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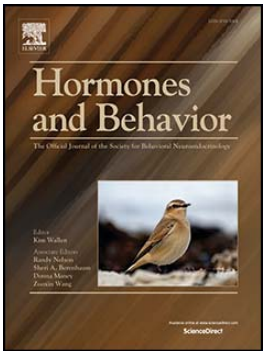


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# Fathers' Decline In Testosterone And Synchrony With Partner Testosterone During Pregnancy Predicts Greater Postpartum Relationship Investment

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Abstract

The transition to parenthood has been associated with declines in testosterone among partnered fathers, which may reflect males' motivation to invest in the family. Moreover, preliminary evidence has found that couples show correlations in hormone levels across pregnancy that may also be linked to fathers' preparation for parenthood. The current study used repeated-measures sampling of testosterone across pregnancy to explore whether fathers' change in T, and correlations with mothers' T, were associated with fathers' and mothers' postpartum investment.

In a sample of 27 couples (54 individuals) expecting their first child, both parents' salivary testosterone was measured multiple times across pregnancy. At approximately 3.5 months postpartum, participants rated their investment, commitment, and satisfaction with their partner. A multilevel model was used to measure change in testosterone over time and associations between mother and father testosterone. Fathers who showed stronger declines in T across pregnancy, and stronger correlations with mothers' testosterone, reported higher postpartum investment, commitment, and satisfaction. Mothers reported more postpartum investment and satisfaction if fathers showed greater prenatal declines in T. These results held even after controlling for paternal investment, commitment, and satisfaction measured prenatally at study entry.

Our results suggest that changes in paternal testosterone across pregnancy, and hormonal linkage with the pregnant partner, may underlie fathers' dedication to the partner relationship across the transition to parenthood.

Keywords: testosterone; couples; pregnancy; transition to parenthood; fathers

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Over the transition to parenthood, many men show a shift from mating-oriented to parenting-oriented behaviors. Although the evolutionary advantages of this shift are clear, its biological mechanisms are not well understood. Testosterone (T), an androgenic steroid hormone, may play a role in men's preparation for fatherhood. High levels of T have been associated with competition and aggression, whereas low levels of T have been associated with nurturance and particularly the care of offspring (van Anders, 2013). Cross-sectionally, lower levels of T have been found in partnered fathers and fathers who are more involved in their children's care as compared to men without children (e.g Alvergne, Faurie & Raymond, 2009; Burnham, Chapman, Gray, McIntyre, Lipson & Ellison, 2003; Gray, Parkin, Samms-Vaughan, 2007; Wynne-Edwards, 2001). Consistent with this, a longitudinal study found that T levels declined among men who became partnered fathers, and greater childcare involvement predicted a steeper decline (Gettler, McDade, Feranil, & Kuzawa, 2011). Generally, research has suggested that decreases in T are associated with sensitive and involved parenting, as well as with men's involvement in committed and long-term relationships (Fleming, Corter, Stallings, Steiner, 2001; Gettler et al., 2011; Storey & Ziegler 2015).

Animal studies suggest that experimentally lowered T (through castration after mating) is associated with decreases in infanticide after birth (in mice: vom Saal, 1983) and increases in caregiving behavior toward the infant (in gerbils: Clark & Galef, 2000). Similarly, marmoset males who carried infants most often show the lowest urinary T levels (Nunes et al., 2001), and the most significant declines in gonadal steroids (Nunes et al., 2000).

In humans, paternal decreases in T around the transition to parenthood may reflect a shift away from pursuing new mating opportunities in favor of investment in offspring and the partner relationship (van Anders, Goldey & Kuo, 2011). Among partnered men, those with lower

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salivary T report higher relationship satisfaction and commitment lower interest in sex outside the marriage (specifically the reported desire for uncommitted sexual activity), and a lower likelihood of divorce, compared to partnered men with higher T (e.g. Booth & Dabbs, 1993; Edelstein et al. 2011; Edelstein et al., 2014; Julian & McKenry, 1989; McIntyre et al. 2006; Perini et al., 2012). In two samples of young males, partnered fathers were found to have lower T than partnered, married and unpartnered men (Burnham et al., 2003; Gray et al., 2007). Similarly, monogamous men had lower T than polygynously married men and unpartnered men (Alvergne et al., 2009). Thus, men who were more invested in the partner relationship showed lower T levels across contexts. Interestingly, differences in desire for uncommitted sexual activity as a function of T level were not found in single men (Edelstein et al., 2011; McIntyre et al., 2006). Thus, it appears that testosterone decreases after becoming partnered is important for the investment in the parenting relationship.

Testosterone has also been associated with paternal behaviors in men. A study using an interactive doll found that men showed T declines in response to infant cries when able to comfort the doll (van Anders et al., 2012), although other studies have not found short-term declines in T among fathers interacting with their children (Gettler et al., Gray et al., Storey et al., & Weisman et al.). However, fathers who showed greater decreases in T during a stressful infant task (the Strange Situation) exhibited more sensitive fathering behavior during a subsequent task (Kuo, et al., 2015), and fathers with lower T overall (and smaller testes volume) also had greater involvement in infant caregiving (Mascaro, Hackett, & Rilling, 2013). Thus, although the evidence to date has been somewhat mixed, multiple studies suggest that partnered fathers with lower testosterone may show greater commitment to, and investment in, the family. There is some evidence that increases in childcare experience predict declines in men's

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testosterone over time (Gettler et al., 2015); however, given that most data on this topic is cross-sectional, the direction of causality in the relationship between testosterone and involvement in the family remains unknown.

Many questions regarding hormonal changes in human fathers remain unanswered. For example, what proximate signals might drive shifts in male testosterone over the transition to parenthood? Among women, many prenatal hormonal changes are precipitated by the placenta, and help to support a viable pregnancy and delivery. However, paternal behavior appears without any obvious biological or hormonal shift that is biologically necessary for the birth of the child. Cross-cultural perspectives suggest that the physical closeness of the two parents around the time of birth may predict paternal involvement after birth (Barry & Paxson, 1977; Whiting & Whiting, 1975; Broude, 1983; Elwood & Mason, 1994). Thus, proximity to a pregnant partner may instantiate hormonal shifts in men transitioning to fatherhood. In fact, animal models have found that in California mice and Djungarian hamsters, biobehavioral cues from females are important for the onset and maintenance of paternal care (Wynne-Edwards & Reburn, 2000; Gubernick & Nelson, 1989). In humans, women's prenatal hormones are associated with the stage of pregnancy and the closeness to the birth. However, one study of expectant couples found that men's hormones were correlated with women's hormones even after controlling for the days until birth, suggesting that fathers were more tuned to their partner than to the pregnancy stage (Storey et al., 2000). This evidence suggests that the father's relationship with the mother and exposure to the mother's hormones may contribute to biological readiness for fatherhood. Long-term behavioral data (from humans and animals) also suggest that direct, conspicuous and frequent paternal care tends to evolve simultaneously with the development of a pair-bond between the mother and care-giving father (Fernandez-Duque, Valeggia & Mendoza, 2009;

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Fraley, Brumbaugh, & Marks, 2005). Thus, associations between maternal and paternal hormones may reflect fathers' investment and commitment to the relationship dyad, specifically in the transition to parenthood context.

A growing literature has reported on physiological linkage among romantic partners (reviewed by Timmons, Margolin, & Saxbe, 2015). Within-dyad linkage (also described as "synchrony," "coregulation," or "covariation") has been observed in multiple physiological systems, including the sympathetic nervous system (e.g., heart rate, electrodermal activity) and the endocrine system (with most research focusing on the hypothalamic pituitary adrenal (HPA) axis, which produces the stress hormone cortisol). These studies have found evidence for both cross-sectional and time-lagged positive associations in couples' physiology using a variety of research designs. For example, synchrony in couples' momentary cortisol changes has been observed in experience sampling studies over several days (Liu et al., 2013; Papp et al., 2013; Saxbe & Repetti, 2010; Saxbe et al., 2015) as well as in the lab (Laurent & Powers, 2007; Saxbe et al., 2014). These associations have been linked to the couples' proximity and time spent together, as well as characteristics of their relationship.

Most of the above-cited studies have not included pregnant couples (excepting Saxbe et al., 2015). However, several studies focused on the transition to parenthood have also reported positive correlations in couples' hormones during this time. Specifically, Edelstein and colleagues (2015) found significant correlations in couples' average levels of prenatal cortisol and progesterone, as well as marginally significant correlations in prenatal testosterone; Storey et al. (2000) found positive associations in prolactin, estradiol, and cortisol among pregnant couples (but did not measure testosterone in women). In contrast, Wynne-Edwards and Berg (2002) did not find correlations in testerosterone and estradiol among couples sampled before and after

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birth, although they did find positive associations in prenatal cortisol and as well as positive correlations between women's cortisol and men's testosterone. However, their sample was small (9 couples).

To our knowledge, only three published studies have tested within-couple linkage in testosterone over time: two studies of pregnant couples (the above-cited Edelstein et al., 2015 and Wynne-Edwards & Berg, 2002 studies) and one study of non-pregnant couples (Schneiderman et al., 2014 measured hormones in newly dating couples during a laboratory conflict discussion). None of these studies found associations between couples' testosterone levels, with the exception that Edelstein et al. (2015) reported marginally significant positive correlations after averaging across time points. Additionally, Booth, Johnson, and Granger (2005) found small positive correlations in testosterone in a sample of 307 couples, although these couples were not pregnant and their testosterone levels were only measured twice, on two successive mornings.

Despite the paucity of prior evidence, the possibility of linkage in couples' testosterone levels during pregnancy may warrant further investigation. First, as discussed above, testosterone appears to drop among partnered males who become fathers, without a clear precipitating influence. Hormonal entrainment between partners is one plausible explanation for these changes. Additionally, within-couple cortisol linkage has now been reported in at least nine published studies (listed in Timmons, et al., 2015), and cortisol and testosterone show bidirectional influences on each other (Mehta, Jones, & Josephs, 2008; Orr & Mann, 1992; Sapolsky, 1985). Moreover, the three studies that examined testosterone linkage in couples shared common statistical limitations. All three studies performed correlations using either aggregate hormone levels (averaged over time) or single levels measured at one timepoint. Given

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that testosterone appears to fluctuate dynamically, particularly during the transition to parenthood, a more appropriate statistical approach would use multilevel modeling to assess partners' trajectories of hormonal change over time across multiple sampling timepoints while simultaneously testing their degree of association with their partner's hormones at each timepoint.

We previously reported on levels of T, cortisol, progesterone, and estradiol in a sample of 29 couples from the current dataset who were followed across four prenatal timepoints (Edelstein et al., 2015). Mothers showed increasing T across the prenatal period, whereas fathers showed decreasing T; partners' average levels of T were weakly correlated with each other, at a marginal level of significance. The current study has two main aims. First, we build on the earlier study and the inconclusive prior literature by using multilevel modeling to test both paternal testosterone changes and within-couple linkages within the same statistical model. This represents an advance over the previous literature because it allows us to capture and control for hormonal change over time and disambiguate this change from within-couple linkage. Next, we examine the functional implications of fathers' testosterone changes over pregnancy and their degree of linkage with partners. Given preliminary evidence that shifts in prenatal testosterone may reflect an orientation away from mating opportunity and toward investment in the stable couple relationship in order to facilitate child-rearing, we focused on fathers' postpartum reports of their investment, commitment, and satisfaction with the couple relationship.

## Methods

### Participants

The study was conducted at a large public university in the Midwest. Couples were recruited via online and print advertisements to participate in a study of hormonal and

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psychological changes among first-time parents (see Edelstein et al., 2015, for more details). Twenty-nine couples (58 individuals) participated in at least two and up to four prenatal visits. However, two couples did not complete the postpartum follow-up assessment, so our final sample consisted of 27 couples (54 individuals). Eligibility criteria included that both partners be adults (between 18-45), and that the couple was cohabiting, expecting their first child, and within the first two trimesters of pregnancy at the first prenatal visit. One male participant had a child from a previous relationship, but this was the first child together for all couples and the first pregnancy for all female participants. Exclusion criteria included current smoking habit, and medical conditions or medications that could influence hormones (e.g., autoimmune disorders, steroid medications).

The majority of couples were married or engaged (91%). Median household income was $50,000-$75,000 and 72% of participants had at least a college degree. Women were, on average, 29.19 years of age (SD = 3.93. range 20-38); men's average age was 30.33 (SD = 4.50, range 20-42). Participants self-reported their race/ethnicity as 70% Caucasian, 4% Black or African American, 7% Asian American, 6% Hispanic, and 6% mixed or other ethnicities (7% did not report their race/ethnicity).

**Procedures**

All procedures were approved by the university's Institutional Review Board (IRB). Prenatal laboratory sessions were scheduled, according to couples' due dates, at approximately 8-week intervals (roughly weeks 12, 20, 28, and 36 gestation). These intervals were modeled after Fleming et al. (1997), with the goal of capturing hormones during each trimester and at the very end of pregnancy. However, due to the difficulty of recruiting couples earlier in the first trimester, our study began at 12 weeks and we targeted the beginning of the ranges used by

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Fleming for subsequent sessions. Several couples began the study during the second trimester of pregnancy, and some did not complete the last session because their baby was born before their scheduled session, so there was some variability in the number of sessions completed by each couple (mean number of sessions =3.62, SD=0.62). On average, couples did their first study visit at 12.86 weeks (range 10-18 weeks, SD = 2.06), their second visit at 21.04 weeks (range 18-25, SD = 1.77), their third visit at 28.74 weeks (range = 24-32, SD = 1.63), and their fourth visit at 36.28 weeks (range = 34-38, SD = 1.17). Three couples completed two sessions, seven couples completed three sessions, and 19 couples completed all four sessions.

Couples came to the laboratory together for each session. Sessions were conducted on the same day of the week at the same time (as possible) for each couple in order to adjust for diurnal and day-to-day variations in hormone levels. Because hormone levels are most stable in the afternoon to evening hours (Schultheiss and Stanton, 2009), all couples were tested in the afternoon, between 12:30 h and 18:30 h. Informed consent was obtained during the initial session and participants were told that they could withdraw from the study at any time without penalty. Sessions included completion of questionnaire measures and hormone sampling. Participants provided two saliva samples to assess hormone levels–the first after a 20-minute adaptation period and the second 20 minutes later–to increase measurement reliability. These two samples were then averaged together.

**Salivary hormone collection protocol**. Participants were asked to refrain from eating, drinking (except for water), smoking, or brushing their teeth for 1 h before the beginning of each session. After rinsing their mouths with water, participants used polypropylene tubes to provide two 7.5 mL saliva samples during each of the in-laboratory sessions. Samples were frozen until further processing at our university's Core Assay Facility. Testosterone was assayed by

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radioimmunoassay (RIA), using commercially available kits from Siemens. The inter-assay coefficient of variation (CV) was 5.26% and 14.97% at high and low levels, respectively; the intra-assay CV was 9.86%. Analytical sensitivity (B022 SD) for testosterone was 1.14 pg/mL.

Samples were assayed in duplicate, and the average of duplicates was taken. Average hormone values were inspected for outliers, separately by gender and session. To maximize the use of all available data, hormone values that were larger than three standard deviations above the mean for each gender and session were replaced with values corresponding to three standard deviations above the mean for that particular variable (i.e., Winsorized; Reifman and Keyton, 2010; see also Edelstein et al., 2014, for a similar approach). Two testosterone values (both male) between 3 and 3.5 standard deviations above the mean were replaced using this approach.

**Postpartum Assessment**. An online postnatal follow-up questionnaire was emailed to participants approximately three months after their scheduled due date (mean of questionnaire completion = 14.36 weeks after birth; SD = 2.52 weeks; range 8.14-23.29). As is common in other studies of the transition to parenthood (e.g., Belsky & Rovine, 1990), we focused on three months postpartum because it reflects the end of the "fourth trimester" (Jennings & Edmundson, 1980), the baby's initial adjustment to life outside the womb and the parents' initial adjustment to the new family dynamic.

We assessed relationship quality with the widely used Investment Model Scale (IMS; Rusbult et al., 1998). The 10-item *satisfaction* subscale ($\alpha$ = .97) includes items such as "My relationship is close to ideal" and "Our relationship makes me very happy". The 7-item *commitment* subscale ($\alpha$ = .79) includes items such as "I want our relationship to last forever" and "I feel very attached to our relationship—very strongly linked to my partner". The 10-item

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*investment* subscale ($\alpha$ = .92) includes items such as "My partner and I share many memories" and "I have invested a great deal into our relationship that I would lose if the relationship were to end". Participants rated the extent to which they agreed with each statement, using a 9-point Likert-type scale, ranging from 1 (*do not agree at all*) to 9 (*agree completely*).

**Data Analysis Approach**. Multilevel modeling (HLM 7.0; Raudenbush et al., 2011) was used to test change in hormone levels and within-partner hormonal correlations across pregnancy. This approach is well suited for data that have a nested structure, such as multiple hormone sampling occasions nested within participants. HLM can also adjust for missing values at Level 1 (e.g. the fact that not all couples participated in all four prenatal visits).

For testing our first aim (paternal testosterone changes and within-couple linkage across pregnancy), a two level HLM model was used. At Level 1 (the visit-level) our outcome variable was fathers' testosterone level from each of the four prenatal visits. We included two predictors: the week of pregnancy that corresponded to each visit, and the mothers' testosterone level from that same visit.

## Results

Table 1 presents descriptive information on the sample and zero-order correlations between key study variables, including the testosterone values for fathers and mothers. As can be seen in the table and in Figure 1, which depicts mean testosterone levels over the four prenatal visits, fathers' mean testosterone levels dropped steadily from the first to the last visit, whereas mothers' testosterone levels increased sharply at the end of pregnancy. Both mother and father testosterone showed more variability (larger range and standard deviations) later in pregnancy. Comparing the first study visit to the last study visit, fathers showed a mean 17% decline in T across pregnancy (median 18%, ranging from a 76% decline to a 26% increase; standard

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deviation 25%). This decline was statistically significant, according to a paired-sample t test comparing first study visit T to last study visit T ($t(22)=3.08$, $p=.01$). Paternal prenatal testosterone was generally negatively associated with the Investment Model Scales, although many correlations were non-significant or marginally significant. Mother and father testosterone were not associated with each other early in pregnancy, but were significantly positively associated later in the pregnancy (the third and fourth prenatal visit), suggesting convergence of testosterone levels within couples over the course of pregnancy. Figure 2 depicts scatterplots showing correlations between mother and father T at each of the four study visits.

First, we tested a model with only these Level 1 coefficients (the week of pregnancy that corresponded to each visit and the mothers' testosterone level from that same visit). We found that fathers' testosterone dropped significantly over the four prenatal assessments ("pregnancy weeks" Level 1 variable = $b(80) = -.46$, $t =-3.86$, $p=.001$). In other words, the later in pregnancy the sample was taken, the lower fathers' testosterone. We also found that fathers' testosterone was significantly positively associated with mothers' when controlling for fathers' intercept (starting value of testosterone, e.g. his level at the first visit) and pregnancy week ($b(80)=.12$, $t=2.30$, $p=.02$). By including the pregnancy weeks predictor in the same model as mothers' testosterone, we are able to examine within-couple effects over and above the overall trajectory of hormonal change across pregnancy. In other words, fathers' testosterone showed linkage with mothers' testosterone at the same prenatal visit, after adjusting for pregnancy week or between-person differences in fathers' starting value of testosterone.

Next, at Level 2 (the couple-level), we included three covariates: the week of the couples' first visit to the lab, the number of weeks elapsed from the first visit and the last visit, and the sampling time of day for testosterone. Controlling for these variables allowed us to adjust for not

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only within-person change in hormone levels over pregnancy, but also between-couple differences in the timing of participation in the study. For example, a couple that did their first visit in week 15 and their last visit in week 32 might be expected to show different patterns of hormonal change than a couple participated first in week 18 and last in week 37. The week of the couples' first visit and the number of weeks elapsed between the first and last visit significantly moderated each of the Level 1 terms, such that the later in pregnancy the first week of study participation occurred, and the more weeks elapsed over total study participation, the more fathers' testosterone dropped from visit to visit and the stronger the relationship between mother and father testosterone. In other words, using the above hypothetical examples, the father that started the study with his partner in week 18 and completed the study in week 37 would show stronger testosterone declines and stronger linkage with mothers' testosterone than the father that started in week 15 and completed the study in week 32. This finding is consistent with expectations because, if testosterone drops across pregnancy, couples that participated for more weeks of pregnancy would be expected to show larger drops. Additionally, hormones appear to be most dynamic in later pregnancy (Fleming et al., 1997), which may explain why we found greater change and greater within-partner coregulation among couples that began the study later in pregnancy.

To test our second aim (the postpartum implications of paternal and within-couple testosterone changes), we added each of fathers' investment model variables to the model that included both the Level 1 and Level 2 covariates. We ran each investment model scale separately, in three different models (one the model for investment, one for satisfaction, and one for commitment). Results for each are shown in Table 2. Results were consistent across the three investment model variables: fathers who went on to report greater postpartum investment,

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commitment, and satisfaction with their partner showed more of a decline in testosterone across pregnancy (b=-.21, t=3.06, p=.003; b=-.38, t=-3.19, p=.002; and b=-.11, t=-1.79, p=.08, respectively) and stronger associations with partners' testosterone at each prenatal visit (b=.05, t=2.22, p=.03; b=.08, t=2.91, p=.01; and b=.04, t=2.52, p=.01, respectively). In other words, fathers' hormonal change and hormonal coregulation with the partner during pregnancy were each predictive of father dedication to the partner relationship during early parenthood. These results are summarized in Figure 3, a panel figure generated within HLM that shows estimated simple slopes at the 25% and 75% percentile for each of the investment model scales.

Next, we repeated these analyses but substituted mothers' postpartum investment model scales for fathers'. The overall results (e.g., for paternal change and within-couple linkage in testosterone) did not change. Consistent with the results for fathers' scores, both mothers' postpartum investment and postpartum satisfaction were associated with a greater decline in fathers' testosterone across pregnancy (b=-.29, t=-2.14, p=.04 and b=-.21, t=-2.65, p=.01, respectively). However, mothers' postpartum commitment was not associated with fathers' prenatal testosterone, and mothers' investment, commitment, and satisfaction were not associated with the within-couple linkage in testosterone across pregnancy.

Next, we added prenatal investment model scales completed at the first pregnancy visit. Including the prenatal scales as a covariate allows us to test whether fathers' prenatal testosterone patterns are associated with their postpartum relationship involvement over and above their prior (prenatal) ratings.

When tested alone (without postpartum investment model scores included in the model), none of the prenatal investment model scales were significantly associated with fathers' testosterone patterns or coregulation with the mother. These findings suggest that prenatal

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relationship quality does not predict fathers' testosterone declines or the extent to which their hormone levels are correlated with their partners. When we added the prenatal investment model scales to the model that also included the corresponding postpartum investment model scale, the postpartum investment model scales all remained significant, with one exception: the association between fathers' satisfaction and declines in T went from marginally significant to non-significant when prenatal satisfaction was controlled. Results are shown in Table 4.

We repeated the analysis with mothers' prenatal and postpartum investment scales entered together. As before, mothers reported more postpartum satisfaction when fathers' testosterone declined more across pregnancy (b=-.31, t=-3.23, p=.002). The association between mothers' postpartum investment and fathers' prenatal decline in testosterone became marginally significant after we controlled for prenatal investment (b=-.61, t=1.83, p=.07). As before, mothers' postpartum commitment was not associated with fathers' testosterone patterns, and mothers' satisfaction, commitment, and investment were not associated with within-couple linkage in testosterone.

Given that infant sex was significantly correlated with paternal postpartum relationship investment and commitment (such that fathers of girls reported greater dedication to the relationship) we also tested infant sex as a moderator on each of the Level 1 coefficients. However, it was not significantly associated with these coefficients and did not affect the investment model scale results for testosterone. Hence, we did not include it in final models for parsimony.

## Discussion

This study followed first-time parents across their pregnancy and into the first few months of the postpartum period. We found that fathers showed significant declines in

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testosterone as the pregnancy progressed, as well as significant positive correlations with their partners' testosterone level at each timepoint. Moreover, both paternal change over pregnancy and the degree of synchrony with mothers predicted fathers' investment, commitment, and satisfaction in the couple relationship at about 3.5 months postpartum. Paternal prenatal hormones predicted postpartum relationship outcomes even after adjusting for fathers' Investment Model Scale scores at the first prenatal assessment, suggesting that hormonal changes and synchrony contributed to greater paternal investment rather than vice versa.

This study is the first to explore within-couple synchrony in testosterone and fathers' hormonal change across pregnancy within the same statistical model, and the first to test whether fathers' prenatal hormonal synchrony with mothers predicts postpartum relationship outcomes. These results are novel, but are consistent with theory and with past research. Our finding that fathers show prenatal declines in testosterone, and that these declines predict postpartum relationship outcomes, replicates prior work but this study is the first, to our knowledge, to test repeated-measures change in prenatal testosterone using a multilevel model. This study is also the first to include partners' testosterone as a predictor of male prenatal testosterone and to connect within-dyad synchrony in prenatal testosterone with postpartum outcomes.

The mechanisms of these effects warrant greater exploration. For example, what might potentiate paternal hormonal change and synchrony with the partner? One possibility is that proximity and intimacy with the partner contribute to greater hormonal linkage. Father-mother relationship quality has been associated with increased paternal involvement in multiple studies (e.g., Harris & Morgan, 1991). Although these associations are usually explained in psychological terms (e.g., fathers who enjoy time with their partners are more likely to engage in family activities), it is also possible that the couples' physical closeness promotes greater

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paternal hormonal change, which in turn motivates greater father investment in the family. Cortisol research has also found that proximity and time together appear to contribute to within-dyad hormonal linkage (Timmons, Margolin, & Saxbe, 2015). Past research focused on cortisol has also found within-dyad linkage to be associated with generally negative relationship characteristics (e.g., marital distress (Saxbe & Repetti, 2010) and intimate partner violence (Saxbe et al., 2015)). This study found the opposite: fathers' degree of synchrony with their partners' testosterone predicted *better* postpartum relationship investment, satisfaction, and commitment. This difference may be due to both to the timing of our data collection (during pregnancy, when within-couple hormonal linkage may be adaptive and help to facilitate paternal preparation for parenthood) and to the hormone under study (cortisol is a hormone that reflects stress and threat, whereas testosterone has been associated with competition, power dynamics, and sexual motivation (Archer, 2006; Carré & Olmstead, 2015; Roney & Gettler, 2015). In addition to testosterone and cortisol, within-couple and parent-child coregulation of oxytocin levels has also been reported (Feldman, 2012); for example, couples appear to have correlated levels of oxytocin in the early postpartum period (Gordon, Zagoory-Sharon, Leckman, & Feldman, 2010). Much more research is needed before reliable associations can be established between within-couple prenatal testosterone synchrony and postpartum outcomes.

The direction of our effects suggests that hormonal change and synchrony predict relationship investment, not the other way around; that is, relationship investment at the first prenatal assessment was not significantly associated with testosterone change or coordination with mothers. The possibility that fathers' testosterone patterns may influence their relationship attitudes over time warrants further study, and raises interesting questions. For example, relationship satisfaction generally declines over the transition to parenthood (Doss, Rhoades,

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Stanley, & Markman, 2009), but these findings suggest that individual differences in paternal testosterone might play a meaningful role. Interestingly, another study of 37 fathers over the transition to parenthood found that men who had higher testosterone levels prenatally *and* who showed larger testosterone declines pre- to postpartum reported larger declines in "tenderness" toward their partner (Perini et al., 2012); other aspects of relationship quality (communication and conflict) were not significantly associated with changes in or baseline levels of men's testosterone. Although this finding seems at odds with the findings reported here, one difference is that we assessed the degree of investment in the relationship, whereas tenderness may reflect affection rather than commitment or stability. Also, Perini and colleagues found the largest decreases in relationship quality among men who started with higher T during the prenatal period, suggesting that lower prenatal T may be linked with better relationship quality.

Although we report a prenatal drop in T here, other studies have reported paternal declines in T in the postpartum period, particularly for fathers spending more time in daily childcare; this suggests that parental caregiving may influence T levels (Gettler et al., 2011). There may also be cultural differences in the timing and extent of fathers' changes in T, linked to normative expectations for father involvement within each culture (Gettler, 2014).

Limitations of this study include the small sample of couples, which limits our statistical power and our ability to do group comparisons, e.g. comparing fathers who showed a prenatal drop in T vs. fathers who did not show a drop. Participants were mostly Caucasian, college-educated, and married or engaged; although this sample reflects the Midwestern university town community from which the sample was drawn, and is demographically similar to previous studies of prenatal hormone changes (e.g., Berg & Wynne-Edwards, 2001), it may not generalize to more diverse populations. We also have only one measurement of T from each study visit (2

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samples collected over one laboratory session), which is a limitation because recent work has suggested that diurnal change in T might be a better correlate of fathering behavior than single-timepoint measures (Endendijk et al., 2016). In addition, although we recruited first-time parents, we did not ask parents whether they had had a previous pregnancy which may have ended in miscarriage or termination; thus we cannot verify that this was the first experience of pregnancy for both parents. Finally, we did not collect postpartum hormonal data, which would have allowed us to test within-couple synchrony into the postpartum period and would have given us a richer perspective on fathers' psychobiological shifts across the transition to parenthood.

Strengths of the study include the repeated-measures, longitudinal design with multiple measures of testosterone taken across pregnancy from both partners, as well as assessments of the couple relationship collected both prenatally and postpartum. Our use of multilevel modeling, a statistical approach that is well-suited for nested dyadic data, represents an advance over previous examinations of prenatal hormonal changes within couples. We were able to analyze fathers' trajectory of testosterone across pregnancy and within-couple synchrony within the same statistical model, allowing us to simultaneously capture two potential influences on fathers' testosterone. The positive association between mothers' testosterone predicting fathers' testosterone tells us that, for every occasion in which mothers have higher testosterone, fathers will also have higher-than-usual testosterone, controlling for their own baseline T values and their overall trajectory of change over pregnancy. This approach to modeling within-couple associations in prenatal hormones has not been previously used in the testosterone literature.

In conclusion, our findings suggest that fathers' patterns of prenatal testosterone may be meaningful to their adjustment to the transition to parenthood. Our findings also suggest that within-couple synchrony in testosterone may predict fathers' involvement in the couple

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relationship after the birth of a child. Children whose parents report better relationship quality appear to show better emotional and cognitive outcomes (Goldberg & Easterbrooks, 1984), so understanding family dynamics over the transition to parenthood is of critical importance.

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Table 1: Sample Descriptives and Zero-Order Correlations

| | Mean | SD | Range | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. First pregnancy week of study participation | 15.33 | 5.25 | 10-28 | 1 | | | | | | | | | | | | |
| 2. Total number of pregnancy weeks elapsed over study | 20.44 | 5.07 | 8-26 | -.77** | 1 | | | | | | | | | | | |
| 3. Father satisfaction (1st prenatal visit) | 8.19 | 0.63 | 6.60-9 | .03 | -.06 | 1 | | | | | | | | | | |
| 4. Father commitment (1st prenatal visit) | 8.67 | 0.53 | 7-9 | .03 | .09 | .37* | 1 | | | | | | | | | |
| 5. Father investment (1st prenatal visit) | 7.56 | 0.91 | 5.40-9 | .07 | -.20 | .71** | .43* | 1 | | | | | | | | |
| 6. Father satisfaction (postpartum assessment) | 7.69 | 1.50 | 2.60-9 | -.02 | -.03 | .57** | .03 | .34+ | 1 | | | | | | | |
| 7. Father commitment (postpartum assessment) | 8.41 | 1.26 | 3.43-9 | -.08 | -.01 | .54** | .40* | .49** | .76** | 1 | | | | | | |
| 8. Father investment (postpartum assessment) | 7.47 | 1.79 | 1.50-9 | -.09 | -.19 | .60** | .47* | .61*** | .71*** | .87** | 1 | | | | | |
| 9. Infant sex | | | | -.02 | -.08 | .33+ | .37+ | .32 | .25 | .41* | .51** | 1 | | | | |
| 10. Father T (Visit 1) | 50.23 | 11.25 | 27.56-68.58 | -.09 | -.09 | -.16 | .06 | .01 | -.28 | -.24 | -.10 | -.23 | 1 | | | |
| 11. Father T (Visit 2) | 49.79 | 16.54 | 23.50-86.56 | .48* | -.30 | -.32 | .04 | -.29 | -.27 | -.23 | -.13 | -.24 | .63** | 1 | | |
| 12. Father T (Visit 3) | 48.45 | 14.32 | 24.47-94.43 | .37+ | -.34+ | -.24 | -.16 | -.28 | -.36+ | -.53** | -.29 | -.19 | .43* | .63** | 1 | |
| 13. Father T (Visit 4) | 47.62 | 17.09 | 21.22-101.84 | .34+ | -.24 | -.55** | -.33 | -.36+ | -.37+ | -.51** | -.35+ | -.33 | .69** | .67** | .69** | 1 |
| 14. Mother T (Visit 1) | 9.89 | 4.80 | 3.78-21.67 | .11 | .10 | .00 | -.17 | -.08 | .18 | .15 | -.04 | .19 | .15 | -.04 | .02 | .23 |
| 15. Mother T (Visit 2) | 16.25 | 7.74 | 3.65-37.19 | .35+ | -.19 | .03 | -.41* | -.13 | .07 | -.04 | -.14 | -.22 | .18 | .21 | .27 | .26 |
| 16. Mother T (Visit 3) | 23.47 | 11.79 | 9.82-50.79 | .09 | .03 | -.00 | -.32 | -.17 | -.19 | .33+ | -.32 | .03 | .28 | .11 | .29 | .21 |
| 17. Mother T (Visit 4) | 54.15 | 24.30 | 20.91-100.84 | .15 | -.03 | -.19 | -.28 | -.30 | .18 | -.44* | -.33 | .09 | .26 | .27 | .39* | .49* |

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Table 2: *Two-Level Model Showing Mother Testosterone As A Predictor of Father Testosterone, With Fathers' Postpartum Investment Model Scales: Fixed Effects With Robust Standard Errors (n = 27 couples; 98 visits)*

| | Investment | | | Commitment | | | Satisfaction | | |
|---|---|---|---|---|---|---|---|---|---|
| Fixed effects | Estimate | (SE) | t ratio | Estimate | SE | T ratio | Estimate | SE | T ratio |
| Testosterone intercept (first visit) | 59.40 | (6.25) | 9.51*** | 57.72 | (6.16) | 9.30*** | 59.87 | (6.38) | 9.38*** |
| *Level 2 covariates*[1] | | | | | | | | | |
| Sampling time | -46.99 | (36.84) | -1.28 | -28.63 | (26.6) | -1.08 | -49.89 | (39.93) | -1.25 |
| Week of first visit | 5.39 | (1.43) | 3.76*** | 5.01 | (1.42) | 3.53** | 4.83 | (1.62) | 2.99** |
| Weeks elapsed | 3.71 | (1.11) | 3.35*** | 3.50 | (1.14) | 3.07** | 3.14 | (1.34) | 2.34* |
| Postpartum Scale | 1.74 | (1.77) | 0.99 | 4.37 | (2.49) | 1.75+ | -0.12 | (1.68) | -0.07 |
| Testosterone slope (pregnancy week) | -0.46 | (.23) | -1.96+ | -0.40 | (.23) | -1.75+ | -0.50 | (.24) | -2.10* |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | -0.25 | (.08) | -3.04** | -0.23 | (.08) | -2.85** | -0.21 | (.08) | -2.54* |
| Weeks elapsed | -0.25 | (.07) | -3.40** | -0.23 | (.07) | -3.29** | -0.20 | (.07) | -2.91** |
| Postpartum Scale | -0.21 | (.07) | -3.06** | -0.38 | (.12) | -3.19** | -0.12 | (.06) | -1.79+ |
| Mothers' testosterone | 0.10 | (.06) | 1.56 | 0.10 | (.07) | 1.53 | 0.12 | (.06) | 1.91+ |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | 0.13 | (.05) | 2.81** | 0.12 | (.04) | 2.76** | 0.12 | (.04) | 2.81** |
| Weeks elapsed | 0.14 | (.05) | 2.95** | 0.13 | (.05) | 2.97** | 0.13 | (.04) | 3.01** |
| Postpartum Scale | 0.05 | (.02) | 2.22* | 0.08 | (.03) | 2.91* | 0.04 | (.02) | 2.52* |

+ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

[1] Level 2 covariates refer to effects tested on each of the bolded Level 1 indices.

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Table 3: *Two-Level Model Showing Mother Testosterone As A Predictor of Father Testosterone, With Mothers' Postpartum Investment Model Scales: Fixed Effects With Robust Standard Errors (n = 27 couples; 98 visits)*

| | Investment | | | Commitment | | | Satisfaction | | |
|---|---|---|---|---|---|---|---|---|---|
| Fixed effects | Estimate | (SE) | t ratio | Estimate | SE | T ratio | Estimate | SE | T ratio |
| Testosterone intercept (first visit) | 59.13 | (6.14) | 9.64*** | 59.13 | (6.68) | 8.85*** | 58.73 | (6.21) | 9.46*** |
| *Level 2 covariates*[2] | | | | | | | | | |
| Sampling time | -45.67 | (44.04) | -1.04 | -29.55 | 34.23 | -0.86 | -35.01 | 26.13 | -1.34 |
| Week of first visit | 5.49 | (1.46) | 3.76*** | 4.91 | (1.58) | 3.10** | 4.89 | (1.38) | 3.53*** |
| Weeks elapsed | 4.13 | (1.17) | 3.53*** | 3.46 | (1.39) | 2.49* | 3.43 | (1.18) | 2.91** |
| Postpartum Scale | 5.19 | (3.15) | 1.65 | -0.17 | (5.77) | -0.03 | 1.95 | (1.19) | 1.65 |
| Testosterone slope (pregnancy week) | -0.45 | (.23) | -1.91+ | -0.47 | (.26) | -1.80+ | -0.43 | (.23) | -1.88+ |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | -0.24 | (.10) | -2.57* | -0.19 | (.10) | -1.93+ | -0.21 | (.07) | -2.80** |
| Weeks elapsed | -0.25 | (.09) | -2.84** | -0.19 | (.08) | -2.34* | -0.21 | (.07) | -3.18** |
| Postpartum Scale | -0.29 | (.13) | -2.14* | -0.18 | (.24) | -0.74 | -0.21 | (.08) | -2.65** |
| Mothers' testosterone | 0.10 | (.06) | 1.60 | 0.11 | (.07) | 1.56 | 0.09 | (.06) | 1.53 |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | 0.13 | (.05) | 2.54** | 0.10 | (.05) | 2.16* | 0.11 | (.04) | 2.79** |
| Weeks elapsed | 0.15 | (.06) | 2.58** | 0.11 | (.05) | 2.43* | 0.13 | (.04) | 3.08*** |
| Postpartum Scale | 0.11 | (.09) | 1.16 | 0.05 | (.05) | 0.85 | 0.05 | (.05) | 1.18 |

[2] Level 2 covariates refer to effects tested on each of the bolded Level 1 indices.

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Table 4: *Two-Level Model Showing Mother Testosterone As A Predictor of Father Testosterone, With Prenatal and Postpartum Investment Model Scales: Fixed Effects With Robust Standard Errors (n = 27 couples; 98 visits)*

| | Investment | | | Commitment | | | Satisfaction | | |
|---|---|---|---|---|---|---|---|---|---|
| Fixed effects | Estimate | SE | t ratio | Estimate | SE | T ratio | Estimate | SE | T ratio |
| Testosterone intercept (first visit) | 60.44 | 5.71 | 10.58*** | 58.29 | 6.63 | 8.79*** | 59.76 | 6.04 | 9.90*** |
| *Level 2 covariates*[3] | | | | | | | | | |
| Week of first visit | 6.89 | 1.58 | 4.35*** | 5.28 | 1.52 | 3.46*** | 5.54 | 1.71 | 3.25*** |
| Weeks elapsed | 4.17 | 1.25 | 3.64*** | 3.50 | 1.63 | 2.15*** | 3.63 | 1.34 | 2.71* |
| Prenatal Scale | -11.96 | 5.10 | 0.90 | -6.15 | 6.74 | -0.91 | -7.40 | 8.53 | -0.87 |
| Postpartum Scale | 7.11 | 2.73 | -2.99 | 6.47 | 2.33 | 1.81 | 3.43 | 0.53 | 0.60 |
| Testosterone slope (pregnancy week) | -0.48 | 0.23 | -2.03 | -0.48 | 0.26 | -1.86 | -0.51 | 0.23 | -2.26* |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | -0.34 | 0.10 | -3.47** | -0.20 | 0.08 | -2.38** | -0.25 | 0.10 | -2.65** |
| Weeks elapsed | -0.30 | 0.08 | -3.64** | -0.19 | 0.08 | -3.28** | -0.24 | 0.08 | -3.14** |
| Prenatal Scale | 0.21 | 0.23 | 0.90 | 0.57 | 0.29 | 2.01* | 0.02 | 0.31 | 0.07 |
| Postpartum Scale | -0.35 | 0.12 | -2.99** | -0.51 | 0.14 | -3.64*** | -0.14 | 0.13 | -1.03 |
| Mothers' testosterone | 0.09 | 0.07 | 1.25 | 0.14 | 0.07 | 2.06* | 0.14 | 0.07 | 1.93+ |
| *Level 2 covariates* | | | | | | | | | |
| Week of first visit | 0.16 | 0.06 | 2.86** | 0.10 | 0.05 | 1.87+ | 0.14 | 0.05 | 2.77** |
| Weeks elapsed | 0.17 | 0.06 | 2.90** | 0.11 | 0.05 | 2.09* | 0.15 | 0.05 | 2.98** |
| Prenatal Scale | 0.01 | 0.09 | 0.07 | -0.35 | 0.19 | -1.87 | -0.07 | 0.12 | -0.62 |
| Postpartum Scale | 0.06 | 0.06 | 2.30* | 0.11 | 0.03 | 4.04*** | 0.05 | 0.02 | 2.70** |

+ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

[3] Level 2 covariates refer to effects tested on each of the bolded Level 1 indices.

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Figure Captions

Figure 1
Panel Figure Depicting Average Change In Testosterone Over The Four Prenatal Visits

Figure 2
Scatterplots Depicting Within-Couple Associations in Testosterone at Each Prenatal Visit

Figure 3
Associations Between Investment Model Scales and Fathers' Testosterone Change Over Pregnancy and Linkage With Mothers' Testosterone
Estimated simple slopes at the 25$^{th}$ and 75$^{th}$ percentile for each of the three investment model scales (investment, commitment and satisfaction) are shown

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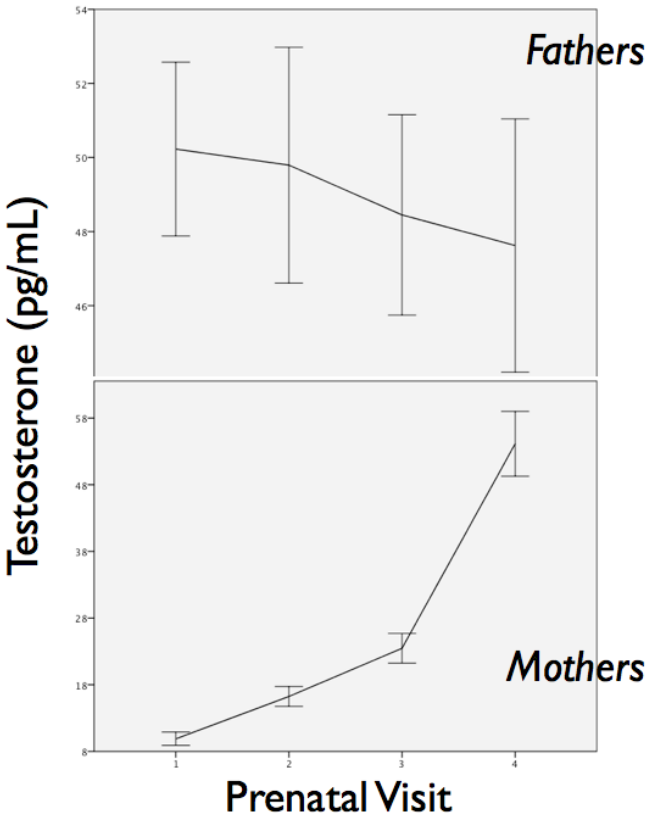


Fig. 1

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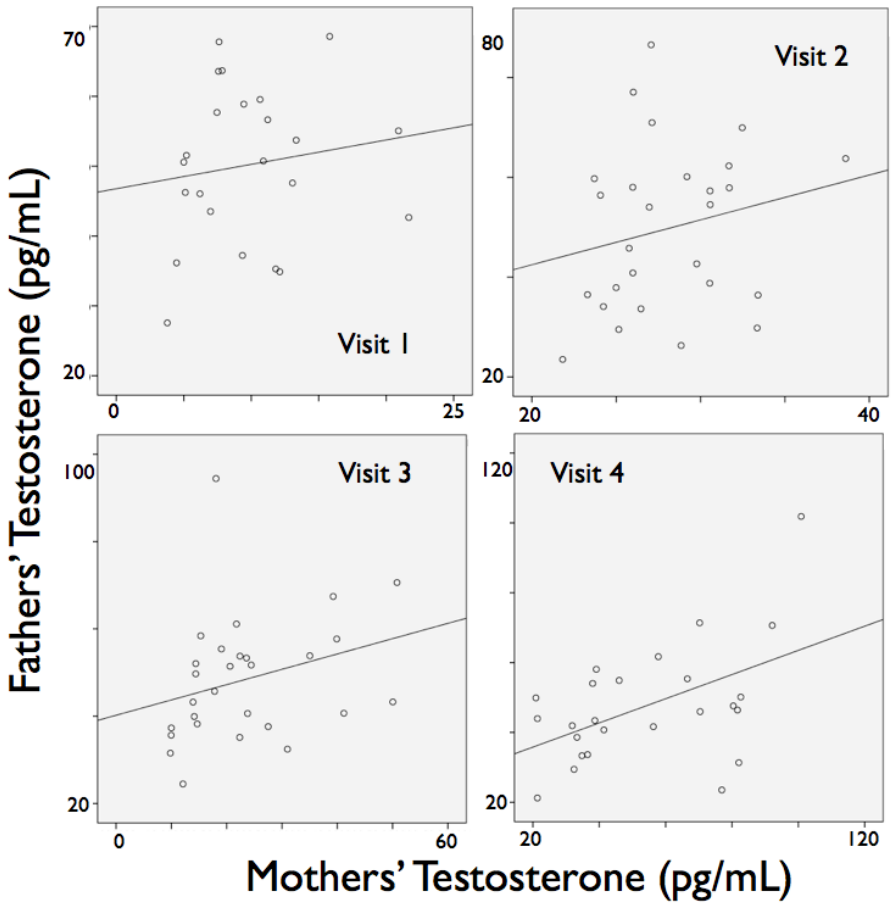


Fig. 2

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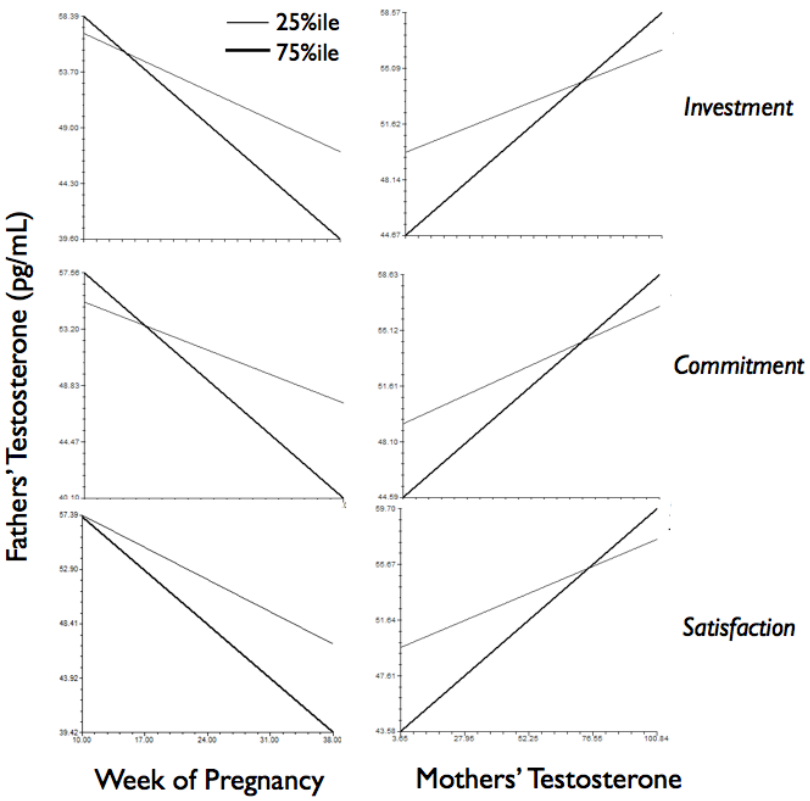


Fig. 3

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## Highlights

- In a sample of 27 couples, testosterone (T) was measured multiple times over pregnancy

- Father T dropped across pregnancy and was correlated with partners' T at each visit

- Father declines in T predicted their postpartum investment in the couple relationship

- Father synchrony with mothers' T also predicted postpartum relationship investment

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