, 2015). Taken together with our findings at Cebu, these studies suggest that cues of familial instability and sibling death do not predict an acceleration of maturity in populations with less abundant nutrition.

Since maternal absence during childhood is less common than paternal absence, fewer studies have examined its relationship with age at menarche. Moreover, from an evolutionary perspective, it is likely that death or absence of the mother would have greatly increased mortality for infants and young children, making it an unlikely ecological cue to which plasticity in life history strategy would be calibrated in humans (Sear & Mace, 2008). However, if pubertal development is responsive to cues of local mortality risk (Chisholm, 1993), then death of a sibling should be an important predictor. Using historical records from Germany, Canada, and Finland, Störmer and Lummaa (2014) reported that death of a sibling during childhood predicted earlier ages of marriage and first birth for both men and women. Interestingly, the data showed cumulative effects of family-level mortality, rather than effects of individual experiences of death among relatives—suggesting that an increasing number of mortality events in a family might exert greater influence on timing of reproductive milestones.

In theory, the lack of evidence for pronounced effects of psychosocial or environmental cues on age at menarche at Cebu could reflect limitations of our study design, such as having relatively low resolution markers of environmental instability or mortality risk. These included 24 subjects in our sample of 807 with maternal absence, and 86 with paternal instability during their infancy or childhood. The latter variable focuses on exposure up to age 11.5, and is a derived category that includes data measured at the time of the participants' births and when they were 1, 2, 8.5, and 11.5 years of age. Use of these variables here was modeled after our previous published work by Gettler and colleagues (2015), using the same markers of adversity, collected

over the same infancy to childhood period, for the males in the cohort to explore the effects of

early life familial instability on maturational tempo and sexual debut. The large gap between

measures at age two (1986 survey) and again at age 8.5 (1991 survey) is worth noting,

particularly in light of literature that indicates that insults in earlier childhood may be more

salient predictors of sexual behavior in adolescence and adulthood (Simpson, Griskevicius, Kuo,

Sung, & Collins, 2012).

   Despite these limitations, our measures of paternal and maternal absence and sibling

death were prospectively collected during childhood, are comparable to measures used in similar

studies in other populations, and were predictive of behavioral aspects of male life history pace

in recent work from this site (Gettler et al., 2015). It is also notable that our findings here

parallel findings of the predictors of the males' maturational tempo and sexual debut. Using self-

assessed Tanner stages as a measure of reproductive maturity in boys, we reported that higher

caloric intake, skinfold thickness, and body size at 8.5 years of age predicted early maturation

among Cebu men (Gettler et al., 2015). In contrast, but similar to our present findings among

females, no psychosocial stressors predicted early maturity among the males independent of

those childhood energetic and growth markers. These stressors did predict earlier sexual debut

and entry into fatherhood, possibly indicating greater impacts of family-based cues of adversity

on social and behavioral, rather than maturational, outcomes (Gettler et al., 2015). Indeed,

previous work in this cohort by Upadhyay & Hindin (2007), as well as more general findings on

adolescent sexual debut behavior, indicate the quality of familial relationships influences age at

first sex, and that females are more responsive to familial cues than males (Upadhyay & Hindin,

2007; Werner-Wilson, 1998). Del Giudice (2014) proposes middle childhood, specifically the

period around adrenarche just prior to puberty may be a particularly sensitive developmental

period, resulting in a "development switch point" when humans become more attuned to environmental and genetic inputs to determine life history strategy (Del Giudice, 2014). Future analyses will be necessary to explore possible links between psychosocial cues of adversity or risk and later life female reproductive behavior at Cebu.

Although we find no evidence that these various psychosocial stress markers accelerate maturational tempo at Cebu, our results are broadly consistent with a long-standing literature documenting the primary importance of energetic conditions in predicting maturational tempo and age at menarche (Eveleth & Tanner, 1990; Tanner, 1966). The historical secular trends in maturational tempo documented in many populations across the globe consistently show that improvements in living conditions, including large reductions in mortality rates and related mortality cues, are accompanied by sustained intergenerational maturational acceleration (Eveleth & Tanner, 1990). Similar effects are also well known among non-human primates, among whom daughters of higher-ranking females tend to experience menarche earlier than those of low-ranking mothers (Altmann & Alberts, 2005; Bercovitch & Strum, 1993; Charpentier, Tung, Altmann, & Alberts, 2008). This is likely explained by higher-ranking females enjoying greater access to resources; if psychosocial stress has any role in determining timing of menarche it is to delay menarche, as is seen in the daughters of lower-ranking females. These offspring are more likely to be subjected to stress, and show a developmental delay rather than acceleration.

In a compelling longitudinal study, girls in the city of Šibenik, a Croatian town exposed to severe war conditions from 1991-1996, exhibited a significant reversal of a previously observed secular trend towards earlier menarche during and just after the war. Increase in mean menarcheal age was observed in all socioeconomic groups, and was more pronounced in girls

whose homes were damaged during the war, with the most significant delays in menarche exhibited by girls who had lost a family member during the war (Prebeg & Bralić, 2000). In this context it appears that nutritional stress and psychosocial stress occurred in tandem, delaying menarche, and illustrating that even large increases in the risk of mortality do not accelerate maturation when nutritional conditions are marginal.

Although nutritional and psychosocial stressors are posited to have opposing effects on maturational tempo, the maturity-delaying effect of nutritional stress appears to be considerably stronger than the putative accelerating effect of psychosocial stressors. To address the relationship between the two, Coall and Chisholm (2003) propose a hierarchical model in which psychosocial maturational acceleration only occurs in settings in which nutrition is adequate. While theoretically possible, it is uncertain whether the conditions necessary for these responses to independently evolve would have been routinely met: during the course of human evolution individuals would have needed to experience social instability and high mortality risk while also being well-nourished, or, conversely, to have been nutritionally stressed but socially stable and with low mortality. The potential for elevated community- and familial-level psychosocial stressors and nutritional short-falls to be experienced together may be particularly common for humans, owing to the central importance of food sharing and cooperative care in raising offspring (Gurven, 2004; Howell, 2010; Hrdy, 2009; Kuzawa & Bragg, 2012, Quinlan, 2007). Paternal absence/instability might be an exception to this, as some have suggested that fathers may have been more likely to be highly invested in energy-restricted ecologies in the evolutionary past (discussed in Ellis et al., 2009). However, cross-cultural analyses tend to refute the notion that fathers are, on average, more involved under harsher conditions (Quinlan, 2007).

What seems clear is that when psychosocial stress is accompanied by some degree of nutritional stress, the maturation-slowing effect of poor nutrition will overcome any more modest accelerating effect of psychosocial stressors and lead to an overall delay in maturation in high stress settings, much as we see at Cebu. As discussed above, and elsewhere (Anderson, 2015; Kuzawa & Bragg 2012), most of the work examining the role of psychosocial stressors as predictors of maturational acceleration has been conducted in populations characterized by adequate or chronically abundant caloric availability, such as in the United States and United Kingdom (Brumbach, Figueredo, & Ellis, 2009; Chisholm et al., 2005; Ellis et al., 1999; Nettle, Coall, & Dickins, 2010). These are also settings in which low income populations, who are likely to face relatively high levels of daily stress and uncertainty (McEwen & Seeman, 1999), tend to have higher rates of overweight and obesity (Banks, Marmot, Oldfield, & Smith, 2006), which themselves lead to earlier maturity. It seems likely that a concordance between caloric abundance and chronic high psychosocial stress is a relatively recent and evolutionarily novel scenario, and one not well suited to providing insights into the evolved, adaptive function of plasticity in human maturational tempo.

As noted above, the predicted link between extrinsic mortality (or cues signaling this) and accelerated maturational tempo is an assumption extrapolated from models developed to explain broad inter-specific variation ("mouse to elephant") in vertebrate life history evolution (e.g. Charnov, 1991). In light of the present findings, it is worth considering whether factors important in explaining evolved between-species differences in life history are relevant for explaining the more nuanced variation attributable to plasticity within a single species. In the case of extrinsic mortality as a driver of maturational plasticity in humans, work by Hill (1996) showed that mortality rates are low enough in human populations, including among the Aché hunter gatherers

studied, that the increased risk of dying due to maturational delay imposes a negligible penalty to reproductive success. They find instead that delaying onset of reproduction has, in females, much larger negative impacts on completed fertility by truncating the duration of the reproductive lifespan owing to the relatively fixed age of the menopause, when reproduction ceases in humans. Thus, although differences in ambient rates of extrinsic mortality faced by different species may help explain broad inter-specific trends in body size, life span and other traits, we speculate that these same forces may be less pertinent when explaining nuanced changes in maturational tempo in human populations.

Based upon our findings, and the above discussion, the concept of extrinsic mortality-motivated plasticity in human life history strategy might better be applied to events in female reproductive life that are not constrained by nutrition and around which women have a greater degree of behavioral agency (Gettler et al., 2015; Kuzawa & Bragg 2012). Age at first sex and age at first birth, other milestones that have been explored in studies examining the effect of psychosocial stressors, are under more variable social and individual control (Gettler et al., 2015; Störmer & Lummaa, 2014). In a Catholic country like the Philippines, where premarital sex is discouraged, cultural constraints on reproduction are likely more important and in theory should be adjustable in response to early life cues. The trade-offs that constrain pubertal timing or that influence onset of reproduction are inherently different: for instance, the latter depends on factors like the need to accrue social capital sufficient to have a successful pregnancy or raise a child to adulthood, while pubertal timing merely reflects biological maturation, which is driven by growth and development.

In the present study we included recalled maternal age at menarche as a proxy for inter-generational genetic correlations in this developmental milestone. While not a perfect control for

genetic influence on age of menarche, the significant relationship between mother's and daughter's age at menarche underscores methodological concerns when evaluating causal inferences concerning life history strategies. The genetic underpinnings of phenotypic/behavioral traits may be erroneously attributed to environmental variance in the relationship between two traits—such as father absence and age of menarche—if genetic variance is not also considered. Alternative explanations for the links between familial instability and earlier menarche, such as genetic correlations between behavioral and biological markers (Barbaro, Boutwell, Barnes, & Shackelford, 2017) warrant additional attention as contributors to these patterns.

Data Availability

The data associated with this research are available at http://www.cpc.unc.edu/projects/cebu.

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Table 1. Characteristics of women from metropolitan Cebu (N = 807)

	All (n=807)	Early maturers (n=582)	Late maturers (n=225)	
	Mean±SD	Mean±SD	Mean±SD	p-value*
Age Menarche	12.8 ± 1.2	12.2 ± 0.8	14.4 ± 0.6	<0.0001
Birthweight (g)	2986 ± 411	2986 ± 402	2983 ± 433	0.9281
Weight velocity birth to 24 months (g/month)	265 ± 41	270 ± 42	254 ± 38	<0.0001
Caloric intake at age 8.5 (kcal/day)	1312 ± 475	1360 ± 491	1190 ± 408	<0.0001
BMI (kg/m^2)	14.6 ± 1.3	14.8 ± 1.4	14.3 ± 1.0	<0.0001
Paternal absence	10.7%	11.5%	8.4%	0.205
Maternal absence	3.0%	2.8%	3.6%	0.545
Sibling death	16.6%	16.0%	18.2%	0.443
Maternal age at menarche (years)	14.1 ± 1.5	13.9 ± 1.5	14.4 ± 1.5	<0.0001

*p-value comparing early to late maturers, 2-sided t-test or Chi^2

Table 2. Multiple regression model output predicting age of menarche in Cebu women[#]

Predictor	Bivariate	p-value	Model 1	p-value	Model 2	p-value	Model 3	p-value	Model 4	p-value
Birthweight (kg)*	-0.07 ± 0.11	0.498	0.04 ± 0.11	0.682			0.04 ±0.11	0.694	0.03± 0.11	0.747
Weight velocity birth to 24 months (kg/month)	-6.51 ± 1.02	<0.0001	-3.91 ± 1.07	<0.0001			-3.89 ±1.08	<0.0001	-3.80 ± 1.07	<0.0001
Caloric intake for body weight (kg)** (1000 kcal/day)	-0.62 ± 0.09	<0.0001	-0.49 ± 0.09	<0.0001			-0.49 ±0.09	<0.0001	-0.47 ± 0.09	<0.0001
BMI (kg/m^2)**	-0.21 ± 0.03	<0.0001	-0.14 ± 0.03	<0.0001			-0.14 ±0.03	<0.0001	-0.14 ± 0.03	<0.0001
Paternal absence	-0.05 ± 0.14	0.729			-0.08 ± 0.15	0.606	-0.02 ±0.14	0.901	0.04 ± 0.14	0.765
Maternal absence	0.09 ± 0.25	0.737			0.15 ± 0.27	0.590	0.10 ± 0.26	0.704	0.11 ±0.26	0.680
Sibling death	0.12 ± 0.12	0.306			0.12 ± 0.12	0.297	0.01 ±0.11	0.962	0.00 ± 0.11	0.991
Maternal age at menarche (years)	0.15 ± 0.03	<0.0001							0.14 ± 0.03	<0.0001

[#] Values are regression model coefficients ± SE

*adjusted for gestational age

**at approximately age 8.5 years

Fig. 1 Relationships between menarcheal age and A) energy intake during childhood (8.5 years), B) body mass index during childhood (8.5 years), and C) infancy weight velocity (birth-2 years). All significant at p<0.0001. Relationships were derived from the fully-adjusted model (Table 2, Model 4) that included childhood energy intake, infancy weight velocity, gestational age-adjusted birth weight, childhood energy intake, sibling death, paternal instability, maternal instability and maternal age at menarche (see Section 2. Methods).