Evidence for an adolescent sensitive period to family experiences influencing adult male testosterone production

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Across vertebrates, testosterone is an important mediator of reproductive trade-offs, shaping how energy and time are devoted to parenting versus mating/competition. Based on early environments, organisms often calibrate adult hormone production to adjust reproductive strategies. For example, favorable early nutrition predicts higher adult male testosterone in humans, and animal models show that developmental social environments can affect adult testosterone. In humans, fathers’ testosterone often declines with caregiving, yet these patterns vary within and across populations. This may partially trace to early social environments, including caregiving styles and family relationships, which could have formative effects on testosterone production and parenting behaviors. Using data from a multidecade study in the Philippines ($n = 966$), we tested whether sons’ developmental experiences with their fathers predicted their adult testosterone profiles, including after they became fathers themselves. Sons had lower testosterone as parents if their own fathers lived with them and were involved in childcare during adolescence. We also found a contributing role for adolescent father–son relationships: sons had lower waking testosterone, before and after becoming fathers, if they credited their own fathers with their upbringing and resided with them as adolescents. These findings were not accounted for by the sons’ own parenting and partnering behaviors, which could influence their testosterone. These effects were limited to adolescence; sons’ infancy or childhood experiences did not predict their testosterone as fathers. Our findings link adolescent family experiences to adult testosterone, pointing to a potential pathway related to the intergenerational transmission of biological and behavioral components of reproductive strategies.

Significance

Testosterone influences how animals devote energy and time toward reproduction, including opposing demands of mating and competition versus parenting. Reflecting this, testosterone often declines in new fathers and lower testosterone is linked to greater caregiving. Given these roles, there is strong interest in factors that affect testosterone, including early-life experiences. In this multidecade study, Filipino sons whose fathers were present and involved with raising them when they were adolescents had lower testosterone when they later became fathers, compared to sons whose fathers were present but uninvolved or were not coresent. Sons’ own parenting behaviors did not explain these patterns. These results connect key social experiences during adolescence to adult testosterone, and point to possible intergenerational effects of parenting style.

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documented lasting effects of early social exposures on HPG function in other mammalian taxa (34–36), pointing to the plausibility of developmental calibration in humans as well.

Any effects of early social experiences on adult T production may be critical to individual variation in reproductive strategies because of T’s important role as a mediator of behavioral trade-offs between competition/mating and parenting effort (13, 37–39). For example, the transition to fatherhood is often accompanied by a decline in T in humans (13, 37, 38). Although rare in other mammals, in which paternal care is typically absent, this biological response is convergent with patterns observed in many bird species in which fathers cooperate with mothers to raise young. In birds, field-based studies have shown that experimental administration of T to males during periods of biparental care leads to substantial reductions in parenting behaviors, such as feeding (11). Instead, males’ behavioral priorities shift toward mate attraction and extra pair mating effort (11, 40). In humans, there is evidence that fathers with lower T provide more nurturant, direct care of children, engage in less marital conflict, and have higher relationship quality with their partners, compared to fathers who have higher T (28, 41–46). These patterns point to the importance of these hormonal shifts as social roles change across life-history transitions, as well as to potential fitness benefits through more cohesive partnerships and improved child health and development outcomes (13, 39, 47).

Although such adaptive modulation of T in the context of committed partnering and parenting has been documented widely in human populations, there is substantial variation in T levels across cultural settings based on fathering roles and marriage systems (37, 39). For example, fathers have been shown to have similar or even higher T compared to nonfathers in settings where fathering requires little direct childcare or where local marriage and family systems involve polygyny and male competition or risk taking (48–51). This cross-cultural variation in the psychobiology of fathering may partially trace to early-life contexts because children acquire information about their future family roles through social learning across our prolonged developmental period, including through their experience of relationships with their own caregivers (52). For example, longitudinal studies have shown that the quality of early-life father–son relationships and fathers’ own parenting styles influence sons’ future fathering, including their later involvement in direct paternal care (26, 27). Although studies have been rare, human research has also explored links between boys’ early social experiences within their families and their later T as adults. In Dominica, males who reported their fathers were absent during their childhoods had lower T than those whose fathers were consistently present (22). In the Philippines, males who experienced an unstable paternal presence during childhood tended to have less of a decline in T as first-time fathers, especially if they also had an earlier age at sexual debut (21). Similarly, in the United States, new fathers’ T rebounded faster in the postpartum period if they had an earlier age at sexual debut, though fathers’ childhood family experiences were not associated with their T during the parenthood transition in this setting (23).

Although this work generally supports the notion that early rearing experiences are predictive of adult T production in human males, to date few studies have attempted to tease apart the relative importance of the specific pathways or sensitive ages during development. In addition to any indirect effects of learned parenting behaviors on adult T, it is also plausible that early family environments and experiences could have direct programming effects on HPG axis function. The plausibility of a direct effect comes from animal models showing that early social experiences influence hormone levels before transitions to adult reproductive or parenting roles. In a range of mammalian species, early-life social contexts—such as being born to larger, denser groups or experiencing maternal separation—were linked to later activity of the HPG axis and androgen production during puberty and adulthood (34–36, 53–55). Although comparable work in humans is rare, one longitudinal study linked early-life poverty to lower adolescent T production (20), hinting at lasting effects of developmental social conditions on the HPG axis and T production that preceded any behavioral changes that would accompany adult parenting duties.

Here, we used data from a multigenerational birth cohort study to investigate whether childhood family experiences predicted sons’ T production in adulthood, including as fathers. We used longitudinal data to explore the possibility of both direct effects on adult T production and indirect effects on T, operating through learned parenting and partnering behaviors. We drew on a large sample of males (n = 966) enrolled in the Cebu Longitudinal Health and Nutrition Survey (CLHNS), which is an ongoing birth cohort study that began in Metropolitan Cebu (Philippines) in 1983 and that has involved a specific focus on male reproductive ecology and physiology since 2005. A key advantage of the study design is that it provides the opportunity to investigate the timing of sensitive period effects, with family-life data collected at multiple stages of the sons’ development (infancy, middle childhood, and adolescence) in addition to three surveys when they were adults (SI Appendix, Fig. S1). We used first-generation fathers’ caregiving roles, presence in the household, and relationships with their sons as predictors of the second-generation sons’ T. We first predicted sons’ T among never-married nonfathers surveyed in young adulthood, prior to major life-history transitions, to help assess whether any influences of developmental social conditions on the HPG axis are independent of endocrine changes that accompany becoming a father and engaging in caregiving in this population. We then predicted T among sons who had themselves transitioned to fatherhood. We included sons’ own parental care data in these models to test whether those behaviors explained associations between sons’ T and their own fathers’ behaviors.

Results

Key Descriptive Statistics. As we describe in Methods, we combined generation one (G1) fathers’ residence status and caregiving for each wave of developmental data (infancy, childhood, adolescence) into respective categorical variables. For each time point, we refer to this categorical variable as “fathering” at that specific wave (Table 1). Across the three infancy waves of data collection (1983 to 1986), it was most common for G1 fathers to reside with their infants (generation two, G2) and spouses and to have some involvement with childcare (~54% of G1 fathers). Later in G2 sons’ development, the most frequent G1 fathering grouping was for fathers to be coresident but not involved in childcare: G2 middle childhood (76%) and adolescence (62%).

We also created a second categorical variable for G2 adolescence that combined G1 paternal residence status with G2 sons’ perceptions of their upbringing. We refer to this categorical variable as “upbringing.” It was most common for G2 adolescents to attribute their upbringing to both of their parents and for their fathers to live with them in the same household
The interactions indicate a significant difference in the slopes of the lines comparing waking (AM) vs. evening (PM) T for the two relevant G1 fathering categories. Follow-up analyses showed that G2 sons whose fathers were not routinely coresident during G2 infancy had lower PM T compared to the reference category, including after the addition of covariates (coefficient [95% CI]: \(-0.28 [-0.50, -0.06]; P = 0.011\)) (SI Appendix, Table S1). The groups did not significantly differ for AM T (P > 0.3). The other categories for G1 fathering during infancy did not meaningfully differ from the reference group (main effects, P > 0.6) (SI Appendix, Table S1). There were also no significant associations between G2 middle childhood or adolescent experiences of G1 fathering and G2 sons’ T during young adulthood (all P > 0.1) (SI Appendix, Tables S2 and S3).

In complementary models for G2 sons’ adolescence, our core predictor was the categorical variable for upbringing (see description above), where the reference group was G2 sons who lived with their fathers and attributed their upbringing to both their parents. We found a significant interaction between G2 upbringing and AMPM (P = 0.035) (Fig. 1 and SI Appendix, Table S4). Compared to the reference group, G2 sons had higher AM T if during adolescence their fathers were not coresident and the sons did not attribute their upbringing to them (coefficient: 0.31 [0.04, 0.58]; P = 0.026) (SI Appendix, Table S4). The two groups did not significantly differ for PM T (P > 0.5). The other categories for G2 upbringing did not meaningfully differ from the comparison group (main effects, P > 0.3) (SI Appendix, Table S4). Please see SI Appendix, Table S4 for the full model results.

Table 1. Key descriptive statistics for the study sample (total n = 966)

| G2 males as fathers | G2 males as never married, nonfathers |
|---------------------|--------------------------------------|
|                     | n | Mean or percentage | SD | n | Mean or percentage | SD |
| G2 males’ T (pg/mL) (Total observations = 2,911) |   |                  |    |   |                  |    |
| AM T 2005           | 135 | 165.52 | 66.16 | 705 | 195.7 | 77.78 |
| PM T 2005           | 136 | 99.99 | 41.79 | 702 | 120.49 | 53.14 |
| AM T 2009           | 440 | 153.06 | 59.31 |   |                  |    |
| PM T 2009           | 439 | 90.29 | 41.63 |   |                  |    |
| AM T 2014           | 529 | 113.65 | 44.93 |   |                  |    |
| PM T 2014           | 528 | 67.15 | 26.27 |   |                  |    |

G2 experiences with their generation one (G1) fathers during development

| G1 fathering in G2 infancy (1983 to 1986) | % |  | % |
|-----------------------------------------|---|---|---|
| Father always coresident, some care     | 302 | 53.83 | — | 349 | 53.45 | — |
| Father not coresident, no care when present | 50 | 8.91 | — | 61 | 9.34 | — |
| Father not coresident, some care when present | 28 | 4.99 | — | 41 | 6.28 | — |
| Father always coresident, no care       | 181 | 32.26 | — | 202 | 30.93 | — |

| G1 fathering in G2 middle childhood (1994) | % |  | |
|-----------------------------------------|---|---|---|
| Father coresident and caregiver         | 65 | 11.15 | — | 76 | 11.26 | — |
| Father coresident, noncaregiver         | 445 | 76.33 | — | 512 | 75.85 | — |
| Father not coresident                   | 73 | 12.52 | — | 87 | 12.89 | — |

| G1 fathering in G2 in adolescence (1998) | % |  |  |
|-----------------------------------------|---|---|---|
| Father coresident and caregiver         | 122 | 21.18 | — | 158 | 24.09 | — |
| Father coresident, noncaregiver         | 362 | 62.85 | — | 398 | 60.67 | — |
| Father not coresident                   | 92 | 15.97 | — | 100 | 15.24 | — |

| G2 upbringing in adolescence (1998) | % |  |  |
|-----------------------------------|---|---|---|
| Father coresident, upbringing by both parents | 412 | 68.55 | — | 481 | 69.41 | — |
| Father not coresident, upbringing by both parents | 30 | 4.99 | — | 39 | 5.63 | — |
| Father coresident, upbringing by father | 16 | 2.66 | — | 25 | 3.61 | — |
| Father coresident, upbringing by others | 83 | 13.81 | — | 88 | 12.70 | — |
| Father not coresident, upbringing by others | 60 | 9.98 | — | 60 | 8.66 | — |

G2 ages by survey*

| Age | n | Mean or percentage | SD |
|-----|---|-------------------|----|
| 1994 | 583 | 11.49 | 0.42 | 675 | 11.46 | 0.41 |
| 1998 | 603 | 16.08 | 0.33 | 694 | 16.06 | 0.33 |
| 2005 | 137 | 21.54 | 0.31 | 707 | 21.47 | 0.29 |
| 2009 | 440 | 26.00 | 0.31 |   |      |    |
| 2014 | 529 | 30.52 | 0.35 |   |      |    |

*G2 infants were born between 1983 and 1984 and the initial survey wave between 1983 and 1986 encompassed their first 2 y of life (Methods).
Models for G2 Males’ T after They Had Transitioned to Fatherhood. We then predicted G2 sons’ T after they had become parents from their experiences of G1 fathering. G2 fathers’ T did not significantly differ based on G1 fathering during infancy (all P > 0.6) (SI Appendix, Table S1). In the model for middle childhood, there was a significant interaction between G1 fathering with AMPM (P = 0.030) (SI Appendix, Table S2). For this comparison, G2 sons whose fathers were not coresident during their middle-childhood period tended to have higher AM T than the reference group, with an effect size comparable to our other key results, but with wider confidence intervals that included zero (coefficient: 0.22 [-0.01, 0.45]; P = 0.062). After inclusion of covariates, the interaction term remained significant (P = 0.043); however, the effect size diminished slightly for the groups’ AM T comparison (coefficient: 0.21 [-0.03, 0.44]; P = 0.093) (SI Appendix, Table S2). The two groups did not differ for PM T (P > 0.3). Other G1 fathering group comparisons were not significant for middle childhood (main effects, P > 0.3) (SI Appendix, Table S2).

In models predicting G2 sons’ T from their adolescent experiences, there was a significant interaction between G1 fathering with AMPM (P < 0.05). Compared to the reference group, G2 fathers had higher PM T (P < 0.05) and AM T (P < 0.001) if their fathers were not coresident, but the effect size was larger for AM T (Fig. 2A, Table 2, and SI Appendix, Table S5). The PM T comparison was no longer significant after inclusion of covariates (P > 0.1) (Table 2 and SI Appendix, Table S5).

There was also a separate, significant main effect, as G2 fathers had higher T if their fathers were coresident but not involved in childcare in comparison to the reference group (P < 0.05). Please see Table 2 and SI Appendix, Table S5 for these full model results. In a post hoc analysis, we also included G1 fathering during middle childhood and adolescence, respectively, as predictors of G2 sons’ T. Adolescent experiences significantly predicted G2 sons’ T (both P < 0.01), whereas middle-childhood experiences did not (both P > 0.6) (SI Appendix, Table S6).

In models focusing on G2 upbringing during adolescence, there was a significant interaction between upbringing and AMPM (P ≤ 0.05) (Table 3). Compared to the reference group, G2 fathers had higher AM T if their fathers were not coresident and they did not credit them with their upbringing, including with covariates added (P < 0.001) (Fig. 2B, Table 3, and SI Appendix, Table S7). The two groups did not significantly differ for PM T (P > 0.1). Comparisons for the other categories of upbringing were not statistically significant (main effects, P > 0.2). Please see Table 3 and SI Appendix, Table S7 for the full model results.

Discussion

In this multidecade longitudinal study, sons who experienced father involvement in care during adolescence had lower adult T when they became fathers themselves, compared to sons whose fathers were less involved or were not coresident during adolescence. These associations were independent of sons’ adult behaviors that could influence T, including their involvement in romantic relationships, parenting, and sexual activity, as well as sons’ energetic condition during development and adulthood (14, 15, 24–33). Furthermore, links between adolescent experiences with their fathers and future T levels were already present among young adults who had not yet entered long-term relationships or become fathers. In contrast, sons’ experiences with fathers at earlier ages, during childhood and infancy, did not consistently predict the sons’ adult T. Taken together, these findings point to adolescence—an age of heightened HPG axis activity—as a potential sensitive period when experiences of paternal involvement could have lasting effects on adult production of T, a key regulator of energetic and behavioral components of adult reproductive strategies (9, 10, 12, 13, 37, 38).

Our results show that boys’ experiences with their fathers during development were predictive of their T in adulthood when they became fathers themselves. We found two complementary links between sons’ adolescent paternal contexts and their later-life T. First, boys whose own fathers lived with them and were involved in childcare during their adolescence had lower T as fathers, compared to those whose own fathers were not coresident during adolescence or who were coresident but not involved in care. Relative to the comparison group (father present and involved in care), the effect size for fathers not being coresident was nearly twice as large as for the father-present but noncaregiver group. These results are consistent with a graded effect of paternal involvement on adult HPG activity.

Second, we also found evidence for the importance of father–son relationships to these T dynamics. Sons had lower T as parents if their own fathers were present when they were teenagers and if the sons credited their fathers for their upbringing, when compared to sons whose fathers were not coresident and were not reported as responsible for their upbringing. We also found that sons who did credit their

Fig. 1. G2 sons’ T during young adulthood prior to life-history transitions, based on their paternal experiences during adolescence. The plots were derived using predictive margins (adjusted predictions) following model 2 in SI Appendix, Table S3. The two upbringing groups featured in Fig. 1 (n = 541) significantly differed for waking T (P = 0.026). Error bars indicate 95% CIs.
fathers with their upbringing, even though they were not coresident, had similar T as those whose fathers were present and considered responsible for their upbringing, pointing to paternal involvement, per se, as important. In the 1980s and 1990s, fathers in the Philippines played consequential, culturally valued roles in families, including as economic providers, moral and social guides to children, and gender-based role models (56, 57). Our results indicate that sons’ later T levels after they were parents themselves were predicted by their adolescent experiences related to these paternal influences and father–son relationships, which can occur under different father–son living arrangements.

Along with evidence for developmental influences on sons’ later T as fathers, their adolescent experiences similarly predicted their T when they were young adults, prior to their transition to marriage and fatherhood. At this life stage, sons whose fathers were parents themselves were predicted by their adolescent experiences related to these paternal influences and father–son relationships, which can occur under different father–son living arrangements.

![Fig. 2. G2 sons’ T after becoming fathers themselves, based on their paternal experiences during adolescence. The plots were derived using predictive margins (adjusted predictions) following model 2 in Tables 2 and 3, respectively. (A) The two fathering groups (n = 214) significantly differed for waking T (P < 0.001). Evening T did not significantly differ between the fathering groups, following adjustment for covariates (P > 0.1) (Table 2). (B) The two upbringing groups (n = 472) significantly differed for waking T (P < 0.001). Evening T did not significantly differ between the upbringing groups (P > 0.2) (Table 3). Error bars indicate 95% CIs.](https://doi.org/10.1073/pnas.2202874119)
were not coresident and were not considered responsible for their upbringing had higher T than sons with coresident fathers who were reported as shaping their upbringing. Since the sons were not yet parents, these findings indicate the association is independent of any effect of learned parenting behaviors on adult T levels. For this set of results that involve sons’ upbringing perceptions, the differences were restricted to waking T. In our prior work, we have likewise found that developmental effects on adult T in Cebu primarily occurred for waking T (14, 21) and other related studies have specifically focused on morning T (23, 58). We have previously suggested that these stronger developmental effects on waking T may be intertwined with HPG axis roles in somatic investment and trade-offs that occur during sleep, although the underlying biology requires further study and clarification (21, 59).

Collectively, these findings point to adolescence as a potential sensitive period for the development of the HPG axis in response to social inputs, and complement prior studies showing that better early-life energetic and growth conditions help calibrate adult HPG axis function (14, 15). Puberty is a period of brain maturation, when rising reproductive steroid levels influence domains of cognitive development, like motivation and decision making, and help prepare the individual for adult social roles (4, 60, 61). Our work contributes to this biosocial perspective by pointing to the likely influence that adolescent social experiences play in shaping adult production of T, a key coordinator of metabolic and behavioral components of adult reproductive strategies that also contributes to trajectories of survival and health (9, 10, 12, 13, 37, 38, 62–65). The effect sizes for our key findings suggest that these adolescent inputs to adult T are likely biologically meaningful for such life-history and health-related phenotypes. For example, the effect sizes we observed here exceed those documented in a recent large-scale meta-analysis linking fatherhood status to T (66; but see also ref. 43).

Because the associations that we observed were minimally changed after adjustment for measures of T-linked adult behaviors, our findings point to a possible direct, programming effect of adolescent experiences on adult HPG function. Research in other mammals is consistent with the idea that social environments prior to adulthood can have lasting programming effects on later T production, although the specific importance of adolescent social experience to later HPG axis function is understudied (34, 36, 53–55). In complementary research, experimental rodent models have shown that social stressors during adolescence contribute lasting, long-term effects on social behavior and brain development (hippocampus), potentially through endocrine (glucocorticoid) pathways (67). These are thought to result from heightened plasticity and sensitivity during the major biological transition that occurs during puberty (68). Related findings in humans indicate that the window of pubertal development is one of recalibration for stress-related physiology in response to current social circumstances (17). This lends support to the idea that social inputs to the developing HPG axis may likewise be important during this period of biological transition.

Table 3. Predicting G2 fathers’ T from their adolescent experiences with their own (G1) fathers: Paternal presence and G2 upbringing attributions (n = 601; Obs = 2,161)

| Model 1 | Model 2 |
|---------|---------|
| Coefficient | Bootstrapped 95% CI | P value | Coefficient | Bootstrapped 95% CI | P value |
| Father not coresident, upbringing by both parents | −0.02 | −0.25, 0.22 | 0.880 | −0.04 | −0.27, 0.19 | 0.744 |
| Father coresident, upbringing by father | −0.01 | −0.20, 0.19 | 0.955 | 0.02 | −0.18, 0.22 | 0.840 |
| Father coresident, upbringing by others | 0.05 | −0.10, 0.20 | 0.521 | 0.02 | −0.14, 0.17 | 0.841 |
| Father not coresident, upbringing by others | 0.12 | −0.05, 0.29 | 0.168 | 0.10 | −0.07, 0.28 | 0.234 |
| AMPM | −0.02 | −0.11, 0.06 | 0.570 | −0.02 | −0.11, 0.06 | 0.560 |

Interaction terms

- Father not coresident, upbringing by both parents × AMPM | 0.11 | −0.24, 0.46 | 0.547 | 0.11 | −0.24, 0.46 | 0.548 |
- Father coresident, upbringing by father × AMPM | 0.19 | −0.12, 0.50 | 0.232 | 0.19 | −0.12, 0.50 | 0.229 |
- Father coresident, upbringing by others × AMPM | −0.10 | −0.30, 0.11 | 0.344 | −0.10 | −0.29, 0.10 | 0.325 |
- Father not coresident, upbringing by others × AMPM | 0.23 | 0.01, 0.45 | 0.038 | 0.23 | 0.01, 0.45 | 0.039 |

G1 household covariates

- Mother coresident in 1998 | 0.03 | −0.11, 0.18 | 0.670 |
- Household female kin (#) | −0.04 | −0.09, 0.01 | 0.123 |
- Household male kin (#) | 0.004 | −0.03, 0.04 | 0.826 |
- Maternal education (SD units) | −0.003 | −0.05, 0.04 | 0.898 |
- Household income (SD units) | 0.07 | 0.01, 0.13 | 0.033 |

G2 adulthood covariates

- Age at sexual debut (y) | −0.02 | −0.03, −0.01 | 0.005 |
- Separated | 0.26 | 0.02, 0.51 | 0.036 |
- Lives with child(ren) | −0.21 | −0.36, −0.06 | 0.006 |
- Hours of weekly childcare (SD units) | 0.02 | −0.03, 0.07 | 0.405 |

Comparison groups for categorical variables: G1 father coresident, upbringing by both parents; PM samples; G1 mother not coresident in 1998; G2 nonseparated males; G2 nonresidential fathers. The models also include a fixed effect for survey year (i.e., 2005, 2009, or 2014). Pairwise comparisons for key groups from Model 2. For AM T (father coresident, upbringing by both parents) vs. (father not coresident, upbringing by others): (coefficient: 0.34 [0.15, 0.52]; P < 0.001); for FM T: (coefficient: 0.10 [−0.07, 0.28]; P = 0.234).
prior United States study relying on retrospective reports, childhood paternal experiences did not predict differences in new fathers’ change in T across the transition to parenthood (23). In Cebu, our earlier work on this topic focused only on father coresidence up to middle childhood and lacked data on other father–son dynamics and on the adolescent period (21). However, we found that boys who grew up with an unstable paternal presence tended to have lower waking T as fathers, which parallels our middle-childhood findings here, though they were not significant (21). Together, these results could indicate a weaker relationship between sons’ later T as fathers and their paternal experiences during childhood, compared to adolescence. Adrenarche involves heightened adrenal androgen production that can have effects on cognitive development and typically occurs during middle childhood. This could represent an additional sensitive period of biological transition, similar to puberty (69). However, in a post hoc test, we found that with the inclusion of sons’ paternal experiences during adolescence, their middle-child experiences no longer significantly predicted their later T, reinforcing our interpretation that our models are picking up an effect of adolescent experience specifically.

Among adult males in Dominica, T was lower if they experienced early childhood paternal absence, compared to those whose fathers were present, which diverges from our core findings (22). In that study, boys who experienced father absence gained less weight during infancy than father-present infants (22), a pattern of constrained early-life energetics and growth that we have shown predicts reduced adult T in Cebu (14). Thus, early-life growth/energetics could potentially help explain the findings in Dominica (14, 15), while in the present analyses our key results for adolescence were not altered by adjustment for developmental and adult energetic measures. Collectively, past work on this topic is consistent with beneficial energetics or nutrition during early life as a positive predictor of adult T (14, 15), while our present findings point to a likely role of parent–child experiences and relationships during adolescence as a distinct influence on HPG axis regulation.

Although our results appear consistent with a direct effect of adolescent social experiences on adult T production, we cannot rule out potential alternative explanations. Parent–child genetic confounding is one possibility (70, 71), as there is a heritable component to T production (72). For example, if fathers who were genetically predisposed to have higher T were less likely to be involved with childcare as a result, their sons could inherit these genes and produce higher T themselves. This would tend to link low caregiving effort in one generation to T levels in the next, not due to a sensitive period in response to care, but because of genetic correlations that influence T and behavior in both generations. Although this is in theory plausible (e.g., ref. 70), we think genetic confounding of this sort is unlikely to fully explain our findings. Such a genetic effect would be predicted to influence paternal behavior at all ages of their child’s development, and not only during adolescence, as would be necessary to explain our present findings from Cebu. Previously, we found that a genetic polymorphism related to androgen receptor function moderated links between T and behavior in Cebu, but this gene is X-linked and did not predict variation in T, making it unlikely to affect the current results for T (73).

In contrast to our findings for sons’ adolescence, we did not find that sons’ infancy or middle-childhood experiences were consistently linked to their later T, including no significant findings for sons’ T as parents. These results could reflect that the HPG axis is largely quiescent during these developmental periods, outside of the early infancy “mini puberty” window, compared to the onset of HPG maturation in adolescence (14, 74). However, early survey rounds of the Cebu study did not collect parent–child relationship quality measures prior to adolescence. Consequently, we could not test for links between family social bonds for these earlier periods and sons’ later T. Past longitudinal work on the quality of early-care environments as an influence on later neurobiological and endocrine function (6, 17, 18, 75) suggests that this merits consideration in future research.

Our study has limitations that warrant discussion. In the earlier waves (2005 and 2009) of data collection, our study design relied upon single measurements of waking and bedtime salivary T. Averaging values from repeated sampling, as we were able to do in 2014, reduces measurement error. Our approach in the present study helps to attenuate this limitation by pooling thousands of data points from the three surveys together in linear mixed models. An additional limitation is that we do not have data on some T-linked adult behaviors that could potentially be affected by childhood conditions. For example, some forms of risk taking and competition, such as for resources or status, are interrelated with T in humans and other species (10, 76).

It is also important to clearly outline the limitations of our measures of fathering and father–son relationships as the sons were growing up. The Cebu study was originally designed to focus primarily on mothers, which limited the breadth/precision of fathering measures. This contributes to the possibility of type II errors with respect to a role of early-life fathering in the current analyses. Our measures of fathers’ caregiving in the 1990s only capture whether mothers reported fathers as one of the household children’s caregivers in the respective surveys. Consequently, these variables cannot not provide information on the range of variation in the quantity of care, types of care, or quality of that care, all of which are relevant to understanding caregiver contributions to trajectories of child development, health, and biology, including across infancy, childhood, and adolescence when offspring needs and fathers’ roles differ in critical ways (6, 47, 75, 77–80). Moreover, fathers who provide high-quality care in developmentally appropriate and culturally relevant forms help shape father–child relationships and children’s perceptions of those relationships (19, 77, 78), which our upbringing measure attempts to capture. A strength of our upbringing measure is that the sons, themselves, reported on it during adolescence, rather than it being a parent-reported or retrospective variable. These son-reported data are a complement to mother-reported data on paternal care, giving us multiple-respondent information on sons’ adolescent experiences. The fact that adolescent sons who credited their fathers with their upbringing were more likely to report feeling close to their fathers suggests to us that the variable likely does reflect at least some aspects of father–son interaction quality and quantity. Still, our upbringing measure is a limited categorical indicator of sons’ perceptions of their relationships to their fathers and other caregivers. A prospective study designed for these issues would likely use extensive, validated surveys, observational methods, and newer technology-based emotional and behavior sampling techniques geared toward measuring different components of parent–child relational qualities and interactions, including during adolescence (77, 81).

Finally, father coresidence was also a key measure in these analyses. The child development and health effects of fathers residing elsewhere and separate from their children varies across populations and societies, based on cultural systems and other socioecological dynamics (22, 79, 82, 83). Moreover, the occurrence of fathers residing elsewhere has been used to pathologize
family forms in some populations (79), so it is important to clarify the local relevance of father coresidence in our study context. In Cebu, fathers residing separate from their children was relatively uncommon during the study time frame, and likely stemmed from multiple sources, such as employment abroad and marital separations (19, 32, 83). Given valued local roles for fathers and Filipino cultural emphases on two-parent households, informed in part by Catholic norms (19, 26, 56, 57, 80), father coresidence was likely meaningful for sons during development. Consistent with this notion, we found that adolescent sons were less likely to attribute their upbringing to their fathers if their fathers resided elsewhere earlier in the sons’ childhood. Nonetheless, a meaningful number of sons also credited their fathers with their upbringing despite not living with them as adolescents, which hints that data on contributions made by nonresidential fathers could have provided further nuance to our analyses (77, 79). Such data were not available in the 1980s or 1990s for nonresidential fathers.

**Conclusion**

In sum, our results from this multidecade longitudinal study indicate that adolescence may serve as a sensitive period when experiences of care and social relationships, especially with fathers, may influence adult T regulation in offspring. These patterns thus link key social experiences during adolescent development to adult production of a hormone that plays a central role as a coordinator of behavioral and biological components of male reproductive strategies.

**Methods**

**Study Population.** Data are from the CLHNS, an ongoing, population representative birth-cohort study of infants and their mothers in the Philippines that began in 1983. In the present study we drew on longitudinal data from families with infants identified as male at birth, who have been followed through multiple survey waves across their lives. Specifically, we used data collected from the G1 mothers during their sons’ infancy (1983 to 1986), middle-childhood (1994), and adolescent (1998) periods, as well as their G2 sons in 1998, 2005, 2009, and 2014. This research was conducted with informed consent and human subject clearance from the institutional review boards of Northwestern University and the University of North Carolina. All participants provided written informed consent. See SI Appendix, Supplemental Methods for further details on the study design and the measures described below. Please also see SI Appendix, Fig. S1, which is a visual guide to the waves of CLHNS data collection and key measures we drew on for this study.

**Data From G2 Infancy, Middle Childhood, and Adolescence.**

*G2 childhood sociodemographic and family life data.* For waves in the 1980s to 1990s, the G1 mother was the main respondent. G2 sons were also interviewed as adolescents between 1998 and 1999 (“hereafter,” 1998”). Mothers provided household sociodemographic data during each survey and also reported on the main caregivers for the children in the household.

In each survey, the CLHNS collected data on who resided in each target household. Individuals were defined as household residents if they had regularly lived together in the past 6 mo in the same structure and had routinely shared meals and household costs. We used these criteria to define G1 fathers as coresident vs. not coresident in each survey. At each stage (infancy, middle childhood, and adolescence), father coresidence was the predominant pattern (∼84 to 87% of households) (Table 1). There also tended to be continuity in coresidence across surveys: for example, ∼82% of sons resided with their fathers in both 1994 and 1998, while ∼9% did not reside with their fathers during either time point. A small percentage (∼2 to 3%) of G1 mothers in the present study sample were widowed in 1994 and/or 1998, making it probable that the G2 sons’ father was deceased. We categorized these G2 participants as “G1 father not coresident.” We also used the household rosters to identify other coresidential potential caregivers, including mothers, grandparents, aunts/uncles, cousins, older siblings, and other kin. We created counts of female kin and male kin, respectively, as indicators of alloparental care. For a small percentage of G2 sons in 1994 (∼3%) and 1998 (∼9%), their (G1) mother was not residing in their household and a caretaker reported the relevant household roster data.

During the 1983 to 1986 data collections, when the G2 sons were 2, 6, and 14 mo of age, mothers reported which household members engaged in caregiving for children <5 y old in the prior week. In 1994 and 1998, G1 mothers responded to questions regarding who in the household was mostly or mainly responsible for caring for the children. Mothers could respond with up to four household members; they were not asked to report on specific caregiving tasks nor the quantity of care provided by household members. In 1994 and 1998, fathers were most frequently characterized as coresident but not involved in childcare (62 to 76%) (Table 1). It was also common in the 1980s and 1990s for families to receive at least some routine alloparental care in the household (83). G1 caregiving data were not reported for nonresident fathers (26, 83).

*G2 sons’ adolescent upbringing perceptions.* In 1998, the G2 adolescents reported who they felt was responsible for their upbringing, including mother, father, both parents, caretaker, and a list of other kin. Over 92% of G2 sons identified one or both parents as responsible for their upbringing, with ∼6.5% choosing “caretaker.” We combined these data into a categorical variable with these upbringing groupings: both parents, father only, and mother or caretaker only. We used this variable as an indicator of the G2 sons’ perceptions of their relationship with their caregivers. Sons only reported these upbringing data in 1998. As we describe in SI Appendix, Supplemental Methods, in 1998, adolescents also reported on whether they felt close to their fathers (26). Sons who felt close to their fathers were substantially more likely to credit their fathers with their upbringing (P < 0.001) (SI Appendix, Table S8), which helps validate the upbringing measure as an indicator of the adolescent-caregiver relationship quality. Closeness data were largely limited to sons living with their fathers. Prior relevant work on young adults has similarly shown strong correlations between perceptions of family closeness and upbringing satisfaction (84). As a complementary indication that the upbringing perceptions were linked to father-son dynamics, adolescent sons were much more likely to attribute their upbringing to their fathers if their fathers lived with them 4 y earlier, during middle childhood (P < 0.001). There was a similar, strong cross-sectional association for adolescent father-son coresidence and sons’ concurrent upbringing perceptions in 1998 (P < 0.001) (SI Appendix, Table S8).

**Key Independent Variables.**

*G2 experiences with their fathers and upbringing perceptions.* We created a set of categorical variables that combined G1 paternal residence status and caregiving involvement for each individual wave of developmental data (infancy, childhood, adolescence). At each respective time point, we refer to this categorical variable as “fathering” for that specific wave (Table 1). During G2 middle childhood and adolescence, respectively, the fathering groupings were: 1) fathers were coresident and involved in care, 2) fathers were coresident but not involved in care, 3) fathers were not coresident. During G2 infancy, between 1983 and 1986, there were three applicable waves of data collection. Consequently, the fathering groupings were: 1) fathers were coresident at all waves and involved in care for 1+ waves; 2) fathers were coresident at all waves and not involved in care at any wave; 3) fathers were not coresident at 1+ waves with some involvement in care when coresident; 4) fathers were not coresident at 1+ waves with no involvement in care when coresident. For each developmental period, we used G2 participants whose fathers lived with them and were involved in care as the reference group (i.e., category 1 for each survey).

We created a separate categorical variable for G2 adolescence that combined G1 paternal residence status with G2 adolescents’ reports regarding who they felt was responsible for their upbringing. We refer to this categorical variable as “upbringing.” It characterizes whether G2 sons lived with their fathers as adolescents (or not) and whether they identified their fathers as contributing to their upbringing. The upbringing groupings were: 1) fathers were coresident and both parents were responsible for upbringing; 2) fathers were not coresident and both parents were responsible for upbringing; 3) fathers were not coresident and were responsible for upbringing; 4) fathers were coresident and were not responsible for upbringing; 5) fathers were not coresident and were not responsible for upbringing. We used G2 adolescents who resided with their
fathers and who attributed their upbringing to both their parents as the reference group, which was the most common grouping (69% of G2 sons in the study).

**G2 infant weight velocity and childhood/adolescent anthropometrics.** Prior CLHNS research found that G2 males’ growth (weight velocity) from birth to 6 mo of age was linked to their T as young adults (14). Adjusting our models for this infancy growth measure, as described in Kuzawa et al. (14), did not meaningfully change our findings but did result in dropping 30 or more participants per model due to missing data. Thus, we did not include this variable in the models presented in the paper.

During the 1994 and 1998 surveys, anthropometric measurements were collected using standard techniques (85). Using these data, we calculated G2 participants’ weight-for-height at each time point. In the relevant models for the respective time points, we included weight-for-height as a covariate, as an indicator of G2 participants’ nutritional experiences and energetic condition. A small number of participants in 1994 and 1998, respectively, were missing anthropometric data, so alongside our core models for our key results, we included an additional model adjusting for weight-for-height for the relevant time period (1994, 1998) along with adult body mass index (BMI, see below).

**Data from G2 in Adulthood**

**G2 salivary testosterone.** We collected saliva in 2005, 2009, and 2014 using similar collection procedures except that in 2014 we included repeated sampling for each subject (four total samples). Participants collected the first sample immediately before bed (PM), and they were instructed to collect the second sample immediately on waking the following morning (AM). We adjusted the T data for time of sample collection, following procedures we have previously reported (24). T concentrations were determined using an enzyme immunoassay protocol (Salimetrics; Kit no. 1-2402). Interassay coefficients of variation for high and low kit-based control samples were as follows: 2005 (7.8% and 17.9%), 2009 (6.6% and 7.2%), 2014 (6.3% and 12.2%).

**G2 paternal care.** In 2009 and 2014, fathers reported the time they spent in the past week on a list of 20 common paternal caregiving behaviors in the Philippines. The domains of caregiving are not mutually exclusive and thus some behaviors could co-occur. In 2005, participants reported time allocation data for both a routine working day and a nonworking day. These data were then coded into activity codes, including specific childcare activities (32, 80). For each survey, we analyzed caregiving time as hours of weekly care.

**Other G2 sociodemographic variables.** In addition to their fathering status, men reported their marital/cohabitation status, including whether they were separated from their partner. They also reported whether they resided with their children. All G2 participants in the analyses focused on fathers who had engaged in sexual intercourse and reported their age at sexual debut. For the analyses focusing on G2 young adulthood, some participants had not yet had intercourse. We created a dichotomous variable indicating whether they had experienced intercourse by the 2005 survey or not.

**G2 anthropometrics.** At each adult survey, anthropometric measurements were collected using standard techniques (85). We calculated BMI as weight (kg) height (m^2) and included BMI as a covariate alongside childhood/adolescent weight-for-height.

**Attrition rates.** During the early years of the project attrition rates ranged between 9% and 11% and have declined to ~5% in the adult surveys, with a majority of the attrition resulting from participants migrating out of Metro Cebu.

Of the original mothers, those who have remained enrolled in the CLHNS tend to come from lower socioeconomic status households, whereas for their sons, those with higher socioeconomic status have tended to be retained (86).

**Statistical Analyses.** We conducted all statistical analyses using Stata v17.0 (Stata Corporation). In our core models focusing on G2 sons’ T, we used linear mixed models with maximum-likelihood estimation (Stata’s ‘mixed’ command). In each model, we included a random intercept effect for each individual participant to account for the structure of the data, with individuals having multiple T observations. In models that had data from multiple waves between 2005 and 2014, we also included a random slope effect for the survey year. We also used bootstrapping (1,000 replicates with bias-correction and acceleration) to calculate confidence intervals (Stata’s ‘bootstrap, bca’ command). In the models predicting T, the linear mixed models included interactions between our key independent variables and a categorical variable (AMMP) that identified T values as waking (AM) or evening (PM) values. The interaction terms between AMMP and the key predictors were modeled because prior research, including from our team (14, 21), has shown stronger links between early-life experiences and waking T, compared to evening T, but the predictions for AM and PM T were analogous and the mixed modeling approach allowed us to include them in the same model. Prior to their inclusion in models together, we converted waking T and evening T, respectively, to SD units. Please see the SI Appendix, Supplemental Methods for further information regarding the covariates, which included G1 and G2 sociodemographic, behavior, and energetic variables as potentially important confounding factors. We converted the following covariates to SD units: G1 maternal education level, G1 household income, G2 weight-for-height, G2 BMI, and G2 weekly paternal care. We generated the figures using predictive margins (adjusted predictions) using Stata’s ‘margins’ and ‘marginsplot’ commands. We evaluated statistical significance at P < 0.05.

**Data Availability.** Data files (Datasets S1 and S2) and codebook data have been deposited in GitHub (https://github.com/leegetter/PNAS2022) (87). Those files and other study data are included in the main text and supporting information.

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