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Hormonal predictors of women's sexual motivation

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ABSTRACT

Women's mating psychology may have evolved to track reproductive conditions, including conception risk, across and between ovulatory cycles. Alternatively, within-woman correlations between mating psychology and ovarian hormones may be byproducts of between-women relationships. Here, we examined associations between steroid hormones and two facets of sexual psychology with putatively different adaptive functions, sociosexual orientation and general sexual desire, in a sample of naturally cycling women (NC; n = 348, 87 of whom completed 2 sessions) and hormonally contracepting women (HC; n = 266, 65 of whom completed 2 sessions). Across two sessions, increases in estradiol predicted elevated sociosexual desire in NC women, and this relationship was stronger in women whose progesterone simultaneously decreased across sessions. Changes in hormones were not associated with changes in general sexual desire. Between-subjects differences in testosterone robustly, positively predicted sociosexuality and general sexual desire among NC women. Hormones were not consistently related to changes or differences in sexual psychology among HC women. The present results are consistent with testosterone contributing to individual differences, or modulating relatively long-term changes, in women's mating psychology. Further, our within-woman findings are consistent with the hypothesis that shifts in women's mating psychology may function to secure genetic benefits, and that these shifts are not byproducts of between-women associations.

1. Introduction

A growing body of research suggests that the steroid hormones estradiol, progesterone, and testosterone modulate aspects of women's sexuality, including general sexual desire (Jones et al., 2018; Roney & Simmons, 2013), in-pair and extra-pair sexual desire (Arslan, Schilling, Gerlach, & Penke, 2018; Grebe, Emery Thompson, & Gangestad, 2016; Roney & Simmons, 2016) and sociosexuality (interest in uncommited sex; Edelstein, Chopik, & Kean, 2011; van Anders, Hamilton, & Watson, 2007). However, findings are mixed (reviewed in Cappelletti & Wallen, 2016; Motta-Mena & Puts, 2017), associations may differ with the specific aspect of sexuality considered (Grebe et al., 2016; Jones et al., 2018; van Anders, Brotto, Farrell, & Yule, 2009), and whether the same hormonal mechanisms modulate within-woman changes and between-women variation remains unclear (Roney & Simmons, 2013).

Relationships between hormones and sexuality are likely to be clarified by understanding their functions. In many vertebrates, testosterone mediates the allocation of time and energy away from parenting and somatic effort (Folstad & Karter, 1992), and toward mating effort by responding to extrinsic indicators of the potential to obtain mates, such as season, diet, availability of mates, and presence of competitors (Muller & Wrangham, 2004; Smith, Brenowitz, Beecher, & Wingfield, 1997; Wingfield, Hegner, Dufty, & Gregory, 1990). In both men (Gettler, McDade, Agustin, Feranil, & Kuzawa, 2015; Gettler, McDade, Feranil, & Kuzawa, 2011; Kuzawa, Gettler, Muller, Mcdade, & Feranil, 2010; van Anders et al., 2007) and women (Barrett et al., 2013; Cashdan, 2008; Kuzawa, Gettler, Huang, & McDade, 2010; van Anders et al., 2007; van Anders & Watson, 2007), higher testosterone has been associated with greater mating effort and lower parenting effort. Though the majority of these studies did not directly assess sexual desire (but see van Anders et al., 2007), it is possible that observed tradeoffs between mating, parenting, and somatic effort are proximally mediated by testosterone-driven changes in mating motivation. However, despite long-held assumptions of a link between testosterone and self-reported sexual desire in women, the causal nature of such a link appears equivocal (reviewed in Cappelletti & Wallen, 2016; Motta-

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Mena & Puts, 2017).

Sexual behavior and desire may also respond to the ovarian hormones estradiol and progesterone (Dennerstein, Burrows, Wood, & Hyman, 1980; Grebe et al., 2016; Jones et al., 2018; Roney & Simmons, 2013; Sherwin, Gelfand, & Brender, 1985). Several adaptive explanations for such hormone-behavior relationships have been proffered, and each makes specific predictions about the nature of these relationships.

The "dual-sexuality" hypothesis postulates that women's mating psychology was shaped by selection to fluctuate across the ovulatory cycle, directing mating effort toward uncommitted sex with males possessing putative indicators of genetic quality during the fertile phase, and promoting long-term relationships with investing males outside of this phase (Thornhill & Gangestad, 2008). The dual-sexuality hypothesis thus predicts increased interest specifically in uncommitted sex during the fertile phase when estradiol is high and progesterone is low (Gangestad, Thornhill, & Garver-Apgar, 2005; Larson, Pillsworth, & Haselton, 2012; Shimoda, Campbell, & Barton, 2017). Because estradiol also increases during the non-fertile luteal phase when progesterone is elevated, this means that estradiol and progesterone would be expected to negatively interact, such that the positive effect of estradiol on interest in uncommitted sex should be greatest when progesterone is lowest.

Alternatively, Roney and Simmons (Roney & Simmons, 2008, 2013) proposed that calibration of sexual motivation and ovarian hormones may function less to facilitate dual-sexuality specifically and more as a means of promoting sexual behavior generally when the fitness benefits (potential conception) exceed the costs (e.g., expenditure of mating effort, risk of sexually transmitted infections). This "sexual motivation" hypothesis predicts that fluctuating hormone concentrations across the ovulatory cycle will produce changes in sexual interest and motivation broadly, as opposed to interest in uncommitted or extra-pair sex specifically (Arslan et al., 2018; Roney & Simmons, 2016).

Roney (2009) further proposed the related but conceptually distinct "between-cycle" hypothesis, suggesting that selection primarily favored increases in sexual motivation during ancestrally rare fertile months when ovulation was not suppressed by pregnancy or lactation. Such an adaptation might secondarily generate within-cycle shifts, but unlike the dual-sexuality hypothesis, the between-cycle hypothesis predicts increased sexual motivation when estradiol is elevated regardless of cycle phase and progesterone concentrations (Roney, 2009; Roney & Simmons, 2013). In other words, whereas the dual-sexuality and sexual motivation hypotheses predict an interaction between estradiol and progesterone, the between-cycle hypothesis predicts only a main effect of estradiol on sexual motivation broadly.

Finally, according to the "spandrel hypothesis," within-woman shifts in psychology are byproducts of adaptations for expressing these phenotypes in relation to between-women differences in reproductive condition (Havlíček, Cobey, Barrett, Klapilová, & Roberts, 2015). Because hormone concentrations track reproductive condition, this hypothesis predicts that the hormones that produce between-women differences in sexual psychology will mediate within-women changes in a similar pattern.

Although the hypotheses outlined above are not mutually exclusive, they make unique sets of predictions (Table 1, ESM Table 1 Rationale). However, few studies have tested these predictions regarding hormone-behavior relationships, even fewer have tested multiple hypotheses in comparison, and none have tested them all. In addition, much work investigating psychological traits across the ovulatory cycle utilizes suboptimal study design, including underpowered between-subjects designs and self-report rather than hormonal validation of ovulatory cycle phase (Gangestad et al., 2016; Gonzales & Ferrer, 2016).

Several studies stand out as being particularly well-designed to test relationships between women's changing hormone levels and sexual psychology. For example, Roney and Simmons (2013) explored relationships between salivary hormones and sexual desire and behavior in 36 naturally cycling (NC) women sampled daily across two cycles and an additional 7 women sampled daily across one cycle. These authors found that estradiol positively, and progesterone negatively, predicted day-to-day fluctuations in daily sexual desire. Grebe et al. (2016) sampled hormones and sexual interests at two time points from 33 romantically involved NC women, finding that estradiol negatively, and progesterone positively, predicted in-pair sexual desire. Moreover, estradiol predicted greater extra-pair sexual interests relative to in-pair sexual interests, whereas progesterone predicted the reverse. Shimoda, Campbell and Barton (2018) sampled 35 romantically involved NC women, using luteinizing hormone test kits to detect ovulation, and found a peri-ovulatory peak in extra-pair sexual desire and a similar trend for in-pair sexual desire. Finally, Jones et al. (2018) examined 337 NC women across 5–15 test sessions, finding that within-woman changes in progesterone negatively predicted general sexual desire, whereas estradiol tended to positively predict solitary sexual desire, and neither hormone predicted changes in sociosexual desire.

Table 1
Predictions by hypothesis regarding associations with ovarian hormones. Check marks and an "x" indicate predictions regarding the presence or absence of associations, respectively, and no symbol indicates no clear prediction.

Hypothesis	Within-subjects			Between-subjects
	Sociosexuality	Sexual desire	E × P	Same as within-subjects
Dual-sexuality	✓		✓	
Sexual motivation		✓	✓	
Between-cycle		✓	✗	
Spandrel				✓

However, these studies excluded women using hormonal contraceptives (HC), even though such women do not experience ovulation and its associated hormonal fluctuations, and can thus serve as a quasi-control group (e.g., Puts, 2006). It is important to note that exogenous hormones obtained through HC may have different effects from those of endogenous ovarian hormones. Some hormone-behavior relationships observed among NC women are not found, or even reversed, among HC women (for reviews, see Fleischman, Navarrete, & Fessler, 2010; Welling, 2013). One explanation for this observation is that less hormonal variation exists within cycles for HC as compared to NC women (De Leo, Musacchio, Cappelli, Piomboni, & Morgante, 2016), and this restricted range and variation makes it difficult to detect hormone-behavior relationships. A second explanation is that exogenous and endogenous hormones have different downstream physiological effects. Progesterone and medroxyprogesterone acetate (MPA), a synthetic progestin found in many hormonal contraceptives, have differential effects on cardiovascular (Hermsmeyer, Thompson, Pohost, & Kaski, 2008), glucocorticoid (Koubovec, Ronacher, Stubsrud, Louw, & Hapgood, 2005), androgenic (Sitruk-Ware, 2004), neuroprotective (Nilsen & Brinton, 2003), and neuroendocrine (Pazol, Northcutt, Patisaul, Wallen, & Wilson, 2009) function, suggesting that they might also differentially affect psychological processes. For example, studies of women's physiological responses to visual sexual stimuli (VSS) have carry-over effects, wherein a woman's hormonal milieu and associated responses influence responses during subsequent sessions. Though some work suggests that NC and HC women exhibit similar hormone-driven carry-over effects in measures of implicit sexual interest (Wallen & Rupp, 2010), they may differ in their carry-over effects in subjective measures of sexual interest (Renfro, Rupp, & Wallen, 2015), rendering it unclear whether identical hormonal effects should be expected for NC and HC women. Nevertheless, consideration of HC women as a quasi-control group may be useful for gauging the false discovery rate, and because juxtaposing hormone-behavior relationships of HC users and NC women can clarify underlying hormonal mechanisms (Arslan et al., 2018).

In addition to excluding HC women, some of the studies mentioned

above may have created demand characteristics related to ovulatory cycle phase, for example by measuring hormone levels daily across an entire cycle, or by scheduling participants according to ovulatory cycle phase (Arslan et al., 2018). Citing these and other methodological issues as motivation for their research, Arslan et al. (2018) collected over 26,000 usable online self-reports in a diary format across 30–35 days from 1043 women who reported being in a heterosexual relationship, of whom 421 were naturally cycling. These authors found increases in both extra-pair and in-pair sexual desire in NC (n = 421), but not HC (n = 622), women across the cycle, but did not assess hormone concentrations. Thus, not only has no study tested all predictions detailed in Table 1, but also no previous study has explored both within- and between-subjects relationships between steroid hormones and women's sexual psychology, utilizing HC users as controls, as well as a design that is unlikely to produce demand characteristics related to cycle phase. Here, we report on such a study. Specifically, we investigated relationships between ovarian hormones and within- and between-women variation in sociosexuality and general sexual desire in a large sample of women (n = 614) to test relationships between hormones and psychosexuality, utilizing these data to discriminate among hypotheses on the evolution of women's sexuality.

2. Method

2.1. Participants

Study procedures were approved by the Pennsylvania State University institutional review board. Six hundred twenty-nine participants were recruited for a study on hormones and psychology via radio, Craigslist, newspaper advertisements, posts on social media sites, the psychology department subject pool, and emails on research volunteer listservs run by the Pennsylvania State University. All participants were between 18 and 45 years of age (M (SD) = 20.04 (0.18) for NC women, M (SD) = 20.00 (0.16) for HC women), fluent in English, and not pregnant. Most participants self-identified as White (76.9%), followed by Asian (13.4%), Black or African American (8.8%), and American native or Pacific Islander (1.0%). Participants received either monetary compensation ($15) or course credit for introductory psychology courses. Only data from women who provided information about current contraceptive use (n = 629; NC n = 353, HC n = 276) were included in the present analyses (three women did not provide this information). Prevalence of HC use in the United States between 2015 and 2017 was 16.6% and 19.5% for women aged 15–19 and 20–29, respectively (Daniels & Abma, 2018). Because HC use is positively associated with education (Daniels & Abma, 2018), its expected prevalence is higher in the present sample. Approximately 44% of women who completed at least one session in the present sample reported current HC use, though we did not explicitly oversample for HC women. As per recent guidelines for sample sizes in ovulatory shift research (Gangestad et al., 2016), both our between-subjects and within-subjects sample sizes exceeded those required to achieve 80% power given anticipated effects of moderate magnitude (Cohen's d = 0.5).

2.2. Procedure

Sessions were scheduled between 09:00 and 12:00 to minimize effects of diurnal decreases in testosterone (Montanini et al., 1988). After giving informed consent, participants provided a saliva sample via passive drool. Subsequently, participants were directed to private computer workstations where they responded to demographic questions, questions on any hormonal contraceptive use in the previous six months, the Revised Sociosexual Orientation Inventory (SOI-R), the Sexual Desire Inventory (SDI-2), as well as instruments not analyzed in the present paper. The SOI-R and SDI-2 have been uploaded as ESM. The SOI-R (Penke & Asendorpf, 2008) consists of nine items measuring willingness to engage in uncommitted sex, with subscales (three items each) targeting sociosexual attitudes (e.g., “Do you agree that sex without love is OK?”), behavior (e.g., “With how many different partners have you had sexual intercourse on *one and only one* occasion?”), and desires (e.g., “How often do you have fantasies about having sex with someone you are *not* in a committed romantic relationship with?”). Participants responded to each item using a 9-point Likert scale, and responses to the three items on each subscale were averaged to produce a composite score for that subscale. All subscales exhibited high internal consistency (Cronbach's α = 0.85, 0.83, and 0.86 for Behavior, Attitude, and Desire, respectively; full-scale α = 0.84), similar to previous results (Penke & Asendorpf, 2008).

The SDI-2 is a 14-item questionnaire assessing general sexual desire (Spector, Carey, & Steinberg, 1996). The SDI-2 consists of 14 items measuring sexual desire, with subscales targeting solitary sexual desire (e.g., “How strong is your desire to engage in sexual behavior by yourself?”) and dyadic sexual desire (e.g., “How strong is your desire to engage in sexual behavior with a partner?”). Participants responded to each item using 8-point and 9-point Likert scales, and responses to the items on each subscale were averaged to produce a composite score for that subscale. Both subscales exhibited high internal consistency (Cronbach's α = 0.92 and 0.89 for solitary and dyadic sexual desire, respectively; full-scale α = 0.90). To facilitate comparisons between the present study and recent studies using a single item to assess sexual desire (e.g., “How much did you desire sexual contact yesterday?” in Roney & Simmons, 2013), we also created a composite variable from SDI items 3, 7, 9, and 11, which correlated highly with the single item assessing general sexual desire reported in Jones et al. (2018). Additionally, we performed analyses using only item 7 from the SDI-2 (“How strong is your desire to engage in sexual activity with a partner?”), as this item correlated most strongly with, and most closely resembles, the single item assessing “current sex drive” from Jones et al. (2018). Methods and data for these correlations and composites can be found online (Jones & Debruine, 2018).

Upon completing the survey, participants provided a second saliva sample via passive drool. Pre- and post-survey saliva samples were combined in equal proportions to minimize the effect of pulsatile secretory patterns on measured hormone concentrations. Samples were then stored at −20 degrees Celsius until being shipped for analysis. Finally, all participants were invited to return for a second, identical testing session (mean length between sessions: 58.28 days, SD = 12.11). Sessions were scheduled according to an aim of the broader study of which the present study is a part, and were scheduled irrespective of cycle day or phase with the requirement that the second session was completed between 1 and 3 months after the first session.

2.3. Hormone quantification

Saliva samples were analyzed at the Nipissing University Biomarkers Lab (Nipissing University, North Bay, Ontario). All samples were assayed using commercially available enzyme immunoassay kits purchased from DRG International. Sensitivities for estradiol, progesterone, and testosterone were 0.5, 3.8, and 1.9 pg/mL, respectively. Estradiol (E) intra- and inter-assay CVs were 11% and 10%, respectively, progesterone (P) intra- and inter-assay CVs were 14% and 12%, respectively, and testosterone (T) intra- and inter-assay CVs were 6.4% and 4.9%, respectively. These CVs are similar to previously published values (Edelstein, Kean, & Chopik, 2012; Grebe et al., 2016; Liening, Stanton, Saini, & Schultheiss, 2010; Schultheiss & Zimni, 2015).

2.4. Data treatment

Descriptive statistics for hormone and survey measures can be found in Table 2. Hormone concentrations were first log-transformed to reduce skew, and because some work suggests that hormones may be nonlinearly associated with other physiological processes relevant to

Table 2
Descriptive statistics showing means and standard errors.

	NC women			HC women		
	Session 1, 1 session	Session 1, 2 sessions	Session 2	Session 1, 1 session	Session 1, 2 sessions	Session 2
Estradiol (pg/mL)	3.39 (0.13)	4.21 (0.28)	4.43 (0.34)	3.82 (0.14)	3.56 (0.26)	3.44 (0.18)
Progesterone (pg/mL)	46.41 (3.44)	40.92 (5.23)	53.91 (7.82)	35.38 (2.99)	21.73 (2.02)	24.28 (3.24)
Testosterone (pg/mL)	28.65 (0.47)	28.02 (0.71)	2918 (0.85)	27.85 (0.84)	26.60 (0.91)	27.67 (0.94)
SOI-R overall	9.31 (0.29)	9.29 (0.48)	9.52 (0.50)	10.90 (0.31)	10.97 (0.55)	11.19 (0.58)
SOI-R behavior	1.98 (0.10)	2.02 (0.14)	2.05 (0.14)	2.48 (0.10)	2.44 (0.18)	2.56 (0.18)
SOI-R attitude	3.99 (0.14)	3.97 (0.26)	4.09 (0.26)	4.79 (0.16)	4.98 (0.28)	5.01 (0.29)
SOI-R desire	3.32 (0.12)	3.30 (0.17)	3.38 (0.18)	3.62 (0.12)	3.55 (0.22)	3.62 (0.24)
SDI-2 overall	7.93 (0.20)	8.38 (0.30)	8.54 (0.32)	8.41 (0.17)	8.66 (0.27)	8.88 (0.32)
SDI-2 solitary	2.76 (0.12)	3.07 (0.20)	3.33 (0.22)	2.63 (0.12)	2.74 (0.20)	3.08 (0.23)
SDI-2 dyadic	5.18 (0.11)	5.31 (0.15)	5.35 (0.16)	5.78 (0.08)	5.92 (0.13)	5.80 (0.15)

Notes: SOI-R = Revised Sociosexual Orientation Inventory; SDI-2 = Sexual Desire Inventory.

women's reproductive function (Sherry, McGarvey, Sesepasara, & Ellison, 2014). NC and HC women were then separated for main analyses. This approach was favored for several reasons. First, the majority of work assessing the link between measured hormone values and psychosexual phenotypes has been conducted among NC women, who presumably are exposed to fluctuations of endogenous, rather than exogenous, hormones. The extent to which exogenous and endogenous hormones have similar effects on downstream processes that modulate sexual psychology has not been elucidated, though there is some reason to suspect they may differ (see Introduction). Second, our primary research questions of interest were whether hormones modulate different aspects of sexual psychology among NC women, not whether hormone-behavior relationships differ between NC and HC women per se. For this reason, models were run separately on NC women and on HC women. However, we also conducted all analyses presented below using a combined sample of NC and HC women, testing for interactions between group (NC versus HC) and all hormone predictors of interest. Summaries of our primary model results, with NC and HC women combined, can be found on ESM Tables 13–16. All of our main findings were robust to the inclusion of NC and HC women in the same models.

Log-transformed hormone values were scaled and centered, separately for NC and HC women. *Z*-scores > 3 SD from the mean were classified as outliers and excluded from our main analyses. Results were generally robust to outliers (ESM Tables 3, 5, 7, 9, and 11). Sample sizes for between-subject analyses with and without outliers were, respectively, 353 and 348 for NC women, and 276 and 266 for HC women. Sample sizes for within-subject analyses with and without outliers were 92 and 87, respectively, for NC women. No outliers were eliminated for within-subject analyses for HC women ($n = 65$).

Two main sets of analyses were performed: between-subjects analyses to elucidate whether between-subject differences in hormones predict differences in psychosexual phenotypes, and within-subjects analyses to elucidate whether within-subject changes across sessions in hormones predict changes in psychosexual phenotypes. Multilevel models using the R packages *lme4* and *lmerTest* were run for between-subjects analyses, nesting observations within participants, and testing the main effects of estradiol, progesterone, and testosterone, and the interaction between estradiol and progesterone. Heterogeneity exists in the literature examining hormones and sexual desire, and ovulatory shifts in sexual desire more generally, as to whether the ratio of estradiol to progesterone (E/P) ratio (e.g., Grebe et al., 2016) or interaction between estradiol and progesterone (Roney & Simmons, 2013) should be utilized. Here, we favor the latter approach. Due to multicollinearity issues, models are unable to calculate point estimates for the main effects of estradiol and progesterone in addition to the E/P ratio. As we were interested in obtaining point estimates for the main effects of estradiol and progesterone in addition to estimating their interaction, we focus on models including estradiol × progesterone interactions. We refer to these as our primary between-subjects models, and to models with the E/P ratio and testosterone entered as predictors as our secondary between-subjects models. We focus on our primary models in the manuscript, but discuss our secondary models in brief, and present secondary model results in ESM Tables 6, 7, 10, and 11. Time of day was additionally entered as a covariate in all between-subjects analyses. For within-subject analyses, we ran multiple linear regressions with changes in estradiol, progesterone, and testosterone, as well as the interaction between change in E and change in P, as predictors of changes in SOI-R and SDI-2 scores. As changes in hormones were unrelated to changes in time of day across sessions (all $p > .204$) time of day was not entered as a covariate in within-subjects analyses.

All analyses were performed in R, and data and R script files have been uploaded as ESM.

3. Results

3.1. Within-subjects analyses: SOI-R

Most participants attended the initial session for course credit rather than monetary compensation, potentially contributing to a minority (152 of 614 women with non-outlier hormone values) agreeing to participate in the second session, for which only monetary compensation was offered (see also Discussion). In a multiple linear regression among NC women who completed two sessions, only changes in estradiol significantly positively predicted changes in overall SOI-R (estimate = 1.18, $t = 2.08$, $p = .041$; Fig. 1, panel a). Subsequent linear regressions using individual SOI-R subscales as predictors revealed a significant positive effect of changes in estradiol on changes in SOI-R Desire (estimate = 1.08, $t = 3.25$, $p = .002$), and a significant negative estradiol × progesterone interaction (estimate = −0.46, $t = -2.29$, $p = .025$; Table 3). As seen in Fig. 1, the effect of increased estradiol on Desire was greater when progesterone simultaneously decreased. No other hormone changes were significantly associated with changes in SOI-R subscale scores or overall SOI-R score (Table 3). These results were robust to the inclusion of outliers (ESM Table 1).

Sixty-five of 266 HC women with non-outlier hormone values completed both testing sessions. Changes in hormones did not predict changes in overall SOI-R, nor did they predict changes in any SOI-R subscale (Table 3).

3.2. Within-subjects analyses: SDI-2

Analyses including the 87 NC women who completed two sessions revealed that changes in hormones did not significantly predict changes in overall SDI-2, nor did they significantly predict changes in SDI-2 Solitary or SDI-2 Dyadic subscales (ESM Table 2). We then analyzed whether changes in hormones predicted changes in our composite of SDI-2 items 3, 7, 9, and 11 (see Method). Again, changes in hormones did not predict changes in the composite, and changes in hormones did

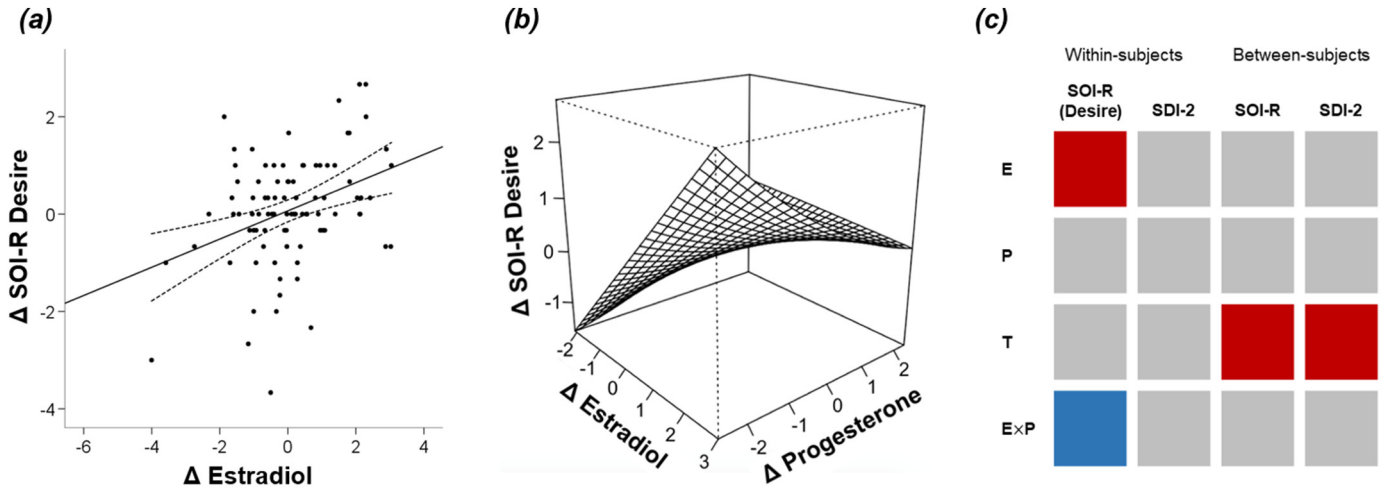

Fig. 1. Results of analyses in normally cycling (NC) women. Changes in estradiol positively predicted changes in SOI-R Desire (result of primary analysis shown in panel a). The interaction between changes in estradiol and changes in progesterone also predicted changes in SOI-R Desire (result of primary analysis shown in panel b; plot calculated from polynomial regression model and created using RSM package in R; Lenth, 2009). Panel (c) summarizes all results for NC women; red and blue indicate a preponderance of significant positive and negative associations across analyses, respectively. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

not predict changes in SDI-2 item 7 (ESM Table 2). These results were robust to the inclusion of outliers (ESM Table 3).

Repeating these analyses with the 65 HC women who completed two sessions, changes in hormones again did not predict changes in overall SDI-2, or in the SDI-2 Solitary and Dyadic subscales. Changes in hormones also did not predict changes in our composite of SDI-2 items 3, 7, 9, and 11, nor did they predict changes in SDI-2 item 7 (ESM Table 2).

3.3. Between-subjects analyses: SOI-R

We next examined interindividual relationships between hormones and SOI-R utilizing data from all sessions from all NC women, nesting observations (n = 443) within women (n = 348). Our primary models included estradiol, progesterone, testosterone, and the estradiol × progesterone interaction as hormonal predictors, while our secondary models included the E/P ratio and testosterone as hormonal predictors. Testosterone marginally significantly and positively predicted overall SOI-R scores in our primary model (estimate = 1.50, t = 1.95, p = .052; ESM Table 4). When outliers were included, the effect of testosterone was statistically significant (estimate = 1.40, t = 2.32, p = .022; ESM Table 5). Results from secondary models were consistent with these findings, such that testosterone significantly positively predicted SOI-R scores when outliers were both excluded and included (estimate = 1.37, t = 2.05, p = .030 and 1.314, t = 2.141, p = .020, respectively; ESM Tables 6 and 7; Fig. 1, panel c). No other hormone predictors were statistically significant. No hormones significantly predicted SOI-R Behavior, with the exception of testosterone significantly positively predicting SOI-R Behavior in our primary model including hormone outliers. No hormones significantly predicted SOI-R Attitude across any models. Testosterone significantly positively predicted SOI-R Desire in our primary (estimate = 0.92, t = 2.48, p = .014; ESM Table 4), and secondary (estimate = 0.94, t = 2.86, p = .005; ESM Table 6) models. These results were robust to the inclusion of outliers (ESM Tables 5 and 7).

The same nested models were run for HC women (observations n = 331, women n = 266). No hormones predicted overall SOI-R or SOI-R subscales, with the exception of testosterone positively predicting

Table 3
Multiple regression models predicting changes across sessions in SOI-R subscales in NC and HC women who completed 2 sessions.

		NC women (n = 87)			HC women (n = 65)		
		Estimate	t	p	Estimate	t	p
Overall SOI-R	ΔE	1.18	2.08	**0.041**	0.20	0.37	0.713
	ΔP	0.01	0.05	0.962	−0.03	−0.12	0.907
	ΔT	0.43	0.52	0.608	−1.58	−1.23	0.225
	ΔE × ΔP	−0.56	−1.65	0.103	−0.21	−0.43	0.671
SOI-R behavior	ΔE	0.07	0.32	0.748	0.14	0.66	0.512
	ΔP	0.02	0.25	0.802	0.04	0.34	0.739
	ΔT	0.10	0.30	0.766	−0.67	−1.34	0.194
	ΔE × ΔP	−0.04	−0.27	0.785	0.02	0.08	0.936
SOI-R attitude	ΔE	0.03	0.07	0.947	< −0.01	> − 0.01	0.998
	ΔP	−0.06	−0.60	0.551	−0.03	−0.15	0.880
	ΔT	−0.06	−0.10	0.917	0.44	0.50	0.620
	ΔE × ΔP	−0.07	−0.29	0.772	0.11	0.32	0.751
SOI-R desire	ΔE	1.08	3.25	**0.002**	0.06	0.16	0.876
	ΔP	0.06	0.61	0.547	−0.04	−0.21	0.838
	ΔT	0.39	0.80	0.429	−1.35	−1.48	0.144
	ΔE × ΔP	−0.46	−2.29	**0.025**	−0.33	−0.95	0.344

Notes: ΔE, ΔP, ΔT, = changes across sessions in estradiol, progesterone, and testosterone, respectively; SOI-R = Revised Sociosexual Orientation Inventory. Values in bold are significant at α = 0.05.

SOI-R Attitude in our primary model (estimate = 1.11, $t = 2.12$, $p = .035$; ESM Table 4), though this effect was not robust to the inclusion of outliers (ESM Table 5).

3.4. Between-subjects analyses: SDI-2

Nested models were run similar to those specified above, with overall SDI-2, SDI-2 Solitary, and SDI-2 Dyadic subscales as outcomes. Among NC women in our primary model, testosterone significantly positively predicted overall SDI-2 scores (estimate = 1.44, $t = 2.49$, $p = .013$; ESM Table 8), and this effect was robust to the inclusion of outliers (ESM Table 9). In our secondary models, testosterone did not significantly predict SDI-2 when outliers were excluded (estimate = 0.89, $t = 1.74$, $p = .083$; ESM Table 10), but this effect was significant when outliers were included (estimate = 0.86, $t = 2.00$, $p = .046$; ESM Table 11; Fig. 1, panel c). No hormones significantly predicted SDI-2 Solitary, with the exception of testosterone positively predicting SDI-2 Solitary when including outliers in our primary model (ESM Table 9). In our primary model predicting SDI-2 Dyadic, testosterone had a significant positive effect (estimate = 0.83, $t = 2.48$, $p = .014$; ESM Table 8), which was robust to the inclusion of outliers (estimate = 0.57, $t = 2.00$, $p = .047$; ESM Table 9). Testosterone was not a significant predictor of SDI-2 Dyadic in our secondary models, though E/P ratio significantly negatively predicted SDI-2 dyadic when outliers were excluded (estimate = −0.15, $t = -2.30$, $p = .023$; ESM Table 10) and included (estimate = −0.17, $t = -2.57$, $p = .012$; ESM Table 11). Progesterone significantly predicted SDI-2 Dyadic (estimate = 0.83, $t = 2.48$, $p = .014$) in our primary model, but this effect was not robust to the inclusion of outliers, nor did E/P ratios predict SDI-2 Dyadic in our secondary models.

Progesterone (estimate = 0.42, $t = 2.28$, $p = .024$) and testosterone (estimate = 0.71, $t = 2.05$, $p = .041$; ESM Table 8) significantly positively predicted the SDI-2 composite of questions 3, 7, 9, and 11, though these effects were not robust to the inclusion of outliers (ESM Table 9), nor were any hormone effects significant our secondary models (ESM Tables 10 and 11). Progesterone (estimate = 0.51, $t = 2.01$, $p = .049$; ESM Table 8) significantly positively predicted SDI-2 question 7, and this effect was not significant when outliers were included in the model (ESM Table 9). In our secondary models, the E/P ratio significantly negatively predicted SDI question 7 when outliers were and were not included (estimate = −0.20, $t = -2.05$, $p = .041$ and estimate = −0.21, $t = -2.28$, $p = .023$, respectively).

Among HC women in our primary models, estradiol negatively (estimate = −2.78, $t = -2.26$, $p = .025$) and the estradiol × progesterone interaction positively (estimate = 0.74, $t = 2.07$, $p = .040$; ESM Table 8) predicted overall SDI-2; neither effect was significant when outliers were included (ESM Table 9). In secondary models, only testosterone significantly negatively predicted overall SDI-2 when outliers were included (estimate = −0.95, $t = -2.01$, $p = .045$; ESM Table 10). In primary models predicting SDI-2 Solitary, estradiol had a negative (estimate = −1.79, $t = -2.13$, $p = .035$) and the estradiol × progesterone interaction had a positive (estimate = 0.50, $t = 2.02$, $p = .044$; ESM Table 8) effect, though neither effect was significant when outliers were included (ESM Table 9). Neither E/P ratios nor testosterone predicted SDI-2 scores in secondary models (ESM Tables 10 and 11). No hormones significantly predicted SDI-2 Dyadic in our primary models (ESM Tables 8 and 9), though testosterone significantly negatively predict SDI-2 Dyadic in models with the E/P ratio and testosterone as predictors both when outliers were excluded (estimate = −0.63, $t = -2.32$, $p = .021$; ESM Table 8) and included (estimate = −0.57, $t = -2.38$, $p = .018$; ESM Table 9). No hormones significantly predicted the SDI-2 composite of questions 3, 7, 9, and 11, though there was a significant negative effect of estradiol when including outliers (estimate = −1.30, $t = -2.16$, $p = .032$; ESM Table 9). In secondary models with the E/P ratio and testosterone as predictors, no hormones significantly predicted this SDI-2 composite, with the exception of testosterone when outliers were included (estimate = −0.55, $t = -2.10$, $p = .037$; ESM Table 11). No hormone terms significantly predicted SDI-2 question 7 in any models.

4. Discussion

By juxtaposing several evolutionary hypotheses, we are able to shed light on relationships between ovarian hormones and women's sexual psychology. Increases in estradiol across sessions predicted increases in sociosexual desire, but not general sexual desire, among NC women, particularly when progesterone simultaneously decreased. As would be expected if hormone-driven changes in sexuality are tracking ovulatory cycle status, changes in hormones did not predict changes in solitary or dyadic sexual desire, or in composites of general sexual desire among HC women. The observed pattern of hormone-driven changes in sexuality in the present study are inconsistent with the predictions of the sexual motivation and between-cycle hypotheses that fluctuating ovarian hormones coordinate broad changes in sexual motivation in order to allocate mating effort to fertile days of the ovulatory cycle or to fertile cycles, respectively.

Likewise, our data do not support the primary prediction of the spandrel hypothesis (Havlíček et al., 2015) that within-subjects hormone-behavior relationships will mirror between-subjects associations. Indeed, within- and between-subjects relationships could scarcely have been more discordant. Estradiol positively predicted only SOI-R Desire and interacted negatively with progesterone in within-subjects analyses, but was unrelated to both SOI-R and SDI-2 composites and subscales, and did not interact with progesterone in our primary between-subjects analyses. Likewise, testosterone positively predicted SOI-R and SDI-2 between subjects, but was unrelated to these measures within subjects. It is therefore unlikely that the within-woman associations between hormones and sexual psychology observed in the present study are byproducts of between-women associations.

Rather, our data are most consistent with the dual-sexuality hypothesis that women's mating strategies have evolved to recruit genetic benefits through promoting uncommitted sexual behavior during the fertile phase of the ovulatory cycle, while directing reproductive effort toward obtaining or retaining partner investment during nonfertile phases (Thornhill & Gangestad, 2008). Indeed, prior work has found elevated sociosexual or extra-pair desire during the fertile phase (Arslan et al., 2018; Gangestad, Thornhill, & Garver, 2002; Grebe et al., 2016; Shimoda et al., 2017). Given that the hormonal changes that predicted elevated sociosexual desire in the present study (increased estradiol and decreased progesterone) correspond with increased conception risk during the ovulatory cycle, it is logical to infer that these hormones jointly mediate cyclic changes in uncommitted and extra-pair sexual interest.

It is important to emphasize that our methods are unlikely to have generated demand characteristics related to ovulatory cycle phase. Participants in previous studies may have anticipated the relevance of ovulatory cycle phase due to self-report or self-collection of cycle-relevant data, scheduling of sessions according to ovulatory cycle data, and/or use of ovulation test kits, and altered their responses accordingly. Indeed, some evidence indicates that women who track their cycle phase may exhibit stronger cycle effects, likely due to demand characteristics (Arslan et al., 2018). Our methods offered little indication of any focus on the ovulatory cycle, yet our results are difficult to explain without reference to the ovulatory cycle. Because other studies have found that in-pair (Arslan et al., 2018; Roney & Simmons, 2016) or general sexual desire (Jones et al., 2018; Roney & Simmons, 2013), but sometimes not sociosexuality (Jones et al., 2018), changed as a function of ovarian hormone fluctuations, it is worth exploring in future research the extent to which demand characteristics may moderate these relationships. Future work should also investigate the influence of other methodological or analytic approaches that differ across this and prior studies. For example, the studies of Roney and Simmons (2013) and

Jones et al. (2018) differed from the present study in the number of sessions and density of the sampling schedule (data collected daily [Roney & Simmons, 2013] or weekly [Jones et al., 2018] vs. approximately 2 months apart in the present study). On the one hand, a greater number of more closely-spaced sessions might facilitate detection of acute within-subjects changes. On the other hand, it is possible that this approach leads to a consolidation of responses, especially on items or instruments designed to assess psychological constructs at the trait level. Differences across items and instruments in such consolidation, coupled with differences across studies in statistical power, could lead to discrepant patterns of results across studies. For example, it could be the case that SOI-Desire is more susceptible to such consolidation, so that Jones et al. (2018) were unable detect a relationship with estradiol even with greater statistical power. If items on sexual desire are less susceptible to such an effect, then the greater number of observations in Roney and Simmons (2013) and Jones et al. (2018) may have enabled them to detect relationships with changes in ovarian hormones, whereas we could not. It is also possible that differences in survey instruments, model-building approaches, and hormones included in the models contributed to the divergent findings across studies. For instance, a high degree of measurement precision may be required to reliably detect intra-individual changes in sociosexual desire, which was measured using 9-point scales in the present study and 5-point scales in Jones et al. (2018). The extent to which such procedural and statistical choices moderate study results should be systematically tested moving forward.

Although they make different sets of predictions, the main hypotheses tested in the present paper are not mutually exclusive, and it is possible that both general sexual desire and sociosexual desire track changes in estradiol and progesterone. However, because changes in ovarian hormones predicted sociosexual desire but not general sexual desire in the present study, our data are most consistent with the dual-sexuality hypothesis.

With regard to between-subjects relationships, ours is the first study to find that testosterone predicts sociosexuality in women, perhaps due to our larger sample size than previous studies (Charles & Alexander, 2011; Edelstein et al., 2011; Puts et al., 2015), as well as sampling over a narrower range of times of day, collecting two saliva samples at each testing session, and controlling statistically for estradiol and progesterone. On their own, the positive associations between testosterone and SOI-R and SDI-2 could be interpreted to reflect both between- and within-subjects variability in testosterone. However, the fact that within-subjects changes in testosterone did not predict changes in SOI-R or SDI-2 makes this interpretation considerably less tenable, instead suggesting that between-individual differences in steroid concentrations contribute to differences in sociosexuality and sexual desire. Further, in both NC and HC women, all hormones significantly correlated across sessions (see ESM Table 12), suggesting that hormone concentrations capture significant between-subjects differences in hormone production.

These findings also add to a debate about the relative importance of estradiol and testosterone in modulating individual differences in trait levels of sexual desire, suggesting a positive role for testosterone but not estradiol. It has been proposed that testosterone influences sexual psychology indirectly by aromatization into estradiol in the brain and subsequent binding to estrogen receptors (Cappelletti & Wallen, 2016), or via binding to sex hormone-binding globulin in the blood, thus increasing biologically active, unbound estradiol (Burke & Anderson, 1972). However, these possibilities appear inconsistent with the lack of relationship with estradiol at the between-subjects level, and the lack of relationship with testosterone at the within-subjects level. If testosterone influences sexual psychology by increasing estrogen receptor binding, then why do within-subject changes in testosterone not predict changes in sexual psychology, and why do individual differences in estradiol not predict differences in sociosexuality? Instead, perhaps ovulatory cycle-related changes in estradiol have relatively acute positive effects on sociosexual desire, and between-subjects differences in testosterone, but not estradiol, influence general sexual interest over the longer term. Indeed, sociosexual desires as indexed by the SOI-R are less temporally stable than behaviors and attitudes (Penke & Asendorpf, 2008; also see ESM Table 12) and may therefore be more susceptible to acute changes in ovarian steroid concentrations. That only sociosexual desires related to estradiol at the within-subjects level, while overall SOI-R related to between-subjects differences in testosterone, is thus consistent with short-term effects of estradiol and long-term effects of testosterone.

4.1. Limitations

Observed differences in estradiol or progesterone across testing sessions could be attributed to factors beyond ovulatory cycle phase, including diet, exercise, stress, pregnancy, and lactation (Ellison, 2003; Jasienska, 2012; Motta-Mena & Puts, 2017). Although participants in our study were not pregnant or lactating, we cannot definitively exclude other factors as potential causes of hormonal changes across sessions. Nevertheless, the primary cause of variation in these hormones across sessions is likely to have been position in ovulatory cycle. Moreover, the negative interaction between estradiol and progesterone in predicting sociosexual desire suggests a within-cycle effect (Table 3), an inference that is bolstered by the absence of these relationships in HC women, in whom ovulatory cycle shifts in ovarian hormones are suppressed. Second, relationship status was not obtained for women in the present study. Though some studies do not report differences in the presence of hormone-behavior relationships or ovulatory shifts between partnered and single women (Jones et al., 2018; Jünger et al., 2018a), many studies find a moderating effect of relationship status on hormone-behavior relationships (Debruine et al., 2019; Jünger, Gerlach, & Penke, 2018b; Jünger, Kordsmeyer, Gerlach, & Penke, 2018c; Marcinkowska et al., 2018; Roney & Simmons, 2016). We are unable to test whether similar hormone-behavior-partnership interactions are present for measures of sexual desire, and believe this would be a fruitful avenue for future work. Third, as most women who completed a first testing session did not return for a second testing session, it is possible that there exist differences between women who completed one versus two sessions, reducing the generalizability of the present results. However, we can think of no reason why differences between women who completed one versus two sessions would translate into differences between the hormone-behavior relationships of interest in the present study. Further, there were no significant differences in SOI-R or SDI-2 scores in women who completed one versus two sessions (see Table 2). Thus, it is unlikely that any selection bias would change the pattern of reported results. Finally, the SOI-R and SDI-2 were not specifically constructed to assess state levels or intraindividual changes in psychosexuality. However, recent studies in personality (Brose, Lindenberger, & Schmiedek, 2013) and sexuality (Goldey & Van Anders, 2012) psychology suggest that affective states significantly modulate participants' self-reports of trait measures. Penke and Asendorpf (2008) found low test-retest reliability over a one-year period for the Desire subscale ($r = 0.39$), suggesting that this measure is indeed labile. In our data, this test-retest correlation was considerably higher ($r = 0.77$ and $r = 0.78$ for NC and HC women, respectively), as might be expected given the shorter interval between sessions ($\leq$ 3 months), but the correlation was lower than those for the Attitude ($r = 0.86$ and 0.90 for NC and HC women, respectively) and Behavior ($r = 0.89$ and $r = 0.88$ for NC and HC women, respectively; ESM Table 12) subscales. Nevertheless, future research investigating changes in psychosexual phenotypes at short time scales should utilize instruments that have specifically been shown to capture acute changes in sociosexual and general sexual desire, and the degree to which sociosexual and general sexual desire fluctuate across different timespans should be characterized.

4.2. Conclusions and future directions

The present work investigates the hormonal predictors of fundamental, yet highly variable, components of women's sexuality, suggesting that hormonally-driven changes in sociosexuality are partly independent of general sexual desire. The absence of robust, hormonally-driven changes in HC women suggests that such changes may track conception risk, and that future work should elucidate whether endogenous and exogenous hormones have similar downstream effects on processes modulating sexual psychology. Our results are most consistent with the dual-sexuality hypothesis that women possess adaptations for recruiting genetic benefits during the fertile phase of the ovulatory cycle. Future work should continue to discriminate between conceptually distinct aspects of women's sexual psychology, such as sociosexual desire and general sexual desire, to clarify the putative adaptive value of within- and between-cycle shifts in women's mating psychology. Objective markers of ovulatory status, such as ultrasonography or luteinizing hormone tests, should be employed alongside ovarian hormone measurements to jointly elucidate the proximate and ultimate modulators of any such shifts. Finally, studies evaluating the proximate mechanisms by which *chronic* or *average* hormone levels and *fluctuations* in hormone levels act to modulate mating psychology in NC women are required to elucidate discrepancies between inter- and intra-individual hormone-behavior relationships. A deeper understanding of both the hormonal mechanisms as well as putative adaptive benefits of cyclic changes in sexual motivation has implications for understanding estrus in other pair-bonding as well as non-pair-bonding animals, and the evolution of female sexuality more broadly.

Author contributions

D. Puts and K. Dawood designed the study. H. Self coordinated data collection. J. C. Carré and T. Ortiz performed hormone assays. T. N. Shirazi and K. Rosenfield performed data management and analysis. T. N. Shirazi and D. Puts drafted the manuscript. All authors provided critical revisions, and have approved the final version of this manuscript for submission.

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Declaration of interest

The authors declare no conflicts of interest.

Open practices statement

The present manuscript's data and code have been uploaded as part of the electronic supplemental material.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.evolhumbehav.2019.02.002.

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