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# Developmental Energetics, Sibling Death, and Parental Instability as Predictors of Maturational Tempo and Life History Scheduling in Males from Cebu, Philippines

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*KEY WORDS* life history theory; developmental plasticity; environmental risk; extrinsic mortality; paternal investment

*ABSTRACT OBJECTIVES:* Cross-species comparisons show that high extrinsic mortality favors the evolution of "faster" life histories. There is interest in applying this principle to human life history plasticity, based on the idea that psychosocial stressors that correlate with extrinsic mortality accelerate reproductive pace. Most prior studies have been conducted in settings in which psychosocial stressors co-occur with the maturation-accelerating influence of nutritional abundance.
*MATERIALS AND METHODS*: We evaluate cues of local mortality (sibling death) or low parental investment (paternal instability; maternal absence) and energetic measures during development as predictors of life history scheduling among males ($n = 754$) in a Philippine population with marginal developmental nutritional.
*RESULTS*: Males who had more favorable nutritional status during childhood, as reflected in linear growth, skinfold thickness, and caloric intake, were more maturationally advanced in adolescence (all $P < 0.05$). Taller stature and higher caloric intake during childhood also predicted earlier ages at first sex (both $P < 0.01$), which persisted after controlling for the effect of nutrition on pubertal maturation. While psychosocial stressors did not predict accelerated maturation, males who as children grew up with an unstable paternal presence had sex earlier ($P < 0.05$) and tended to become fathers sooner than those with a stable fatherly presence. Those who had a sibling die became fathers sooner than those who did not ($P < 0.05$).
*DISCUSSION*: Our findings point to important energetic constraints on the onset of reproductive maturity, while psychosocial stressors accelerate entry to parenthood, which may be comparatively more socially, rather than biologically, constrained. Am J Phys Anthropol 158:175–184, 2015. 

Timing of reproductive maturity is a key event in an organism's life history, as it reflects the age when growth ceases and productivity is diverted from growth into reproduction (Charnov and Berrigan, 1993; Hill and Kaplan, 1999; Kuzawa, 2007). At a species level, age at reproductive maturity is viewed as being shaped by natural selection to balance the reproductive and survival benefits of attaining a larger adult size against the mortality risk associated with deferring reproduction to continue growing (Stearns, 1992; Charnov and Berrigan, 1993). As such, high mortality environments generally favor the evolution of fast life history strategies characterized by earlier maturation and increased investment in early reproduction, which come at a cost to maintenance and reproductive lifespan (Promislow and Harvey, 1990; Stearns, 1992; Charnov and Berrigan, 1993).

There is much interest in applying tenets of this cross-species framework to help explain within-species variation, tracing to developmental and behavioral plasticity (Belsky et al., 1991; Chisholm, 1993; Coall and Chisholm, 2003; Ellis et al., 2009; Nettle, 2011; Nettle et al., 2011). This work is premised on the assumption that selection pressures would favor the capacity for

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context-sensitive responses in which cues reflecting local risk of mortality, or correlated cues related to environmental harshness or unpredictability, are tracked during development (Ellis et al., 2009). Human research has generally supported the hypothesis that human maturation and reproductive scheduling are accelerated when harsh psychosocial conditions are experienced during infancy or childhood (Ellis and Garber, 2000; Belsky et al., 2007; Nettle et al., 2011).

These ideas have primarily been investigated, and gain most empirical support, among females (Ellis et al., 2009). Numerous studies have reported earlier reproductive maturation among girls raised in settings characterized by low parental investment or other conditions contributing to elevated psychosocial stress (Ellis et al., 1999; Ellis and Garber, 2000; Quinlan, 2003; Chisholm et al., 2005; Belsky et al., 2007). Although findings vary, past work has also shown that women raised in stressful developmental contexts are more likely to first have sex or give birth earlier and to have more sex partners (Quinlan, 2003; Chisholm et al., 2005; Nettle et al., 2011). To date, comparably few studies have evaluated whether similar principles apply to males, for whom the optimal timing of maturity is likely constrained by similar tradeoffs. As is true in females, delayed maturity increases the risk of dying before maturing, but also affords males with reproductive benefits such as larger body size (Bribiescas, 2001; Ellison, 2003), which some studies suggest increase attractiveness and genetic fitness (Pawlowski et al., 2000), along with enhanced knowledge, skill, wealth, social prestige, and other measures of embodied capital that influence suitability as a potential mate (Hill and Kaplan, 1999; Kaplan et al., 2000; Gurven and Walker, 2006; von Rueden et al., 2011).

Past studies have shown that energetic deficits during early development slow growth and delay human male reproductive maturation (Kulin et al., 1982; Campbell et al., 2004, 2005; Kuzawa et al., 2010), similar to what has been found in females. However, there is conflicting evidence as to whether paternal absence, which is often modeled as a developmental stressor or marker of early-life social instability, affects reproductive maturation and adult reproductive behavior in males. In two prior studies, boys with marginal paternal relationships did not show accelerated maturational tempo (Malo and Tremblay, 1997; Belsky et al., 2007), while a third study found no effect of paternal death on the age when males became fathers (Winking et al., 2011). Elsewhere, males raised with low fatherly involvement or paternal absence matured earlier (Bogaert, 2005; cf. James et al., 2012), had sex earlier (Stern et al., 1984; Dittus et al., 1997; Alvergne et al., 2008), and/or became fathers at younger ages (Jaffee et al., 2001; Hofferth and Goldscheider, 2010; Sheppard and Sear, 2012). Many studies have linked earlier sexual debut and earlier fatherhood to more generalized measures of psychosocial stress, such as living under low socioeconomic status (SES) conditions or being born to a teenage mother (Nelson, 2004; Hawes et al., 2010). Finally, across cultures, human fathers' investments are more variable and their absence is more common, compared to mothers (Hewlett, 1991; Geary and Flinn, 2001; Gray and Anderson, 2010; Gettler, 2014), and maternal absence likely would have had severe implications for child mortality during the evolutionary past (Sear and Mace, 2008). These issues might underlie the more common focus on paternal instability in prior studies, but maternal effects should likewise be considered in contemporary populations in which other family members (e.g., fathers, grandmothers) and nonfamilial or institutionalized care can buffer children from extreme outcomes.

One challenge to testing evolutionary models of the timing of maturity and reproductive scheduling, in either sex stems from the fact that psychosocial stress and nutritional stress, which are experienced variably across individuals and populations, have opposing effects on growth and maturational tempo (Coall and Chisholm, 2003; Ellis et al., 2009; Kuzawa and Bragg, 2012). This is important because long-term energy abundance, such as occurs with overweight and obesity, is itself among the strongest accelerators of maturational tempo, while growth studies show that the effects of nutritional stress in slowing maturity are much larger than the accelerating effect of psychosocial stress (Eveleth and Tanner, 1976; Eveleth and Tanner, 1990). For instance, in countries like Norway, Finland, and the United States, improvements in nutrition and hygiene are thought to explain why menarcheal age has declined at a pace of 0.3 years per decade, or approximately 7 months per generation, leading to cumulative declines in onset of menarche from 17 to 14 years between the mid-19th and mid-20th centuries, with further and continued acceleration up to the present (Parent et al., 2003). While the effect sizes are not directly comparable, putative psychosocial cues of mortality risk typically predict pubertal acceleration of several months (Kuzawa and Bragg, 2012).

During the evolution and prehistory of modern humans, cooperation and food sharing were likely integral to foraging subsistence success (Hawkes et al., 1998; Hill and Hurtado, 2009; Howell, 2010; Kramer and Ellison, 2010). As such, social strife or unrest and nutritional stress are likely to have co-occurred among members of our genus (Fuentes et al., 2010; Antón and Snodgrass, 2012; Kuzawa and Bragg, 2012). Under such circumstances, any accelerating effect of psychosocial stress on pubertal timing would likely be swamped by the opposing and larger decelerating effect of poor nutrition (Kuzawa and Bragg, 2012). While the dominant effect of nutrition in setting maturational tempo has been acknowledged by scholars in this field (Coall and Chisholm, 2003; Ellis et al., 2009), the majority of human work testing life history-inspired models of maturational tempo and reproductive scheduling have been conducted in high-income countries with high inequality, where severe psychosocial stressors commonly co-occur with nutritional abundance and a high prevalence of overweight (Mackenbach et al., 2008; Siervo et al., 2009; but see Quinlan, 2010; Caudell and Quinlan, 2012). In particular, in these settings maturational acceleration associated with stressful life experiences may be confounded by the co-occurrence of maturation-accelerating overweight and other metabolic disturbance (Ellis and Essex, 2007).

While maturational tempo and pubertal timing are under strong nutritional constraint, subsequent reproductive milestones, such as age at first sex or entry into parenthood, are in theory less tied to physical maturity. Cues of local mortality or environmental stability could thus have an accelerating effect on the timing of sexual activity and parenthood independent of pubertal maturation, which would be less open to confounding by differences in nutritional status or overweight. With the exception of a recent paper that considered psychosocial

adversity and childhood body mass index (BMI) in a British sample (Sheppard and Sear, 2012), to date there has been little attempt to simultaneously model early-life energetic and psychosocial stressors as predictors of maturational tempo or reproductive scheduling in studies of human males (Ellis et al., 2009).

Here, we seek to clarify the role of environmental influences on pubertal maturation and reproductive scheduling in humans, with a focus on a cohort of young adult males from a population in Metropolitan Cebu City, the Philippines, in which early-life nutrition was marginal and growth faltering common (Adair, 1999; Adair et al., 2011). We test a series of models aimed at teasing apart the influences of energetic and social inputs to maturational tempo and reproductive behavior in this population. Specifically, we use height-for-age *z*-score (HAZ), triceps skinfold thickness, and dietary calorie intake (per kilogram body weight) at 8.5 years of age as markers of developmental energy sufficiency. Building from past work in this field (Chisholm, 1993; Ellis et al., 2003), we use death of a sibling, maternal absence, and instability of paternal presence in the household, occurring prior to the late childhood survey (11.5 years of age), as cues of mortality or environmental instability. Using a multivariate framework, we evaluate the independent role of these nutritional and psychosocial stressors as predictors of three key indicators of life history strategy and reproductive scheduling: maturational tempo, age at first sex, and age at transition into fatherhood.

## METHODS

### Study population

Data were compiled throughout the subjects' lives from multiple surveys spanning 1983 (birth) to 2009 (age 26.0 ± 0.3 years) conducted as part of the Cebu Longitudinal Health and Nutrition Survey (CLHNS), a population-based birth cohort study that originally enrolled 3,327 mothers and their infants in 1983–1984 (Adair et al., 2011). After birth, subjects were followed at 2-month increments for 24 months. Subsequent follow-up surveys took place in 1991–1992, 1994–1995, 1998–1999, 2002, 2005, and 2009 (Adair et al., 2011). Here, we focus on data collected from surveys between 1983 and 1994, and surveys in 1998 and 2009. Sociodemographic and behavioral data were collected using questionnaire-based, in-home interviews administered by Cebuano-speaking interviewers (Adair et al., 2011). Of the original cohort, a final sample of 754 individuals had complete early life and adult data available for the purpose of the present analyses. Data collection was conducted under conditions of written informed consent, with human subjects oversight by the IRB of Northwestern University and the University of North Carolina, Chapel Hill. The maternal, childhood, and adolescent instruments, codebooks, and data that were used in the present analyses are freely available for download (http://arc.irss.unc.edu/dvn/dv/cebu).

### Anthropometry, energy intake, and pubertal maturation

Our core analyses include body weight (kg), height (cm), and triceps skinfold thickness (mm) measurements from the childhood follow-up (8.5 ± 0.04 years of age). We also report anthropometric measurements from adolescence (16.1 ± 0.3 years of age) and adulthood (26 years of age). At each time point, measurements were collected using standard anthropometric techniques (Lohman et al., 1988). Adolescent BMI was calculated as the ratio of weight (kg)/height ($m^2$), and subjects with BMI ≥ 25 were categorized as overweight/obese. Childhood energy intake was calculated with a quantitative food frequency questionnaire, with energy content of foods estimated using the Philippines Food Composition Tables developed by the Food and Nutrition Research Institute (FNRI, 1990). At age 16.1 years, participants reported pubic hair development by comparing themselves with line drawings of five pubic hair stages that were physician-validated among Filipino youth (Adair et al., 2011). Men were considered more maturationally advanced if they classified themselves as being in the two most advanced pubic hair stages (Kuzawa et al., 2010).

### Psychosocial stressors and key demographic-life history variables

Men who were classified as having experienced "paternal instability" up to age 11.5 (±0.4) years were those whose father was deceased or absent or whose mother was unmarried during their first year of life or beyond, or whose mother remarried during their childhood-juvenile period. Subjects were classified as having experienced maternal absence if they lived in a separate household than their mother at 8.5 or 11.5 years of age. Our maternal absence variable differs somewhat from "paternal instability" due to the explicit mother–infant dyadic focus of the CLHNS (Adair et al., 2011).

Sibling death and maternal fertility were assessed via maternal reports of her children's births and deaths after 1984 up to when the subjects were age 11.5 years. Seventeen subjects with otherwise complete data did not have maternal fertility data available at age 11.5 years. For these subjects, we substituted maternal fertility data from the surveys when subjects were at age 16.1 years ($n = 7$) or 8.5 years ($n = 10$). Men's age at first sexual intercourse and fatherhood status (having at least one biological child) were reported in 2009 when the men were 26.0 (±0.3) years old (Gettler et al., 2011, 2013).

### Statistical analyses

All analyses were conducted using version 12.1 of Stata (Stata Corporation, College Station, TX). Subjects' triceps skinfold thickness and daily food intake per unit of body weight (kcal/kg) were entered into models as standard deviation scores to allow comparison of unitless effect sizes. HAZs were calculated using the World Health Organization's (WHO) AnthroPlus software in Stata, which provides nutritional status information for children between 5 and 19 years of age (WHO, 2007). SES was modeled using education (highest grade completed) and household income (per household member) and the variables were treated as continuous variables. Paternal instability, maternal absence, and sibling death were treated as dichotomous variables.

We first used logistic regression to predict which males were relatively advanced maturationally, defined as being in the two most advanced pubic hair stages (measured in 1998), controlling for age at the time of maturity assessment (Kuzawa et al., 2010). We next

*TABLE 1. Sample characteristics*[a]

| Demographic characteristics | Full sample ($n = 754$) | | Paternal instability ($n = 77$) | | Maternal absence ($n = 30$) | | Sibling death ($n = 123$) | |
|---|---|---|---|---|---|---|---|---|
| | Mean | (SD) | Mean | (SD) | Mean | (SD) | Mean | (SD) |
| Paternal instability (% yes) | 10.2 | – | – | – | 29.9 | – | 7.8 | – |
| Maternal absence (% yes) | 4.0 | – | 76.7 | – | – | – | 10.0 | – |
| Sibling death (% yes) | 16.3 | – | 4.9 | – | 2.4 | – | – | – |
| Age (years) | 26.0 | (0.3) | 26.0 | (0.3) | **26.1*** | **(0.3)** | **25.9*** | **(0.3)** |
| Education (highest grade) | 10.8 | (3.3) | 10.9 | (2.9) | 9.8 | (3.2) | **9.1*** | **(3.6)** |
| Maternal education (highest grade)[b] | 7.5 | (3.8) | 7.8 | (3.5) | 7.2 | (3.7) | **6.2*** | **(3.7)** |
| Per capita household income (pesos) (1998)[c] | 549 | (478) | 509 | (645) | 519 | (369) | 478 | (413) |
| Birth order | 3.3 | (2.3) | 3.0 | (2.7) | 2.6 | (2.6) | **4.0*** | **(2.5)** |
| Total number of siblings (1994) | 5.3 | (2.6) | **4.7*** | **(2.8)** | 4.8 | (2.5) | **6.8*** | **(2.6)** |
| Adult anthropometric characteristics | | | | | | | | |
| Adult weight (kg) | 60.7 | (11.3) | 61.3 | (11.5) | 58.5 | (9.3) | **57.5*** | **(9.5)** |
| Adult height (cm) | 163.1 | (5.8) | 163.7 | (5.8) | 163.6 | (4.7) | **161.8*** | **(5.5)** |
| Childhood anthropometric characteristics[d] | | | | | | | | |
| Weight (kg) | 20.7 | (3.1) | 20.5 | (2.3) | 20.7 | (2.7) | **20.1*** | **(2.2)** |
| Childhood height (cm) | 117.7 | (5.4) | 117.8 | (4.9) | 118.2 | (4.6) | **116.3*** | **(5.0)** |
| Triceps skinfolds (mm) | 6.3 | (2.0) | 6.3 | (1.6) | 6.5 | (1.7) | **5.9*** | **(1.5)** |
| Energy intake for weight (kcal/kg) | 72.2 | (27.1) | **78.3*** | **(30.7)** | 80.3 | (2.4) | **65.7*** | **(24.9)** |
| Body mass index (kg/m$^2$) (1998)[c] | 18.6 | (2.5) | 18.5 | (2.3) | 18.5 | (2.4) | 18.4 | (2.4) |
| % Overweight/obese (1998)[c] | 2.3 | – | 1.0 | – | 3.3 | – | 1.6 | – |
| Reproductive characteristics | | | | | | | | |
| Had sexual intercourse (% yes) | 92.4 | – | 94.8 | – | 93.3 | – | 90.2 | – |
| Father (% yes) | 49.6 | – | 59.7 | – | 56.7 | – | 57.7 | – |
| Age at first sex (years)[e] | 18.4 | (2.9) | **17.7*** | **(3.1)** | 18.3 | (2.8) | 18.3 | (3.0) |
| Age at fatherhood transition (years)[f] | 22.9 | (2.2) | 22.9 | (2.3) | 22.8 | (2.1) | 22.7 | (2.4) |

[a] Descriptive statistics from men at age 26.0 (±0.3) years, unless otherwise noted. * and bold font: $P < 0.05$. Results from unpaired *t*-tests.
[b] At the time of subjects' birth in 1983–1984.
[c] Measured at age 16.1 (±0.3) years.
[d] Measured at age 8.5 (±0.04) years.
[e] Restricted to males who reported having had sexual intercourse by age 26.0 (±0.3) years (n = 687).
[f] Restricted to males who had become fathers by age 26.0 (±0.3) years ($n = 369$).

used survival time hazard regression (Weibull distribution) to model the predictors of ages at first sex and ages at which men first became fathers, starting with models that included only sibling death, paternal instability, and maternal absence (Model 1), then adding childhood nutrition variables (Model 2), and subsequently adding household income measured in childhood (Model 3). In analyses for which sibling death was a significant predictor after Model 3, we additionally included subjects' total number of siblings (i.e., maternal fertility) at age 11.5 years and the subjects' birth order in a subsequent model to assess covariation with parental life history characteristics. To evaluate the possible role of timing of puberty as a pathway linking nutritional variables with age at first sex, we also adjusted the models predicting age at first sex for maturational tempo, modeled as self-assessed pubic hair stage, age at maturity assessment (mean of 16.1 years), and their interaction (see Kuzawa et al., 2010). We set statistical significance at $P < 0.05$, with $P < 0.1$ considered a statistical trend. We produced the adjusted values for our figures using predictive margins and Stata's marginsplot command, following our full statistical models.

## RESULTS

Table 1 reports descriptive statistics for the entire sample and also stratified according to paternal instability, maternal absence, and sibling death. There was minimal overlap between subjects experiencing the parent-related social stressors and sibling death, whereas there was more overlap between paternal instability and maternal absence. For example, approximately 77% of subjects experiencing maternal absence also had unstable paternal presence. In unadjusted bivariate analyses (Table 1), males who experienced sibling death during childhood were shorter, had thinner triceps skinfolds, and had lower caloric intake (kcal/kg) compared to the rest of the sample (all $P \leq 0.01$) consistent with the lower SES of sibling death households (maternal education, $P < 0.0001$). Subjects experiencing sibling death also had a higher number of siblings, on average ($P < 0.0001$). In contrast, subjects who experienced paternal instability and maternal absence did not significantly differ from the broader sample for most childhood growth-energetic markers or household SES indicators ($P > 0.05$).

We next ran multivariate models to evaluate the early-life predictors of each life history outcome (maturational tempo, age at first sex, age at becoming fathers). In a logistic regression model adjusting only for the age at maturity assessment (Table 2), males who experienced the death of a sibling had a lower odds of being maturationally advanced compared to other subjects, while paternal instability and maternal absence were unrelated to maturational tempo. All three markers of childhood nutritional status were significant and independent predictors of early maturity (Fig. 1), and once these were added to the model sibling death was no longer a significant predictor of maturational tempo (Table 2).

*TABLE 2. Logistic regression models predicting advanced maturational status at 16 years of age based on developmental experiences (*n = *754)*[a]

| | Model 1 | | *P* | Model 2 | | *P* |
|---|---|---|---|---|---|---|
| Sibling death | 0.62 | (0.41, 0.93) | 0.022 | 0.77 | (0.50, 1.19) | 0.244 |
| Paternal instability | 1.03 | (0.60, 1.78) | 0.907 | 1.04 | (0.60, 1.82) | 0.885 |
| Maternal absence | 0.61 | (0.26, 1.46) | 0.267 | 0.53 | (0.21, 1.29) | 0.160 |
| Height-for-age *z*-score | | | | 1.65 | (1.37, 1.99) | 0.0001 |
| Tricep skinfolds | | | | 1.34 | (1.10, 1.63) | 0.003 |
| Kcal/kg | | | | 1.22 | (1.05, 1.43) | 0.011 |

[a] Values are odds ratio (95% CI). Models adjusted for age at maturational status assessment (16.1 ± 0.3 years). Childhood height, skinfold thickness, and calorie intake were measured at 8.5 ± 0.04 years and modeled as SD scores.

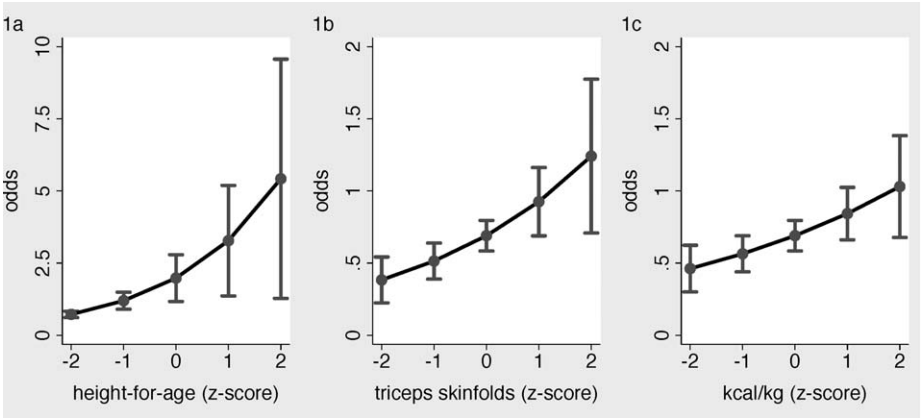


**Fig. 1.** Adjusted odds showing the likelihood of males being maturationally advanced during adolescence based on childhood nutritional status. Males had a higher odds of being maturationally advanced at age 16.1 (± 0.3) years if they experienced favorable childhood energetic conditions, based on measures of (**a**) linear growth (HAZ), (**b**) skinfold thickness, and (**c**) caloric intake (kcal/kg) (all $P < 0.05$; Table 2). All nutritional status predictors modeled as continuous variables and converted to SD scores, with models adjusted for age at maturational status assessment and exposure to psychosocial stressors (sibling death, paternal instability, and maternal absence). Note the *y*-axis range for (a) differs from (b) and (c). Error bars represent 95% CI.

In survival analyses, sibling death and maternal absence were not significant predictors of earlier age at first sex (Table 3 and Fig. 2a). In the initial model, subjects who experienced paternal instability had earlier ages at sexual debut (Fig. 2b). Consistent with the models predicting pubertal timing, being taller or having higher caloric intake during childhood predicted earlier sexual debut (Fig. 3). Adjusting for childhood nutritional variables had no effect on the coefficients linking sibling death or maternal absence to age at first sex, while the effect of paternal instability became a trend ($P = 0.057$) with little change to the effect size (Table 3). In contrast to the other energetic markers, greater childhood adiposity, as reflected in skinfold thickness, predicted a later age at first sex (Table 3 and Fig. 3). In light of the possibility that overweight/obese subjects primarily drove this effect, we ran an additional set of models in which we included a dichotomous variable for adolescent overweight/obesity. Subjects who were overweight/obese tended to have later ages at sexual debut ($P = 0.055$), and neither childhood adiposity nor adolescent overweight/obesity were significant predictors when they were included in the same model (both $P > 0.1$; Supporting Information Table 1). Because favorable childhood nutrition predicted both early pubertal maturation (Table 2) and earlier age at first sex (Table 3), we also evaluated the extent to which early maturity helped explain the earlier sex among well-nourished

*TABLE 3. Survival analysis relating men's age at first sexual intercourse to developmental experiences (*n = *754)*[a]

| | Model 1 | | *P* | Model 2 | | *P* | Model 3 | | *P* |
|---|---|---|---|---|---|---|---|---|---|
| Sibling death | 0.92 | (0.75, 1.13) | 0.449 | 0.99 | (0.81, 1.22) | 0.948 | 0.99 | (0.81, 1.22) | 0.953 |
| Paternal instability | 1.32 | (1.01, 1.73) | 0.042 | 1.30 | (0.99, 1.70) | 0.057 | 1.31 | (1.00, 1.71) | 0.053 |
| Maternal absence | 0.85 | (0.56, 1.29) | 0.444 | 0.80 | (0.53, 1.21) | 0.292 | 0.80 | (0.53, 1.22) | 0.300 |
| Height for age *z*-score | | | | 1.17 | (1.07, 1.27) | 0.0003 | 1.16 | (1.07, 1.26) | 0.0006 |
| Triceps skinfolds | | | | 0.92 | (0.85, 0.99) | 0.023 | 0.91 | (0.84, 0.98) | 0.013 |
| Kcal/kg | | | | 1.23 | (1.14, 1.32) | 0.0001 | 1.21 | (1.13, 1.31) | 0.0001 |
| Household income[b] | | | | | | | 1.06 | (0.99, 1.14) | 0.109 |

[a] Values are hazard ratios (95% CI). Childhood height, skinfold thickness, and calorie intake were measured at 8.5 ± 0.04 years and modeled as SD scores.
[b] Household income at age: 16.1 (± 0.3) years. Modeled as a SD score.

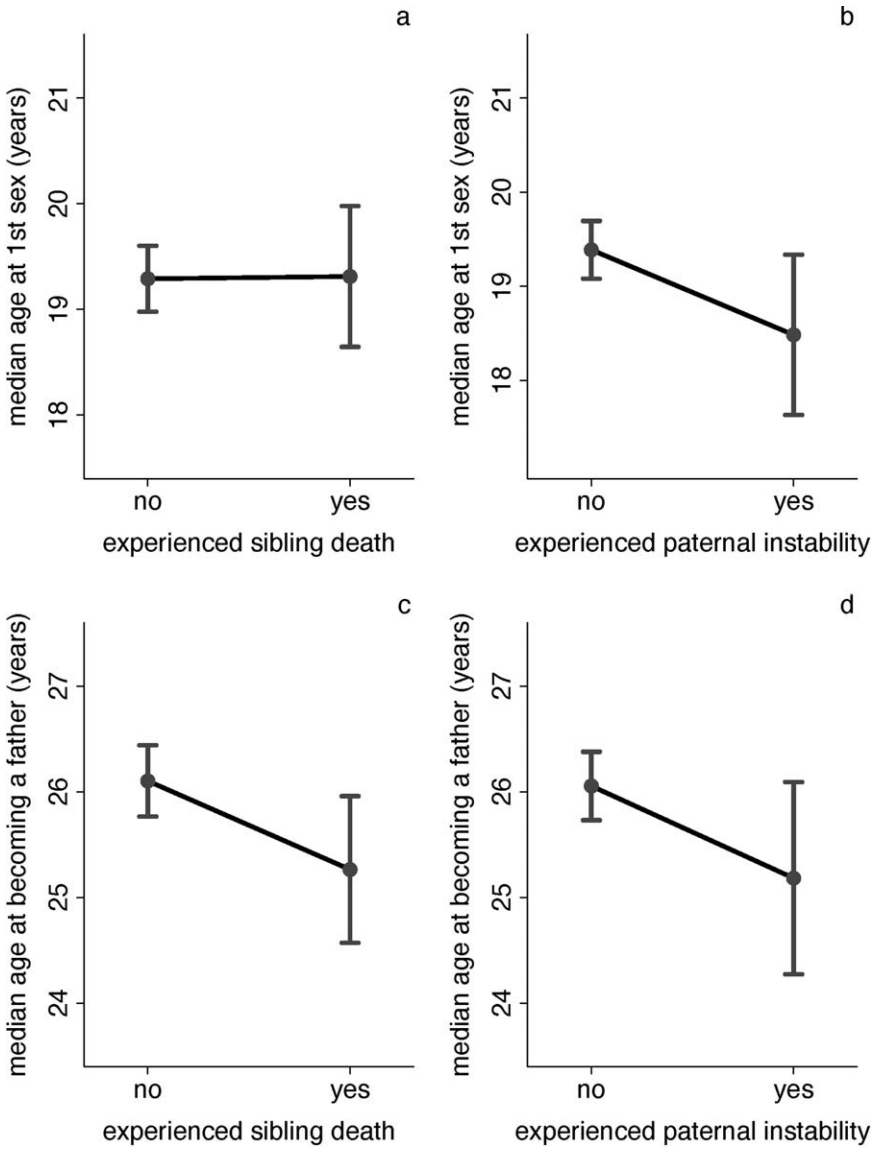


**Fig. 2.** Adjusted median ages at which males first had sexual intercourse and became fathers based on developmental exposures to psychosocial stressors. Males had earlier ages at sexual debut if they experienced paternal instability ($P \leq 0.05$) but not sibling death ($P > 0.9$; Table 3). Men became fathers at younger ages if they experienced the death of a sibling ($P < 0.05$) with a similar trend for those who experienced paternal instability ($P < 0.1$; Table 4). We present adjusted median ages, yielded using predictive margins following survival analysis models that included childhood nutritional status, maternal absence, and adolescent SES. Error bars represent 95% CI.

individuals. After adjustment for maturational tempo, the hazard ratios linking nutrition to age at first sex were minimally attenuated and remained equivalently significant (results not shown).

Men who experienced sibling death in childhood made the transition into fatherhood at a younger age, with a similar trend for those experiencing paternal instability. The effect for sibling death was largely independent of maternal absence, childhood nutrition variables, and adolescent SES (Fig. 2c,d), none of which were significant predictors of age at fatherhood (Table 4). When we added subjects' birth order and their total number of siblings to the model, sibling death remained a significant predictor of age at the transition to fatherhood, with little change to the effect size (Table 4).

## DISCUSSION

In this Philippine population, males who had more favorable nutritional status during childhood, as reflected in childhood linear growth, skinfold thickness, or caloric intake, were more maturationally advanced in adolescence. Taller boys and those with higher caloric intake also had sex at an earlier age compared to males experiencing poorer nutritional status. These findings thus support a role of favorable energetic conditions as an accelerator of life history scheduling. We found that men who experienced paternal instability had sexual intercourse at younger ages and also tended to become fathers at younger ages. Men raised in households in which a sibling had died also became fathers earlier. Our findings support a primary effect of energetic

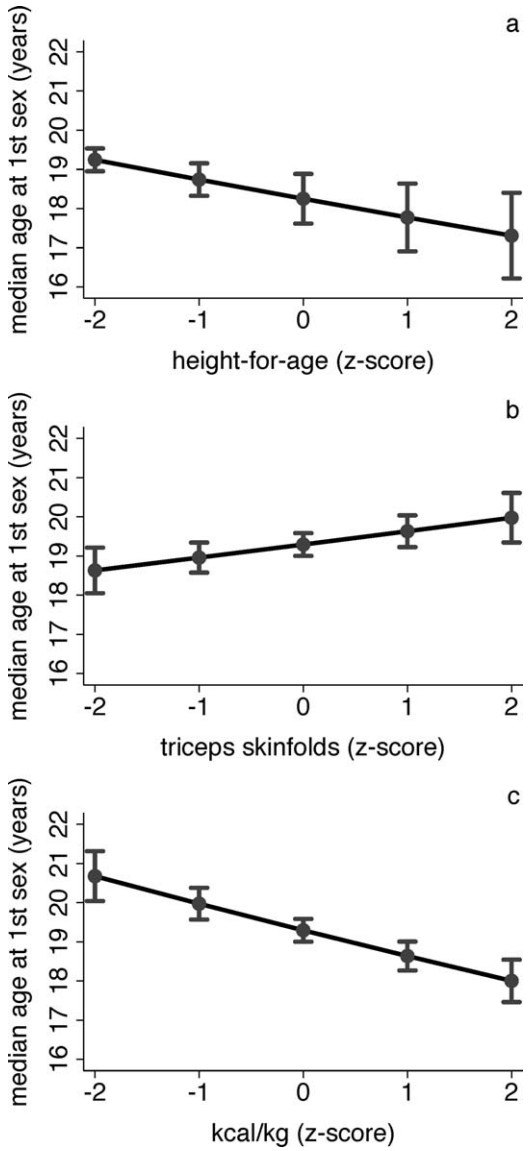


**Fig. 3.** Adjusted median ages at which males first had sexual intercourse based on childhood nutritional status (presented as SD scores). Males' ages at sexual debut were lower if they were (**a**) taller (HAZ) and (**c**) consumed more calories (kcal/kg) (both $P < 0.01$). Males became sexually active at older ages if they had (**b**) thicker childhood triceps skinfold measures ($P < 0.05$; Table 3). We treated the nutritional status predictors as continuous variables in our survival analysis models and adjusted for age at maturational status assessment and exposure to psychosocial stressors (sibling death, paternal instability, and maternal absence) and adolescent SES. Error bars represent 95% CI.

conditions in calibrating maturational tempo and the onset of sexual activity in Cebu, with psychosocial stressors playing a role in motivating scheduling of reproductive behaviors (Coall and Chisholm, 2003; Ellis et al., 2009; Kuzawa and Bragg, 2012).

Our finding that favorable childhood energetic conditions accelerate the onset of puberty is similar to results from some prior studies of human males (Kulin et al., 1982; Campbell et al., 2004; Sheppard and Sear, 2012), and parallels the more extensive literature documenting earlier reproductive maturity among well-nourished girls (Eveleth and Tanner, 1976, 1990; Parent et al., 2003; Ellis and Essex, 2007). The finding that favorable early nutrition also predicted an earlier age at first sex suggests that initiation of sexual activity among these males is biologically constrained by the transition to sexual maturity and opportunities that then emerge thereafter. While all three markers of nutritional status predicted earlier puberty, it is notable that taller stature and higher caloric intake predicted earlier age at first sex, whereas greater adiposity was linked to later sexual debut. These results remained significant and were minimally changed after adjustment for maturational tempo, suggesting that early-life energy conditions also predict the timing of first sex through other pathways. One possibility is that larger body size and greater physical strength increase male intrasexual competitiveness, self-confidence, and social standing during the pubertal years, contributing to enhanced reproductive opportunities at younger ages for early-maturing males (James et al., 2012). Conversely, children with higher body fat tend to have smaller social networks (Daniels, 2006), and overweight-obese male teenagers start dating and having sexual intercourse at later ages (Cawley et al., 2006). While it is not presently clear whether these US-based results apply to young males in Cebu, they are generally consistent with our finding that overweight/obese adolescents tended to experience delayed sexual debut relative to their leaner peers. In that vein, it is important to acknowledge the salience of social and cultural factors in shaping the extent to which early sexual maturity and/or somatic characteristics related to favorable childhood energetics can affect the dynamics of sexual debut.

For example, recent research in Cebu found that males' sexual debut was influenced by the sexual behavior of their peer group (Gipson et al., 2014). As boys enter adolescence, and continuing into adulthood, these peer groups or *barkadas* take on increasingly significant roles for camaraderie and as outlets for male social activity. Because interactions in those groups can often involve overt competition, such as being intertwined with physical activities like competitive sports (Dumont, 1993; Guggenheim, 1994; Cagas et al., 2014), this could provide a pathway through which being taller and stronger could increase male social standing and dominance. However, the Philippines is also a predominately Catholic society, in which cultural norms regarding sexual behavior before marriage are traditionally conservative. Families have historically exerted much control over young adults' dating behaviors and romantic opportunities (Medina, 2001). While this has changed somewhat in recent decades (Medina, 2001), these conservative sexual mores likely increase the time gap between pubertal maturation and onset of sexual activity, compared to what might be observed in a less conservative setting.

Finally, favorable early-life energetic exposures may also affect neurobiological-endocrine networks (e.g., the hypothalamic-pituitary-gonadal axis that produces testosterone) that have implications for reproductive and competitive behaviors (Kuzawa, 2007; Hinde and Capitanio, 2010; Kuzawa et al., 2010; Hinde, 2013; Gettler, 2014). For example, in Cebu, we have previously shown that males who experience rapid weight gain during infancy have higher testosterone during adulthood and are more sexually active (Kuzawa et al., 2010) and that young adult single men with elevated testosterone are

*TABLE 4. Survival analysis relating the age that men first become fathers to developmental experiences (*n = *754)*[a]

| | Model 1 | | *P* | Model 2 | | *P* | Model 3 | | *P* | Model 4 | | *P* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sibling death | 1.36 | (1.05, 1.76) | 0.021 | 1.37 | (1.05, 1.78) | 0.020 | 1.36 | (1.05, 1.77) | 0.022 | 1.34 | (1.02, 1.77) | 0.039 |
| Paternal instability | 1.37 | (0.97, 1.94) | 0.074 | 1.36 | (0.96, 1.92) | 0.085 | 1.35 | (0.95, 1.91) | 0.091 | 1.36 | (0.96, 1.93) | 0.086 |
| Maternal absence | 1.03 | (0.60, 1.79) | 0.904 | 1.02 | (0.59, 1.76) | 0.944 | 1.02 | (0.59, 1.76) | 0.944 | 1.01 | (0.58, 1.75) | 0.973 |
| Height for age *z*-score | | | | 1.03 | (0.92, 1.15) | 0.611 | 1.03 | (0.92, 1.16) | 0.582 | 1.04 | (0.92, 1.17) | 0.550 |
| Triceps skinfolds | | | | 0.95 | (0.85, 1.06) | 0.389 | 0.96 | (0.85, 1.07) | 0.432 | 0.96 | (0.86, 1.07) | 0.446 |
| Kcal/kg | | | | 1.04 | (0.95, 1.15) | 0.400 | 1.05 | (0.95, 1.16) | 0.355 | 1.05 | (0.95, 1.16) | 0.361 |
| Household income[b] | | | | | | | 0.97 | (0.88, 1.08) | 0.635 | 0.98 | (0.88, 1.09) | 0.656 |
| Number of siblings | | | | | | | | | | 1.01 | (0.95, 1.08) | 0.672 |
| Birth order | | | | | | | | | | 0.99 | (0.92, 1.06) | 0.765 |

[a] Values are hazard ratio (95% CI). Childhood height, skinfold thickness, and calorie intake were measured at 8.5 ± 0.04 years and modeled as SD scores.
[b] Household income at age: 16.1 (± 0.3) years. Modeled as a SD score.

more likely to become fathers in their 20s, compared to men with lower testosterone (Gettler et al., 2011). In general, the identification of the mechanistic neurobiological-endocrine pathways through which developmental nutritional and social contexts (e.g., Flinn et al., 1996) might influence later reproductive behavior would lend further empirical support to the model and is a worthwhile target for future research in this area.

In contrast to the effects of favorable childhood nutrition, we found mixed evidence for an accelerating effect of developmental psychosocial stressors on life history schedules. Counter to the notion that social cues of high extrinsic mortality speed up life history tempo (Chisholm, 1993; Ellis et al., 2009), men who as children experienced sibling death—perhaps the most direct and reliable cue available of local mortality risk—did not mature earlier or initiate sexual activity at younger ages, compared to other subjects. Individuals raised in households with an unstable paternal presence did not exhibit accelerated maturity but tended to have earlier ages of first sex (see further discussion below).

The predictors of age at fatherhood in Cebu were generally concordant with past work documenting an accelerating effect of psychosocial cues of environmental risk or of low paternal investment (Draper and Harpending, 1982; Belsky et al., 1991; Chisholm, 1993; Ellis et al., 2009; Nettle et al., 2010), although the effect sizes for our findings were modest. In Cebu, there was a trend for males who grew up with an unstable paternal presence to become fathers sooner than those who experienced paternal stability. Cebuano fathers commonly play important authoritative, disciplinarian, and provider roles and divorce/remarriage is uncommon (Medina, 2001; Hindin, 2005; Harper, 2010), suggesting that the experience of paternal instability could be stressful or socially challenging during childhood. Given the important role of Filipino fathers in their sons' socialization, Cebuano boys growing up without fathers may have social developmental experiences that predispose them toward younger ages at sexual debut and earlier transitions to fatherhood, perhaps through increased risk-taking behavior (Ellis et al., 2009), such as not using family planning methods to prevent pregnancy. For example, a nationally representative study of Filipino young adults found that individuals with strict fathers were significantly less likely to engage in premarital sex, and that boys experiencing paternal instability had less at-home supervision during adolescence (Cruz et al., 2001; Medina, 2001; Marquez, 2008). While these socialization experiences might be pathways through which life history strategy is calibrated, specifically in the cultural context of Cebu, the correlation between paternal instability and sons' later reproductive behaviors could also reflect alternative explanations, such as father–son shared genetic effects on behavior. Presently, we cannot distinguish between these possibilities.

Notably, we found no effects of maternal absence on any life history variables in this sample. Although humans exhibit plasticity in response to variation in maternal care (Wells, 2014), there was likely little opportunity for selection to shape plastic life history responses to prolonged maternal *absence* during hominin evolution due to the high likelihood of child mortality (Sear and Mace, 2008). Given prior research showing that generalized measures of psychosocial stress predict men's ages at first sex and the transition to fatherhood (Nelson, 2004; Hawes et al., 2010), it is somewhat surprising that the stressor of maternal absence does not predict life history measures among Cebu males.

We found that subjects who experienced the death of a sibling became fathers at younger ages, independent of the nutritional status variables that are strongly linked to maturational tempo and sexual debut in this sample. It is notable that this outcome is consistent with observations in populations in which mortality cues tend to co-occur with nutritional abundance and overweight (males: Jaffee et al., 2001; Sheppard and Sear, 2012; females: see review in Ellis et al., 2009). Our findings are broadly consistent with the idea that psychosocial stress can accelerate aspects of life history tempo (Ellis et al., 2009), such as the timing of parenthood, although the effect sizes we documented for the "rate of acceleration" were modest, given humans' long lifespans. There is often a substantive lag between the timing of sexual debut and the transition to parenthood, reflecting the influence of a range of social, cultural, and economic pathways (Pears et al., 2005; Hofferth and Goldscheider, 2010; Scelza, 2010). Our results specifically suggest that the transition to fatherhood in Cebu is relatively independent of any effects of developmental energetics on maturational tempo.

Our study has limitations that merit discussion. The structure of the surveys, particularly the ~6.5 year gap between the infancy and mid-childhood surveys, and the fact that few individuals experienced the death of a sibling between ages 8.5 and 11 years, prevented us from pinpointing the timing of men's exposure to paternal absence or sibling death. Thus, we were unable to distinguish whether there are key sensitive periods when these exposures have greatest impact on life history variables (Wells, 2014). In addition, although some psychosocial variables were significant predictors of age of

sexual debut or entry into fatherhood, the magnitude of predicted change was often small. For example, men who experienced sibling death became fathers roughly 0.9 years earlier than individuals whose siblings did not die. Given human's long reproductive careers, this difference might have relatively modest fitness implications. However, the relationships that we document are stronger than those reported in other similar studies of other populations (Kuzawa and Bragg, 2012), and likely underestimate the true effects of mortality cues, given the coarse nature of our available proxy measures.

In summary, our findings raise questions about the adaptive significance of psychosocial acceleration of puberty in males, which is primarily nutritionally constrained. In this sample, age at first sex is relatively strongly tied to nutritional influences and also likely influenced by corollaries of physical maturity and paternal presence or investment. Our finding that age at fatherhood was accelerated by psychosocial conditions, but was unrelated to childhood energetic conditions, also suggests that other behavioral milestones are less biologically/energetically constrained, particularly in males, and may be plastically shaped by local social and cultural dynamics. Compared to life history events tied to the slowing effects of early-life energetic exposures, there may have been greater opportunity for adaptive linkages to emerge between this milestone and responsiveness to psychosocial cues.

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