9-2018

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Published in *Physiology & Behavior* 193 (Part A) (September 2018), pp. 82–89;
doi: 10.1016/j.physbeh.2017.11.029

Submitted July 3, 2017; revised November 27, 2017; accepted November 28, 2017; published online November 29, 2017.

Supplemental information follows the references.

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Abstract

Early life cues of environmental harshness and unpredictability have been hypothesized to influence within-species variation in the timing of life history transitions and the dynamics of reproductive strategies, such as investments in mating and parenting. It is also believed that adolescence is an influential developmental period for male reproductive strategies, with those who achieve greater social and sexual success during that period maintaining faster life history strategies into adulthood. If correct, such early life and post-pubertal experiences could also help shape the psychobiological pathways that mediate reproductive strategies, including the well-documented physiological shifts

that occur when some men become parents. Drawing on a large sample of Filipino men (n = 417), we evaluate whether men who experienced cues of harshness or unpredictability in childhood or have earlier ages at sexual debut have elevated testosterone (T) as fathers. We also test whether males who experienced a combination of early life experiences of harshness or unpredictability and had earlier ages of sexual debut during adolescence had the most elevated T as fathers. We found that fathers who experienced early life harshness and who engaged in sex at an earlier age had elevated waking T. Among men transitioning to fatherhood across the 4.5-year follow-up period of this study, those who experienced unpredictability and who engaged in sex at an earlier age showed attenuated declines in waking T between baseline and follow-up. Complementing these findings, we found that fathers who first engaged in sex at later ages had greater acute declines in T when they played with their toddlers. We suggest that these patterns could reflect programming effects of sociosexual experiences during the years following the marked biological transitions that accompany puberty, which occur along with the better-studied effects of earlier life exposures to stressors. Overall, our results support the hypothesis that early life circumstances and social and sexual experiences, from early life to young adulthood, help calibrate physiological axes as key mechanisms coordinating dynamic life history strategies.

Keywords: testosterone, acute reactivity, life history theory, fatherhood, developmental plasticity

1. Introduction

Evidence from research in diverse cultural settings indicates that human male biology has the capacity to flexibly respond to the transition to marriage and fatherhood with hormonal changes [10, 14–16, 20, 22, 28, 29, 41]. Lower testosterone (T) in fathers has been linked to greater participation by fathers in direct childcare, heightened affectionate contact, and attunement in interactions with children as well as greater sensitivity to infant cries [16, 20, 24, 40, 60]. Fathers also exhibit acute hormonal reactivity to parenting that is both context- and stimuli-dependent. For example, fathers' T spikes when they listen to recorded infant cries [16], and men's ability to soothe infant crying and distress in such contexts might contribute to acute T declines [34, 57].

It is hypothesized that aspects of these physiological adjustments, which are interpreted as helping enable invested caregiving among fathers, reflect past selective pressures on human biology during hominin evolution [23, 26, 30, 31, 48, 55, 56, 58, 62, 63]. Yet, human paternal psychobiology is not a rigid feature of human biology. Rather, it is a flexible and facultative capacity. Cultural norms and expectations of fathers vary widely across societies, which contributes to variation in male psychobiological responses to fatherhood [23, 26, 58]. For instance, T declines with fatherhood in settings in which males are commonly involved with childcare [10, 20]. In contrast, fathers' T was comparable to that of non-fathers in a polygynous society in which there is minimal direct paternal care [41]. Accordingly, it is widely assumed that men's familial and social behaviors and experiences influence these patterns, contributing to population-level differences between groups as well as local, within-group variation in the psychobiology and neurobiology of fatherhood [23, 26, 58, 64, 65]. As noted above, there is evidence to support this proposition. Within societies, T is lower among fathers who report spending more time in direct care [20, 40, 60] or who co-sleep with their children [21, 39].

In addition to the well-documented effects of a man's current partnering and parenting roles, early life experiences could also play a role in shaping the nature of a male's bio-behavioral response to becoming a father [15, 26, 58], as has been shown in a number of other mammalian species in which fathers care for young [4]. The capacity for early environmental experience to have lasting effects on neural development and physiological function are examples of developmental plasticity [18, 36, 61], and may have particular relevance for understanding both cross-cultural and within-societal variation in the bio-behavioral transitions that accompany fatherhood [26]. Evolutionary and life history theoretical approaches to developmental plasticity assume that organisms have an ability to adaptively calibrate their behavior and allocation priorities to their early ecological niche [2, 8, 13, 18, 36, 61], and in particular to environmental cues that correlate with extrinsic mortality, which predicts life history pace across species [45]. Broadly, there is support for the notion that certain stressful psychosocial conditions during early life can lead to earlier human maturation (only when paired with energy abundance) and accelerated reproductive scheduling [2, 7, 11, 25, 36, 38, 46, 53, 54].

Cues reflecting environmental harshness and unpredictability are key dimensions of ecological risk theorized to influence the life history trajectory that an individual adopts [2, 13, 53]. Harshness, which reflects levels of extrinsic mortality and morbidity, is commonly operationalized through measures of socioeconomic status (e.g., [2, 53]), because most prior work has focused on populations in which extrinsic mortality rates are low. Less commonly, harshness is characterized through more direct measures of local mortality rates, such as death of a sibling [25] or community-level infant mortality [46]. Frequently used markers of environmental unpredictability, defined as spatiotemporal variation in harshness, commonly include traits like fathers' absence or instability in paternal presence, variation in the quality of parental investment, and childhood household moves [2, 13, 53]. Consistent with existing theory [13], recent findings suggest that experiences of harshness and unpredictability contribute unique effects to an accelerated life history strategy, such as greater number of sexual partners, earlier sexual debut, and earlier entry into parenthood [2, 25, 53].

Evidence that these early experiences influence the timing and pace of human reproduction raises the question of whether they might have long-term impacts on the endocrine axes that influence the partitioning of male reproductive effort between mating and parenting, such as the hypothalamic-pituitary-gonadal (HPG) axis that produces T. As initial support for this idea, prior research has shown that exposure to harshness and unpredictability during early life has durable effects on neuroendocrine function, including the HPG axis, during childhood and into adulthood [11, 17]. Early experiences likely also shape cognitive and affective processing of social cues [42], which can influence the output of axes such as the HPG. These neuroendocrine pathways then contribute to psychosocial functioning and social behavioral development across childhood and young adulthood.

Experiential impacts on endocrine set points are not limited to the earliest life stages, but continue into later life. This is especially true during the marked biological transitions that accompany puberty, including the dramatic increase in circulating T among males, which has effects on neural development and connectivity [43, 47]. These programming

effects of T during adolescence can affect social behavior [52], but social context also influences the magnitude of those effects. Because male reproductive success tends to be heavily influenced by intra-sexual competition and status attainment, it is thought that males who achieve greater social and sexual success during early adolescence may be inclined to maintain a faster life history strategy into adulthood [8, 33]. Consistent with this perspective, males with earlier ages of sexual debut also have sex more frequently and with a greater number of partners [50, 51]. Less considered is whether social and sexual success following puberty contributes to psychobiological function in the long-term.

Here, we explored a series of questions aimed at clarifying the potential role of developmental exposures during early life and adolescence as influences on male psychobiology, particularly in the context of parenting. Working with data from a longitudinal birth cohort study in Cebu, the Philippines, we tested three primary hypotheses. First, we tested whether men who experienced cues of harshness or unpredictability in childhood had elevated T as fathers. Second, we tested whether males who had an earlier age at sexual debut, consistent with a "faster" life history pace, had comparatively heightened T as fathers. Third, we tested for interactions between childhood harshness or unpredictability and age at first sex in predicting men's T as fathers. For example, we specifically hypothesized that if males experienced early life harshness *and* were younger when first having sex in adolescence they would have elevated T as fathers, compared to men with other combinations of harshness and adolescent sexual experiences. Complementing these longer-term perspectives, we drew on a smaller subsample of Cebuano fathers to test whether young adult sociosexual experiences correlated with short-term T hormonal reactivity during parent-child interaction. Although few of these men experienced early life cues of harshness/unpredictability, there was variability in age at sexual debut in this group. Using these data, we hypothesized that men who experienced a younger age at sexual debut would exhibit an attenuated decline in T while playing with their children.

2. Materials and methods

2.1. Study population

Data from adult males were collected in 2005, 2009, and 2010 from men (n = 417) enrolled in the Cebu Longitudinal Health and Nutrition Survey (CLHNS), a population-based birth cohort that began in 1983–84 [1]. The infants enrolled in the study in 1983–84 have been followed up at multiple waves of data collections between their births and 2010. Our measures of early life harshness and unpredictability include data collected between 1983–84 and 1994 [1, 25]. At the time of data collection in 2005, men were 21.5 ± 0.3 (SD), in 2009 men were 26.0 ± 0.3 (SD), and in 2010 men were 26.6 ± 0.3 (SD) years old. Using questionnaire-based, in-home interviews administered by Cebuano-speaking interviewers, data collected included socioeconomic, demographic, health, and general behavioral information. All research was conducted under conditions of informed consent with human subjects' clearance from the Institutional Review Boards of the University of North Carolina, Chapel Hill and Northwestern University.

From the original cohort of 1633 live-born males, 1008 (2005) and 908 (2009) males were located and interviewed for the 2005 and 2009 surveys, respectively. For the 2010 father-

toddler interaction study, subjects were eligible if they were living with at least one biological child (older than 1 year of age and less than 4 years of age) and the mother of that child, had no adopted or stepchildren, and had full data from both the 2005 and 2009 CLHNS surveys. A total of 45 men agreed to participate in the interaction study.

2.2. Early life unpredictability and harshness

Men were defined as having experienced early environmental harshness if they were exposed to the death of a sibling (n = 77; 18.6%) at the age of 11.5 ± 0.4 (SD) years old or younger [25]. They were characterized as experiencing environmental unpredictability if they grew up with paternal instability (n = 50; 12.1%), which was defined as having a father who was deceased or absent, a mother who was unmarried during the males' first year of life or beyond, or mother who remarried during the males' childhood or juvenile period, measured up to age 11.5 ± 0.4 (SD) years [25]. If the original mothers (1983–84) were cohabitating with a partner but not legally married, those partners were recorded as spouses during interviews, thus "unmarried" in this context refers to mothers without a long-term partner in their household.

2.3. Age of sexual debut

The age at which men first had sexual intercourse was used as an indicator of sexual activity during adolescence and young adulthood. In 2009, participants reported their age of sexual debut. We have previously shown that across the full sample, which includes both fathers and non-fathers, men who experienced childhood paternal instability had sex at younger ages, whereas their experiences of sibling death were not predictive of significantly earlier sexual debut [25].

2.4. 2005 and 2009 saliva collection protocol

Participants were given instructions and two polypropylene tubes for saliva collection. They were asked to collect the first sample immediately before bed (PM), with mean sampling times of 10:31 PM ± 2:35 (SD) in 2005 and 10:05 PM ± 2:15 in 2009. They then collected the second sample immediately on waking the following morning (AM). Mean AM sampling times were 6:40 AM ± 1:50 in 2005 and 6:43 AM ± 1:53 in 2009. All 2005, 2009, and 2010 (below) samples were transported to the University of San Carlos in Metro Cebu (Philippines) where they were frozen at –35 °C. All samples were later shipped on dry ice to Northwestern University, where they were stored at –80°C.

2.5. 2010 father-child interaction protocol

Interviewers arrived at the subjects' homes in the early afternoon. In the first hour of the home visit, men gave consent for the study and participated in the questionnaire-based interview. After this preliminary hour, men provided an initial set of saliva samples, and then were asked to play with their child for up to 30 min. Fathers were instructed to use a medium-sized plastic ball for the interaction. Following the interaction, interviewers continued with the interview process, collecting additional samples timed to 30 min and 60 min after the initial baseline sample.

2.6. 2010 saliva collection protocol

Prior to the father-child interaction, men provided a saliva sample in a polypropylene tube collected by the interviewer following standard collection procedures. The average time for the first saliva collection was 1:46 PM ± 43.6 min. Upon completion of the sample collection, the interviewer set a timer for 30 min during which fathers were asked to play with their child. The duration of the father-child interaction averaged 24.1 ± 5.5 (SD) minutes (range: 15–30 min). When the 30-min timer finished, men provided a second saliva sample. The average time between baseline and second samples was 41.4 ± 4.0 min. The subjects again provided a subsequent saliva sample 30 min later, targeted to 60 min after the first sample (73.9 ± 6.9 min after baseline). We calculated percentage change in T between samples 1 and 2 and 1 and 3, respectively.

2.7. Salivary T (2005, 2009, and 2010)

Salivary T (pg/mL) assays were run at the Laboratory for Human Biology Research (LHBR) at Northwestern University using enzyme immunoassay protocols developed for use with saliva samples (Salimetrics, State College, PA; T: Kit No. 1-2402). Interassay coefficients of variation were 13.7% and 11.5% for high and low kit-based control samples, respectively, for 2005 T, 7.8% and 17.9% for high and low control samples, respectively, for 2009 T, and 6.4% and 7.2% for high and low control samples, respectively, for 2010 T. One male was eliminated from these analyses on the basis of a T value that was 6 + SD above the mean, indicating potential blood contamination. A small number of males had waking T values but not evening T, thus sample sizes for waking T analyses are slightly larger.

2.8. Potential covariates

For each set of analyses, to avoid over-fitting the models, we separately considered theoretically motivated potential covariates derived from psychosocial acceleration frameworks, life history theory, and psychobiological theory. We retained covariates in the final models if they were correlated ($p < 0.1$) to our measures of T (our dependent variables) and either harshness, unpredictability, or age of sexual debut (our core independent variables). For harshness, unpredictability, and age of sexual debut, we report the full model results for these covariate analyses in Supplemental Table 1. Relevant results for T are indicated in Supplemental Table 1 and full model results are generally found in Gettler and colleagues' prior work on these data [19, 20, 21, 22, 24, 27].

We used men's educational attainment, which they reported in 2009, as a marker of adult SES, and controlled for it in relevant models in Results. As measures of childhood SES, maternal education (the men's own mothers) and household income (adjusted for inflation) were averaged from multiple data collections between 1983 and 1994 [1]. Neither childhood SES variable met the criteria to be included as covariates. In our prior, related work on this topic [25], we found that males with better long-term childhood energetic status, indexed by height-for-age *z* scores (HAZ), had earlier ages of sexual debut (see [25] for review, including calculations of HAZ). Early life growth and energetics may also help calibrate HPG axis function and is linked to adult T production in men from this sample [35]. Here, we found no significant relationships between childhood HAZ and men's T (indicated in Supplemental Table 1).

As potential covariates related to familial demographics and men's parenting, we tested men's marital status, age of oldest and youngest child, total number of children, daily paternal care [20, 27], residence status (whether fathers resided with their children or not), and cosleeping (whether fathers coslept with their children or not; [21]) (see Supplemental Table 1). In relevant models, we controlled for marital status, daily paternal care, residence status, and the ages of fathers' youngest and oldest children (see Supplemental Table 1 and Results). In addition, as potential covariates for the 2010 short-term reactivity analyses, we considered fathers' educational attainment, number of children, ages of their oldest and youngest children, and daily paternal care, as well as the total time of the observed interaction with the child. None of these were correlated with T reactivity at $p < 0.1$, and therefore were not included as covariates in the models.

3. Statistical analyses

Analyses were conducted using version 14 of Stata (Stata Corporation, College Station, Texas). In our core analyses, we treated T (pg/ml), longitudinal change in basal T (2005 to 2009), percent change in T (2010), age of sexual debut, men's educational level, and ages of men's children as continuous variables.

All hormonal measures were adjusted for the time of sampling prior to analysis and prior to the calculation of change scores (i.e., 2005 to 2009 for basal T; reactivity change scores for 2010 T). These adjustments were conducted by separately regressing the hormone on the time of sample collection, predicting the model's residuals, and adding the original dependent variable's mean to the residuals, which removes the effect of the independent variable on the dependent variable [21]. Similarly, we analyzed change in men's T between 2005 and 2009 as "baseline adjusted" (i.e., we removed the effect of baseline T). These adjustments were conducted by separately regressing change in AM and PM T on their baseline values, predicting the model's residuals, and adding the original dependent variable's mean to the residuals [22].

To test whether age of sexual debut and unpredictability/harshness interacted to predict differences in basal T after men became fathers and across the transition to fatherhood, we conducted a series of OLS regression models with separate interaction terms for (paternal instability × age of sexual debut) and (sibling death × age of sexual debut). In the absence of significant interactions, we ran a subsequent model without interaction terms, predicting T from paternal instability, sibling death, and/or age of sexual debut as main effects that were not conditional on interaction terms.

Using that framework, we first tested for differences in 2009 T for all men who were fathers in 2009. In the next set of models, we then focused on men who were non-fathers at baseline and became fathers by follow-up and predicted their change in T over the study period. For all models we conducted analyses with relevant covariates, as described above and indicated in Supplemental Table 1.

Finally, we examined whether age of sexual debut predicted reactivity in T to father-toddler interaction using OLS linear regression. T reactivity scores (percent change) from before the interaction began to 30-min later and before the interaction to 60-min later were the dependent variables. Because of the smaller subsample size for these reactivity data

(n = 45), and limited statistical power, we did not conduct moderation analyses (harshness/unpredictability × age at sexual debut) for these hormonal outcomes.

4. Results

Among men in this sample, 29.5% had experienced harshness and/or unpredictability, measured through the death of a sibling or paternal instability, respectively (Table 1). On average, they first had sexual intercourse at 18.0 (± 2.6) years of age. As fathers, the large majority were married/cohabitating (~90%) and living with their children (~87%). Fathers reported being relatively involved with day-to-day care of their child, with more than 70% reporting that they spent more than one hour in daily childcare.

4.1. Models focusing on fathers at follow-up (2009)

In models predicting fathers' AM T at follow-up, there was a significant interaction for age at sexual debut × sibling death (p = 0.03; Table 2). Fathers who experienced sibling death and who engaged in sex at an earlier age had elevated AM T, on average, while fathers who had a sibling die and who were older when they first had sex had comparatively reduced T (Fig. 1). In a subsequent model without interaction terms, there was a main effect of paternal instability on AM T, though it did not reach statistical significance (p = 0.08; Table 2). There were no significant interactions or main effects for PM T (p's > 0.1).

4.2. Models focusing on men who became fathers between baseline and follow-up

For men who transitioned to fatherhood between 2005 and 2009, there was a significant interaction (age at sexual debut × paternal instability) predicting men's change in AM T (p = 0.02; Table 3). New fathers who experienced paternal instability and who engaged in sex at earlier ages showed attenuated declines in AM T over the follow-up period, compared to other new fathers (Fig. 2). The interaction for age at sexual debut × sibling death was not statistically significant (p = 0.08). However, the predicted results for change in AM T were in the same direction as the significant results above for 2009 AM T, as new fathers who had a sibling die and who were older when they first had sex tended to exhibit large, biologically meaningful decreases in T, compared to other new fathers. For change in AM T, there was no significant main effect for sibling death (p > 0.5). There were no significant interactions or main effects for change in PM T (p's > 0.1).

Table 1. Descriptive statistics for the total sample[a,b]

	2005–2009 sample (n = 413)		2010 reactivity sample (n = 45)	
Demographic characteristics	Mean	*SD*	Mean	*SD*
Age (years)	26.00	0.31	26.63	0.27
Education (highest grade)	10.36	3.19	9.00	3.06
Married/cohabitating (% y)	90.1%	—	100%	—
Father (% y)	100.0%	—	100%	—
Residential father (% y)	86.7%	—	100%	—
Number of biological children	1.57	0.80	2.33	1.02
Age of youngest child (years)	1.91	1.61	1.65	1.03
Age of oldest child (years)	3.18	2.17	4.45	2.15
Hours of daily childcare				
0–1 h	26.9%	—	11.1%	
1–3 h	42.1%	—	44.4%	
3+ h	31.0%	—	44.4%	
Cosleeping (among residential fathers)	94.4%	—	91.1%	
Childhood unpredictability and harshness				
Paternal instability (% y)	12.1%	—	7.3%	—
Sibling death (% y)	18.6%	—	17.4%	—
Experienced paternal instability or sibling death (% y)	29.3%	—	24.4%	—
Adolescent/young adulthood sexual activity				
Age at first sex (years)	18.00	2.63	17.58	2.25
Basal testosterone (T)				
AM T 2005[c] (pg/ml)	199.11	75.70	—	—
PM T 2005[c] (pg/ml)	126.46	54.67	—	—
AM T 2009 (pg/ml)	153.69	59.38	—	—
PM T 2009 (pg/ml)[d]	89.71	42.51	—	—
Reactivity T (2010)				
Baseline (B) T (pg/ml)	—	—	75.98	20.15
30 min T (pg/ml)	—	—	74.63	20.51
60 min T (pg/ml)	—	—	73.29	21.95
% change in T (B to 30 min)	—	—	0.44	21.69
% change in T (B to 60 min)	—	—	–1.75	23.05

a. See methods for definitions of categorical variables.
b. Values are from fathers at follow-up (2009) unless otherwise noted.
c. Values are restricted to the non-fathers at baseline (2005); n = 253 (AM T); n = 250 (PM T).
d. n = 412

Table 2. Predicting fathers' AM T at follow-up (2009) from paternal instability, sibling death, and age at sexual debut (n = 413)[a]

	Model 1			Model 2		
	b	95% CI	p	b	95% CI	p
Main effects[b]						
Paternal instability	108.10	(2.40, 213.81)	0.05	15.55	(–2.05, 33.15)	0.08
Sibling death	110.68	(–11.38, 0.65)	0.03	—	—	—
Age at sexual debut	1.58	(–1.09, 4.25)	0.25	—	—	—
Interaction terms						
Age at sexual debut × paternal instability	–5.39	(–11.38, 0.63)	0.08	—	—	—
Age at sexual debut × sibling death	–6.16	(–11.77, –0.64)	0.03	—	—	—
Covariates[b]						
Marital status	19.62	(–12.08, 51.32)	0.22	18.43	(–13.23, 50.08)	0.25
Daily paternal care						
1–3 h of care	–9.96	(–27.37, 7.45)	0.26	–9.33	(–26.76, 8.09)	0.29
3+ h of care	–2.04	(–20.41, 16.33)	0.83	–2.92	(–21.22, 15.38)	0.75
Residence status	–33.56	(–65.09, –2.02)	0.04	–31.85	(–63.32, –0.38)	0.05
Model R^2		0.054			0.036	

a. b (95% CI) from OLS regression.
b. Comparison groups for categorical variables: men who did not experience childhood paternal instability; men who did not experience sibling death; fathers who were not married/ cohabitating; fathers engaging in 0–1 h of daily childcare; fathers not residing with their children.

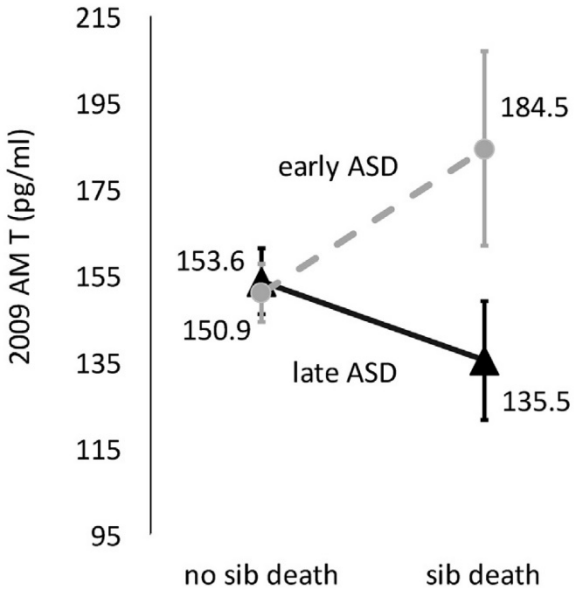

Figure 1. 2009 waking (AM) T among fathers (n = 413), stratified according to experiences of early life sibling (sib) death and age of sexual debut (ASD). For visual purposes, we categorize men as early ASD (–1 SD or lower) and late ASD (+1 SD or higher). We treated ASD as a continuous variable in our models (see Results and Table 2). The interaction term for (sib death × ASD) was significant (p = 0.03). Error bars indicate SE. The y-axis is centered on the mean and ranges from approximately –1 SD to +1 SD of 2009 AM T.

Table 3. Predicting new fathers' change in AM T from baseline (2005) to follow-up (2009) based on paternal instability, sibling death, and age at sexual debut (n = 253)[a]

	Model 1			Model 2		
	b	95% CI	p	b	95% CI	p
Main effects[b]						
Paternal instability	148.77	(31.63, 265.92)	0.01	—	—	—
Sibling death	99.00	(–14.00, 212.86)	0.09	–3.39	(–21.28, 14.51)	0.71
Age at sexual debut	1.77	(–1.37, 4.91)	0.27	—	—	—
Interaction terms						
Age at sexual debut × paternal instability	–7.53	(–14.00, 1.06)	0.02	—	—	—
Age at sexual debut × sibling death	–5.51	(–11.58, 0.57)	0.08	—	—	—
Covariates[b]						
Daily paternal care						
1–3 h of care	–12.08	(–34.27, 10.10)	0.28	–11.62	(–34.01, 10.77)	0.30
3+ h of care	–12.53	(–35.61, 10.55)	0.29	–13.56	(–36.68, 9.56)	0.25
Residence status	–22.78	(–48.94, 3.39)	0.09	–24.24	(–50.33, –1.85)	0.07
Model R^2		0.094			0.057	

a. b (95% CI) from OLS regression.

b. Comparison groups for categorical variables: men who did not experience childhood paternal instability; men who did not experience sibling death; fathers engaging in 0–1 h of daily childcare; fathers not residing with their children.

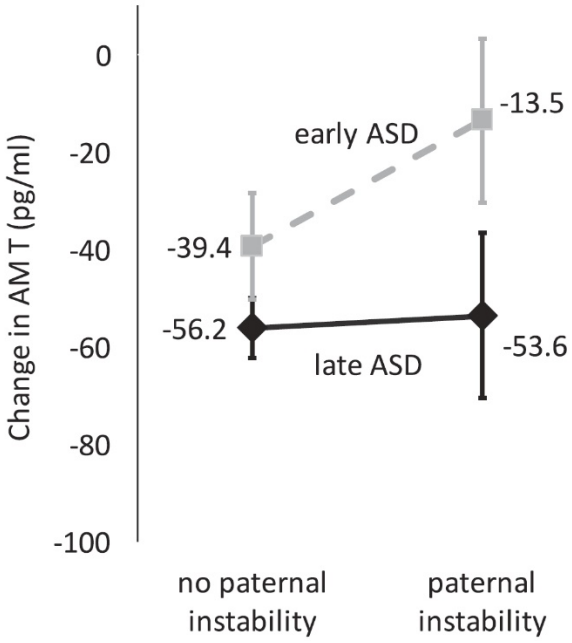

Figure 2. Change in waking (AM) T between 2005 and 2009 among men transitioning from being non-fathers (2005) to new fathers (2009) (n = 253), stratified according to experiences of childhood paternal instability and age of sexual debut (ASD). For visual purposes, we categorize men as early ASD (–1 SD or lower) and late ASD (+1 SD or higher). We treated ASD as a continuous variable in our models (see Results and Table 3). The interaction term for (paternal instability × ASD) was significant (p = 0.02). Error bars indicate SE. The y-axis is centered on the mean and ranges from approximately –1 SD to +1 SD of change in AM T. Values for change in AM T are adjusted for baseline T values (see Methods).

4.3. Assessing relationships between age at sexual debut and short-term T responses to father-child interaction

Finally, we tested whether T reactivity to father-toddler interaction was predicted by age at sexual debut. We found that fathers who had older ages of sexual debut had significantly greater decreases in T from baseline to 30 min later when interacting with their toddlers, compared to fathers who first had sex at younger ages (b = –3.30, 95% CI = (–6.06, –0.52), p = 0.021, model R^2 = 0.12; Fig. 3). Similarly, we found that fathers who first had sex when they were older had greater decreases in T from baseline to 60 min later (b = –3.00, 95% CI = (–6.01, –0.01), p = 0.051, model R^2 = 0.09; Fig. 3). Due to the sample size, we lacked statistical power to test whether age of sexual debut interacted with harshness or unpredictability to predict T reactivity.

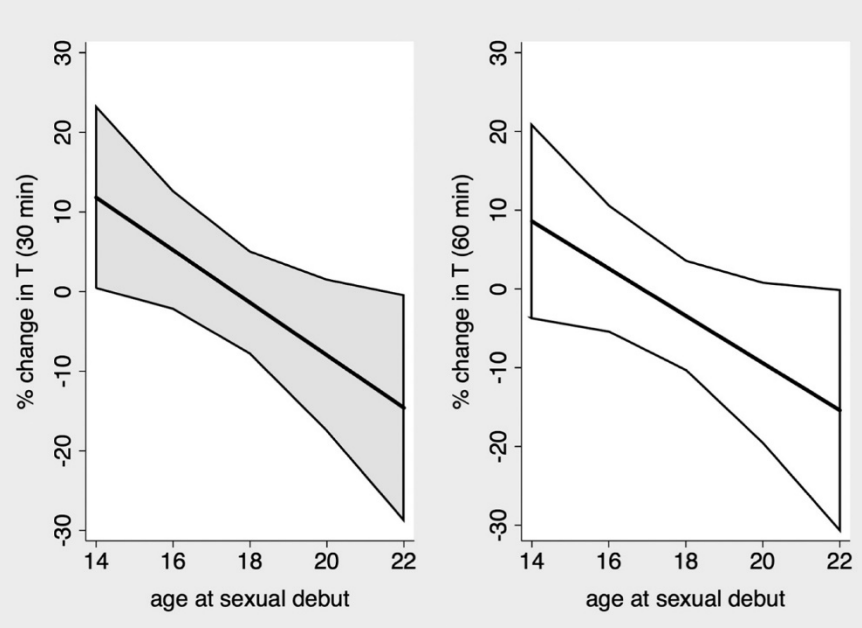

Figure 3. Percentage change in T during (30 min) and following (60 min) father-child interaction based on fathers' ages at sexual debut (n = 45). Percentage change reflects shifts in T between samples collected just before father-child interaction and 30 min and 60 min later. Men who had earlier ages at sexual debut tended to experience increases (or milder decreases) in T (see Results). CIs indicate 95% CI.

5. Discussion

There is growing interest in individual-level factors (e.g., personality, genetics) that predict differences in physiological mediators of life history strategies, including variability in human paternal psychobiology (e.g., [15, 20, 27, 32, 44]). Here, drawing on a long-running project in the Philippines, we present one of the few studies to examine how social experiences and behaviors before fatherhood, measured from infancy until young adulthood, relate to men's adult psychobiology as fathers. Specifically, we focused on whether indicators of environmental harshness and unpredictability and a marker of sexual activity in adolescence and young adulthood (i.e., age at sexual debut) interacted to predict elevated T among fathers and less of a decline in T when men transitioned to fatherhood. Our findings provide some support for these hypotheses.

Among all men who were fathers by follow-up, those who had experienced the death of a sibling and who had first engaged in sex at an earlier age had comparatively elevated waking T. Somewhat similarly, men growing up with paternal instability in childhood and who were younger at sexual debut also experienced less pronounced declines in morning T during the transition to fatherhood across the 4.5-year study period, relative to other new fathers. Broadly, our results suggest that experiencing unpredictability or harshness during childhood, when paired with heightened sexual activity in the years following pubertal maturation, is predictive of paternal T profiles that are consistent with faster life history

trajectories and reduced parenting effort. In that sense, these findings for T complement the social and behavioral patterns we have previously linked to paternal instability and sibling death at this site [25]. We do note that the follow-up T (all fathers) and change in T (new fathers) findings are somewhat divergent regarding interactions between harshness-unpredictability and post-pubertal sociosexual experiences in predicting paternal psychobiology. However, while not statistically significant, the (instability × age at sexual debut) interaction for 2009 fathers and (sibling death × age at sexual debut) interaction for new fathers are in the expected directions for AM T, hinting at commonalities across our analyses.

In contrast to the patterns above, fathers had lower waking T if they experienced the death of a sibling and they first engaged in sexual activity at a later age. In addition, among men transitioning to parenthood, fathers who experienced childhood paternal instability and had later ages of sexual debut experienced declines in T that were similar to men who had a stable childhood paternal presence and also had later ages of sexual debut. In other words, the potential T-elevating effects of childhood unpredictability were not observed for men who first had sex at older ages. When considered alongside the results above, these patterns may be consistent with developmental models that focus on individual differences in phenotypic plasticity and variable sensitivities to context [3, 12]. Potentially, there are between-male differences in responses to early life stressors, as some respond with a faster life history phenotype (earlier age of sexual debut; elevated T as fathers), while others, in response to similar experiences, exhibit a slower life history phenotype (later age of sexual debut; reduced T as fathers). Finally, though not directly comparable, our results for paternal instability do diverge somewhat from prior findings from a Caribbean population, in which men experiencing paternal absence during childhood had lower T in adulthood [17], thus pointing to the need for further exploration of the ecological- and cultural-contextual dependence of these effects. Overall, we think this type of developmental biosocial perspective merits further testing in future studies of paternal psychobiology and hope our contribution here spurs research in this area [26].

Our results linking early life harshness/unpredictability and age of sexual debut to paternal T are potentially complementary to Kuzawa and colleagues' [35] findings from this sample linking higher T production in adulthood to greater somatic growth during early infancy, which includes the "mini puberty" window of HPG activity in males. The authors speculate that favorable nutritional and growth conditions during this early sensitive period in HPG development helps calibrate HPG output for adult males [35]. The present findings highlight the potential role that early life social cues play in shaping paternal T, which is a mechanism that commonly mediates trade-offs between mating and parenting effort among fathers in a range of species with paternal care [23, 26, 30, 31, 48, 49, 55]. In both our results and Kuzawa et al. [35], the effects of developmental programming were restricted to waking T. Elsewhere, males with higher birth weight also had elevated morning T in adulthood, though evening T was not measured [59]. There are relatively few studies that focus on developmental programming of men's HPG function, but if the present findings are replicated, they may indicate that the effects of early life and childhood experiences on HPG function may be most pronounced for waking T production. Complementing that idea, we have previously hypothesized that the overnight rise in T, and T

levels upon waking, may be most important in facilitating the HPG's role in regulating the somatic component of male reproductive effort during sleep [37].

Focusing on the smaller father-child interaction subsample from this site, we found that the fathers who engaged in sex at an earlier age experienced increases in T (or comparatively attenuated decreases) during 30 min of playing with their child and afterward (60–70 min later). These patterns are generally complementary to the longer-term T (2005 and 2009) findings above, though we could not test similar interaction models because of the relatively small sample size of 45 participants. It is possible that men's faster life history experiences following pubertal maturation could have helped program the HPG axis, further influencing acute reactivity of T in later life when men became fathers. An alternative possibility is that trait-like differences between men that are consistent across the lifespan shape sexual activity in adolescence and young adulthood and similarly impact later life paternal behavior and psychobiology. Researchers have long recognized that individual-level differences in traits such as dominance and power motivations, sensation seeking, extraversion, and anxiety interrelate with or moderate the function of neuroendocrine axes, with implications for behavioral and endocrine variation [5, 6, 9, 44].

Although the longitudinal design is a distinct strength of our study, our analyses have several limitations that merit discussion. Notably, the specific early life experiences we evaluated here are not the only variables representing unpredictability or harshness in the environment (e.g., [2, 53].), and as such we cannot rule out that our results might have been different had we had other measures of harshness and predictability available. While not the central focus of our analyses, we did consider other relevant indices of early life experiences as covariates, such as two measures of childhood SES, and they were not significantly associated with men's T profiles. We also assessed exposure to unpredictability and harshness up until when males were 11–12 years old and did not distinguish between different periods of exposure during childhood. Recent work that made such timing distinctions found that exposure to harshness and unpredictability earlier in development had more profound effects on life history trajectories [53]. Unfortunately, our study is underpowered to conduct similar analyses. This is due to the 6–8 year gap between the infancy and mid-childhood surveys and the relatively few men who experienced paternal instability (~12%) or sibling death (~19%), particularly between ages 8.5 and 11 years.

In summary, we demonstrated that the interaction of early life harshness and unpredictability with sociosexual experiences during adolescence is linked to later-life paternal psychobiology. Our results specifically indicate that when males experience cues of harshness and unpredictability during childhood, post-pubertal sociosexual experiences are predictive of fathers' HPG axis production of T. We suggest that these patterns could reflect a programming effect of sociosexual experiences during the years following the marked biological transitions that accompany puberty. Alternatively, there may be individual differences between males in their responses to early life harshness/unpredictability that then influence both of these outcomes (sociosexual behavior and paternal psychobiology). These explanations are not mutually exclusive and both may make contributions to paternal physiology profiles. These novel insights on factors shaping individual differences in fathers' neuroendocrine profiles and acute reactivity are salient given general psychobiological frameworks for the importance of hormonal profiles and reactivity to behavior,

mood, and cognition and the significance of fathers to family function as well as child health and development outcomes.

Funding and role of the funding sources – Work supported by: Wenner Gren Foundation (Gr. 7356; Gr. 8186), National Science Foundation (BCS-0542182; BCS-0962212), the Interdisciplinary Obesity Center (RR20649), and the Center for Environmental Health and Susceptibility (ES10126; project 7-2004-E). LTG was supported by the Wenner-Gren Foundation's Hunt Postdoctoral Writing Fellowship, and MSS was supported by a National Science Foundation Graduate Research Fellowship. The funding sources played no role in the conduct of the research or the preparation of this article.

Acknowledgments – Linda Adair, Judith Borja, and Thom McDade played central roles in designing and implementing components of the CLHNS study from which a portion of these data and samples were obtained. Jeffrey Huang helped with lab work. We thank the Office of Population Studies, University of San Carlos, Cebu, Philippines, for their role in study design and data.

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Supplemental Information

Supplemental Table 1. Testing for relevant covariates related to sibling death, paternal instability, age at sexual debut, and T

	Sibling death			Paternal instability			Age at sexual debut			2009 T		Change in T (2005–2009)	
	Odds ratio	95% CI	p	Odds ratio	95% CI	p	Hazard ratio	95% CI	p	AM	PM	AM	PM
Participants' educated (2009)	0.84	(0.78, 0.90)	0.001	1.08	(0.98, 2.10)	0.12	0.96	(0.93, 0.99)	0.003				X
Participants' mothers' education	0.89	(0.82, 0.95)	0.001	1.05	(0.97, 1.13)	0.22	1.00	(0.98, 1.03)	0.86				
Childhood household income[a]	0.81	(0.52, 1.26)	0.36	1.03	(0.66, 1.59)	0.91	1.16	(1.02, 1.32)	0.03				
Childhood height-for-age[b]	0.72	(0.54, 0.97)	0.03	1.08	(0.77, 1.52)	0.64	1.09	(0.98, 1.21)	0.10				
Marital status in 2005	1.30	(0.77, 2.17)	0.32	0.99	(0.53, 1.85)	0.97	2.05	(1.65, 2.54)	0.001				
Marital status in 2009	1.22	(0.52, 2.85)	0.64	0.65	(0.27, 1.54)	0.32	0.76	(0.56, 1.04)	0.09	X	X		
Fatherhood variables (2009)													
High paternal care (3+ hrs per day)[c]	2.26	(1.13, 4.50)	0.02	1.04	(0.50, 2.17)	0.92	0.77	(0.60, 0.99)	0.04	X	X	X	X
Residence status	2.14	(0.88, 5.17)	0.09	0.43	(0.21, 0.89)	0.02	0.73	(0.55, 96)	0.03	X	X	X	X
Cosleeping[d]	1.47	(0.74, 2.94)	0.27	0.53	(0.27, 1.04)	0.07	0.72	(0.57, 0.93)	0.01	X	X	X	X
Age of youngest child[e]	0.88	(0.74, 1.04)	0.12	0.96	(0.80, 1.17)	0.71	1.19	(1.12, 1.26)	0.001				X
Age of oldest child[e]	0.99	(0.88, 1.11)	0.88	1.01	(0.88, 1.15)	0.92	1.27	(1.21, 1.33)	0.001		X		X
Number of children	1.19	(0.89, 1.59)	0.24	0.93	(0.64, 1.37)	0.72	1.49	(1.31, 1.68)	0.001				

a. Converted to *z* scores.
b. Height-for-age measured at age 8.5 (± 0.04) years in 1991.
c. Compared to fathers who reported engaging in 0–1 hours of daily childcare. Results not shown for fathers who reported 1–3 hours of daily childcare.
d. Cosleeping status is collinear with residence status so we did not include it in relevant models.
e. $n = 8$ fathers did not report their children's ages, hence analyses including these variables have slightly smaller sample sizes.