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A comparison of testosterone and cortisol levels between gay fathers and non-fathers: A preliminary investigation

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ABSTRACT

Humans are unique among great apes and most other mammals, in that our wide range of offspring investment behaviors includes significant paternal care and provisioning of children. Moreover, hormones play an important role in modulating male paternal investment. Despite a growing body of research on the hormonal associations with paternal care in humans, fathers who self-identify as gay have not received the same level of research attention. We explore associations between hormones that are central to reproductive effort in American gay couples (n = 48 pairs, mean age 36 ± 11 SD years) with and without children. Building on previous investigations of paternal investment, we focus on testosterone and cortisol given their primary roles in the behavioral and metabolic aspects of male reproductive effort. We provide preliminary evidence that gay fathers have lower cortisol levels compared to gay non-fathers. Cortisol and testosterone also positively co-varied in all couples, independent of potential covariates. We did not find evidence for differences in testosterone levels between gay fathers and non-fathers, although sample sizes were limited. Based on this preliminary evidence, we suggest that psychosocial stress among gay fathers may differ compared to gay couples without children, or that the stress response in gay fathers is mitigated in some way compared to non-fathers. These data underscore the importance of human paternal care diversity and the value of inclusivity in human evolutionary behavior research.

1. Introduction

1.1. The behavioral endocrinology of paternal behavior

Hormone variation modulates paternal behavior in numerous organisms, including humans. For example, human fathers exhibit lower testosterone levels compared to age matched non-fathers in a variety of sociocultural contexts suggesting biological underpinnings that engage natural selection, social factors, and gene/environment interactions [43,46,49,50,52,65,70]. Moreover, differences in testosterone levels between fathers and non-fathers can be affected when fathers contribute to childcare [5,41,69,77], when fathers are married or otherwise pair-bonded to the mothers of their children [22,50] and when they cosleep with their children [44].

Recently, paternal associations with testosterone were shown longitudinally: North American men's testosterone declined over the course of their partners' pregnancy [11,35,108,128]. In Filipino men aged 21, those who had high baseline testosterone were more likely than those with lower baseline levels to be fathers at age 26. The group of fathers also had lower testosterone levels at age 26 than those men without children [42]. While attention for cross-cultural and longitudinal changes in hormones such as testosterone has grown, investigations that engage the neuroendocrinology of fatherhood and paternal investment across the spectrum of human male sexuality remains understudied.

1.2. Testosterone and gay fathers

To-date, investigations of hormonal associations between fatherhood and paternal engagement with their children have not considered variation in paternal sexual orientation or identity, as virtually all studies have focused on heterosexual men. van Anders and Watson [120] investigated partner status and testosterone in men and women and reported no effect of partnered status on testosterone for those partnered with men (non-heterosexual men or heterosexual women). However, they reported a significant decrease in testosterone in heterosexual men and non-heterosexual women. They concluded that the relationship between partner status and testosterone is only seen in individuals who partner with women. van Anders and Watson's results lead to the question of whether certain affiliative behaviors and their

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neuroendocrine correlates are gender-specific within a pair-bond. The only other study to examine hormone levels in gay male couples found no statistically significant differences in oxytocin levels between heterosexual mothers, fathers, and gay fathers [1]. Most interestingly, there were no differences in oxytocin levels between gay biological and non-biological fathers. However, neither testosterone nor cortisol were assessed.

1.3. Cortisol and fatherhood

The glucocorticoid cortisol is regulated by the hypothalamic-pituitary-adrenocortical axis (HPA), and is commonly recognized as a hormone that is associated with various forms of stress, or departures from physical and/or mental homeostasis. Cortisol is involved in increasing cellular glucose availability, immune function and inflammation, metabolism, neurobiology, electrolyte balance, and reproductive physiology [26,97].

Cortisol can alter reproductive function and behavior, often through the suppression of the hypothalamic-pituitary-gonadal (HPG) axis as demonstrated extensively in rodent models [45], but also non-rodent vertebrates [24,111] including humans [30,92]. Variation in cortisol and its interaction with testosterone can therefore inform our understanding of the behavioral endocrinology of fatherhood and paternal care. Most salient for this study, cortisol has been demonstrated to have suppressive effects on testosterone levels [30] and gonadotropin releasing hormone (GnRH) and luteinizing hormone (LH) production [24,45,111].

In contrast to testosterone, the association between cortisol and fatherhood is mixed. Research has shown that positive paternal responsiveness to offspring is associated with either increased cortisol levels or reactivity in humans ([11,40,105,106,108]; but see [35]). Additionally, experimental manipulations demonstrate that cortisol declines compared to baseline after father/offspring play are greater in experienced fathers compared to inexperienced fathers, suggesting that the hormonal response can be modulated by prior paternal experience [40,107].

However, evidence for a complementary relationship between cortisol and testosterone in the context of parenting remains unclear. In certain primate species, males exhibit a positive association between parental and mate seeking investment ([31,68,82]; reviewed in [78]). In humans, a handful of studies have failed to demonstrate an inverse association indicative of a trade-off between cortisol and testosterone [43,51,55].

1.4. Cortisol and gay fathers

The role of cortisol in the parenting experiences of gay fathers has not been previously explored. The majority of studies relating sexuality to cortisol levels has mostly focused on gay men with HIV/AIDS. However, in the last few decades more attention has centered on the role of psychosocial stress in people who belong to sexual minority groups and often experience social marginalization. Some studies have explored cortisol's relationship to disclosing one's sexual orientation and arrived at conflicting results that suggest disclosing *or* concealing one's sexual orientation might be stressful states [58,61]. Another study demonstrated that gay men displayed lower cortisol concentrations compared to heterosexual men when confronted with an experimental stressor [60]. Similar results were found in a study of lesbian, gay and bisexual young adults where structural stigma led to a blunted cortisol response following an experimental stressor [56], however other studies did not find this effect [7].

Gay fathers can receive more social support from multiple sources, including their families (who sometimes had not previously supported the men's sexual orientation), general parenting support groups and LGBTQ-specific parenting groups [12,130]. Having this extended support network could mitigate psychosocial stress levels. On the other hand, sexual-minority headed families are still unusual in the United States, and gay fathers might experience more elevated cortisol than heterosexual parents because of the additional pressures of often not being considered socially normative [6]. Moreover, the road for a gay man to become a father can be more stressful than that of a heterosexual father. With the exception of gay men who had children in previous opposite-sex couple marriages, most gay fathers must embark upon a lengthy and costly adoption, fostering or surrogacy process when creating their families. Often this process is made more difficult by agencies, social workers or birth parents who object to gay father-headed families [13,83,109]. Remarkably, given the presumed pyschobiological toll such challenges might take, the dynamics of stress in gay families remains quite understudied.

1.5. Objectives of the current study

The objectives of this study are to investigate potential differences between fathers and non-fathers who self-identify as gay in hormones that have been previously shown to be associated with fatherhood and paternal care in heterosexual men as well as other organisms. The hormones in question are testosterone and cortisol. We also examine potential co-variates with paternal status that may contribute to hormone differences including age, anthropometrics, mate seeking, and socioeconomic status.

Our null hypothesis was that there are no statistically significant differences in fathers' and non-fathers' testosterone levels. The alternative hypothesis is that, similar to heterosexual fathers, gay fathers would exhibit lower testosterone levels compared to non-fathers. Because of the complexities and interactions of the HPA and HPG systems, this study poses the question of whether any significant difference between cortisol levels exists between fathers and non-fathers without any *a priori* assumptions about what direction the differences in these groups should be. In very basic terms, if these men experience more stress, cortisol levels could be increased, or the cortisol response could be blunted. This study addresses whether or not there are differences in the stress experienced by gay fathers than non-fathers, as measured by cortisol. We also investigated whether cortisol and testosterone co-varied in fathers and non-fathers, and whether the relationship between these two hormones was affected by the demographic and psychosocial co-variates mentioned above.

2. Materials and methods

2.1. Participant recruitment

This study was approved by the Yale University Institutional Review Board (Yale Human Subjects Protocol number 1306012152). Written, informed consent was obtained from all human subjects. Healthy adult American men from across the United States, between the ages of 21 and 60 in same-sex couples were asked to participate. Recruitment efforts included word of mouth, recruitment booths at LGBTQ events in various cities, and social media. Potential participants were screened for substances or health issues that could interfere with hormone assessments. These included the use of exogenous testosterone, DHEAS/DHEA, or androstenedione, other forms of hormonal therapy, medications that might alter hormone levels, metabolic disorders with known hormonal effects (e.g. diabetes, Cushing's Syndrome, etc.), blood borne infection (HIV/AIDS and Hepatitis B), having a pacemaker (to avoid interference caused by the Tanita scale used for body fat measurement), or not being a U.S. citizen (to restrict the sample demographics and to more easily compensate participants). The screening was done in such a way that participants did not have to reveal any personal medical information (e.g. HIV status) as they simply checked “yes” or “no” to whether any of these excluding circumstances applied, without indicating which applied specifically. Participants were compensated $50 per couple. In total, the final sample comprised 48 individuals: 18

fathers and 30 non-fathers.

2.2. Body composition, demographic and parenting data collection

Age, weight, height, and body fat percentage were recorded for all participants since body composition can affect testosterone and cortisol levels (e.g. obesity: [131]). Participants filled out a survey with questions about their income, education, partner relationships, children, and childcare activities. Childcare questions were adapted from [132] and [50] and modified after an initial pilot study. Modifications included the addition of language to ensure the accuracy of family formation information. This included questions regarding the biological and non-biological relatedness of parents and children. These modifications were based on feedback from pilot participants [21]. Sexual feelings towards men were assessed using a self-assigned Kinsey rating [133].

2.3. Study population characteristics

The men in this study population were mostly well-educated (the majority held advanced degrees), received over 7 h of sleep per night and exercised 4 h per week. Fathers and non-fathers did not differ in any measures except for age and body fat; fathers were both older and had higher percentages of body fat than non-fathers (Table 1).

All participants were in a relationship, and both members of the couple were required to participate to be included in the study. Couple-level characteristics are reported in Table 2. Most fathers reported being married, while most non-fathers reported being unmarried (not married, not in a civil union). However, participants were recruited both before and after the U.S. Supreme Court ruling on *Obergefell v. Hodges*, which ruled state-level bans on same-sex marriage unconstitutional [81]. Couples who reported being unmarried may not have been able to marry legally.

2.4. Paternal caregiving

The average age of fathers in this study was 42, but the average age at fatherhood was 37 years. This is later than the Center for Disease Control's reported national average of 25 years for first time fathers between the ages of 15 and 44 (National Center for Health Statistics). Thus, most men had older children in the home, not infants, as reflected by the average age of the youngest child in the household at 6.75 years. The majority of fathers in this sample were not biologically related to their children. The majority of families had adopted children, or only one partner was biologically related to the couple's children. Families comprised of either biological children of one partner, or adopted or foster children who were not biologically related to either partner. No couples reported that their families were composed of biological children of both spouses, nor were families composed of both non-biological and biological children (Table 3).

Table 1
Characteristics of the sample population, stratified by fatherhood status. Fathers and non-fathers compared using Mann-Whitney *U* test.

Measure	All men	Fathers	Non-fathers	*p*-Value
	$n = 48$	$n = 18$	$n = 30$	
Age (SD)	36 (11)	42 (8)	30 (9)	$2.63e^{-10}$
Body fat percentage (SD)	20.8% (8.7%)	27.3% (10.1%)	16.9% (4.7%)	$4.36e^{-5}$
Education (mode)	Advanced degree	Advanced degree	Advanced degree	0.81
Individual income (SD)	$82,494 ($74,807)	$118,400 ($73,528)	$60,950 ($67,971)	0.20
Kinsey score (mode)	6	6	6	0.29
Average hours of sleep per night (SD)	7.4 h (0.93)	7.2 h (0.96)	7.5 h (0.91)	0.30
Average hours of exercise per week (SD)	4.2 h (3.7)	4.4 h (5.6)	4.1 h (2.1)	0.77

Table 2
Couple characteristics. Mean comparisons conducted using Mann-Whitney *U* test.

Measure	All men	Fathers	Non-fathers	*p*-value
	$n = 24$	$n = 9$	$n = 15$	
Length of relationship (SD)	9 years (8.1)	15.8 years (6.7)	5 years (5.9)	0.0002
Cohabitation (SD)	92% (28.2%)	100% (0)	83.3% (36.2%)	0.22
Length of cohabitation (SD)	7.4 years (8.3)	15.2 years (6.5)	2.4 years (4.3)	0.0002
Sexually monogamous	66.7% (48.2%)	66.7% (50%)	66.67% (49%)	0.98
Legal status (mode)	Unmarried (not married, not in a civil union)	Married	Unmarried (not married, not in a civil union)	0.03

2.5. Salivary sample collection for testosterone and cortisol assessment

Salivary samples were collected using passive drool methods [66]. Approximately two milliliters of saliva were collected twice daily to account for diurnal variation for a period of three consecutive days. Participants were instructed to collect the morning sample upon waking and before eating, drinking or brushing their teeth. The evening sample was collected immediately before going to bed, at least 1 h after eating, drinking or brushing their teeth. Kraemer et al. [63] demonstrated that calculating slope on two samples, one taken at waking (which they recommend over 30 minute post-waking) and one in the evening (they used a 9 PM sampling time) provided almost as good an estimate of slope as sampling five times over the course of the day. They concluded that two samples are sufficient to measure slope and that the number of days is a much more important influence on accuracy than the number of samples per day.

Participants wrote the time and date of sample collection on their vials and froze samples immediately upon collection in their home freezers. All salivary samples were transported in person via portable freezer or by mail packed in dry ice to the Reproductive Ecology Laboratory (YREL) within the Yale Department of Anthropology. All samples arrived frozen and upon arrival were kept at −20 °C until analysis. These steps ensured that samples were never defrosted prior to analysis.

2.6. Salivary testosterone measurement

Salivary testosterone was measured using eight separate Expanded Range High Sensitivity salivary testosterone enzyme immunoassay (EIA) kits (Salimetrics, Inc., Carlsbad, CA). According to the manufacturer, cross reactivity with other endogenous androgens is non-detectable or below 1% with the exception of dihydrotestosterone (DHT, 36%) which is found in marginal levels in saliva. Twenty-five microliters of undiluted sample were assayed in duplicate according to the kit protocol. The inter-assay CV for the high control was 9.7% and the CV for the low control was 11.5%. Mean intra-assay CV was 7.4% ± 1.87 SD. Two evening outliers were identified because they fell outside of three times the interquartile region and were removed from all subsequent analyses. Twenty-nine samples had to be discarded because of a lack of adequate sample volume for assay.

Table 3
Father characteristics.

Measure	Percent		
Fathers with biological children	17%		
Families in which one partner is biologically related to his children	33.3%		
Measure	Mean or mode	Standard deviation	Range
Average number of children per couple	2.28	1.0	1–4
Average age of youngest child	6.75 years	5.5	9 months to 19 years
Average age man became a father	37 years	7.9	22–46
Average number of years a man has been a father	7.8 years	4.9	2–19
Average percentage of waking hours spent in childcare activities during the working week	19%	20.5%	0–71%
Average percentage of waking hours spent in childcare activities during a day off	29%	30%	0–100%
Average percentage of waking hours spent in playtime activities during the working week	10%	7.2%	0–28%
Average percentage of waking hours spent in playtime activities during a day off	31%	36.8%	0–100%
Percentage of couples reporting a 50/50 split of childcare responsibilities	22.2%	–	–
Percentage of families whose children are also cared for by an additional caretaker (nanny, relative, etc.)	44.4%	–	–
Percentage of fathers who co-sleep with children	11%	–	–

2.7. Salivary cortisol measurement

Samples were assayed for cortisol at the Yale Center for Clinical Investigation in New Haven, CT using an MP Biomedicals coated tube ^{125}I cortisol radioactive immunoassay (RIA) kit (MP Biomedicals, LLC, Santa Ana, CA). The sensitivity of this RIA is 0.03 μg/mL. All samples were run in a single assay. Two hundred microliters of undiluted sample were assayed in duplicate according to the kit protocol. The intra-assay CV for the low control was 1.9% and the CV for the high control was 10.2%. Two evening sample outliers were removed from all analyses because they fell outside of three times the inter-quartile region.

2.8. Cortisol indices

For our first set of analyses, only samples that had complete sample days were included, meaning only days in which a participant completed both a morning and evening sample. We attributed missing complete days to one of the following: 1) a participant incorrectly collecting their first sample in the evening, 2) samples with insufficient volume for assay measurement, 3) participant noncompliance and 4) the two discarded outliers. Unusable samples were missing at random. This resulted in one to three complete sample days per participant used in the subsequent analyses. Altogether due to sample quality control, we omitted 32 out of 294 cortisol samples (10.8%) from the dataset.

Once we isolated complete sample days in the dataset, we calculated three different indices of cortisol: 1) time-of-day specific mean levels were calculated by centering cortisol levels for each day at 6 hour post-waking; 2) day-specific slopes, calculated using morning and evening levels; and 3) area under the curve with respect to ground (AUC_G) values for each complete day of sampling. We assessed these three indices separately as they represent different constructs [63].

Time-of-day specific cortisol allows the comparison of levels at the same time post-waking across individuals. This was done because participants were instructed to take samples at their natural waking and sleeping times, as opposed to standardized times throughout the day. As a result, individuals in this study varied in the number of hours between sampling, but all were awake for at least 6 h. Accordingly, we standardized the sample to 6 hour post-waking. Levels of cortisol for each complete sample day were determined by anchoring the waking samples at time = 0 (the y-intercept) and using the slope between morning and evening samples to determine the cortisol level at 6 hour post-waking. Because only two data points across the day were taken, slopes were calculated as a simple linear function of the time between the two samples and their untransformed cortisol values [59,63]. Box-Cox transformation was used to check for normality for all hormone data going forward. This data was transformed by taking the natural log of the cortisol measurements to normalize the distribution of the data. Slope also represents the rate of decline in cortisol over time from waking to sleeping, and thus captures the magnitude of diurnal change in any single day. Slope was anchored at the morning sample and calculated using a simple linear function of the time between the two samples and their untransformed cortisol values. Slopes were inspected for normality, determined to be normal, and were not transformed.

The AUC_G represents total exposure to cortisol over the course of the waking day, and is a function of both cortisol levels and the number of waking hours of an individual. In this way, AUC_G accounts for waking cortisol exposure because each participant collected their first sample at waking and the last sample right before bed, and the amount of time between these measures was allowed to vary for each participant and for each complete sample day. In this study, AUC_G was calculated using the trapezoid formula [86] which has been used extensively to calculate total daily cortisol secretion [89,91]. This index was then transformed by taking the natural log.

Slope, AUC_G and time-of-day specific measures were analyzed separately since two individuals may be identical in terms of slope or time-of-day specific levels, but very different in terms of total cortisol exposure, and vice versa. For example, research has found that slope and AUC_G are not particularly strongly associated with each other [2], and that some groups differ in their slopes, but not their total AUC_G [87]. For the analysis of cortisol and testosterone covariation, full days were not necessary since we measured the covariation of these two hormones in a single sample. Therefore, all samples that had both a valid cortisol and testosterone measurement were used. This resulted in 128 morning samples and 131 evening samples available for statistical analysis.

2.9. Multi-level modeling

Cortisol values were analyzed using multi-level modeling (MLM) techniques. Multi-level modeling (also known as mixed-level or hierarchical linear modeling) was chosen because of its ability to deal with nested data; in this study, sampling days are nested within participants, who are nested within couples, who are assigned to fixed groups of fathers and non-fathers [102]. Multi-level modeling is more appropriate for these data than repeated-measure ANOVA because it is better able to handle missing data, imbalanced designs, and numeric predictors measured at multiple levels [59]. This is especially necessary when dealing with hormone measures because circulating levels commonly fluctuate considerably within subjects. As a result of these advantages, multi-level modeling is used extensively for cortisol analyses [32,59,63,91,98,99]. All models in this study were fit using SAS PROC MIXED.

Cortisol indices were modeled as linear mixed effects models with random intercepts by couple and by participant within couple. Because individual observations (complete day measurements) are nested within participants, which are then nested within couples, we refer to

Table 4
Results of the unconditional model for time-of-day specific cortisol means. AIC and BIC provided to assess the quality of these models.

Covariance parameter estimates					
Covariance parameter	Subject	Estimate	Standard error	Z value	Pr > Z
Couple-level variance (v_{00k})	Couple	0.06	0.03	2.04	0.02
Participant-level variance (u_{0jk})	Participant	0.04	0.02	1.79	0.04
Residual variance (e_{ijk})		0.08	0.01	6.27	< 0.0001

Solution for fixed effects					
Effect	Estimate	Standard error	DF	*t*-Value	Pr > |t|
Intercept (γ_{000})	−0.60	0.07	23	−9.15	< 0.00

Fit statistics	
AIC	121.5
BIC	125.0

this as a 3-level nested model. The unconditional three-level model is writing as follows:

Level 3 model:

$$Y_{ijk} = \gamma_{000} + v_{00k} + u_{0jk} + e_{ijk} \quad (1)$$

In this equation, *Yijk* is the cortisol measurement for day *i* of participant *j*, in couple *k*, γ_{000} is the grand mean of cortisol of all couples, v_{00k} is the difference between couple *k*'s mean cortisol level and the grand mean for all couples (it therefore represents the variation between couples), u_{0jk} represents the variation between participants within couples and e_{ijk} represents the variation within participants.

To assess whether fatherhood affected cortisol, we added the fixed effect of fatherhood status at the third (couple) level. The mixed model equation, in which the time-of-day specific cortisol level is expressed as a function of couple-level fatherhood status with a fixed slope, γ_{01}, is written as:

$$Y_{ijk} = \gamma_{000} + \gamma_{01}\, FATHERHOOD_k + v_{00k} + u_{0jk} + e_{ijk} \quad (2)$$

One can compute how much of the variation is explained by fatherhood status by calculating how much the variance changed between the two models:

$$\frac{\sigma_2^2 - \sigma_1^2}{\sigma_2^2} \quad (3)$$

where σ_1^2 is the between couple variance in the model containing the variable for fatherhood and σ_2^2 is the individual variance without it.

To further explore whether cortisol differences were empirically associated with fatherhood status, we ran the model controlling for several potential covariates (age, body fat percentage, sleep, exercise, sexual monogamy, and income) first by themselves and then simultaneously in a single model (Supplemental materials). First, age was controlled by adding it to the model as level two fixed effect because cortisol levels are known to increase with age [80]. Since body fat percentage between fathers and non-fathers were significantly different, we controlled for this variable as well. Cortisol is commonly sensitive to the sleep-wake cycle [67], and fathers might experience sleep disruption as a result of caring for children, though fathers and non-fathers had similar self-reported hours of sleep. We therefore modeled self-reported average nightly hours of sleep in the same manner as age and body fat. Acute physical activity can elevate cortisol [25,34,54], so to control for activity levels we added self-reported average weekly hours of exercise as a fixed effect to the fatherhood status model. Socio- economic status (SES) is negatively correlated with cortisol, as higher cortisol measures are associated with lower lifetime SES across multiple studies [33]. To control for SES, we modeled both individual and couple-level annual income. Although the effect is unclear but often evident, we controlled for mating effort by adding sexual monogamy to the model.

3. Results

3.1. Cortisol time-of-day mean measures

The following analysis tests the hypothesis that cortisol levels will differ between fathers and non-fathers. The average natural log of cortisol at 6 hour post-waking of couples in the study is given by the estimate for the fixed effect intercept, γ_{000}, and is −0.60, which is equivalent to 15.22 nmol/L (Table 4). The variance estimates for couple and participant are 0.06 and 0.04, respectively. These effects are modest but significant at the $p < .05$ level.

The addition of the fixed effect of fatherhood status improved the model fit, as indicated by both Akaike Information Criterion (AIC) and Baysian Information Criterion (BIC) decreasing from the unconditional to the fixed-effect model (Table 5; Fig. 1). AIC and BIC are estimates of model fit based partially on the likelihood function, while penalizing the model complexity (number of parameters). The lower the AIC or BIC, the better fit the model. The intercept, −0.49, estimates the average couple's cortisol when the predictor of fatherhood status is zero (the subject was a non-father). This is equivalent to 16.83 nmol/L. The estimate for the fixed effect of fatherhood is −0.27. This indicates that fathers have a log cortisol mean that is 0.27 lower than non-fathers. This is significant at the $p < .05$ level which supports the hypothesis that cortisol means differ between fathers and non-fathers.

In the model with the fixed effect of fatherhood, the variances become conditional components. The variance component representing variation between couples diminished, decreasing from 0.06 to 0.05. This indicates the predictor fatherhood explained a substantial portion of the participant-to-participant variation in cortisol slopes.

Using equation 3, we calcualted the amount of variation explained by fatherhood status. In this case, 33% of the total explainable variation in cortisol at the couple level (which was calculated as 33.5% of the total variation) was accounted for by fatherhood status.

3.2. Controlling for covariates in the time-of-day specific cortisol means model

When controlling for potential covariates, age did not exert a strong effect on cortisol levels. When controlling for fatherhood, the effect of

Table 5
Results of the fixed effect model of fatherhood on time-of-day specific cortisol means.

Covariance parameter estimates					
Covariance parameters	Subject	Estimate	Standard error	Z Value	Pr > Z
Couple-level variance (v_{00k})	Couple	0.05	0.03	1.75	0.04
Participant-level variance (u_{0jk})	Participant	0.04	0.02	1.84	0.03
Residual variance (e_{ijk})		0.08	0.01	6.31	< 0.00

Solution for fixed effects					
Effect	Estimate	Standard error	DF	*t*-Value	Pr > |t|
Intercept (γ_{000})	−0.49	0.08	22	−6.45	< 0.00
Father status ($\gamma_{01}FATHERHOOD_k$)	−0.27	0.13	83	−2.15	0.03

Fit statistics	
AIC	119.4
BIC	122.9

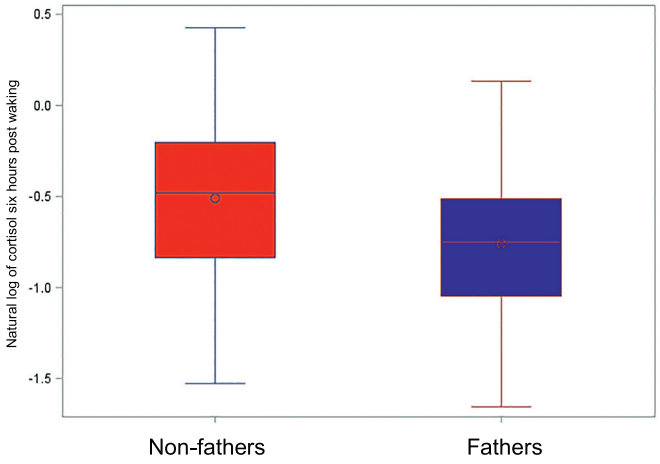

Fig. 1. Transformed cortisol stratified by fatherhood status. Natural log of time-of-day specific cortisol by fatherhood status.

age was 0.01 and not statistically significant. Nevertheless, father status remained significant and its effect actually increased, from −0.27 to −0.35, meaning that controlling for age, fathers had a log 6 hour post waking cortisol level that is 0.35 lower than non-fathers. Moreover, the model fit was worse with age than without it. The AIC and BIC increased from 119.4 and 122.9 to 126.9 and 130.5, respectively. Since body fat percentages between fathers and non-fathers were significantly different, we controlled for this variable. However, body fat itself was not significantly associated with cortisol and it did not improve model fit.

Neither individual nor combined income were significantly associated with variation in cortisol. After fatherhood, sexual monogamy had the strongest association with cortisol, at −0.17, but this was not statistically significant. However, sleep and exercise both accounted for a significant amount of between participant variance and improved the model fit. Consequently, we combined the significant fixed effects—father status, sleep and exercise—in one model (Table 6). Fatherhood status still exerted a significant effect on cortisol levels, with sleep and exercise controlled. However, when comparing the AIC and BIC fit statistics, the model that included father status and exercise fixed effects but excluded sleep was a better fit and therefore the best model of these data. We conclude that fatherhood status decreases cortisol at 6 hour post-waking independent of other covariates.

Table 6
The fixed-effect models for father status, sleep and exercise on time-of-day specific cortisol levels.

Covariance parameter estimates	
Couple-level variance (v_{00k})	0.08 (0.03)*
Participant-level variance (u_{0jk})	0.00 (0.01)
Residual variance (e_{ijk})	0.09 (0.01)*
Solution for fixed effects	
Intercept (γ_{000})	−0.07 (0.39)
Father status	−0.30 (0.13)*
Exercise	0.04 (0.01)*
Sleep	−0.08 (0.05)
Fit statistics	
AIC	114.7
BIC	118.3

Standard errors in parentheses.
* Significant at $p < .05$.

3.3. Cortisol slope measures

The average slope of couples in the study is given by the estimate for the fixed effect in the unconditional model, γ_{00}, and is −0.06. The effect of both couple and participant-level variances were small and not statistically significant, indicating that there was more day-to-day variation among cortisol slopes for single individuals than at the level of participants or couples.

Modeling the fixed effect of fatherhood modestly decrease the variation among couples. Fatherhood status accounted for 54.9% of the total explainable variation in cortisol slopes. The fixed effect model reveal that the average cortisol slope of non-fathers was −0.06, while fatherhood slopes decreased −0.05, indicating that fathers experienced less cortisol change over the course of the day than did non-fathers. The fixed effect of fatherhood was noteworthy but not significant ($p = .06$). The potential covariates of age, body fat, sleep, exercise, individual and combined income were added as fixed effects. Father status, age, body fat, and exercise had modestly large effects, but combined none of these factors was significant. This indicates insufficient evidence to support an independent effect by fatherhood on cortisol slope over the course of the day.

3.4. Total cortisol exposure (AUC_G) measures

The average AUC_G in this dataset is calculated by the intercept, and equals 16.98 nmol/L. Adding fatherhood as a fixed effect in the conditional model failed to explain more of the between-couple variance. In this model, the participant-level variance increased and the fixed

effect of fatherhood was not significant. The model fit also diminished, with both the AIC and BIC increasing. Fatherhood did not influence total cortisol exposure. Potential covariates were modeled separately which identified age, exercise and sleep as variables to control in the combined model. When these covariates were added, the couple and participant level variances became significant, with exercise and sleep, the model approached significance at $p = .05$ and 0.05, respectively. However, father status and age were not significant. Therefore evidence did not support that these two factors actually influence cortisol AUC_G at the couple level.

3.5. Testosterone indices

Four different indices of testosterone were evaluated. Consistent with the methodology of other studies, morning and evening samples were analyzed separately. Morning and evening results were transformed to their square roots for normality. Morning and evening levels were used calculate day-specific slopes and area under the curve with respect to ground (AUC_G) using the same methods described earlier. Slopes were determined to be normal so were not transformed. AUC_G was transformed to the square root value for normality. Testosterone values were analyzed using multi-level modeling (MLM) techniques as described earlier (e.g. [8,134,135,136]).

3.6. Modeling morning testosterone

Average morning participant testosterone levels were determined by the intercept and were equal to 8.41, or 70.66 pg/mL. In the unconditional model, the majority of the variance was found at the observation level. Interclass coefficients at the residual level was 83.4%. The couple-level variance accounted for the second largest portion of the variance, at 16.5%. The participant-level variance was zero and not significant.

To assess the effect of fatherhood on testosterone, we added the fixed effect of fatherhood status at the third (couple) level (Table 7). In this model, fatherhood was not significantly associated with variation in morning testosterone ($p = .68$). Fixed-effect models that included fatherhood and each of the following variables: body fat, age, exercise, sleep, monogamy, education, relationship length, legal status, cohabitation and cohabitation length (results not shown) were assessed. These variables served as proxies for pair-bonding which are known to be associated with testosterone levels. None of these covariates were significant in any of the models tested, though they did improve the model fit (Table 7). The null hypothesis that morning testosterone levels are the same between fathers and non-fathers could not be rejected.

3.7. Modeling evening testosterone

Evening testosterone was modeled in the same way as morning testosterone (Table 8). The average participant evening testosterone level is given by the intercept of the unconditional model and is 40.79 pg/mL. As with morning testosterone, the majority of the variance occurred at the observation level. The interclass coefficients at the residual level was 69%. Couple-level correlation was not significant. However, it accounted for considerably less of the variance. There was a sizable amount (23%) of variation in testosterone at this level.

To assess if there was an effect of fatherhood on testosterone, we added the fixed effect of fatherhood status at the third (couple) level. Fatherhood was not significantly associated with variation in evening testosterone ($p = .30$). Potential covariances that may mask the effect of fatherhood did not have an effect on any of the models tested, nor did they improve the model fit. Therefore, we cannot reject the null hypothesis that evening testosterone levels are the same between fathers and non-fathers.

Table 7
Results of the morning testosterone models.

Model	Unconditional model	Father status fixed effect model	Father status and covariates
Covariance parameter estimates			
Couple-level variance (v_{00k})	0.86 (0.54)	0.93 (0.57)	0.99 (0.80)
Participant-level variance (u_{0jk})	0 (−)	0 (−)	0 (−)
Residual variance (e_{ijk})	4.35 (0.61)*	4.35 (0.61)*	4.52 (0.66)*
Solution for fixed effects			
Intercept (γ_{000})	8.41 (0.26)*	8.32 (0.34)*	10.01 (2.90)*
Father status	–	0.23 (0.56)	−1.66 (1.64)
Age	–	–	0.02 (0.05)
Body fat	–	–	−0.01 (0.04)
Exercise	–	–	0.02 (0.08)
Sleep	–	–	−0.25 (0.30)
Monogamy	–	–	−0.89 (0.87)
Education	–	–	−0.22 (0.33)
Relationship length	–	–	−0.01 (0.02)
Legal status	–	–	0.49 (0.41)
Cohabitation	–	–	0.44 (0.9)
Cohabitation length	–	–	0.02 (0.02)
Interclass correlation coefficient			
ICC_{Couple}	0.17	–	–
$ICC_{Participant}$	0	–	–
$ICC_{Residual}$	0.83	–	–
Fit statistics			
AIC	576.9	576.1	570.8
BIC	579.3	578.4	573.1

* Significant at $p < .05$.

3.8. Testosterone slopes

The average slope of couples in the study is given by the estimate for the fixed effect in the unconditional model, γ_{00}, and is −2.51 pg/mL/h. The effect of both couple and participant-level variances was zero, indicating that there is more day-to-day variation within testosterone slopes of a single individual than there is at the participant or couple level. ICC_C and ICC_P equal zero, while approximately 100% of the variance occurs at the residual, or sample, level.

Fatherhood status was not significantly associated with variation in testosterone slopes, nor were any of the other covariates (Table 9). Thus, there was no support for a significant relationship between participants' fatherhood status and their testosterone slopes, although sample sizes were modest.

3.9. Total testosterone exposure (AUC_G)

The average AUC_G was 272.90 pg/mL. AUC_G measures were not significantly correlated within couple or participant. The ICC_c of this model is 0.01 and the ICC_P is 0.22, meaning that 1% of the variation in total cortisol is found between couples, while only 22%, is found at the participant level, but since these measures are not statistically significant, one cannot rule out chance as an explanation for the observed ICC results. The residual makes up 76.4% of the variance. Adding fatherhood as a fixed effect in the conditional model did not explain more of the between-couple variance. In this model, the participant-level variance decreased and the fixed effect of fatherhood was not significant. Fatherhood does not appear to influence the total testosterone exposure of this group.

Each potential covariate was modeled separately. The only variable that was significantly associated with the AUC_G was sleep, since waking hours are a function of how this measure was calculated. However,

Table 8
Results of the evening testosterone models.

Model	Unconditional model	Father status fixed effect model	Father status and covariates
Covariance parameter estimates			
Couple-level variance (v_{00k})	0.28 (0.45)	0.26 (0.44)	0.36 (0.60)
Participant-level variance (u_{0jk})	0.80 (0.53)	0.78 (0.52)	0.66 (0.12)
Residual variance (e_{ijk})	2.40 (0.36)*	2.41 (0.37)*	2.43 (0.38)*
Solution for fixed effects			
Intercept (γ_{000})	6.39 (0.22)*	6.21 (0.27)*	6.92 (2.71)*
Father status	–	0.46 (0.44)	−0.60 (1.04)
Age	–	–	0.03 (0.03)
Body fat	–	–	0.02 (0.03)
Exercise	–	–	0.05 (0.06)
Sleep	–	–	−0.24 (0.27)
Monogamy	–	–	−0.87 (0.59)
Education	–	–	−0.04 (0.34)
Relationship length	–	–	−0.01 (0.01)
Legal status	–	–	0.02 (0.22)
Cohabitation	–	–	0.56 (0.77)
Cohabitation length	–	–	0.01 (0.02)
Interclass correlation coefficient			
ICC_C	0.08	–	–
ICC_P	0.23	–	–
ICC_R	0.69	–	–
Fit statistics			
AIC	541.7	540.4	542.0
BIC	545.2	544.0	545.4

Standard errors in parentheses.
* Significant at $p < .05$.

Table 9
Results of the testosterone slope models.

Model	Unconditional model	Father status fixed effect model	Father status and covariates
Covariance parameter estimates			
Couple-level variance (v_{00k})	0 (−)	0 (−)	0.73 (1.17)
Participant-level variance (u_{0jk})	0 (−)	0 (−)	0 (−)
Residual variance (e_{ijk})	8.67 (1.16)*	8.75 (1.18)*	8.64 (1.36)*
Solution for fixed effects			
Intercept (γ_{000})	−2.51 (0.28)*	−2.50 (0.35)*	−5.98 (4.12)
Father status	–	−0.04 (0.58)	−0.54 (1.71)
Age	–	–	−0.02 (0.06)
Body fat	–	–	0.07 (0.05)
Exercise	–	–	0.02 (0.10)
Sleep	–	–	0.02 (0.43)
Monogamy	–	–	0.92 (1.06)
Education	–	–	0.95 (0.47)
Relationship length	–	–	0.00 (0.02)
Legal status	–	–	−0.31 (0.50)
Cohabitation	–	–	−1.14 (1.20)
Cohabitation length	–	–	0.00 (0.03)
Interclass correlation coefficient			
ICC_C	0	–	–
ICC_P	0	–	–
ICC_R	1	–	–
Fit statistics			
AIC	561.4	560.7	564.5
BIC	562.6	561.9	566.8

Standard errors in parentheses.
* Significant at $p < .05$.

when all other variables were controlled, no single covariate is significant (Table 10). Thus, no strong evidence exists that these factors, including fatherhood, is associated with testosterone AUC_G.

3.10. Morning cortisol/testosterone relationship

To determine whether testosterone and cortisol were co-regulated, we modeled cortisol as a predictor of testosterone using the same structure of multi-level model as in the previous sections. Morning and evening values of testosterone and cortisol were each transformed to their natural log in order to be on comparable scales. We then used cortisol levels as predictors of testosterone from the same saliva sample for both morning and evening, separately.

Cortisol strongly predicts testosterone levels in the morning (Table 11). The estimate for the morning cortisol slope (fixed effect) is 0.44, meaning that for every 1 nmol/L increase in cortisol, testosterone increases 0.01 nmol/L. This correlation was maintained when the fixed effects of fatherhood, sexual monogamy, age, body fat, relationship length, cohabitation, cohabitation length, and the legal status of the couple (married, unmarried, civil union) were added to the model. Income, combined income, and education were modeled as proxies for socioeconomic status. However no significant association with morning or evening testosterone was found with these covariates. The only covariate with even a modestly strong association with testosterone was fatherhood, with a fixed effect in the morning model of 0.21. Controlling for fatherhood moderately increased the fixed effect of cortisol on testosterone, but the fixed effect of fatherhood remained insignificant at $p = .15$ (Table 11). However, it did account for approximately 5% of the variance at the couple level. Notably, sexual monogamy, which could represent mating effort, was not significantly associated with testosterone (its fixed effects = 0.001, $p = .97$).

3.11. Evening cortisol/testosterone relationship

As in the morning, cortisol strongly predicted testosterone levels in the evening. The estimate for the evening cortisol slope was 0.35, meaning that for every 1 nmol/L increase in cortisol, testosterone increases 0.0049 nmol/L. In the evening model, fatherhood, sexual monogamy and cohabitation had moderately large fixed effects compared to the other variables modeled, but their standards errors were also large, and thus their effects were not significant (Table 12).

In summary, this population of American gay men, time-of-day specific cortisol was significantly different between fathers and non-fathers, while there appeared to be no group differences between both slope and total diurnal exposure to cortisol. Testosterone values for fathers did not differ significantly from those of non-fathers. Additionally, both morning and evening measures of cortisol and testosterone from the same sample positively co-vary. Taken together, these results suggest that fathers had lower cortisol levels, but not necessarily different cortisol reactivity or exposure, from non-fathers, that testosterone levels did not differ and that testosterone and cortisol are associated regardless of fatherhood status.

4. Discussion

4.1. Cortisol

Cortisol results from the present study are novel compared to previous investigations that did not uncover differences between heterosexual fathers and non-fathers. Fatherhood was consistently and independently associated with lower cortisol. While this may be due to a factor that was not accounted for in this investigation, the robustness of the associations between fatherhood status and cortisol are noteworthy. The effect of fatherhood was accentuated when body fat and age, which

Table 10
Results of the testosterone AUC_G models.

Model	Unconditional model	Father status fixed effect model	Father status and covariates
Covariance parameter estimates			
Couple-level variance (v_{00k})	0.29 (3.45)	0 (−)	2.01 (4.07)
Participant-level variance (u_{0jk})	5.97 (5.27)	5.92 (3.62)*	2.95 (4.78)
Residual variance (e_{ijk})	20.75 (3.64)*	20.91 (3.66)*	21.29 (3.83)*
Solution for fixed effects			
Intercept (γ_{000})	16.52 (0.58)*	16.00 (16.00)*	21.33 (7.35)
Father status	–	1.32 (1.17)	−1.38 (2.97)
Age	–	–	0.06 (0.11)
Body fat	–	–	−0.02 (0.09)
Exercise	–	–	−0.05 (0.19)
Sleep	–	–	−1.18 (0.78)
Monogamy	–	–	−1.78 (1.90)
Education	–	–	0.56 (0.85)
Relationship length	–	–	−0.02 (0.04)
Legal status	–	–	1.55 (0.88)
Cohabitation	–	–	−0.71 (2.15)
Cohabitation length	–	–	0.02 (0.05)
Interclass correlation coefficient			
ICC_C	0.01	–	–
ICC_P	0.22	–	–
ICC_R	0.77	–	–
Fit statistics			
AIC	686.5	681.1	662.5
BIC	690.1	683.5	665.9

Standard errors in parentheses.
* Significant at $p < .05$.

Table 11
Results of the morning cortisol/testosterone models.

Model	Morning testosterone with morning cortisol fixed effect	Morning testosterone with morning cortisol and father status fixed effects
Covariance parameter estimates		
Couple-level variance (v_{00k})	0.07 (0.04)*	0.07 (0.04)*
Participant-level variance (u_{0jk})	0	0
Residual variance (e_{ijk})	0.24 (0.03)*	0.24 (0.03)*
Solution for fixed effects		
Intercept (γ_{000})	4.33 (0.08)*	4.26 (0.09)*
Cortisol	0.44 (0.11)*	0.47 (0.11)*
Father status	–	0.21 (0.15)
Fit statistics		
AIC	211.3	211.2
BIC	213.7	213.5

Standard errors in parentheses.
* Significant at $p < .05$.

are known to co-vary positively with cortisol, were controlled [90,121]. The fathers in this sample were significantly older and had higher body fat percentages than non-fathers, yet still exhibited lower cortisol levels.

The association between psychosocial stress and cortisol is commonly observed as being positive, although there are clearly mitigating factors that often affect this association. A starting interpretation of our results is that gay fathers experience less stress compared to gay male couples without children. This may reflect extended social support, particularly for fathers from multiple parenting or familial communities, which could mitigate stress levels ([12,130]). Lower stress could also be attributed to a stronger sense of "family" among fathers because having children indicates a more secure attachment. It also may reflect a greater dedication and motivation to have a family despite the social challenges that are often faced by this community. In support of this notion, the variable of children is independent of other relationship "strength" indicators, such as the length of the relationship or sexual monogamy. A secure family attachment might be particularly salient to decreasing cortisol levels for members of the gay community, where legal and social opposition to gay marriage and family-making are still prevalent.

However, the relationship between stress and cortisol is complex. Chronic stress can lower morning cortisol, increase afternoon/evening cortisol, lead to a flatter diurnal rhythm, and increase daily volume of cortisol output [75]. In this context, being a parent could be considered a chronic stressor. Parents of children with autism spectrum disorder and cancer, and parents of adult children with serious mental illness can experience hypocortisolism [10,38,76]. Though these are extreme examples, it may be that belonging to a sexual minority group similarly results in hypocortisolism with gay fathers exacerbating this stress response above and beyond that of straight fathers. Our findings are partially consistent with compromised HPA function: in fathers time-of-day specific cortisol was significantly lower and there was a trend towards lower cortisol slopes, while AUCg did not seem to be affected by fatherhood.

Lower cortisol in our population of fathers might also indicate investment in mating behavior. We hypothesized that participants in non-monogamous relationships would have elevated cortisol because of its association with mate-seeking behavior, and tested this hypothesis by comparing monogamous couples with those who reported extra-pair relationships. Note that the study participants could not be separated into discrete "mate seeking" and "parenting-oriented" groups, as three out of the nine couples with children also report being non-monogamous. This means that, in contrast to other studies where "mate seeking" usually refers to non-pair-bonded, non-fathers [43], our subjects fell into one of four categories: 1) pair-bonded non-fathers in a monogamous relationship; 2) pair-bonded non-fathers in non-monogamous relationships; 3) pair-bonded fathers in monogamous relationships; and 4) pair-bonded fathers in non-monogamous relationships. Therefore, a father can be described as both mating- and parenting-oriented and we could test the effects of both mating and parenting effort. Nonetheless, sexual monogamy and fatherhood were not associated with differences in cortisol (fatherhood: $p = .08$; monogamy: $p = .24$).

4.2. Testosterone

The null hypothesis that testosterone values for fathers did not differ significantly from those of non-fathers could not be rejected. These results contrast with those from heterosexual fathers [43,46,52,65]. Given the contrast with heterosexual fathers, the negative results from gay fathers merits attention. Gay fathers' testosterone levels might have resulted from protective paternal behavior, which could increase testosterone levels [118]. Muller [78] points out that infant protection is the primary form of paternal investment in nonhuman primates, and testosterone does not inhibit this behavior. Muller notes that male testosterone levels increase around the time of parturition in species such as lemurs [82], sifakas [20], and colobus monkeys [137] in which infants are at a high risk for infanticide.

In gay fathers, protective behavior may emerge from the stigmatization of gay headed families, perhaps leading to a protective effect that represents unique social challenges compared to heterosexual fathers [88,125]. Ethnographic studies highlight a theme of maintaining a "certain level of vigilance to protect the family" ([125], p. 167). In support of this idea, in the Netherlands gay fathers who experienced more rejection of themselves as fathers felt more concerned about their children [14].

Table 12
Results of the evening cortisol/testosterone models.

Model:	Evening testosterone with evening cortisol fixed effect	Evening testosterone with evening cortisol and father status fixed effect	Evening testosterone with evening cortisol, father status, sexual monogamy and cohabitation
Covariance parameter estimates			
Couple-level variance (v_{00k})	0.06 (0.05)	0.05 (0.05)	0.06 (0.05)
Participant-level variance (u_{0jk})	0.06 (0.05)	0.05 (0.11)	0.06 (0.05)
Residual variance (e_{ijk})	0.27 (0.04)*	0.27 (0.04)*	0.27 (0.04)*
Solution for fixed effects			
Intercept (γ_{000})	4.07 (0.16)*	3.989 (0.17)*	3.95 (0.29)*
Cortisol	0.35 (0.10)*	0.35 (0.21)*	0.34 (0.10)*
Father status	–	0.21 (0.15)*	0.18 (0.16)
Cohabitation status	–	–	0.13 (0.25)
Sexual monogamy	–	–	−0.13 (0.17)
Fit statistics			
AIC	246.5	246.6	248.5
BIC	250.0	250.1	252.2

Standard errors in parentheses.
* Significant at $p < .05$.

This defensiveness might extend to not only protecting children, but protecting one's status and identity as a father. Just by having children, gay fathers are challenging the notion of what it means to be a father, and are often put in the position of defending their choice to build a family [14,125]. This has been labeled a “minority stress” in the psychological literature ([14], p. 366). In the Netherlands, lesbian mothers who faced stigmatization and homophobia more strongly defended their status as mothers [15]. Stigma does not always have to be expressed by someone else towards an individual, as internalized stigma sensitivity (such as thinking others judge one on their sexual orientation) and stress were correlated for North American gay fathers of adopted children [113]. Dutch gay fathers' also felt less competent at child-rearing than straight fathers [14]. Future studies of gay fathers should incorporate questions regarding experiences of stigma, protectiveness of children and defensiveness to determine if there is a correlation with testosterone.

Testosterone results could also be because of extra-pair sexual interest. For men in a relationship, willingness to engage in extra-pair sex significantly predicted testosterone levels [72]. Although we explicitly asked if men were in a monogamous relationship, extra-pair interest was not explored. Also, couples often filled out their surveys together, so if one member of the couple was engaging in extra-pair sex without their partner's knowledge, this might not have been reported. Future studies might incorporate questions about extra-pair sexual interest in a confidential way to capture this variable.

4.3. Cortisol/testosterone covariation

Covariation between morning and evening cortisol and testosterone levels was consistent with co-elevation of cortisol and testosterone during mating in other nonhuman primates and mammals (reviewed in [127]). To test whether testosterone and cortisol were co-elevated in our sample, we examined whether “mating-oriented” men were more likely to experience co-elevated testosterone and cortisol than “parenting-oriented” men. Both morning and evening samples of cortisol and testosterone positively co-varied. This relationship remained virtually unchanged when fatherhood status, sexual monogamy, legal status of relationship, cohabitation, age and body fat were controlled. We used individual and combined income and education as proxies for social status, but these variables also were not significantly associated with the relationship between cortisol and testosterone.

The results of our model for testosterone and cortisol are similar to those reported in Filipino men who exhibited significant co-elevation of testosterone and cortisol [43]. However, in the Filipino study, “mating oriented” men (who were not pair-bonded and not fathers) had co-elevated cortisol and testosterone in the evening, while in our sample, mating effort predicted evening testosterone, but so did fatherhood status. Further testing with a larger sample size will be necessary to clarify the effects of fatherhood.

4.4. Study limitations

A reasonable interpretation of these results is that childcare mitigates the stress experienced by gay couples. However, perceived social stress was not measured. Therefore, we could not assess the interactions between parenting, perceived stress, cortisol and testosterone. Additionally, the direction of the relationship between general stress and parenting in gay fathers is not known. Furthermore, only two samples per day were collected, which might not effectively capture cortisol diurnal cycle. Ideally in a more clinical setting, acute cortisol reactivity might have elucidated whether or not gay fathers experience hypocortisolism as a result of chronic stress.

The sample size of this preliminary investigation presented a challenge, particularly for interpretation of testosterone results. Subject recruitment proved to be challenging, similar to attempts to engage with the gay community on early HIV studies as well as with other historically marginalized communities [29,62]. Although we made robust attempts to engage with LGBTQ community groups and consult with academics who are involved with humanistic areas of LGBTQ research, such as faculty from the Yale University Department of Women's, Gender, and Sexuality Studies, it is clear that a deeper personal and prolonged engagement is necessary to create and maintain community trust as well as research effectiveness [4].

Also, research of LGBTQ individuals is on the rise in many social science disciplines. This has led to evidence of research fatigue within the LGBTQ community [28]. Individuals have been asked to participate in numerous studies in the last 40 years, in particular as related to HIV/AIDs public health research, but also more recently in studies about youth and adult wellbeing. As a result, even well-meaning research might be met with reluctance [27]. For example from our own experience recruiting participants for this investigation, the largest LGBTQ organization in New Haven, Connecticut where Yale University is located, made the decision to bar researchers from posting advertisements for studies at their offices or on their website. This reflected the challenges we faced. Although this also illustrated the importance of building community relationships when conducting research with marginalized communities.

Heterogeneity within our subject sample presented challenges and limitations. Fathers were in longer relationships with their partners, including cohabitation. Our results suggest that this was not a factor in

our results but this may be the result of small sample sizes. Similarly, the children of our subjects were older which likely affected factors such as father sleeping patterns and parenting strategies. The effects of these factors, specifically the age of the child, on testosterone and cortisol levels is not well understood, even in heterosexual fathers.

The relative scarcity of same sex parents was also a challenge. According to the most recent U.S. census data, there are < 10,000 same-sex male couples with children in the United States, compared to the 23 million husband-wife households with children [115]. New census reporting points to an improvement to include sexual orientation data but there are still significant gaps in our knowledge of the demographics of gay fathers. It is therefore difficult to determine if this study captures a representative sample of gay fathers, and whether or not these findings can be generalized to American gay fathers as a whole. However, our participants do align with nation-wide demographic trends. For example, a recent survey shows 40% of gay men hold advanced degrees compared to 26% of all straight Americans [84]. In our study, we also had a high proportion of participants with advanced degrees (60%).

The relationship between stress and reproduction in male same sex couples is interesting since reproduction is not tied to the metabolic costs that are present in opposite-sex couples, such as gestation and lactation. Our findings suggest that the behavioral endocrinology of paternal care can and does vary in association with sexual orientation. It also shows that paternal care is associated with differences in key hormone levels in the absence of a female sexual partner.

Paternal care is rare in the class *Mammalia*; only a few male primates, carnivores and some rodents are known to naturally parent their offspring [129]. B*ipaternal* care, where two males raise offspring, is even rarer in the animal kingdom. The only known instances of bi-paternal care in non-human mammals are found in the carnivores: paired male lions may sometimes look after lost cubs, and one percent of foster parenting in a particular group of cheetas was by a pair of males [9]. The majority of bipaternal cases come from bird species, where pair-bonded males (who may or may not engage in sex with each other) raise offspring together [9]. Human males not only exhibit the rare trait of paternal investment, but they also display the broadest range of variability in their engagement with paternal care, from complete dedication to offspring care, partial care, to infanticide [19]. The incorporation of sexual orientation and male same sex pairing in association with paternal investment adds a new and vibrant illustration of the adaptability that is characteristic of our species, as well as an underserved area of research. Reproductive hormones, particularly androgens, have been extensively studied as they relate to same sex behavior in both non-human mammals and birds (primate examples reviewed in [122]), but this is the first time that they have been studied in humans in the context of gay paternal care. Clearly additional research is warranted to fully elucidate the biosocial dynamics of human parenting.

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Conflicts of interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.physbeh.2018.03.011.

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