Hormones and Behavior 95 (2017) 103–112

Contents lists available at ScienceDirect

Hormones and Behavior

journal homepage: www.elsevier.com/locate/yhbeh

High paternal testosterone may protect against postpartum depressive symptoms in fathers, but confer risk to mothers and children

Darby E. Saxbe[a,*], Christine Dunkel Schetter[b], Clarissa D. Simon[c], Emma K. Adam[d], , & Madeleine U. Shalowitz[c,e]

[a] Department of Psychology, University of Southern California, Los Angeles, CA 90089, USA
[b] Department of Psychology, University of California, Los Angeles, CA 90095, USA
[c] NorthShore University HealthSystem Research Institute, Evanston, IL 60208, USA
[d] School of Education and Social Policy and Institute for Policy Research, Northwestern University, Evanston, IL 60208, USA
[e] University of Chicago, Pritzker School of Medicine, Chicago, IL 60637, USA

ABSTRACT

Following the birth of an infant, decreases in testosterone and increases in depressive symptoms have been observed in fathers. Paternal testosterone may reflect fathers' investment in pair-bonding and paternal caregiving and, as such, may be associated with maternal and familial well-being. This study tests associations between paternal testosterone, paternal and maternal postpartum depressive symptoms, and subsequent family functioning.

Within 149 couples, fathers provided testosterone samples when infants were approximately nine months old and both parents reported on postpartum depressive symptoms at two, nine, and 15 months postpartum. Fathers with lower aggregate testosterone reported more depressive symptoms at two and nine months postpartum. Mothers whose partners had higher evening testosterone reported more depressive symptoms at nine and 15 months postpartum. Maternal relationship satisfaction mediated this effect, such that mothers with higher testosterone partners reported more relationship dissatisfaction, which in turn predicted more maternal depressive symptoms. Higher paternal testosterone and paternal depressive symptoms at nine months postpartum each independently predicted greater fathering stress at 15 months postpartum. Higher paternal testosterone also predicted more mother-reported intimate partner aggression at 15 months postpartum. In addition to linear relationships between testosterone and depression, curvilinear relationships emerged such that fathers with both low and high testosterone at nine months postpartum reported more subsequent (15-month) depressive symptoms and fathering stress.

In conclusion, whereas higher paternal testosterone may protect against paternal depression, it contributed to maternal distress and suboptimal family outcomes in our sample. Interventions that supplement or alter men's testosterone may have unintended consequences for family well-being.

1. Introduction

The transition to parenthood is marked by a series of dramatic hormonal and neural changes that may facilitate parental adaptation to caregiving. Biopsychosocial mechanisms of postpartum changes in mood and behavior have been primarily investigated in mothers. However, there is intriguing evidence that both animal and human fathers show dynamic changes in hormones in early parenthood (Saltzman and Ziegler, 2014). Testosterone, a steroid hormone from the androgen group that stimulates the development of male secondary sex characteristics, appears to show parenting-related declines among human and animal fathers (Gettler et al., 2011).

Men's depressive symptoms increase during the early years of parenthood, with evidence that new fathers report depressive symptoms at higher rates than men in the general population (Garfield et al., 2014; Gettler and Oka, 2016; Paulson and Bazemore, 2010) Men's postpartum depression can affect the well-being of mothers and children, so understanding the causes of paternal mood disorders is important not just to father but to family mental health (Ramchandani et al., 2005). It has been theorized that low paternal testosterone may contribute to paternal depressive symptoms following the birth of a child (Kim and Swain, 2007), but no empirical studies have yet tested associations

* Corresponding author.
E-mail address: dsaxbe@usc.edu (D.E. Saxbe).

http://dx.doi.org/10.1016/j.yhbeh.2017.07.014
Received 7 January 2017; Received in revised form 21 July 2017; Accepted 26 July 2017
Available online 31 August 2017
0018-506X/

between paternal testosterone and postpartum depression.

1.1. Testosterone and depressive symptoms in men

Low testosterone has been identified as a risk factor for depression in men (Vogel et al., 1978), although research findings have been mixed (Amiaz and Seidman, 2008). Several studies have reported higher depression among men with lower testosterone (Almeida et al., 2008; Barrett-Connor et al., 1999; Schweiger et al., 1999) and reductions in depressive symptoms after testosterone supplementation (Zarrouf et al., 2009). However, other investigations have found no associations between men's testosterone and depression (Giltay et al., 2012; O'Connor et al., 2004; Seidman et al., 2001). Testosterone may be most strongly linked with subthreshold depression or dysthymia (Seidman et al., 2002). To date, the literature on testosterone and depression has been limited by a lack of longitudinal research, a constricted age range (with most studies focused on older men), and neglect of contextual variables such as parenting status.

1.2. Testosterone and fatherhood

Biparental mammals including primates, rodents, and humans show significant drops in testosterone during the postpartum period (Saltzman and Ziegler, 2014). Both cross-sectional and longitudinal human studies report low or decreasing testosterone in partnered fathers compared to single men. For example, fathers of young children have significantly lower testosterone than similarly-aged males who are not fathers (Berg and Wynne-Edwards, 2001; Gray et al., 2006). Testosterone appears to decline over time among partnered men who become fathers, and steeper declines have been associated with greater childcare involvement (Gettler et al., 2011).

From an evolutionary perspective, lowered testosterone in fathers may reflect a shift away from pursuing new mating opportunities and towards investment in the family (van Anders et al., 2012). Animal studies have linked lower testosterone levels with reduced aggression towards conspecific infants and more time caring for offspring (Clark and Galef, 2000; Perrigo et al., 1991). Consistent with this, men's prenatal and postpartum decreases in testosterone have been associated with sensitive caregiving, as well as greater commitment to the partner relationship (Fleming et al., 2002; Gettler et al., 2011; Storey and Ziegler, 2016). For example, men whose testosterone decreased more over the course of their partner's pregnancy subsequently reported greater postpartum investment in the relationship with the child's mother (Saxbe et al., 2016). Similarly, married men with lower evening testosterone reported greater spousal investment (Gray et al., 2002). In summary, men in early parenthood appear to have lower testosterone levels than at other periods in the lifespan, and this lowered testosterone may reflect fathers' involvement with the family.

1.3. Research integrating testosterone, depression, and family status

Only a few researchers have considered testosterone and depression in conjunction with life history variables. Booth et al. (1999) noted that, while, low testosterone has been implicated in men's depression, high testosterone in males has also been associated with known risk factors for depression, such as aggression, antisocial behavior, and being unmarried or divorced. Within a sample of male veterans, they found a curvilinear relationship between depression and testosterone, such that symptoms were higher at both low and high levels of testosterone. However, after controlling for antisocial behavior, marital status, and employment, high testosterone was no longer associated with depression. This finding supports a “social mediation” model where both low and high testosterone may be problematic and in which high testosterone indirectly affects depression via social and behavioral factors. In another study of couples with school-aged children, neither husband nor wife testosterone was directly associated with marital quality, but role overload interacted with husbands' testosterone to predict marital quality, such that marital distress was higher among high-testosterone men also reporting greater role overload (Booth et al., 2005). In other words, high testosterone may compromise the partner relationship under stressful life conditions, suggesting again that psychosocial factors may modulate links between testosterone and family outcomes. Similarly, Gettler et al. (2017) found that both low and high levels of a genetic polymorphism linked with testosterone functioning and “androgenicity” predicted relationship instability and low levels of paternal caregiving, although this polymorphism was not associated with paternal depression.

Gettler and Oka (2016) found that men's testosterone interacted with socioeconomic status and relationship and parenting status to predict depression. Within this study, men were identified as fathers if they resided with any children under the age of 18. Low SES, low testosterone, partnered non-fathers had the highest depression rates. Among men living with children, low SES, high testosterone men had mildly elevated depression risk, whereas high SES, high testosterone, partnered men had the lowest depression rates. Partnered men residing with children also had lower testosterone than non-partnered, childless men. Although this is the most comprehensive study of depression, testosterone, and family factors to date, it did not focus specifically on the postpartum period, and men in the sample ranged from 20 to 60 years of age.

No studies have yet examined mothers' depressive symptoms or other aspects of postpartum family functioning in conjunction with fathers' testosterone. Partner social support is known to buffer maternal depression risk during the transition to parenthood (Misri et al., 2000; Stapleton et al., 2012). If fathers with lower testosterone are more invested in the family, higher paternal testosterone might compromise maternal well-being and increase fathers' stress related to parenting. High testosterone in males has also been associated with intimate partner aggression (IPA; McKenry et al., 1995; Pinto et al., 2010). However, the literature on testosterone and IPA has not focused on aggression during the postpartum period (Beydoun et al., 2012).

1.4. Testosterone measurement

Testosterone has a diurnal rhythm, peaking in the morning and declining across the day. Some studies have examined only morning levels (e.g., Almeida et al., 2008; Gray et al., 2006), some only afternoon or evening levels (Fleming et al., 2002; Storey et al., 2000), and some have aggregated both AM and PM measures of testosterone (Giltay et al., 2012). Although many researchers use only morning samples because of testosterone's peak, testosterone sampled later in the day might better reflect exposure to daily social contexts. In a meta-analysis of research on testosterone and aggression, testosterone-behavior relationships were higher for samples taken later in the day (Book et al., 2001). Similarly, Gray et al. (2002) found that evening, but not morning, testosterone levels were associated with relationship and parenting status in men. Collecting multiple measures of testosterone across the day may help elucidate which sampling timepoints best reflect psychosocial processes.

1.5. Current study

The current study examines salivary testosterone levels in fathers, sampled three times over one day, approximately nine months after the birth of their child. We also measured depressive symptoms reported by both parents at three timepoints during the 18 months after birth (approximately two months, nine months, and 15 months postpartum) and assessed fathering stress and mother-reported intimate partner aggression at 15 months postpartum. Our design allowed us to test both cross-sectional relationships (testosterone and depression measured at the same timepoint) as well as longitudinal relationships (whether testosterone predicts depressive symptoms and other outcomes at the

following visit).

We tested four hypotheses. First (Hypothesis 1), we predicted that fathers' testosterone would be inversely associated with father postpartum depressive symptoms. Specifically, we expected that fathers with lower testosterone at nine months postpartum would report more depressive symptoms, both cross-sectionally and longitudinally.

Second (Hypothesis 2), since low paternal testosterone may reflect father investment in the family, and partner support is known to buffer postpartum depression in women, we expected fathers' testosterone would be positively associated with maternal depressive symptoms, both at the same visit and at preceding and following visits. Additionally (Hypothesis 3), we expected that maternal relationship satisfaction would mediate the association between fathers' testosterone and maternal postpartum depressive symptoms.

Next (Hypothesis 4), we planned to test whether testosterone and depressive symptoms at nine months postpartum predicted two family outcomes at 15 months postpartum: fathering stress and intimate partner aggression. Social mediation models of testosterone suggest that both high and low testosterone can be problematic for men. Although low testosterone might accompany depression, high testosterone could predict aggression and reduced investment in the family. Depression also may contribute to maladaptive family functioning. As such, we expected that testosterone and depression would make unique contributions to adverse family outcomes.

Finally, as an additional exploratory aim, we planned to test curvilinear relationships between testosterone and outcome variables for fathers (paternal depressive symptoms and paternal parenting stress), by substituting a curvilinear term (testosterone squared) for the linear term used in our analyses. Given the findings of Booth et al. (1999), we expected that the curvilinear term might be positively associated with depression and paternal parenting stress, such that greater risk would emerge for fathers who were both particularly low and particularly high in testosterone relative to fathers with average testosterone.

2. Methods

2.1. Overview

Data used in this study were drawn from the Lake County, IL site of the larger Child Community Health Network (CCHN) study, a five-site, multi-investigator community-based study funded by the *Eunice Kennedy Shriver* National Institute for Child Health and Human Development (NICHD). Recruitment, eligibility, and demographics for the CCHN study are described in Ramey et al., 2015, and Dunkel Schetter et al., 2013. In brief, biological mothers were recruited at the birth of their first, second, or third child and followed at several visits over the first two years postpartum. Eligible mothers were between 18 and 40 years of age, and self-identified as African-American, White, or Latina. Fathers were invited to participate in the study with mothers' consent. Study sites included Baltimore, MD; Lake County, IL; Los Angeles, CA; eastern North Carolina; and Washington, D.C. Salivary testosterone data was only collected at the Lake County, IL site. The current study uses data from three visits: at approximately 1 to 3 months postpartum (T1), at 6 to 9 months postpartum (T2), and at 12 to 16 months postpartum (T3). Testosterone was collected only at T2 and only from fathers (with the exception of a small subgroup of mothers, not included in this study).

2.2. Participants

Testosterone data were available from 149 fathers. The CCHN investigators sought to recruit a diverse, low-income sample of parents. Within the full sample, 38% of mothers and 30% of fathers reported their household income as below the federal poverty line. Compared with the full sample of CCHN participants, fathers who participated in testosterone sampling and were therefore included in this study were older ($t = 4.29$, $p = 0.001$), more likely to be cohabitating with the baby's mother ($t = -10.41$, $p = 0.001$), more educated ($t = -3.08$, $p = 0.002$), more likely to be born outside the U.S. ($t = -3.62$, $p = 0.001$), and more likely to be white ($t = -12.92$, $p = 0.001$). However, there were no differences in income, mother or father depressive symptoms at T1, T2, or T3, paternal parenting stress, or intimate partner aggression between the full CCHN sample and the sample included in this paper (all p values > 0.25). Within this sample, 95% of participating couples were cohabiting at the T2 visit.

2.3. Procedures

Fathers and mothers participated in three CCHN study visits within the first 18 months postpartum. At T1, infants were approximately two months old (*Mean* = 1.77 months, *SD* = 0.84 months, *Range* = 0.5–7 months); at T2, infants were approximately nine months old (*Mean* = 8.91 months, *SD* = 1.48 months, *Range* = 5.65–13 months), and at T3, infants were approximately 15 months old (*Mean* = 14.70 months, *SD* = 1.66 months, *Range* = 9.28–20.25 months). Visits consisted of in-person interviews conducted in English or Spanish in the participants' homes. Structured interview protocols were developed through community collaboration and pre-tested, then implemented with extensive interviewer training and monitoring. Interviewers included experienced community members and research staff.

2.4. Testosterone sampling

At the second study visit (T2), fathers sampled saliva three times over the course of a day scheduled within 1–2 weeks of the study visit (mean 10 days). Research staff provided saliva sampling kits along with verbal and written instructions for collecting saliva upon waking, 30 min after waking, and bedtime. Participants were asked to self-collect samples by expelling saliva through straws into sterile 2 mL cryogenic vials. Participants also logged the time of each collection (*Mean waking collection time* = 7:50 am, *SD* = 1.5 h, *Range* 3:20 am–12:30 pm; *Mean waking + 30 time* = 8:20 am, *SD* = 1.5 h, *Range* 3:50–1 pm; *Mean evening time* = 10:47, *SD* = 1 h 12 min, *Range* 8 pm–2:30 am).

Completed materials were mailed back to each study office, where they were stored at −80 °C. Saliva samples were subsequently shipped to ZRT Laboratories (Beaverton, OR) and assayed for testosterone (reported detection limits of 5–3333.3 pg/mL and inter-assay coefficients of variance of 10.2%–14.9%). ZRT's testosterone assay results have been found to correlate very highly ($R^2 = 0.9995$) with results from 10 other labs that participate in a biannual salivary proficiency testing program (ZRT Laboratories, 2017).

As outliers in testosterone can bias results, we took a conservative approach to handling extreme values and dropped values > 3 *SD*s from the sample mean. In total, five outliers, or about 3% of the sample, was dropped; two waking samples, two wake + 30 samples, and one bedtime sample. Aggregate testosterone output over the day was estimated by calculating area under the curve with respect to ground, using the trapezoidal formula provided by Pruessner et al. (2003). This formula uses the three testosterone samples and adjusts for time elapsed between samples to estimate overall testosterone across the day. Aggregate testosterone output (AUC) was positively correlated with waking (r (148) = 0.90, $p = 0.001$), waking + 30 (r (148) = 0.93, $p = 0.001$), and evening (r (148) = 0.53, $p = 0.001$) testosterone levels. The two morning testosterone measures were highly correlated with each other (r (152) = 0.94, $p = 0.001$) and positively but more weakly correlated with evening testosterone (r (152) = 0.22, $p = 0.01$ with waking T; r (148) = 0.17, $p = 0.04$ with waking + 30 T). Sampling collection time was not significantly correlated with either of the morning testosterone measures (r (148) = −0.02, $p = 0.81$ and r (148) = −0.06, $p = 0.47$) for waking and waking + 30 T, respectively, but was positively correlated with evening testosterone (r (149)

= 0.19, p = 0.02). None of the three sampling collection times were significantly associated with aggregate testosterone (all p values > 0.15).

Because of the high correlations between the three testosterone measures and aggregate testosterone, we used aggregate testosterone in our analyses. However, based on evidence that evening testosterone might be more strongly associated with social behavior than morning testosterone (Gray et al., 2002), we ran follow-up analyses testing family-level outcomes (maternal depression, parenting stress, and intimate partner aggression) using evening testosterone levels and controlling for evening testosterone sampling time.

3. Measures

3.1. Postpartum depression

The Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1987) is a widely used 10-item measure of postpartum depressive symptoms, validated in both men and women. Participants report depressive symptoms over the previous 7 days, with scores ranging from 0 to 30. We used continuous scores rather than clinical cut-off scores in analyses because of evidence that subthreshold depression can affect infant and parent well-being, and that low testosterone may be associated with mild mood disturbance rather than severe depression (Seidman et al., 2002). Additionally, the majority of this sample did not meet clinical cut-off for depression. Within this sample, mothers' EPDS scores averaged 4.64, *SD* = 4.55, *Range* = 0–21 (at T1); 5.20, *SD* = 3.85, *Range* = 0–16 (at T2); and 5.11, *SD* = 4.09, *Range* = 0–17 (at T3). Fathers' EPDS scores averaged 3.56, *SD* = 3.80, *Range* = 0–20 (at T1); 4.29, *SD* = 3.70, *Range* = 0–25 (at T2); and 4.25, *SD* = 3.80, *Range* = 0–23 (at T3).

3.2. Relationship satisfaction

The Dyadic Adjustment Scale (DAS; Spanier, 1976), administered at T2, is a well-validated measure of relationship functioning, with 32 items that assess areas of disagreement, degree of closeness and affection, and overall satisfaction with the relationship. Within this study, fathers' mean DAS score was 124.17 (*SD* = 13.94, *Range* = 69–150) and mothers' mean DAS score was 121.78 (*SD* = 13.28, *Range* 71–144).

3.3. Parenting stress

The Parenting Stress Index-Short Form (PSI; Abidin, 1995), administered at T3, is a 36-item measure that assesses parents' experiences of parenting and stress related to parenting. Items include "I feel trapped by my responsibilities as a parent," "My child makes more demands on me than most children," and "I expected to have closer and warmer feelings for my child than I do and this bothers me." This measure was normed on 800 parents and found to have good test-retest reliability and internal consistency. No significant gender differences have been observed, making it appropriate for use with fathers as well as mothers. Scores for fathers in this sample ranged from 37 to 142, with a mean of 65.12, *SD* = 18.32. Scores for mothers ranged from 36 to 132, with a mean of 66.63, *SD* = 16.42.

3.4. Intimate partner aggression

At T3, the HITS (for Hurt, Insult, Threaten, Screamed at; Sherin et al., 1998) was administered to mothers. This measure assesses the frequency of physical hurt, insult, threats, and screaming occurring over the past year on a 5-point scale (never = 1 to frequently = 5). Any response other than "never" was followed with the question "Was it your partner/spouse, another family member, or someone else in your household?" Only partner/spouse responses were scored. In line with other studies (e.g., O'Campo et al., 2010), an item on emotional control was added: "How often does your partner/spouse restrict your actions? By actions we mean things such as spending money, visiting with family or friends, or going places that you need to go." Within this sample, scores ranged from 5 to 17, mean = 6.32, *SD* = 2.11.

3.5. BMI

Father BMI (mean = 29.23, *SD* = 5.45, range 18.96–48.69) was derived from height and weight measures obtained in person at T2. Body mass index (BMI) was calculated using the established formula (BMI = weight (lb) / [height (in)]^2 × 703).

3.6. Other covariates

At recruitment, mothers and fathers identified their own ethnic/racial background from a set of standard options; for the present analyses, this was coded dichotomously as racial/ethnic minority (African American or Latino) or non-Hispanic White. Within the current sample, 53% of fathers identified as members of a racial or ethnic minority group and 47% of fathers identified as white. Fathers also reported their current age (mean = 31.01, *SD* = 6.92, range = 18.25–49.64). Infant sex was obtained from birth records. Additional demographic data collected at T1 included education (coded from 1 = less than high school to 4 = completed 4-year college or higher; mean = 2.57, *SD* = 1.24); and U.S. or foreign birth (35% of fathers in the sample were foreign born; most (90%) of these fathers identified as Latino and were from Mexico or other Latin American countries). Finally, we created a "lag" variable to reflect time elapsed between father T2 and T3 visits (mean = 175.11, *SD* = 38.29, range = 82–255).

3.7. Statistical approach

All analyses were performed in SPSS 24.0 (IBM). First, we examined correlations between testosterone and other variables in order to identify significant covariates. Next, we conducted a series of linear regression models to test our hypotheses. We conducted a bootstrapping test to test mediation (Hypothesis 3) via the INDIRECT macro developed by Preacher and Hayes (2008), which uses bias-corrected bootstrapping techniques to estimate confidence intervals. In order to investigate curvilinear relationships between testosterone and our study outcomes (Hypothesis 5), we centered the aggregate testosterone variable and then squared it, and substituted this variable for aggregate testosterone in our linear regression analyses. We tested eight basic regression models in Hypotheses 1–4 and then re-tested the three outcomes for fathers (T2 and T3 depressive symptoms and T3 parenting stress) using the curvilinear term, yielding 11 regression models in all. In order to correct for multiple comparisons, we used the Holm-Bonferroni method (Holm, 1979), in which the alpha value is adjusted such that the lowest p-value (i = 1) is expected to fall below a/k (where k is the number of analyses), and the higher values to progressively less restrictive thresholds (a/(k − i + 1). Therefore, for 11 planned analyses, we would require at least one model be significant at p = 0.0045 (0.05/11); one model significant at 0.005 (0.05/10); one at 0.006; and so forth. All of the regression models that included significant values for testosterone met this adjusted p-value threshold, with one exception that is noted in the text. We also calculated standardized effect sizes for all of the regression models (Cohen's F^2) and included them in the tables.

4. Results

4.1. Zero-order correlations

Table 1 depicts bivariate correlations between study variables. As shown, more educated fathers, fathers with a lower BMI, and fathers with older infants at T2 had higher morning testosterone levels. Infant

Table 1
Zero-order correlations between fathers' testosterone, covariates, and family outcomes.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Testosterone AUCg	1																		
2. Wake T	0.90**	1																	
3. Wake + 30 T	0.93**	0.94**	1																
4. Bedtime T	0.53**	0.22**	0.17*	1															
5. Child age	0.25**	0.28**	0.22**	0.14 +	1														
6. Father age	0.05	0.11	0.12	− 0.18*	0.17*	1	.												
7. Father education	0.16 +	0.24**	0.21*	− 0.06	0.05	0.38**	1												
8. US/foreign-born	− 0.01	− 0.11	− 0.11	0.18*	− 0.01	− 0.12	− 0.49**	1											
9. Father BMI	− 0.14 +	− 0.17*	− 0.17*	− 0.02	− 0.05	0.06	0.04	− 0.05	1										
10. Father minority	− 0.05	− 0.13	− 0.14 +	0.21**	− 0.13 +	− 0.30**	− 0.52**	0.62**	− 0.05	1									
11. Infant sex	0.01	− 0.07	− 0.03	0.12	0.06	− 0.04	− 0.15 +	− 0.03	− 0.07	0.01	1								
12. Father T2 DAS	− 0.01	0.04	0.01	− 0.06	− 0.03	− 0.10	− 0.07	0.14 +	− 0.11	− 0.15 +	− 0.10	1							
13. Mother T2 DAS	0.01	0.10	0.05	− 0.09	0.06	0.04	0.06	0.14 +	− 0.06	0.04	− 0.01	0.43**	1						
14. Mother T1 EPDS	0.04	0.05	0.02	0.07	− 0.01	− 0.13 +	0.09	− 0.01	0.08	− 0.06	− 0.18*	− 0.01	− 0.15 +	1					
15. Mother T2 EPDS	0.03	0.01	− 0.05	0.19*	− 0.01	− 0.18*	− 0.02	− 0.02	0.01	0.03	0.03	− 0.16*	− 0.42**	0.46**	1				
16. Mother T3 EPDS	0.14	0.11	0.07	0.21**	− 0.08	− 0.17*	0.04	0.01	0.09	0.10	− 0.06	− 0.11*	− 0.26**	0.49**	0.56**	1			
17. Father T1 EPDS	− 0.23**	− 0.24**	− 0.26**	− 0.02	− 0.15 +	− 0.08	− 0.18*	− 0.01	0.18*	0.10	0.02	− 0.24**	− 0.08	0.18*	0.06	0.05	1		
18. Father T2 EPDS	− 0.15 +	− 0.16 +	− 0.19*	0.08	− 0.03	− 0.19*	− 0.10	0.03	0.15 +	0.21**	0.06	− 0.32***	− 0.14+	0.02	0.21**	0.08	0.50**	1	
19. Father T3 EPDS	− 0.11	− 0.16 +	− 0.15 +	0.07	− 0.14 +	− 0.23**	− 0.11	0.03	0.05	0.15 +	0.04	− 0.35***	− 0.17*	0.14	0.18*	0.17*	0.48**	0.56**	1
20. Father T3 PSI	0.08	− 0.01	0.02	0.16 +	0.002	− 0.07	− 0.24*	0.21**	− 0.01	0.19*	0.10	− 0.29***	− 0.28***	0.07	0.19*	0.10	0.23**	0.28***	0.36***
21. Mother T3 PSI	0.10	0.05	0.06	0.14 +	0.002	− 0.17*	− 0.04	0.13	0.01	0.10	0.01	− 0.03	− 0.30**	0.24**	0.32**	0.41**	− 0.02	− 0.04	− 0.15 +
22. Mother T3 IPV	0.05	0.01	0.03	0.04	− 0.12	− 0.22**	− 0.06	− 0.10	0.16*	0.15+	− 0.05	− 0.25**	− 0.40***	0.24**	0.35***	0.42***	0.20*	0.21**	0.15 +

*, **, *** $p < 0.05$.

Table 2
Cross-sectional associations between fathers' testosterone and father depressive symptoms at nine months postpartum.

	Model 1: Fathers' T2 T AUCg associated with father T2 depressive symptoms r (132, 7) = 0.42, F = 3.92, p = 0.001 Effect size (Cohen's F^2) = 0.22		Model 2: Fathers' T2 T AUCg and father T2 relationship satisfaction associated with father T2 depressive symptoms r (130, 8) = 0.52, F = 5.72, p = 0.001 Effect size (Cohen's F^2) = 0.37	
	Beta	t	Beta	t
(Constant)		2.17*		14.06
Father T2 BMI	0.16	1.88 +	0.12	1.48
Child age	0.01	0.07	0.03	0.37
Father age	− 0.22	− 2.40*	− 0.27	− 3.02**
Father minority	0.21	1.79†	0.22	2.00*
Father education	− 0.03	− 0.26	0.01	0.12
Father foreign born	− 0.11	− 1.03	− 0.08	− 0.71
Father T2 T AUCg	− 0.17	− 2.02*	− 0.19	− 2.34*
Father T2 DAS	–	–	− 0.32	− 4.02***

† $p < 0.10$.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

sex was not associated with father T. Fathers with higher morning and aggregate testosterone reported fewer depressive symptoms at T1 and, at a marginal level of significance, T2 and T3. Younger fathers, non-white fathers, and foreign-born fathers had higher evening T. The partners of fathers with higher evening testosterone reported more depressive symptoms at T2 and T3. At a marginal level of significance, fathers with higher evening testosterone at T2 reported more paternal parenting stress at T3, and their partners reported higher maternal parenting stress at T3 as well.

Hypothesis 1. Associations between fathers' testosterone and father depressive symptoms.

To test our first hypothesis, we examined cross-sectional associations between testosterone and depressive symptoms (both measured at T2) in a regression model that included

covariates that were correlated with testosterone (infant and father age, father education,

minority status, and foreign birth, and father BMI). As shown in Table 2, fathers with lower aggregate testosterone at approximately nine months postpartum also reported higher depressive symptoms at the same study visit. These results held when we controlled for fathers' T2 relationship satisfaction, and both T2 relationship satisfaction and T2 aggregate testosterone were significantly negatively associated with T2 paternal depressive symptoms. There was no significant curvilinear relationship between father T2 testosterone and father T2 depressive symptoms.

Fathers' aggregate testosterone at T2 did not predict their subsequent (T3) depressive symptoms. However, there was a curvilinear result such that the aggregate testosterone-squared term predicted father T3 depressive symptoms, r (125) = 0.24, t = 2.64, p = 0.009. The positive coefficient for the testosterone-squared term suggests a U-shaped relationship, such that both particularly low and particularly high levels of T2 testosterone predicted more depressive symptoms. However, note that the test statistic for this model (F (125, 7) = 2.28, p = 0.022) did not meet our adjusted p-value threshold for multiple comparisons.

Hypothesis 2. Associations between fathers' testosterone and mother depressive symptoms.

Aggregate T2 testosterone was not associated with mother depressive symptoms at T2. However, as shown in Table 3, father evening testosterone and maternal depressive symptoms were cross-sectionally associated at T2, and this result remained significant after we added paternal T2 relationship satisfaction to the model. Controlling instead for maternal T2 relationship satisfaction made this result marginally significant, and T2 maternal relationship satisfaction was a significant predictor of concurrent T2 maternal depressive symptoms.

Next, we tested longitudinal associations between maternal depressive symptoms and paternal testosterone. Both father evening testosterone and aggregate testosterone at T2 predicted higher maternal T3 depressive symptoms (b (130) = 0.22, t = 2.35, p = 0.02 and b (130) = 0.23, t = 2.52, p = 0.01, respectively), and both relationships held whether we included maternal T2 relationship satisfaction, which was negatively associated with T3 depression (b (138) = − 0.22, t = − 2.59, p = 0.01).

Hypothesis 3. Relationship satisfaction as a mediator of the association between fathers' testosterone and maternal depression.

To test whether maternal relationship satisfaction would mediate

Table 3
Cross-sectional associations between fathers' evening testosterone (T) and mother postpartum depressive symptoms (PDS) at nine months postpartum.

	Model 1: Fathers' evening T associated with mother T2 PDS r (135, 8) = 0.27, F = 1.22, p = 0.30 Effect size (Cohen's F^2 = 0.08)		Model 2: Fathers' evening T associated with mother T2 PDS, with father T2 relationship satisfaction r (133, 9) = 0.28, F = 1.14, p = 0.34 Effect size (Cohen's F^2 = 0.09)		Model 3: Fathers' evening T associated with mother T2 PDS, with mother T2 relationship satisfaction r (133, 9) = 0.43, F = 3.07, p = 0.002 Effect size (Cohen's F^2 = 0.23)	
	Beta	t	Beta	t	Beta	t
(Constant)		1.55		1.61*		3.42*
Father T2 BMI	0.02	0.25	0.01	0.07	0.01	0.02
Child age	0.01	0.09	0.02	0.23	0.02	0.36
Father age	− 0.12	− 1.20	− 0.14	− 1.40	0.03	− 1.64
Father minority	− 0.02	− 0.19	− 0.04	− 0.28	− 0.15	− 0.33
Father education	− 0.11	− 0.96	− 0.11	− 0.91	− 0.04	− 0.73
Father foreign born	− 0.06	− 0.46	− 0.05	− 0.44	− 0.08	− 0.04
Father evening time	− 0.07	− 0.81	− 0.06	− 0.67	− 0.12	− 1.42
Father evening T	− 0.21	2.22*	− 0.19	2.08*	0.15	1.74†
T2 DAS			− 0.07	− 0.79	− 0.35	− 4.09**

† $p < 0.10$.
* $p < 0.05$ level.
** $p < 0.01$.

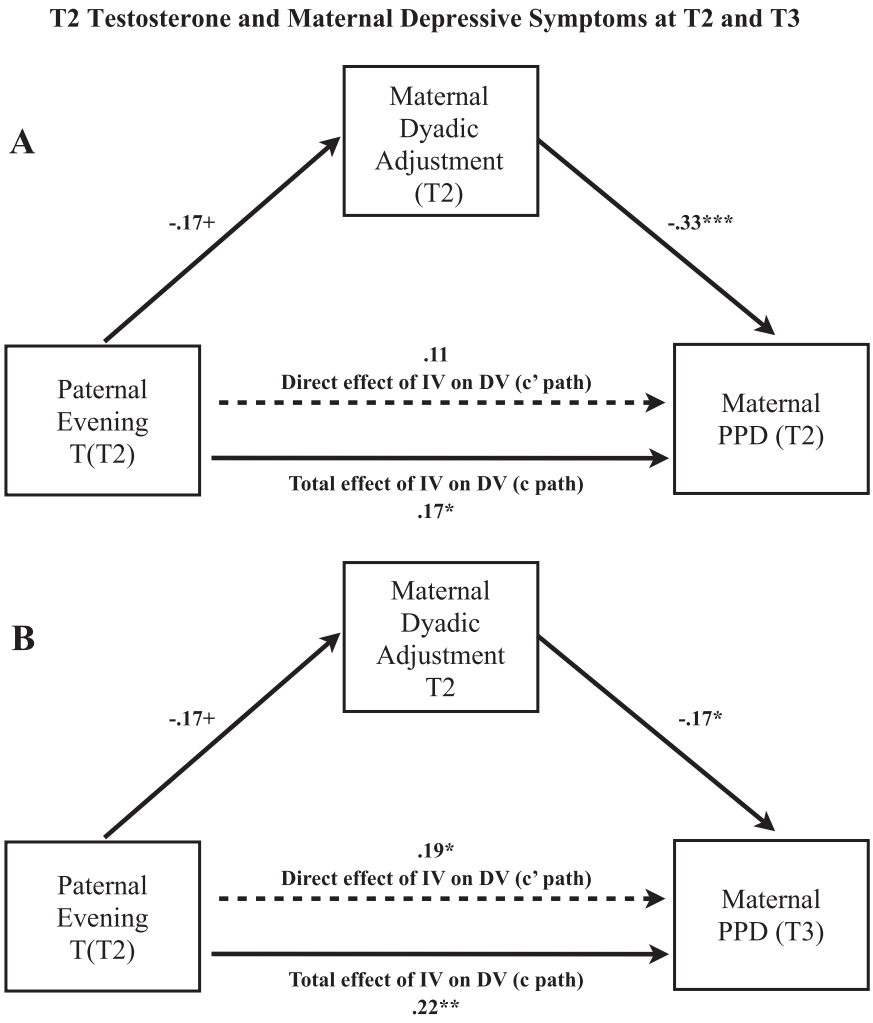

Fig. 1. Maternal dyadic adjustment mediating associations between paternal T2 testosterone and maternal depressive symptoms at T2 and T3.

the association between father T2 evening testosterone and maternal T2 and T3 depressive symptoms, we conducted a bootstrapping test of mediation, including the same covariates as in the regression models (child age, father age, father BMI, father education, father minority status, and father foreign birth). Results are depicted in Fig. 1. Cross-sectionally, we found that maternal relationship satisfaction fully mediated the relationship between father T2 evening testosterone and maternal depressive symptoms at T2 yielding 95% bias-corrected and accelerated bootstrap confidence intervals not containing zero; CI = 0.01 to 0.53, with a relative indirect effect (ratio of indirect effect to the total effect) of 0.51. Longitudinally, we found that maternal T2 relationship satisfaction fully mediated the association between father evening testosterone at T2 and maternal T3 depressive symptoms (CI = 0.01 to 0.37, relative indirect effect = 0.16). However, maternal T2 relationship satisfaction did not mediate the association between father aggregate testosterone and maternal T3 depressive symptoms (CI = −0.03 to 0.40, relative indirect effect = 0.12).

Hypothesis 4. Associations between fathers' testosterone, fathering stress and intimate partner aggression.

To test our fourth hypothesis, we ran a stepwise regression model to predict whether fathers' testosterone and father depressive symptoms at T2 predicted fathering stress at T3. First, we tested only father T2 testosterone as a predictor; next, we tested a model that included both testosterone and father T2 depressive symptoms. As shown in Table 4, fathers' testosterone was only a significant predictor of paternal parenting stress in the model that also included depressive symptoms, and both testosterone and depressive symptoms were independent predictors of paternal parenting stress, in the same direction: higher testosterone and higher depressive symptoms at T2 both predicted greater fathering stress approximately six months later. Father T2 testosterone was not a significant predictor of maternal T3 parenting stress in a model including these same covariates, r (124) = 0.16, t = 1.63, p = 0.11. A curvilinear relationship also emerged between father T2 testosterone and T3 fathering stress: substituting the curvilinear (aggregate testosterone-squared term) for the linear aggregate testosterone term, father T2 testosterone-squared predicted father T3 parenting stress, r (118) = 0.20, t = 2.26, p = 0.026, such that parenting stress was heightened for fathers at both low and high levels of testosterone.

Next, we tested whether fathers' testosterone predicted mothers' report of intimate partner aggression (IPA) at T3. As shown in Table 4, higher father T2 testosterone predicted greater T3 IPA, whether or not the model included father T2 depressive symptoms.

5. Discussion

Fathers' testosterone measured approximately nine months after the birth of a child was associated with both maternal and paternal postpartum depressive symptoms. Interestingly, these associations were in opposite directions. Fathers with lower testosterone at the nine month visit reported more depressive symptoms at the same visit, and their partners reported *fewer* symptoms at both nine and 15 months postpartum. Maternal relationship satisfaction fully mediated the association between father evening testosterone and both nine-month and 15-

Table 4
Fathers' testosterone (T) and postpartum depressive symptoms (PDS) at nine months postpartum predicting family outcomes at 15 months postpartum.

Models predicting T3 father parenting stress				
	Model 1: Fathers' T2 T AUC predicting T3 father parenting stress r (118, 8) = 0.38, F = 2.36, p = 0.022 *Effect Size (Cohen's F^2)* = 0.17		Model 2: Fathers' T2 T AUC and father T2 PPD predicting T3 father parenting stress r (118, 9) = 0.46, F = 3.17, p = 0.002 Effect size (Cohen's F^2) = 0.27	
	Beta	*t*	Beta	*t*
(Constant)		3.91***		3.50
Father T2 BMI	0.03	0.31	− 0.01	− 0.11
Child age	− 0.10	− 0.96	− 0.09	0.97
Father age	0.06	0.64	0.12	1.23
Father minority	0.09	0.73	0.04	0.33
Father education	− 0.28	− 2.37*	− 0.27	2.36*
Father foreign born	0.02	0.16	0.06	0.51
Lag from T2 to T3	− 0.10	− 0.98	− 0.07	− 0.74
Father T2 T AUCg	0.14	1.50	0.18	1.99*
Father T2 PDS		–	0.27	2.89**

Models predicting T3 IPV				
	Model 3: Fathers' T2 T AUC predicting T3 mother-reported IPV r (127, 8) = 0.47, F = 4.30, p = 0.001 Effect size (Cohen's F^2) = 0.29		Model 4: Fathers' T2 T AUCg and father T2 PPD predicting T3 mother-reported IPV r (127, 9) = 0.48, F = 4.01, p = 0.001 Effect size (Cohen's F^2) = 0.31	
	Beta	*t*	Beta	*t*
(Constant)		2.16*		1.92+
Father T2 BMI	0.15	1.81+	0.13	1.54
Child age	0.01	0.07	0.01	0.14
Father age	− 0.14	− 1.48	− 0.11	− 1.21
Father minority	0.38	3.22**	0.35	2.97**
Father education	− 0.17	− 1.51	− 0.17	− 1.58
Father foreign born	− 0.39	− 3.49**	− 0.38	− 3.41**
Lag from T2 to T3	0.21	2.21*	0.22	2.32*
Father T2 T AUCg	0.18	2.10*	0.20	2.30*
Father T2 PDS	–	–	0.11	1.22

+ $p < 0.10$.
* $p < 0.05$ level.
** $p < 0.01$.
*** $p < 0.001$.

month maternal postpartum depression. Higher paternal testosterone predicted adverse family outcomes, specifically fathering stress and intimate partner aggression at 15 months postpartum. Our findings suggest that both low and high testosterone are associated with heightened risk for fathers and for the family, but through different pathways. In keeping with this conclusion, a curvilinear relationship between nine-month aggregate testosterone and 15-month postpartum depression and parenting stress for fathers also emerged, such that fathers with both particularly low and particularly high levels of testosterone subsequently reported more depressive symptoms and greater parenting stress.

Our results are consistent with previous studies identifying low testosterone as a correlate of depression in males, but also suggest new directions for research. Although fatherhood has been associated with lowered testosterone in men, and it has been theorized that declines in testosterone might play a role in postpartum depression (Kim and Swain, 2007), this study provides the first empirical evidence in support of this theory. It is also possible that a third variable (such as sleep disturbance or childcare involvement) influenced both testosterone and depression. Indeed, in another investigation involving both mothers and fathers from this study, we found bidirectional associations between parents' sleep problems and their depressive symptoms (Saxbe et al., 2017). We did not collect a detailed measure of childcare involvement, attitudes, and experiences in this study, but we would expect associations between these variables and testosterone might emerge, consistent with other studies that have found links between parenting behavior and testosterone in fathers (Kuo et al., 2016).

Intriguingly, while low testosterone was linked with males' depression risk, *high* testosterone actually appeared to contribute to their partners' depression risk as well as downstream risks to the family, such as more fathering stress and more mother-reported intimate partner aggression. How might these seemingly contradictory findings be reconciled? On the one hand, testosterone has been linked with competition, dominance, and aggression (Booth et al., 2006), qualities that are traditionally valued in men and that may be protective against depression. On the other hand, low testosterone in a parenting context may be adaptive, potentially facilitating men's nurturant behavior and investment in the family. Social contextual variables are clearly relevant to interpreting men's testosterone and its functional role across the lifespan. Our curvilinear results support this idea, replicating Booth et al. (1999) in finding that both low and high levels of testosterone predict depression in fathers. Similarly, the linear association between fathers' testosterone and subsequent parenting stress only emerged as significant when we controlled for fathers' postpartum depressive symptoms, suggesting a complex relationship between testosterone, depression, and parenting. Consistent with this, we also found a curvilinear association between testosterone and parenting stress, such that fathers who had both low and high levels of testosterone subsequently found parenting to be more stressful.

Our results support a social mediation model for maternal depression, with maternal relationship satisfaction mediating the association between paternal evening testosterone and maternal depressive

symptoms at both nine months and 15 months postpartum. Higher-testosterone fathers might be less invested in the partner relationship (Saxbe et al., 2016), which could translate into greater relationship distress for women that contributes to depression. Indeed, our results converge with a literature showing that a lack of social support, specifically partner support, is a known risk factor for postpartum depression in women (Stapleton et al., 2012). In addition to potentially reduced investment and commitment to the relationship, our results suggest that high testosterone men might also behave more aggressively with their partners, potentially further contributing to maternal relationship dissatisfaction and depression. Testosterone supplementation has been recommended as a treatment for depression in males (Zarrouf et al., 2009), but our results suggest that treatment providers may need to take a more nuanced view of the role of testosterone within the family system.

Some of our findings were driven by evening testosterone, such as the links between fathers' testosterone and maternal depressive symptoms, as well as the mediating effect of maternal relationship satisfaction. Evening testosterone levels may reflect social experiences throughout the day and therefore, might be more strongly linked with family dynamics than morning levels (Book et al., 2001; Gray et al., 2002). Although many studies of men's testosterone and depression have focused only on morning levels, examining both morning and evening levels may provide a clearer picture of testosterone in its daily social context.

Study limitations include that testosterone was only measured at the nine-month postpartum visit, and only in fathers. Women's testosterone and mother-father associations in testosterone may also contribute to depression risk and family functioning. Furthermore, multiple measures of testosterone could have helped infer the direction of causality and provided information about whether testosterone levels were stable or declining during the postpartum period. Another limitation regarding measurement is that the full relationship satisfaction scale was only administered at the nine-month postpartum visit, whereas intimate partner aggression and paternal parenting stress were measured only at the 15-month postpartum visit. Paternal depression, aggression, and parenting stress may be partially overlapping constructs, all reflecting poor underlying mental health. Ideally these measures would have been administered at multiple waves of the study to allow us to test additive or unique effects. However, the larger CCHN study sought to balance the goal of measuring multiple constructs with the need to reduce participant burden. These concerns are offset by the unusually racially and ethnically diverse low-income sample. Most (95%) of fathers in our sample were residing with mothers at the T2 visit, preventing us from exploring differences between residential vs. non-residential or partnered vs. non-partnered fathers, despite evidence that these groups might differ (Garfield et al., 2014; Gettler et al., 2015). Although the repeated measures of postpartum depression from both mothers and fathers is a strength of the study, it also raises the issue of multiple comparisons. We evaluated our results using a corrected p value threshold, adjusted for the number of analyses, and one model fell below this threshold (the curvilinear model predicting fathers' 15-month depressive symptoms), suggesting that it should be interpreted with caution.

In conclusion, this paper is the first to show that paternal testosterone levels are linked with both maternal and paternal depressive symptoms, fathering stress, and intimate partner aggression following the birth of a child. The postpartum period is a time of dynamic fluctuations in hormones and mood that may have critical importance to the long-term health and well-being of the family. Our results contribute to our knowledge of the biological correlates and consequences of men's adjustment to parenthood, and suggest important new directions for testosterone research in males.

References

Abidin, R.R., 1995. Parenting Stress Index, Third Edition. Psychological Assessment Resources, Inc., Odessa, FL Professional Manual.

Almeida, O.P., Yeap, B.B., Hankey, G.J., Jamrozik, K., Flicker, L., 2008. Low free testosterone concentration as a potentially treatable cause of depressive symptoms in older men. Arch. Gen. Psychiatry 65 (3), 283–289.

Amiaz, R., Seidman, S.N., 2008. Testosterone and depression in men. Curr. Opin. Endocrinol. Diabetes Obes. 15 (3), 278–283.

Barrett-Connor, E., von Mühlen, D.G., Kritz-Silverstein, D., 1999. Bioavailable testosterone and depressed mood in older men: the Rancho Bernardo study. J. Clin. Endocrinol. Metab. 84 (2), 573–577.

Berg, S.J., Wynne-Edwards, K.E., 2001. Changes in testosterone, cortisol, and estradiol levels in men becoming fathers. Mayo Clin. Proc. 76 (6), 582–592.

Beydoun, H.A., Beydoun, M.A., Kaufman, J.S., Lo, B., Zonderman, A.B., 2012. Intimate partner violence against adult women and its association with major depressive disorder, depressive symptoms and postpartum depression: a systematic review and meta-analysis. Soc. Sci. Med. 75 (6), 959–975.

Book, A.S., Starzyk, K.B., Quinsey, V.L., 2001. The relationship between testosterone and aggression: a meta-analysis. Aggress. Violent Behav. 6 (6), 579–599.

Booth, A., Granger, D.A., Mazur, A., Kivlighan, K.T., 2006. Testosterone and social behavior. Soc. Forces 85 (1), 167–191.

Booth, A., Johnson, D.R., Granger, D.A., 1999. Testosterone and men's depression: the role of social behavior. J. Health Soc. Behav. 130–140.

Booth, A., Johnson, D.R., Granger, D.A., 2005. Testosterone, marital quality, and role overload. J. Marriage Fam. 67, 483–498.

Clark, M.M., Galef, B.G., 2000. Why some male Mongolian gerbils may help at the nest: testosterone, asexuality and alloparenting. Anim. Behav. 59 (4), 801–806.

Cox, J.L., Holden, J.M., Sagovsky, R., 1987. Detection of postnatal depression. Development of the 10-item Edinburgh postnatal depression scale. Brit. J. Psych. 150, 782–786.

Dunkel Schetter, C., Schafer, P., Lanzi, R.G., 2013. Shedding light on the mechanisms underlying health disparities through community participatory methods: the stress pathway. Perspect. Psychol. Sci. 8, 613–633.

Fleming, A.S., Corter, C., Stallings, J., Steiner, M., 2002. Testosterone and prolactin are associated with emotional responses to infant cries in new fathers. Horm. Behav. 42 (4), 399–413.

Garfield, C.F., Duncan, G., Rutsohn, J., McDade, T.W., Adam, E.K., Coley, R.L., Chase-Lansdale, P.L., 2014. A longitudinal study of paternal mental health during transition to fatherhood as young adults. Pediatrics 836–843.

Gettler, L.T., McDade, T.W., Agustin, S.S., Feranil, A.B., Kuzawa, C.W., 2015. Longitudinal perspectives on fathers' residence status, time allocation, and testosterone in the Philippines. Adaptive Human Behavior and Physiology 1 (2), 124–149.

Gettler, L.T., McDade, T.W., Feranil, A.B., Kuzawa, C.W., 2011. Longitudinal evidence that fatherhood decreases testosterone in human males. Proc. Natl. Acad. Sci. 108 (39), 16194–16199.

Gettler, L.T., Oka, R.C., 2016. Are testosterone levels and depression risk linked based on partnering and parenting? Evidence from a large population-representative study of US men and women. Soc. Sci. Med. 163, 157–167.

Gettler, L.T., Ryan, C.P., Eisenberg, D.T., Rzhetskaya, M., Hayes, M.G., Feranil, A.B., Bechayda, S.A., Kuzawa, C.W., 2017. The role of testosterone in coordinating male life history strategies: the moderating effects of the androgen receptor CAG repeat polymorphism. Horm. Behav. 87, 164–175.

Giltay, E.J., Enter, D., Zitman, F.G., Penninx, B.W., van Pelt, J., Spinhoven, P., Roelofs, K., 2012. Salivary testosterone: associations with depression, anxiety disorders, and antidepressant use in a large cohort study. J. Psychosom. Res. 72 (3), 205–213.

Gray, P.B., Kahlenberg, S.M., Barrett, E.S., Lipson, S.F., Ellison, P.T., 2002. Marriage and fatherhood are associated with lower testosterone in males. Ev. Hum. Behav. 23, 193–201.

Gray, P.B., Yang, C.F.G., Pope, H.G., 2006. Fathers have lower salivary testosterone levels than unmarried men and married non-fathers in Beijing, China. Proc. R. Soc. Lond. B Biol. Sci. 273 (1584), 333–339.

Holm, S., 1979. A simple sequentially rejective multiple test procedure. Scand. J. Stat. 6 (2), 65–70.

Kim, P., Swain, J.E., 2007. Sad dads. Psychiatry 36–47.

Kuo, P.X., Saini, E.K., Thomason, E., Schultheiss, O.C., Gonzalez, R., Volling, B.L., 2016. Individual variation in fathers' testosterone reactivity to infant distress predicts parenting behaviors with their 1-year-old infants. Dev. Psychobiol. 58, 303–314.

McKenry, P.C., Julian, T.W., Gavazzi, S.M., 1995. Toward a biopsychosocial model of domestic violence. J. Marital. Fam. Ther. 307–320.

Misri, S., Kostaras, X., Fox, D., Kostaras, D., 2000. The impact of partner support in the treatment of postpartum depression. Can. J. Psychol. 45 (6), 554–558.

O'Campo, P., Caughy, M.O., Nettles, S.M., 2010. Partner abuse or violence, parenting and neighborhood influences on children's behavioral problems. Soc. Sci. Med. 70 (9), 1404–1415.

O'Connor, D.B., Archer, J., Wu, F.C., 2004. Effects of testosterone on mood, aggression, and sexual behavior in young men: a double-blind, placebo-controlled, cross-over study. J. Clin. Endocrinol. Metab. 89 (6), 2837–2845.

Paulson, J.F., Bazemore, S.D., 2010. Prenatal and postpartum depression in fathers and its association with maternal depression: a meta-analysis. JAMA 303, 1961–1969.

Perrigo, G., Belvin, L., Vom Saal, F.S., 1991. Individual variation in the neural timing of infanticide and parental behavior in male house mice. Physiol. Behav. 50 (2), 287–296.

Pinto, L.A., Sullivan, E.L., Rosenbaum, A., Wyngarden, N., Umhau, J.C., Miller, M.W., Taft, C.T., 2010. Biological correlates of intimate partner violence perpetration.

Aggress. Violent Behav. 15 (5), 387–398.
Preacher, K.J., Hayes, A.F., 2008. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav. Res. Methods 40 (3), 879–891.
Pruessner, J.C., Kirschbaum, C., Meinlschmid, G., Hellhammer, D.H., 2003. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. PNEC 28 (7), 916–931.
Ramchandani, P., Stein, A., Evans, J., O'Connor, T.G., ALSPAC Study Team, 2005. Paternal depression in the postnatal period and child development: a prospective population study. Lancet 365, 2201–2205.
Ramey, S.L., Schafer, P., DeClerque, J.L., 2015. The preconception stress and resiliency pathways model: a multi-level framework on maternal, paternal, and child health disparities derived by community-based participatory research. Matern. Child Health J. 19 (4), 707–719.
Saltzman, W., Ziegler, T.E., 2014. Functional significance of hormonal changes in mammalian fathers. J. Neuroendocrinol. 26 (10), 685–696.
Saxbe, D.E., Edelstein, R.S., Lyden, H.M., Wardecker, B.M., Chopik, W.J., Moors, A.C., 2016. Fathers' decline in testosterone and synchrony with partner testosterone during pregnancy predicts greater postpartum relationship investment. Horm. Behav. 90, 39–47.
Saxbe, D.E., Schetter, C.D., Guardino, C.M., Ramey, S.L., Shalowitz, M.U., Thorp, J., Vance, M., 2017. Sleep quality predicts persistence of parental postpartum depressive symptoms and transmission of depressive symptoms from mothers to fathers. Ann. Behav. Med. 50 (6), 862–875.
Schweiger, U., Deuschle, M., Weber, B., Korner, A., Lammers, C.H., Schmider, J., Gotthardt, U., Heuser, I., 1999. Testosterone, gonadotropin, and cortisol secretion in male patients with major depression. Psychosom. Med. 61 (3), 292–296.
Seidman, S.N., Araujo, A.B., Roose, S.P., Devanand, D.P., Xie, S., Cooper, T.B., McKinlay, J.B., 2002. Low testosterone levels in elderly men with dysthymic disorder. Am. J. Psychiatry 159, 456–459.
Seidman, S.N., Araujo, A.B., Roose, S.P., McKinlay, J.B., 2001. Testosterone level, androgen receptor polymorphism, and depressive symptoms in middle-aged men. Biol. Psychiatry 50 (5), 371–376.
Sherin, K.M., Sinacore, J.M., Li, X.Q., Zitter, R.E., Shakil, A., 1998. HITS: a short domestic violence screening tool for use in a family practice setting. Fam. Med. 30 (7), 508–512.
Spanier, G.B., 1976. Dyadic Adjustment Scale (DAS). J. Marriage Fam. 38, 15–28.
Stapleton, L.R., Dunkel Schetter, C., Westling, E., Rini, C., Glynn, L.M., Hobel, C.J., Sandman, C.A., 2012. Perceived partner support in pregnancy predicts lower maternal and infant distress. J. Fam. Psychol. 26 (3), 453–463.
Storey, A.E., Walsh, C.J., Quinton, R.L., Wynne-Edwards, K.E., 2000. Hormonal correlates of paternal responsiveness in new and expectant fathers. Evol. Hum. Behav. 21 (2), 79–95.
Storey, A.E., Ziegler, T.E., 2016. Primate paternal care: interactions between biology and social experience. Horm. Behav. 77, 260–271.
van Anders, S.M., Goldey, K.L., Kuo, P.X., 2012. The steroid/peptide theory of social bonds: integrating testosterone and peptide responses for classifying social behavioral contexts. PNEC 36 (9), 1265–1275.
Vogel, W., Klaiber, E.L., Broverman, D.M., 1978. Roles of the gonadal steroid hormones in psychiatric depression in men and women. Prog. in Neuro-Psychopharmy 2 (4), 487–503.
Zarrouf, F.A., Artz, S., Griffith, J., Sirbu, C., Kommor, M., 2009. Testosterone and depression: systematic review and meta-analysis. J. Psychiatr. Pract. 15 (4), 289–305.
ZRT Laboratories, 2017. Technical Specifications for Testosterone Assay. Retrieved April 27, 2017 from. http://www.zrtlab.com/images/documents/Testosterone_Saliva_Test_Specifications.pdf.