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Menstrual cycle phase predicts women's hormonal responses to sexual stimuli

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ABSTRACT

A robust body of research has demonstrated shifts in women's sexual desire and arousal across the menstrual cycle, with heightened desire and arousal coincident with heightened probability of conception (POC), and it is likely that ovarian hormones modulate these shifts. However, studies in which women are exposed to audiovisual sexual stimuli (AVSS) at high POC (mid-follicular) and low POC (luteal) phases have failed to detect significant differences in genital or subjective arousal patterns based on menstrual cycle phase. Here, we tested whether hormonal responsivity to AVSS differs as a function of cycle phase at testing, and whether phase during which participants were first exposed to AVSS influences hormonal responsivity in subsequent test sessions. Twenty-two naturally cycling heterosexual women were exposed to AVSS during the follicular and luteal phases, with phase at first test session counterbalanced across participants. Salivary samples were collected before and after AVSS exposure. Estradiol increased significantly during both follicular and luteal phase sessions, and increases were higher during the follicular phase. Testosterone (T) increased significantly only during the follicular phase session, while progesterone (P) did not change significantly during either cycle phase. Session order and current cycle phase interacted to predict P and T responses, such that P and T increased during the follicular phase in women who were first tested during the luteal phase. These data suggest that menstrual cycle phase influences hormonal responsivity to AVSS, and contribute to a growing body of clinical and empirical literature on the neuroendocrine modulators of women's sexuality.

1. Introduction

Menstrual cycle shifts in sexual behavior have been reported for nearly 40 years. Autosexual or solitary (Brown et al., 2011; Burlison et al., 2002; Van Goozen et al., 1997) and female-initiated (Adams et al., 1978; Bancroft et al., 1983; Harvey, 1987; Matteo and Rissman, 1984; Sanders et al., 1983) sexual behaviors increase near ovulation, though some studies have failed to detect changes in sexual behavior across the cycle (Brewis and Meyer, 2016; Elaut et al., 2016; Roney and Simmons, 2013). Trends in partnered sexual behavior may be more heavily dependent upon external factors; for example, several studies have found the strongest predictors of partnered sexual activity to be the day of the week ('the weekend effect,' Caruso et al., 2014; Palmer et al., 1982; Roney and Simmons, 2013; Wilcox et al., 2004). Further, as partnered sexual behavior typically requires the sexual interest of both members of a copulatory pair, its occurrence is not solely a reflection of women's desires. Putative menstrual cycle shifts in sexuality should therefore be more apparent in constructs and behaviors that are more dependent on internal motivation, as opposed to the availability and

interest of sexual partners. Unlike other mammals in which sexual activity is strictly modulated by hormonal condition and confined to high fertility periods (Wallen, 1990), humans are able to mate independently of hormonal condition, allowing for the unique decoupling of sexual interest and sexual behavior.

Indeed, women's sexual desire and arousal show relatively more robust cyclic patterns such that desire and arousal are heightened when probability of conception (POC) is highest, during the late follicular and ovulatory phases, and decrease when POC decreases, during the early follicular and luteal phases (Wilcox et al., 1995). Peaks in self-reported sexual desire (Brown et al., 2011; Diamond and Wallen, 2011; Englander-Golden et al., 1980; Graham et al., 2000; Pillsworth et al., 2004; Röder et al., 2009; Roney and Simmons, 2016, 2013), frequency of sexual fantasies (Dawson et al., 2012; Matteo and Rissman, 1984; Slob et al., 1996), and degree to which fantasies are rated as arousing (Dawson et al., 2012) have been reported in women prior to ovulation. Shifts across the menstrual cycle in sexual desire and arousal may have therefore evolved to promote sexual behavior and the saliency of sexual stimuli when POC is heightened, during the mid-follicular and

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ovulatory phases (Roney, 2015; Roney and Simmons, 2013). In contrast, men do not exhibit cyclic changes in steroid hormone concentrations, conception status, or sexual desire and arousal.

Due to their fluctuating patterns across the menstrual cycle, the ovarian steroid hormones estradiol (E_2), progesterone (P), and testosterone (T) have been evaluated as moderators of menstrual cycle shifts in women's sexual desire and arousal. The single study measuring both sexual desire and salivary hormones for complete menstrual cycles found that within women, sexual desire was positively associated with E_2 , negatively associated with P, and unassociated with T levels (Roney and Simmons, 2013), mirroring patterns observed in rhesus macaques (Wallen et al., 1984; for review of the roles of estrogens and androgens in modulating women's sexual desire, see Cappelletti and Wallen, 2016; Motta-Mena and Puts, 2017).

E_2 , P, and T may further be implicated in women's sexuality, as these hormones may be acutely responsive to external sexual stimuli and concomitantly increase sexual desire, arousal, and behavior. T increases as a result of orgasm (Exton et al., 1999) and partnered sexual behavior (van Anders et al., 2007), as may E_2 (van Anders et al., 2009). Studies in which women were exposed to videotaped courtship interactions (Lopez et al., 2009), pictures of opposite-sex faces (Zilioli et al., 2014), and studies in which women were instructed to imagine a sexual social interaction (Goldey and van Anders, 2011) have reported significant increases in T. Interestingly, several studies in which participants were exposed to visual sexual stimuli (VSS) or audiovisual sexual stimuli (AVSS) have reported no increases in T (Goldey and van Anders, 2016; Hamilton et al., 2008; Heiman et al., 1991; van Anders et al., 2009), while others have shown that the magnitude and direction of T and E_2 changes may differ substantially among women (Garcia et al., 2015). Research on the factors modulating hormonal responses to mating-related and sexual stimuli is sparse, and no studies have systematically examined the effects of menstrual cycle phase on such responses. As menstrual cycle shifts in psychology and behavior generally involve phenotypes related to mating and sexuality, and given that E_2 , P, and T modulate these phenotypes, it follows that hormonal responsivity to sexual stimuli may differ as a function of menstrual cycle phase.

Though hormonal responsivity to mating-related and sexual stimuli across cycle phases has not yet been examined, eye gaze patterns, subjective ratings, and genital arousal patterns have, with results differing as a function of experimental design. Eye tracking studies employing a within-subjects design, wherein menstrual cycle phase at first testing session is counterbalanced across women, have failed to detect significant effects of current menstrual cycle phase on eye gaze patterns and subjective reports of arousal to VSS (Rupp and Wallen, 2007; Wallen and Rupp, 2010); when only data from the first testing session are considered, however, significant differences based on current menstrual cycle phase emerge. Similarly, when genital responses to and subjective ratings of AVSS are measured repeatedly across the cycle in women, differences as a function of menstrual cycle phase are not detected (Bossio et al., 2014; Meuwissen and Over, 1992; Slob et al., 1991, 1996; Suschinsky et al., 2014), but differences are detected when analyzing data from the first session in isolation (Slob et al., 1991, 1996; but see Meuwissen and Over, 1992). How could significant effects of menstrual cycle phase be detected in cross-sectional, but not within-subjects designs? Accounting for this phenomenon in part is the 'carry-over effect,' or significant effect of menstrual cycle phase at initial stimuli exposure, first reported by Slob et al. (1991). Women who were first tested in the follicular phase exhibited heightened genital and subjective responses to AVSS as compared to women tested first in the luteal phase, and continued to exhibit such heightened responses during subsequent test sessions. These results suggest that cycle phase may modulate genital and visual responses to AVSS, though intra-individual menstrual cycle shifts may be masked by the magnitude of such carry-over effects. Though significant order effects of testing have been reported for genital measures (Slob et al., 1996) as well as for eye gaze

patterns (Wallen and Rupp, 2010) in within-subjects studies, some studies have not found significant effects of session order on genital arousal patterns (Bossio et al., 2014; Suschinsky et al., 2014) and subjective reports (Slob et al., 1996). Whether hormonal responses to AVSS would exhibit current cycle phase and session order effects has not been systematically examined.

Given increases in cognition and behavior related to mating and sexual desire when conception is more probable, it is likely that hormonal responses to AVSS would be modulated by cycle phase. Here, we present the first empirical evaluation of this hypothesis. Naturally cycling women were recruited for sexual psychophysiology test sessions during the mid-follicular (when POC > 0) and luteal phases (when POC = 0), with session order counterbalanced across participants to detect any order effects. During sessions, participants were exposed to AVSS, and pre- to post-stimuli levels of E_2 , P, and T were measured. We hypothesized that greater hormone responsivity to AVSS would be observed in sessions during the follicular phase as compared to those during the luteal phase. Further, consistent with work suggesting carry-over effects (Slob et al., 1991, 1996; Wallen and Rupp, 2010), we hypothesized that the magnitude of hormone responsivity would be modulated by cycle phase at initial test session, with women tested first during the follicular phase exhibiting greater hormone responsivity to AVSS across test sessions.

2. Method

2.1. Participants

Women were recruited via flyers posted on a university campus, and were screened via telephone to determine study eligibility. All participants were required to be between the ages of 18 and 40, naturally cycling (i.e., not be on a hormonal contraceptive regimen or pregnant), and to have regular menstrual cycles between 27 and 33 days long. No participants had a history of sexually transmitted infections or sexual dysfunction, and were required to have past experience with vaginal penetration. Experience with vaginal penetration was required, as genital arousal data were collected via a gauge inserted into the vagina, analyzed in other studies (Bossio et al., 2014; Suschinsky et al., 2014). Only women who reported being exclusively or predominantly androphilic, or attracted to men (rating of 0 or 1 on the Kinsey Sexual Fantasy Scale; Kinsey et al., 1953), were included. All study procedures were IRB approved, and all participants provided informed consent.

Of the 37 women initially recruited for the study, 22 were included in the present analyses (M age = 21.9, SD = 4.8). Participants were excluded if: a) they did not attend a second testing session ($n = 6$); b) cycle phase could not be confirmed by hormonal analyses ($n = 7$); c) issues arose with freezing hormone samples ($n = 1$); or d) equipment malfunctions were experienced during the session ($n = 1$).

2.2. Experimental stimuli

Audiovisual stimuli used in the present study were neutral and erotic videos, which have previously been shown to elicit subjective and genital arousal among androphilic women (Chivers et al., 2007). The eight AVSS categories, with two exemplars of each category presented, were as follows: female nude exercise, female masturbation, female-female intercourse, male nude exercise, male masturbation, male-male intercourse, female-male intercourse, and landscapes. Presentation order was randomized for all participants, and films were separated by intertrial intervals to allow for physiological sexual response return to baseline levels.

2.3. Procedure

Procedures in the present study are identical to those of Bossio et al. (2014) and Suschinsky et al. (2014), though the hormone data in the

present study have not been explored previously. Briefly, participants were randomly scheduled to complete their first testing session during either the follicular or luteal phase. Following Puts' (2006) method for assessing menstrual cycle phase, we used forward-counting to estimate the onset of participants' next menstrual cycles, and backward-counting to estimate ovulation. This was used to schedule participants' first test sessions, half of which were during the follicular phase (follicular first, FF), and the other half of which were during the luteal phase (luteal first, LF). More specifically, follicular phase sessions were scheduled zero to four days prior to estimated ovulation, and luteal phase sessions were scheduled four to 11 days after estimated ovulation. After completion of the first session, menstrual cycle phase was reassessed, and a second session was scheduled approximately two weeks later ($M = 13.8$ days, $SD = 4.1$) in the opposite phase of the menstrual cycle phase at first testing. Women were included in analyses if P levels obtained during the presumptive luteal phase session were greater than P levels during the presumptive follicular phase session. Employing a stricter cutoff and repeating our analyses on the subset of women who had a progesterone difference of 30 pg/mL or more between follicular and luteal phase sessions ($n = 18$) did not considerably alter the pattern of significant results. Analyses using this subset of women can be found in the ESM.

Participants were instructed to refrain from using medications that may interfere with sexual arousal, as well as from solitary and partnered sexual activity for 24 h prior to test sessions, from aerobic activity 3 h prior to test sessions, and from using caffeine, alcohol, and recreational drugs on the day of testing. Upon arrival, participants filled out questionnaires on demographic, sexual history, and menstrual cycle characteristics. Two saliva samples (~1 mL each) were obtained via passive drool approximately 30 min apart prior to testing. Sexual physiological testing and measures of subjective arousal, as described in Bossio et al. (2014) and Suschinsky et al. (2014), were obtained while participants viewed AVSS in a private testing room. AVSS included 16 90-s videos shown in a randomized order, separated by approximately 60-s inter-trial intervals. A final saliva sample was obtained immediately after testing. Procedures during the second session were identical to those of the first.

2.4. Salivary assays

Saliva samples were frozen at -80°C until assay. All samples were assayed for salivary E_2 , P, and T, in duplicate using a highly-sensitive enzyme immunoassay (Cat. No. 1-1502, Salimetrics LLC, State College PA). For E_2 , the test used 225 μL of saliva per determination, had a lower limit of sensitivity of 0.1 pg/mL, standard curve range from 1 pg/mL to 32 pg/mL, an average intra-assay coefficient of variation of 7.1%, and an inter-assay coefficient of variation of 7.5%. Method accuracy determined by spike recovery averaged 105.1%, and linearity determined by serial dilution averaged 99.9%. For P, the test used 50 μL of saliva per determination, had a lower limit of sensitivity of 5.0 pg/mL, standard curve range from 10 pg/mL to 2430 pg/mL, an average intra-assay coefficient of variation of 6.2%, and an inter-assay coefficient of variation of 7.6%. Method accuracy determined by spike recovery averaged 99.6%, and linearity determined by serial dilution averaged 91.8%. For T, the test used 25 μL of saliva per determination, had a lower limit of sensitivity of 1.0 pg/mL, standard curve range from 6.1 pg/mL to 600 pg/mL, an average intra-assay coefficient of variation of 4.6%, and an inter-assay coefficient of variation of 9.8%. Method accuracy determined by spike recovery averaged 104.3%, and linearity determined by serial dilution averaged 102.4%.

2.5. Data preparation and analyses

As hormone concentrations in the two samples obtained prior to stimuli exposure were highly correlated (all $r_s > 0.96$), for participants who produced one pre-stimuli salivary sample (2 instances for E_2 ; 5

instances for P), the existing value for that sample was used for analyses; for participants who produced both pre-stimuli salivary samples, the mean was used as the pre-stimuli hormone concentration. Raw hormone concentrations were used for analyses. The decision to use raw or log-transformed hormone concentrations did not affect our primary findings, and analyses with log-transformed hormone concentrations are provided in the ESM. Following prior analytical strategies examining menstrual cycle and order effects, we submitted data from the first test session only to a univariate ANOVA with cycle phase at testing as a between-subjects factor, and baseline hormone concentration as a covariate, wherein a significant effect of cycle phase would provide cross-sectional support for putative menstrual cycle effects. Separate ANOVAs were run for E_2 , P, and T. Next, we performed a 2 (Session order: Follicular First [FF], Luteal First [LF]) \times 2 (Cycle phase at testing: Follicular [F], Luteal [L]) \times 2 (Time: Pre-test, post-test) mixed ANOVA, where cycle phase and time were within-subject factors, and order was a between-subjects factor. Following the individual-differences approach of Garcia et al. (2015), we also examined and present the variability in hormonal responsiveness across subjects. Figures are displayed using mean untransformed hormone values for interpretability. Analyses were conducted using IBM SPSS Statistics software (Armonk, NY). Mean raw hormone concentrations are presented with standard errors.

3. Results

3.1. Estradiol

E_2 data were available for all women described in the methods. When we utilized E_2 data from session 1 only, the pre- to post-stimuli change in E_2 concentrations was significantly different from 0 ($F(1,21) = 5.45$, $p = 0.031$, $\eta^2 = 0.22$). The effects of cycle phase ($F(1,21) = 1.16$, $p = 0.296$, $\eta^2 = 0.06$) and baseline E_2 ($F(1,21) = 1.32$, $p = 0.265$, $\eta^2 = 0.07$) were not significant. In the subsequent repeated-measures ANOVA utilizing E_2 data from both sessions, there was a significant main effect of cycle phase ($F(1,21) = 11.09$, $p = 0.003$, $\eta^2 = 0.6$), such that mean E_2 concentrations were higher during the luteal phase (4.023 ± 0.22 pg/mL) than during the follicular phase (3.36 ± 0.22 pg/mL). The cycle phase \times session order interaction was not significant ($F(1,21) = 0.14$, $p = 0.714$, $\eta^2 = 0.01$), suggesting that differences in follicular and luteal phase session E_2 concentrations did not vary as a function of session order. There was a significant main effect of time ($F(1,21) = 36.57$, $p < 0.001$, $\eta^2 = 0.65$), with higher E_2 concentrations post-stimuli (4.14 ± 0.24 pg/mL) than pre-stimuli (3.25 ± 0.18 pg/mL). Twenty-one out of 22 women tested during the follicular phase, and 20 of 22 women tested during the luteal phase, exhibited pre- to post-stimuli E_2 increases (see Fig. 1). Time and session order did not interact ($F(1,21) = 0.24$, $p = 0.627$, $\eta^2 = 0.01$), nor was the time \times session order \times cycle phase interaction significant ($F(1,21) = 0.47$, $p = 0.502$, $\eta^2 = 0.02$). There was a significant interaction between cycle phase and time ($F(1,21) = 7.00$, $p = 0.018$, $\eta^2 = 0.25$; see Fig. 2). Posthoc paired samples t -tests revealed greater pre- to post-stimuli differences in E_2 during the follicular phase as compared to during the luteal phase ($t(21) = 2.62$, $p = 0.012$, Cohen's $d = 0.72$).

3.2. Progesterone

P data were available for all FF women and 10 LF women. When we utilized P data from session 1 only, the pre- to post-stimuli change in P concentrations was not significantly different from 0 ($F(1,20) = 1.20$, $p = 0.287$, $\eta^2 = 0.06$). The effects of cycle phase ($F(1,20) = 0.01$, $p = 0.910$, $\eta^2 = 0.01$) and baseline P ($F(1,20) = 0.36$, $p = 0.0558$, $\eta^2 = 0.02$) were not significant. In the subsequent repeated-measures ANOVA utilizing P data from both sessions, there was a significant main effect of cycle phase ($F(1,20) = 23.53$, $p < 0.001$, $\eta^2 = 0.55$), such

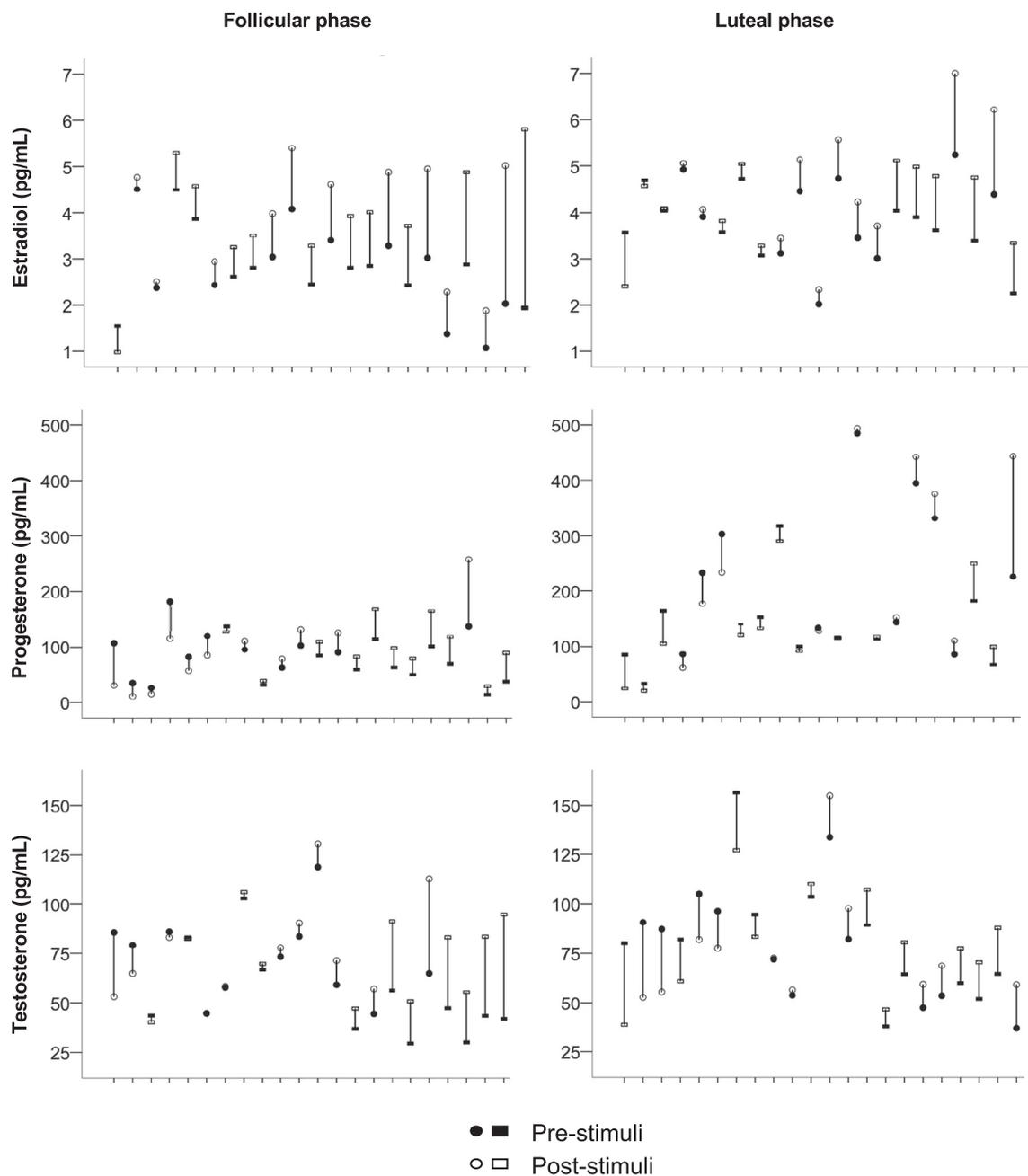


Fig. 1. Individual-level hormonal responses to stimuli during follicular and luteal phase sessions. Individual participants are ordered along the x-axes according to the magnitude of their hormonal responses. Circles represent data from women tested first in the follicular phase, and rectangles represent data from women tested first in the luteal phase.

that mean P concentrations were higher during the luteal phase (190.65 ± 24.67 pg/mL) than during the follicular phase (86.66 ± 9.87 pg/mL). There was also a significant cycle phase \times session order interaction ($F(1,20) = 7.82$, $p = 0.012$, $\eta^2 = 0.29$) such that luteal phase P was higher in women tested first in the follicular phase (252.16 ± 35.71 pg/mL) as compared to women tested first in the luteal phase (129.14 ± 34.05 pg/mL); follicular phase P, however, did not differ between these two groups (88.24 ± 14.29 and 85.09 ± 13.62 , respectively). The main effect of time ($F(1,20) = 1.94$, $p = 0.180$, $\eta^2 = 0.09$) was not significant. Fifteen of 22 women tested during the follicular phase, and 10 of 21 women tested during the luteal phase, exhibited pre- to post-stimuli P increases. The time \times session order interaction ($F(1,20) = 0.01$, $p = 0.943$, $\eta^2 < 0.01$), time \times cycle phase interaction ($F(1,20) = 0.70$, $p = 0.412$, $\eta^2 = 0.04$; see Fig. 2), and time \times cycle \times session order interaction (F

($1,20) = 3.33$, $p = 0.084$, $\eta^2 = 0.15$) were not significant. This interaction was significant when log-transformed values were used, as well as when raw and log-transformed values were used in the subset of women with P differences 30 pg/mL or more between follicular and luteal phase sessions. For women tested first during the luteal phase, pre- to post-stimuli p values differed during the follicular phase, but not during the luteal phase. For women tested first during the follicular phase, pre- to post-stimuli P were not significant during follicular or luteal test sessions phases (see Fig. 3). As it has been previously suggested that E_2 concentrations at initial viewing sessions may account for the putative order effects or carry-over effects observed in subsequent sessions, we re-ran this model with session 1 estradiol entered as a covariate. Time and session 1 E_2 did not significantly interact ($F(1,20) = 0.84$, $p = 0.371$, $\eta^2 = 0.05$), and the three-way interaction between time, current cycle phase, and session 1 E_2 was not significant

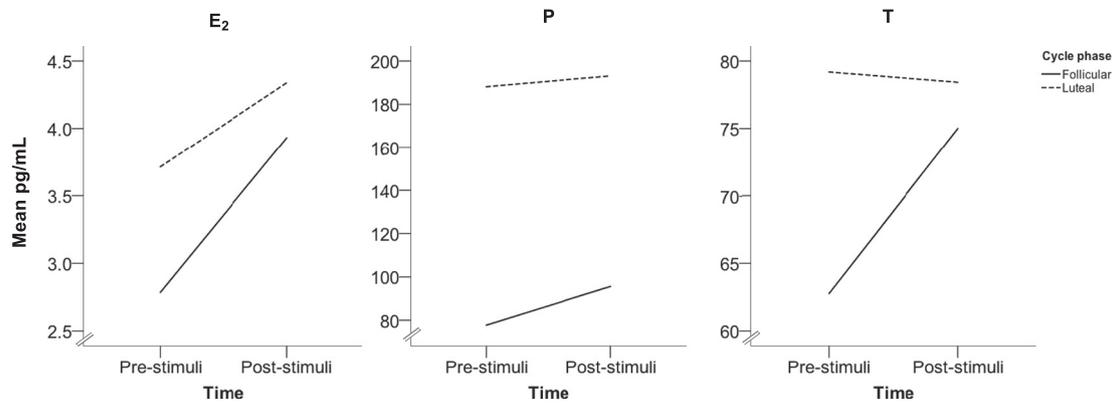


Fig. 2. Pre- vs. post-stimuli hormone concentrations by cycle phase. Main effect of time (pre- vs post-stimuli) was significant for estradiol (E₂), but not for testosterone (T) or progesterone (P). Cycle phase and time interacted significantly for E₂ and T, but not for P.

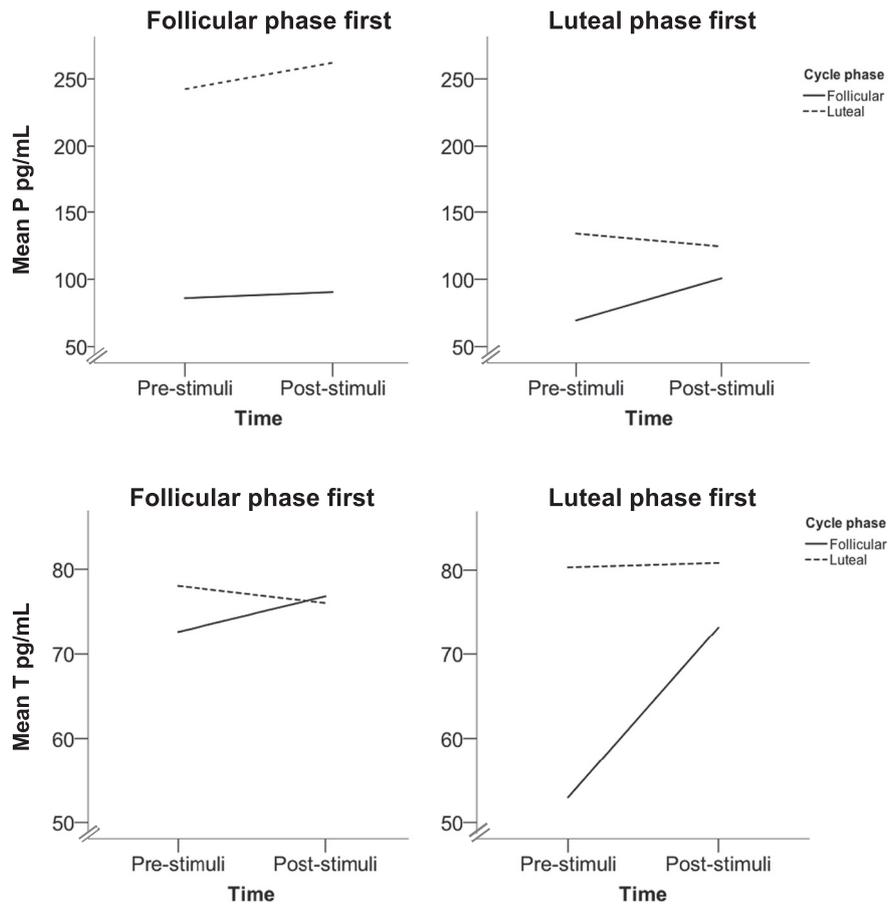


Fig. 3. Interactions between time (pre- vs. post-stimuli), session order (follicular vs. luteal first), and current cycle phase (follicular vs. luteal) in P (top row) and T (bottom row) concentrations.

($F(1,20) = 0.03, p = 0.860, \eta^2 < 0.01$).

3.3. Testosterone

T data were available for all women described in the methods. When we utilized T data from session 1 only, the pre- to post-stimuli change in T concentrations was not significantly different from 0 ($F(1,21) = 4.04, p = 0.059, \eta^2 = 0.175$). The effects of cycle phase ($F(1,21) = 0.02, p = 0.893, \eta^2 < 0.01$) and baseline T ($F(1,21) = 4.15, p = 0.056, \eta^2 = 0.18$) were not significant. In the repeated-measures ANOVA including data from both test sessions, there was a significant main effect of cycle phase ($F(1,21) = 7.56, p = 0.012, \eta^2 = 0.27$), such that T

concentrations were higher during the follicular phase (78.81 ± 5.78 pg/mL) than during the luteal phase (68.89 ± 4.50 pg/mL). There was a significant cycle phase \times session order interaction ($F(1,21) = 4.42, p = 0.048, \eta^2 = 0.18$) such that follicular phase T was higher in women tested first in the follicular phase (74.69 ± 6.37 pg/mL) as compared to women tested first in the luteal phase (63.08 ± 6.37 pg/mL); luteal phase T, however, did not differ between these two groups (77.03 ± 8.18 and 80.59 ± 8.18 , respectively). The main effect of time was not significant ($F(1,21) = 2.32, p = 0.144, \eta^2 = 0.10$). Time and session order did not interact ($F(1,21) = 1.52, p = 0.233, \eta^2 = 0.07$). There was a significant interaction between cycle phase and time ($F(1,21) = 7.60, p = 0.012, \eta^2 = 0.28$; see Fig. 2).

Eighteen out of 22 women tested during the follicular phase, and 16 of 22 women tested during the luteal phase, exhibited pre- to post-stimuli T increases. Posthoc paired samples *t*-tests revealed that T responsivity to AVSS was greater during the follicular phase compared to the luteal phase ($t(21) = 2.69$, $p = 0.014$, $d = 0.63$). The time \times session order \times cycle phase interaction was not significant ($F(1,21) = 2.03$, $p = 0.169$, $\eta^2 = 0.09$). This interaction was significant when log-transformed values were used, as well as when raw and log-transformed values were used in the subset of women with P differences 30 pg/mL or more between follicular and luteal phase sessions. For women tested first during the luteal phase, pre- to post-stimuli T values differed during the follicular phase, but not during the luteal phase. For women tested first during the follicular phase, pre- to post-stimuli changes in T were not significant during both follicular and luteal phase sessions (see Fig. 3). We then re-ran this model with session 1 E₂ entered as a covariate. The interaction between time and session 1 E₂ was not significant ($F(1,21) = 1.55$, $p = 0.229$, $\eta^2 = 0.08$), nor was the three-way interaction between time, current cycle phase, and session 1 E₂ ($F(1,21) = 1.42$, $p = 0.248$, $\eta^2 = 0.07$).

4. Discussion

The aim of the present study was to explore whether hormonal responsivity to AVSS differs as a function of menstrual cycle phase and cycle phase at initial exposure (order of testing). Concentrations of E₂ increased in response to AVSS during both the follicular and luteal phases, though the magnitude of this increase was greater during the follicular phase, whereas T increased only in test sessions occurring in the follicular phase. Increases in both E₂ and T were robust across participants, contrasting with prior work suggesting either no changes, or a lack of interindividual consistency, in the magnitude and direction of E₂ and T changes in response to AVSS (Garcia et al., 2015; Goldey and van Anders, 2016; Hamilton et al., 2008; van Anders et al., 2009). Significant increases in P were not observed in response to AVSS exposure at either cycle phase. Whereas the majority of participants exhibited increases in E₂ and T in response to stimuli, notable individual differences in the direction of P responses were observed. In contrast with prior work finding significant effects of current menstrual cycle phase using cross-sectional but not within-subject designs, here we detected significant effects of current menstrual cycle phase only when analyzing longitudinal, within-subjects data.

That E₂ increased in response to AVSS across luteal and follicular cycle phases is consistent with a positive association between E₂ and sexual desire (Jones et al., 2018; Roney and Simmons, 2013; reviewed in Cappelletti and Wallen, 2016), and with previous research on AVSS and E₂ responsivity (van Anders et al., 2009) wherein E₂ but not T changed significantly in response to eight-minute erotic films. More central to the present study's aims, however, was the interaction between E₂ responsivity and cycle phase. In within-subjects analyses, we observed greater increases in E₂ during presumably mid-follicular phase sessions (when expected POC > 0); that this was not replicated when data were assessed cross-sectionally may be attributable to the reduced statistical power in cross-sectional versus within-subjects designs (Gangestad et al., 2016). Heightened E₂ responsivity during the mid-follicular phase accords with the hypothesis that mating effort should increase with conception probability. Though in most mammal species mating does not occur outside of estrus and its concomitant estrogenic milieu (Nelson, 2011), this is not the case in most humans and non-human primates (Wallen, 1990), a phenomenon referred to as “extended sexuality” (Thornhill and Gangestad, 2008). As sexual desire, arousal, and behavior are not restricted to fertile portions of the cycle, it is perhaps unsurprising that a hormonal modulator of sexuality that is present across the full cycle would change as a function of exposure to sexual stimuli. A robust body of literature suggests that E₂ interacts with dopaminergic reward processing systems in humans (Dreher et al., 2007) and rodents (Jackson et al., 2006; Lynch et al., 2001),

suggesting that rapid increases in E₂ in response to sexual stimuli may function to enhance the reward value assigned to that stimuli. It is also possible that E₂ increases vaginal lubrication during sexual arousal, though the positive effect of E₂ has been assessed only through studies of chronic, rather than acute, administration (Laan et al., 2001). As sexual neuroendocrinology has largely focused on the long-term effects of steroid hormones, driven by genomic mechanisms (see Balthazart et al., 2018), future work elucidating the rapid, non-genomic effects of hormones is needed.

This line of reasoning, however, does not extend to our findings of T responsivity to AVSS, which was positive during the follicular phase and unchanged during the luteal phase when analyzed using within-subjects data. T has been suggested to be an important modulator of women's sexual desire and behavior (Davis, 2000; Davis and Tran, 2001; Guay and Davis, 2002). However, most studies assessing T and sexuality have analyzed associations at relatively long time scales, for example, after several months of chronic T administration (see Cappelletti and Wallen, 2016 for review), or after sampling daily T across two months (Roney and Simmons, 2013), though some work has assessed the effects of T administration on sexuality after several hours (Tuiten et al., 2000). There is a relative paucity of studies, however, on acute effects of T (as well as E₂ and P) on women's sexuality, and results from the present study suggest that such relationships require further examination. For example, it is possible that variables such as age, life history events, health, and diet contribute to chronic T levels, which influence more trait-like levels of sexual motivation. By contrast, sexual stimuli may elicit acute T increases, which temporarily elevate sexual desire and focus attention on these stimuli. That these increases were evident only during the follicular phase suggests that they may function to direct mating effort when conception is possible. More work is required to elucidate the effects of T on various time-scales, and our findings suggest that future studies looking at acute hormone-sexuality relationships should account for cycle phase at testing.

Though there is some evidence that P exerts inhibitory effects on sexual desire in humans (Jones et al., 2018; Roney and Simmons, 2013) and nonhuman primates (Wallen et al., 1984), we did not find evidence that P responds to AVSS, nor did this change as a function of cycle phase in within-subjects and cross-sectional analyses. The single study that assessed changes in P as a function of masturbation-induced orgasm did not find significant changes in P (Exton et al., 1999), consistent with our results. Despite over 30 years since initial reports of P's inhibitory role in female sexuality, it has largely been neglected in studies of human behavioral endocrinology. It is possible that P influences sexual desire, but sexual stimuli do not influence P. As such, future studies on women's sexual responses (and particularly studies of AVSS) ought to consider changes in P in addition to changes in E₂ and T to better clarify the individual contributions of each hormone (or lack thereof) in modulating sexual desire and arousal.

Though previous work has suggested order effects on sexual responses and visual attention to sexual stimuli (Slob et al., 1991, 1996; Suschinsky et al., 2014; Wallen and Rupp, 2010; but see Bossio et al., 2014), no previous studies systematically investigated order effects on hormonal responses to AVSS. In the first empirical test of this, we did not observe significant effects of testing order. We did, however, observe interactions between session order and cycle phase at testing. Women who were first tested during the luteal phase exhibited significant increases in P and T during sessions in the follicular phase, but not the luteal phase, whereas women who were tested first during the follicular phase did not exhibit changes in P or T across either test session. Though E₂ concentrations at initial testing sessions have been hypothesized as the proximate mechanism driving observed order effects (Wallen and Rupp, 2010), within-subjects models evaluating the effect of E₂ concentrations at the initial testing session did not provide support for this. Future work examining order effects, as well as work examining the physiological encoding of sexual stimuli more broadly, should therefore aim to elucidate the mechanisms that contribute to

observed order effects.

Using the same dataset as the current study, [Suschinsky et al. \(2014\)](#) observed order effects on genital measures for a subset of experimental stimuli, such that only AVSS portraying acts that could lead to conception elicited greater genital responses among women who were tested first during the follicular phase. Also in the same dataset, however, [Bossio et al. \(2014\)](#) did not observe an order effect on genital or subjective responses to AVSS varying in sexual valence, attributing this to the intensity of stimuli. As some of the AVSS categories in the present study were greater in intensity than those used previous studies (e.g., still images were used in [Wallen and Rupp, 2010](#)), it is possible that a ‘ceiling effect’ on hormonal responsivity was induced based on the potency of the AVSS displayed, thereby eliminating any order effects. To evaluate this explanation, future work should assess order effects on hormonal responsivity to AVSS by utilizing images and videos varying in the acts portrayed and in intensity.

Interpretations of putative order effects in the present study should be made with caution. Women tested first in the follicular phase exhibited significantly greater P levels (when averaging pre- and post-stimuli levels) during the luteal phase session as compared to women tested first in the luteal phase; women tested first in the follicular phase also exhibited greater T during the follicular phase as compared to women tested first in the luteal phase, though this is driven primarily by low pre-stimuli T. As P is highly variable across the luteal phase, it is possible that luteal phase sessions for women tested first in the follicular phase more frequently coincided with days of peak P production; however, T does not exhibit fluctuations of similar magnitude across the cycle. The low T levels during the follicular phase in women tested first during the luteal phase cannot be attributed to T pulsatility, as two saliva samples were obtained 30 min apart and averaged, presumably minimizing the effect of pulsatile secretion on hormone measures. Future work may benefit from sampling women multiple times per cycle phase to reduce potential differences in the timing of scheduled sessions.

Using a within-subjects design, [Bossio et al. \(2014\)](#) and [Suschinsky et al. \(2014\)](#) observed no effects of cycle phase on genital and subjective measures of arousal to AVSS. The fact that cycle phase influenced hormonal responsivity in the same sample, in a manner consistent with predictions drawn from evolutionary biology, thus highlights the relevance of endocrine responses to human mating. In addition, the variability in relationships among genital, subjective, and hormonal responses suggests that these measures capture different aspects of arousal, and that hormonal responsivity should therefore be measured alongside genital and subjective responses to more fully characterize sexual arousal. Whereas correlational studies have made it clear that baseline hormone measures are associated with baseline measures of sexual desire and arousal ([Roney and Simmons, 2013](#)), the interpretation of short-term changes of hormones is less clear, and relatively few studies have assessed whether they are associated with short-term changes in desire or arousal. Incorporation of fine-tuned physiological, neuroimaging, and self-report data should be utilized to elucidate the rapid, presumably non-genomic effects of hormones on sexual psychology and physiology.

4.1. Limitations

The use of counting methods in cycle phase studies, as we used initially to schedule laboratory visits, has been criticized as imprecise ([Blake et al., 2016](#); [Gangestad et al., 2016](#); [Gonzales and Ferrer, 2016](#)). Though we used progesterone concentrations to validate cycle phase, denser sampling schedules or luteinizing hormone tests might increase precision. Any imprecision in our estimates of cycle phase may therefore have added noise, causing us to underestimate the magnitude of cycle effects on E_2 and T responsivity.

As discussed in [Suschinsky et al. \(2014\)](#), not all women in the present sample were tested within the same menstrual cycle. It has been

suggested that adaptive menstrual cycle shifts in women's sexuality occur both within-individuals across a single cycle, as well as within-individuals across different cycles ([Roney, 2015](#); [Roney and Simmons, 2013](#)). Future work should sample women multiple times across a single menstrual cycle, as well as across several menstrual cycles to elucidate both inter- and intra-cycle shifts in sexuality.

Larger samples than ours would better elucidate the inter-individual variability in hormone responses to sexual stimuli and would confer greater statistical power to detect subtle effects. Although our sample size could have contributed to the lack of statistically significant effect of order, significant order effects have been observed with samples of similar size ([Slob et al., 1991, 1996](#) with $n = 12$ and 20 , respectively; [Wallen and Rupp, 2010](#) with $n = 15$). Additionally, we were able to detect interactions between session order and cycle phase at testing with moderate effect sizes. Recent power analyses have suggested that, depending on correlations across phases and validity of cycle phase measures, sample sizes similar to that of the present study may be sufficient to detect within-subject menstrual cycle shifts ([Gangestad et al., 2016](#)). However, such samples may be insufficient to detect menstrual cycle shifts in cross-sectional designs, which may in part explain the lack of significant effect of current menstrual cycle phase on hormonal responsivity when data from the present study were analyzed in a cross-sectional manner.

Finally, it is unknown whether the observed hormonal responsivity can be attributable to AVSS exposure alone. As genital arousal was measured during sessions using a vaginal probe, it is possible that the combination of the insertion of a vaginal probe and exposure to AVSS simulated the act of intercourse for participants. Hormone changes could then be interpreted as functional responses to what is physiologically perceived as actual sexual behavior, rather than to AVSS alone. This is unlikely to fully account for the observed results, as prior studies without vaginal probes have observed hormonal changes when participants were told to imagine sexual scenarios ([Goldey and van Anders, 2011](#)). Nonetheless, the lack of a non-probe control group should be considered when evaluating our findings. Similarly, as all participants viewed the same sexual stimuli and there was no non-sexual stimuli control group, hormonal responsivity to sexual versus non-sexual stimuli cannot be deduced from the present study. While these two limitations do not pertain to our findings on whether hormone responsivity differs as a function of cycle phase or session order, they should be considered when evaluating the magnitude of hormone changes as a result of AVSS exposure.

4.2. Conclusions and future directions

The present study contributes to our understanding of menstrual cycle shifts in women's sexuality by elucidating patterns of hormonal responsivity to sexual stimuli; namely, that E_2 increases in response to AVSS, regardless of cycle phase, whereas T increases specifically in the mid-follicular, or fertile, part of the menstrual cycle. Further, the direction of these effects was similar across participants, suggesting that these patterns may be more interindividually robust than has been previously suggested ([Garcia et al., 2015](#)). These results also suggest several avenues for future work. It has been shown that AVSS depicting different sexual acts and activities that differ in their sexual valence are associated with varying levels of genital and subjective arousal ([Bossio et al., 2014](#); [Chivers et al., 2007](#); [Goldey and van Anders, 2016](#); [Suschinsky et al., 2014](#)), as well as amygdala reactivity ([Hamann et al., 2004](#)), but it is unknown whether exposure to different categories of AVSS differentially affect hormone concentrations. Thus, future work should aim to characterize patterns of hormonal reactivity to different types of AVSS. Whereas most work assessing the effect of hormones on AVSS has been correlational, experimental administration of exogenous hormones would demonstrate causal relationships between hormones and responses to AVSS. Experimental data may further differentiate between the functional effects of baseline hormone concentrations

versus *changes* in hormone concentrations in modulating women's sexual arousal and desire. More generally, as variability between genital, subjective, neural, and now, hormonal, measures of arousal have been documented, it is imperative to understand the significance of each of these disparate responses. Rather than measuring and collecting responses in isolation, future studies should aim to collect multimodal responses to AVSS to best assess how women's sexuality is affected by the interplay between psychological and physiological variables.

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Declarations of interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.yhbeh.2018.05.023>.

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