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Associations between oxytocin and cortisol reactivity and recovery in response to psychological stress and sexual arousal☆

Jenna Alley[a,*], Lisa M. Diamond[a], David L. Lipschitz[a], Karen Grewen[b]

[a] University of Utah, United States
[b] Karen Grewen, University of North Carolina, Chapel Hill, United States

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ABSTRACT

Previous research suggests a dynamic regulatory relationship between oxytocin and cortisol, but the specific nature of this relationship and its context-specificity have not been fully specified. In the present study, we repeatedly assessed both salivary oxytocin and salivary cortisol during two experimental sessions (one inducing sexual arousal and one inducing psychological stress), conducted two weeks apart with the same group of 63 female participants. Baseline cortisol and baseline oxytocin were significantly correlated in both sessions. Cortisol levels showed significantly different patterns of change during the stress assessment than during the sexual arousal assessment, but oxytocin showed similar patterns of change across both assessments. Greater cortisol stress reactivity predicted higher oxytocin levels immediately after the stressor, but a different pattern emerged during the arousal assessment: Greater oxytocin arousal reactivity predicted attenuated post-arousal reductions in cortisol. For both cortisol and oxytocin, individual differences in women's reactivity to sexual arousal did not predict their reactivity to psychological stress. These findings contribute new insights regarding associations between cortisol and oxytocin reactivity and recovery in different psychological contexts.

Previous research suggests a dynamic regulatory relationship between oxytocin and cortisol, but the specific nature of this relationship has not been fully specified. Both oxytocin and cortisol have been observed to increase in response to psychological stress, and some studies suggest that stress-induced increases in oxytocin function to buffer stress-induced release of cortisol (Ditzen et al., 2009; Neumann et al., 2000; Quirin et al., 2011). However, other research has found that increases in cortisol lead to increases in oxytocin (Tops et al., 2007a). Overall, the timescale and ordering of the oxytocin/cortisol relationship remains unclear. The degree to which this relationship is context-specific also requires investigation. Previous studies have examined associations between cortisol and oxytocin by way of intranasal administration (Cardoso et al., 2013), or during stress or conflict tasks (Heinrichs et al., 2003; Ditzen et al., 2009). However little is known about the relationship between these two hormones during non-stressful psychological contexts. Sexual arousal may be a particularly important context for examining this relationship, given that oxytocin typically *increases* in response to sexual arousal (as it does during stress), whereas cortisol typically declines during sexual arousal. Hence, the interplay between oxytocin and cortisol may take a different form during stress versus sexual arousal, and investigating this possibility can enhance our understanding of each hormone.

1. Oxytocin and cortisol during stress

Both cortisol and oxytocin have been observed to increase during stress. Psychological stress triggers the hypothalamic pituitary axis (HPA) to release adrenocorticotropic hormone, which subsequently signals the adrenal cortex to release cortisol (reviewed in Dickerson and Kemeny, 2004). Oxytocin release is regulated, in part, by some of the same hypothalamic nuclei and hippocampal projections which regulate HPA axis activity (Herman et al., 2002; Risold and Swanson, 1996), and although it has been most widely studied for its role in social and sexual behavior, oxytocin is also implicated in the human stress response (Carter and Lightman, 1987; Cox et al., 2015; de Jong et al., 2015; Kumsta and Heinrichs, 2013). Stress-induced increases in oxytocin are thought to regulate HPA axis activity and potentially to motivate pro-social behaviors such as support-seeking (Carter and Lightman, 1987; Cox et al., 2015; de Jong et al., 2015; Kumsta and Heinrichs, 2013; Olff et al., 2013; Seltzer et al., 2014; Taylor et al., 2006).

A number of studies suggest that increases in oxytocin are related to decreases in cortisol (Ditzen et al., 2009; Heinrichs et al., 2003;

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* Corresponding author at: Department of Psychology, University of Utah, 380 South 1530 East, Room 502, Salt Lake City, UT 84112-0251, United States.
E-mail address: jenna.alley@psych.utah.edu (J. Alley).

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Pierrehumbert et al., 2010), but other studies have failed to find an inverse relationship (Altemus et al., 2001; McQuaid et al., 2016; Taylor et al., 2006) or have found positive associations between oxytocin and cortisol during stress (de Jong et al., 2015; Engert et al., 2016; Yuen et al., 2014). One possible explanation for these conflicting findings may concern the time scale of the oxytocin/cortisol relationship. One notable study found that stress reactivity in oxytocin was related to lower levels of cortisol only *after* the task had been completed, during recovery (Engert et al., 2016). Thus, it is important to investigate how stress-related changes in one hormone relate to subsequent recovery in the other across a sufficiently lengthy time frame capable of capturing slower-acting recovery effects.

It is also important to examine bidirectional effects. Although some studies have observed changes in cortisol after intranasal administration of oxytocin (Cardoso et al., 2013), other studies have failed to replicate these changes (Tops et al., 2007b; Wirth et al., 2015, and some have found *increases* in oxytocin after orally administering cortisol (Kalin et al., 1985). Hence, in order to fully characterize the relationship between oxytocin and cortisol during stress, it is important to examine how oxytocin relates to current and subsequent levels of cortisol *and* likewise to examine how cortisol relates to current and subsequent levels of oxytocin.

2. Oxytocin and cortisol during sexual arousal

Previous research on the potential regulatory relationship between cortisol and oxytocin has focused largely on psychological stress, yet the relationship between these two hormones may operate differently under different psychological contexts, as argued by Cardoso, Kingdon, and Ellenbogen on the basic of a meta-analytic review (2014). Sexual arousal offers a particularly intriguing context for investigating this possibility, given that oxytocin and cortisol appear to play notably different roles during sexual arousal. Oxytocin typically increases during sexual arousal, and is thought to play a key role in sexual arousal and orgasm (Blaicher et al., 1999; Carmichael et al., 1994; de Jong et al., 2015; Veening et al., 2015). In contrast, cortisol typically declines during sexual arousal, although the magnitude of decline varies across studies using different sexual stimuli and different experimental protocols (Exton et al., 2000; Hamilton et al., 2008; Heiman et al., 1991), and elevated cortisol levels are thought to hinder sexual arousal and functioning (Hamilton et al., 2008).

Hence, whereas cortisol and oxytocin show *similar* patterns of change in response to stress (with both showing increases), they show *divergent* patterns of change in response to sexual arousal (with oxytocin increasing and cortisol decreasing). This raises the possibility that the cortisol-regulating function of oxytocin may operate differently during stress than during sexual arousal. Given that oral administration of cortisol has been shown to produce subsequent increases in oxytocin (in a neutral context), it is not clear how changes in oxytocin and cortisol might relate to one another over time during and after an experimental induction of sexual arousal, and our study will systematically investigate this question.

Another unanswered question concerns the stability of interindividual patterns of reactivity and recovery across different psychological states. In other words, do women with the highest levels of oxytocin reactivity to sexual arousal *also* show the highest oxytocin reactivity to psychological stress? Extensive previous research has documented individual differences in both oxytocin and cortisol reactivity to a variety of laboratory tasks (Bruce et al., 2002; Kirschbaum et al., 1992, 1998; McQuaid et al., 2016; Pierrehumbert et al., 2010; Quirin et al., 2008; Schlotz et al., 2011; Spinrad et al., 2009; Tops et al., 2007a, b; Yuen et al., 2014). However, no existing research has directly tested whether these individual differences in reactivity are consistent across different types of tasks (such as stress tasks vs. arousal tasks). Testing this possibility is important for understanding the context-specificity of response profiles for each hormone.

3. The current study

We assessed salivary oxytocin and salivary cortisol during two different experimental sessions, conducted two weeks apart, using the same participants. All experimental procedures were identical for the two sessions, except that the first session involved the induction of sexual arousal and the second session involved the induction of psychological stress. This design allows us to examine how reactivity in one hormone relates to recovery in the other, and how patterns of change in each hormone differ across tasks. Our specific predictions were as follows:

Hypothesis 1. We expected to find correlations between baseline oxytocin and baseline cortisol prior to both tasks, based on previous meta-analytic findings (Brown et al., 2016).

Hypothesis 2. Based on previous research showing that both psychological stress and the administration of cortisol can provoke increases in oxytocin, we predicted that high levels of stress-reactivity in cortisol would predict elevated oxytocin levels during the first post-stress recovery.

Hypothesis 3. Based on research suggesting that stress-induced release of oxytocin functions to regulate cortisol levels, we predicted that higher levels of oxytocin during the stressor and or during the immediate post-stress recovery period would predict lower levels of cortisol during the second post-stress recovery.

Exploratory Question 1. We planned to explore Hypotheses 2 and 3 for the sexual arousal assessment, but given the lack of previous research on the interplay between cortisol and oxytocin during sexual arousal, we consider these tests exploratory.

Exploratory Question 2. We planned exploratory tests of whether the magnitude and direction of cortisol and oxytocin reactivity and recovery differed significantly for the arousal assessment versus the stress assessment, based on patterns observed in previous research. We also planned to examine whether the moderating effects outlined in Hypothesis 2 and 3 differed significantly for the arousal assessment versus the stress assessment.

Exploratory Question 2. Given previous research suggesting stable interindividual differences in task reactivity for both oxytocin and cortisol, we planned to explore whether a woman's reactivity to the arousal task predicted her reactivity to the stress task, for each hormone separately.

4. Methods

4.1. Participants

Participants were 63 women between the ages of 20 and 35 (mean age = 27.2, *SD* = 4.7, mean BMI = 27.5, *SD* = 6.1). In all, 35% (n = 22) of the women identified as heterosexual, 44% (n = 28) as bisexual, and 21% (n = 13) as lesbian. In all,84.1% (n = 53) of the women were White, 12.7% (n = 7) identified as Latina, and 3.2% (n = 2) identified as Asian/Pacific Islander, 2.2%.In all, 35% (n = 22) considered themselves religious, and of these participants, approximately one third affiliated with the Church of Jesus Christ of Latter Day Saints, one third described their religion as "other," and the remaining third affiliated with Catholicism, Protestantism, or Judaism. Regarding education, less than 5% (n = 3) had only a high school degree, 33% (n = 21) had completed some college, 39.7% (n = 25) had completed college or an associates degree, and 22.2% (n = 14) had some graduate training. Regarding income, 39.7% percent (n = 25) had a household income less than $25,000, 34.9% (n = 22) had a household income between $25,000 and $50,000, and 23.8% (n = 15) had a household income greater than $50,000. 79.4% (n = 50) had a regular romantic/

sexual partner. Participants were recruited through Facebook ads that described the study as an investigation of sexuality and stress hormones, and was approved by the University of Utah Institutional Review Board. The Facebook ads were targeted to all women living in the immediate Salt Lake City area, but were not targeted to any specific interest groups. Participants were excluded from participation if they were using prescribed cardiovascular or endocrinological medications, anti-depressants, or if they were currently pregnant. Participants were compensated $80 for their participation.

4.2. Procedures

Women completed two laboratory sessions, two weeks apart. They were aware that the first session would involve an experimental induction of sexual arousal, and that the second session would involve an experimental induction of stress. We elected not to randomize the ordering of the sessions because it was unclear whether there might be carryover from one assessment to the other, even after a two-week interval. Hence, we held the ordering constant to equalize any such effects. To control for variability in oxytocin levels that occurs throughout the menstrual cycle (Salonia et al., 2005), we scheduled the first experimental session to occur 10 days after the start of their most recent period (i.e., the follicular phase). Hence, the second session occurred during the luteal phase. Sessions were always scheduled between 3 and 7 pm to control for diurnal variation in cortisol, and women were instructed to refrain from smoking, eating, or drinking caffeinated beverages for at least two hours before the laboratory session (confirmed upon their arrival at the laboratory). The specific ordering and timing of laboratory activities, including the timing of salivary hormone assessments, is displayed in Fig. 1 (the timing was exactly the same for the stress assessment and the arousal assessment). During session one, they underwent informed consent and completed a detailed questionnaire including questions pertaining to their sexual behavior and history (Diamond et al., 2019). The questionnaire took 15 min. After completing their questionnaires, women were escorted to a private room to participate in the arousal induction task. Set-up and instructions took approximately 5 min, and then they underwent a 15 min baseline period. During the first 5 min, they sat quietly. During the second 5 min, they rated their liking of a set of landscape photographs, in order to engage their attention in a restful pleasant task (Jennings et al., 1992). During the remainder of the baseline period, they paced their breathing slowly in response to a timer (4 s of inhalation, 4 s of exhalation). We then collected the first cortisol and oxytocin samples (more detail provided below). We used a paced breathing task because we were also collecting data on tonic respiratory-sinus arrhythmia (reported elsewhere), which is sensitive to the pace of breathing. This task typically produces enhanced feelings of relaxation, given that it typically slows respondents' rate of breathing.

Next, the sexual arousal task began. Participants listened (through headphones) to 8 different stories describing, from a first-person perspective, the unfolding of an interaction with a woman or a man. Half of the stories were sexually explicit, including detailed descriptions of genital and non-genital sexual activity. The other half of the stories were neutral, describing non-erotic interactions with women and men. There was a one-minute recovery period between each story, so that the total amount of time listening to the stories was 20 min. These stories were designed and validated in previous research assessing women's subjective and genital responses to auditory stimuli (Chivers and Timmers, 2012) and we used the same stories (and the same recordings, all of which used the same female voice) as this previous research. While listening to each story, women provided continuous ratings of their sexual arousal. After the arousal session, we collected a second sample of cortisol and oxytocin.

The first recovery session involved an 11-minute guided relaxation and mindfulness task. Women listed through headphones to a series of instructions asking them to focus on their breathing and to focus on feeling each part of their body as it made contact with the couch, the floor, etc. They were instructed to keep their attention on their breathing, and that if their attention wandered, they should gently bring their focus back to their breath and to the present moment. This task lasted 11 min, and we collected their salivary samples immediately after they completed this task. Next, they spent 15 min filling out additional questionnaires. We collected the final saliva samples at the end of the 15 min period. Hence, each of the four saliva samples were collected 15–20 minutes apart.

The second laboratory assessment occurred two weeks later. The baseline procedure was identical to the first session. Next, women underwent a modified version of the Trier Social Stress Test (Kirschbaum et al., 1993). First, they spent 5 min preparing and 5 min performing a speech to a male research associate (who they had not seen before and who was dressed in a white lab coat), in which they had to assume the role of a job applicant aimed at convincing the manager of their qualifications. For logistical reasons, we elected to have participants deliver their speech to a single individual rather than a committee, but in order to enhance the participants' experience of evaluation, participants were informed that their performance would be evaluated by two professors in the department, and hence they were instructed to speak directly into a video camera which was set up in front of them (which was not running). The lab-coated experimenter stood next to the camera and took detailed notes during the speech. If the participant slowed down or stopped, the experimenter said "*You must speak for the full* 5 min. *Please continue*." After the speech concluded, the experimenter asked the participant to discuss "*any information or photos on your Facebook page, or on the Facebook pages of your friends and associates, which could potentially be a source of embarrassment, or which present you in a less-than-professional manner*" and to describe what course of action they planned to take in response to this material. We added the Facebook-related questions after informal consultation with undergraduates about the types of interview questions that they considered most realistic and stressful and worrisome. Immediately after the speech task, the experimenter administered a math task. The math task consisted of participants mentally subtracting 17 from 2023 until they reached zero.

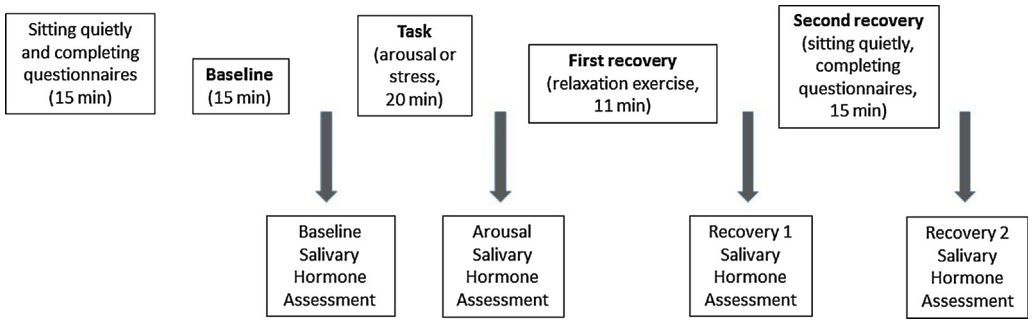

Fig. 1. Timing and ordering of laboratory activities for both the sexual arousal assessment ad the stress assessment.

Three different times during the task, the experimenter interrupted the participant to tell them that they needed to go faster. During three additional times, the experimenter forced the participant to go back to a previous number and start again. The task lasted approximately 5 min, so that the total time for the stress task (including instructions) was approximately 20 min. One minute after the stress task was concluded, saliva samples for oxytocin and cortisol were collected. The procedures for the two subsequent recovery sessions were identical to the sexual arousal session. Hence, as with the sexual arousal assessment, the four saliva samples were taken 15–20 min apart.

Salivary cortisol samples were taken using Salivettes (Sarstedt, Germany), consisting of a plastic tube with a cotton insert. The participant was instructed to lightly chew on the insert to thoroughly soak it with their saliva. All samples were kept frozen at -25 C until being shipped on dry ice to be assayed by the laboratory of Dr. Kirschbaum at the Technical University of Dresden, which uses a time-resolved immunoassay with fluorometric end point detection (see Dressendorfer et al., 1992) with intra- and inter-assay precision of 3.0% and 4.2%. In all, 7% of cortisol samples were either missing or could not be assayed, and follow-up analyses detected no systematic patterns of missing samples (i.e., individuals who had missing samples did not differ from those with complete samples on age, sexual-minority status, BMI, or any demographic characteristics).

For the collection of salivary oxytocin data, participants collected approximately 1 mL of saliva in their mouths and released the saliva into a glass centrifuge tube using the "passive drool" technique. Samples were immediately frozen at -25 C until shipped on dry ice to the University of North Carolina. The OT enzyme-immunoabsorbance (EIA) method used to assay salivary OT is identical to that reported previously (Holt-Lunstad et al., 2008). Salivary OT levels were measured using the OT EIA (Enzo Life Sciences, Farmingdale, NY). After correcting for concentration produced by extraction, the lower limit of sensitivity was 1.5 pg/mL, with intra- and inter-assay variations of 4.8% and 8%.

4.3. Measures

Participants completed additional questionnaire measures assessing sexual history, mental health, and state affect, reported elsewhere. During the sexual arousal task, participants used rating dials to indicate their degree of sexual arousal (on a 1–10 scale). Before and after the stress task, women completed a 10-item state affect measure (a reduced version of the PANAS, Watson, Clark, & Tellegan) to indicate, on a 1–5 Likert scale, their degree of stress and anxiety versus comfort and calm.

4.4. Analytical plan

The multivariate module of HLM, known as HMLM (Bryk & Raudenbush, 1992), was used for all data analysis. To analyze both the arousal assessment and the stress assessment simultaneously while controlling for within-person dependency, we used a parallel process model (Raudenbush et al., 1995). This technique is designed for multilevel data structures in which observations at one level of analysis (in this case, the sequential episodes of baseline, task, first recovery, and second recovery) are nested within higher levels of analysis (type of assessment – arousal versus stress), which are then nested within persons (since each participant completed both assessments). This model begins with a Level 1 equation that predicts the outcome variable (cortisol or oxytocin) from two dummy codes, one for the arousal assessment and one for the stress assessment. The example below uses cortisol as the outcome, but the same model structure was used for oxytocin.

$$\text{Cortisol}_{\text{episode i, participant j, assessment g}} = \pi_{1ja}\,(\text{Arousal}) + \pi_{2js}\,(\text{Stress}) + e_{ijg}$$

Note that this equation does not contain an intercept, so that the coefficients for the stress/arousal dummy codes, π_{1ja} and π_{2js}, actually represent the true scores for participant j's cortisol during episode i of either the arousal assessment or the stress assessment. These true scores become the dependent variables for subsequent levels of analysis. This particular type of multivariate multilevel model uses an unrestricted level 1 variance structure, which allows for a unique error variance for the stress assessment and the arousal assessment and a covariance between them. This structure is analogous to computing two multilevel models and allowing the DV's to be correlated at a constant amount over time. The model fit is parallel to a standard HLM model in which the error variance is fixed over time.

Level 2 of the model focuses on episode-to-episode changes within each experimental assessment. At this level, a regression equation is calculated for each assessment, predicting the DV (cortisol during episode i for participant j) from dummy codes modeling episode-to-episode changes. To assess *reactivity* (i.e., changes from baseline to task), we used the baseline episode as the base category. Hence, the Level 2 model takes the following form:

π_1 Cortisol during **Arousal** Assessment $_{\text{episode i, individual j}} = \beta_{10\ ij} + \beta_{11}$ Task Dummy $\text{Code}_{ij} + \beta_{12}$First Recovery Dummy $\text{Code}_{ij} + \beta_{13}$Second Recovery Dummy Code_{ij}

π_2 Cortisol during **Stress** Assessment $_{\text{episode i, individual j}} = \beta_{20\ ij} + \beta_{21}$ Task Dummy $\text{Code}_{ij} + \beta_{22}$First Recovery Dummy $\text{Code}_{ij} + \beta_{23}$Second Recovery Dummy Code_{ij}

The β_{10} and β_{20} coefficients are the intercepts of the model, representing the predicted value of the DV (in this example, cortisol) during the baseline episode for each assessment (i.e., when all of the dummy codes representing the other episodes of the experiment are zero). The slope terms β_{11} (for the arousal assessment) and β_{21} (for the stress assessment) represent the baseline-to-task *change* in cortisol.

Level 3 tests whether an individual's baseline-to-task changes in one hormone are moderated by baseline and task levels of the other hormone (during the same experimental assessment). Hence, for the reactivity model with cortisol as the DV, the coefficient β_{11} (representing cortisol reactivity for the arousal task) is predicted at Level 3 by baseline and task levels of oxytocin for the arousal task (along with participant's age and BMI) and the coefficient β_{21} (representing cortisol reactivity for the stress task) is predicted at Level 3 by baseline and task levels of oxytocin for the stress task (along with participant's age and BMI). Not that we include both baseline and task levels of oxytocin in these models as predictors, rather than change scores (task minus baseline). We adopted this "residualized" approach for modeling change to allow us to fully characterize potential relationships between baseline levels of one hormone and epoch-to-epoch changes in the other, and also to account for the fact that baseline levels may constrain the degree of potential task-related change.

To calculate recovery, the task level is used as baseline, to estimate changes from task to the first recovery and from task to the second recovery. Hence, for the recovery model with cortisol as the DV, the coefficients β_{12} (change in cortisol from the arousal task to the first recovery) and β_{13} (change in cortisol from the arousal task to the second recovery) are predicted at Level 3 by baseline and task levels of oxytocin for the arousal assessment (along with participant's age and BMI). Correspondingly, the coefficients β_{22} (change in cortisol from the stress task to the first recovery) and β_{23} (change in cortisol from the stress task to the second recovery) are predicted at Level 3 by baseline and task levels of oxytocin for the stress assessment (along with participant's age and BMI).

A unique advantage of this parallel process multilevel modeling approach is that it permits us to directly test whether specific coefficients in the model *differ between the stress and arousal assessments*, controlling for within-person dependency. This allows us to explore questions such as the following: Is the magnitude of oxytocin or cortisol

reactivity different for the arousal assessment versus the stress assessment? Is the *association* between reactivity for one hormone and recovery for the other different for the arousal assessment versus the stress assessment? These contrasts take the form of one-degree-of-freedom chi-square tests. Because we had no a priori hypotheses for these tests, we use a Bonferonni correction for multiple tests.

5. Results

We used an alpha level of .05 for all significance tests. Following established guidelines (Smyth et al., 1998), data points that were more than 4 standard deviations from the mean were discarded (n = 5). Follow-up analyses showed that this did not change the results. In accordance with norms for handling positively skewed data, raw hormone values were transformed using the natural log procedure. Given that most existing research on oxytocin uses plasma levels, it also bears noting that an increasing body of research supports the validity of salivary measures of oxytocin (Grewen et al., 2010; Holt-Lunstad et al., 2011) and their correlation with plasma changes in oxytocin, especially in response to experimental tasks (Valstad et al., 2017). Means, standard deviations, and ranges for raw hormone values at each of the measurement episodes for each assessment are displayed in Table 1. Correlations between baseline measures, baseline-to-task changes, and task-to-final recovery changes (using log transformed values) are displayed in Table 2.

We conducted preliminary analyses using the self-report measures to confirm that participants believed that the sexual arousal task succeeded in inducing sexual arousal and that the stress task succeeded in inducing psychological stress. Given that we included women of diverse sexual orientations and that we included erotic stories involving both genders, we averaged women's self-reported continuous ratings of sexual arousal during all of the stories featuring their self-reported *preferred* gender (as they had indicated on the questionnaires that they completed prior to the study –i.e., male-centered stories for self-described heterosexual participants, female-centered stories for self-described lesbian participants, and both male and female stories for self-described bisexual participants). We averaged responses to the stranger narratives and the relationship narratives in order to provide the most reliable index of women's self-reported arousal. The arousal task produced significant increases in arousal (relative to baseline), $t = 13.0$, $p < .001$. The stress task produced significant increases in state anxiety (relative to baseline), $t = 14.7$, $p < .001$.

Although previous research using these narratives has found that among all women (those with and without romantic partners), the narratives involving strangers were rated as most arousing (Chivers and Timmers, 2012) we conducted several ancillary analyses to check for diffrences between women with and without romantic partners. We detected no differences between partnered and unpartnered women in self-reported arousal (mean difference = .07, $t = .22$, $p = .83$) or oxytocin levels in response to the arousal task (mean difference = .18, $t = .09$, $p = .94$).

Table 1
Descriptive Statistics for Study Variables.

	Arousal Assessment		Stress Assessment	
	M (SD)	Range	M (SD)	Range
Baseline Cortisol	8.5 (5.2)	2.0, 32.9	9.0 (5.6)	1.0, 24.1
Task Cortisol	6.8 (4.1)	1.0, 32.2	9.6 (5.5)	1.3, 24.5
First Recovery Cortisol	6.4 (4.3)	1.4, 20.4	9.3 (4.6)	3.2, 23.1
Second Recovery Cortisol	6.2 (5.0)	1.0, 32.3	8.5 (4.7)	2.1, 23.5
Baseline Oxytocin	10.6 (6.9)	1.0, 32.4	9.2 (5.5)	2.3, 24.3
Task Oxytocin	11.0 (6.2)	1.4, 27.9	10.4 (4.9)	1.6, 38.3
First Recovery Oxytocin	10.1 (6.3)	1.4, 29.8	9.1 (6.2)	1.2, 24.0
Second Recovery Oxytocin	11.1 (6.2)	1.9, 23,6	9.3 (5.0)	1.2, 24.0

5.1. Overall patterns of reactivity and recovery

Figs. 2–5 display mean levels (with standard errors) for raw cortisol and oxytocin, during each epoch of the arousal assessment and the stress assessment. Figs. 2 displays variation in cortisol across the arousal versus the stress assessment. Fig. 3 displays variation in oxytocin levels across the arousal versus the stress assessment. Fig. 4 displays variation in *both* cortisol and oxytocin across the arousal assessment, and Fig. 5 displays variation in *both* oxytocin and oxytocin across the stress assessment (i.e., the same values are shown across the 4 figures, but they are organized different to facilitate within-hormone comparisons across tasks, in Figs. 2 and 3, and to facilitate within-task comparisons across hormones, in Figs. 4 and 5).

Arousal Assessment As evident in Figs. 2 and 4 and displayed in Table 3, participants showed a significant decline in cortisol from baseline to task for the arousal assessment, $b = -.23$, $p < .001$, followed by a significant decline from task to the first recovery ($b = -.38$, $p = .02$) and the second recovery ($b = -.43$, $p = .009$). As evident in Figs. 3 and 4 and displayed in Table 3, participants showed a significant increase in oxytocin from baseline to task for the arousal assessment ($b = .11$, $p = .02$), but no significant change from task to either the first recovery ($b = .02$, $p = .87$) or the second recovery ($b = .10$, $p = .46$).

*Stress Assessment*As evident in Figs. 2 and 5 and displayed in Table 3, participants showed no significant baseline-to-task change in cortisol during the stress assessment, $b = .08$, $p = .19$. There was a trend-level increase in cortisol from the stress task to the first recovery, $b = .36$, $p = .08$, but no significant change in cortisol from the stress task to the second recovery, $b = .26$, $p = .22$. As evident in Figs. 3 and 4 and displayed in Table 3, participants showed a significant increase in oxytocin from baseline to task for the stress assessment ($b = .10$, $p = .02$), no significant change from the stress task to the first recovery, $b = -.22$, $p = .12$, and a significant change from the task to the second recovery, $b = -.30$, p = .03.

5.2. Hypothesis tests

Hypothesis 1 predicted a significant association between baseline oxytocin and baseline cortisol across both assessments: This hypothesis was confirmed, $b_{arousal} = .33$, $p < .001$, $b_{stress} = .31$, $p < .001$.

Hypothesis 2 predicted that women with higher levels of cortisol stress reactivity would show elevated oxytocin levels during the first post-stress recovery. To test this hypothesis, we predicted the coefficient estimating change in oxytocin from the stress task to the first recovery from women's cortisol levels during the stress task (this model included women's baseline cortisol levels as a covariate, so that the task level represents reactivity). As shown in Table 3, this hypothesis was confirmed. To ensure that we fully characterized the oxytocin/cortisol relationship, we followed-up with ancillary tests for *concurrent* associations between cortisol and oxytocin stress reactivity, and found no such association, $b = -.08$, $p = .33$. There was also no current association between oxytocin and cortisol levels during the first recovery $b = -.09$, $p = .42$, but a trend level negative association during the second recovery, $b = -.15$, $p = .50$.Hypothesis 3 predicted that higher levels of oxytocin during the stressor or the first post-stress recovery would predict lower levels of cortisol during the second recovery. To test this hypothesis, we predicted the coefficient estimating change in cortisol from the stress task to the second recovery from women's oxytocin levels during the stress task and during the first recovery period. Hypothesis 3 was not confirmed: As shown in Table 3, higher oxytocin stress reactivity was associated with lower levels of cortisol during the second recovery, but this effect was only significant at the trend level, $b = -.31$, $p = .07$. Oxytocin levels during the first recovery episode were not related to cortisol levels during the second recovery, $b = -.01$, $p = .96$.

Table 2
Correlations Among Baseline, Baseline-to-Task Changes, and Task-to-Recovery Hormone Changes for the Sexual Arousal and Stress Assessments.

	1	2	3	4	5	6	7	8	9	10	11
1. Baseline Cortisol, Arousal Assessment											
2. Baseline Cortisol, Stress Assessment	.51***										
3. Baseline Oxytocin, Arousal Assessment	.40*	.47**									
4. Baseline Oxytocin, Stress Assessment	.45**	.40**	.77***								
5. Baseline-to-Task Change in Cortisol, Arousal Assessment	−.24	.03	.16	.18							
6. Baseline-to-Task Change in Cortisol, Stress Assessment	−.24	−.45**	−.17	−.15	.02						
7. Baseline-to-Task Change in Oxytocin, Arousal Assessment	−.26*	−.21	−.57**	−.38**	−.04	.10					
8. Baseline-to-Task Change in Oxytocin, Stress Assessment	−.17	.08	.04	−.18	.01	−.10	−.04				
9. Task-to-Recovery Change in Cortisol, Arousal Assessment	−.18	−.01	−.05	−.19	−.14	.04	.22	−.01			
10. Task-to- Recovery Change in Cortisol, Stress Assessment	.002	−.39**	−.12	−.05	.03	−.13	.10	−.13	−.02		
11. Task-to-Recovery Change in Oxytocin, Arousal Assessment	.15	.13	.08	.11	−.20	−.17	−.50	−.01	−.19	−.06	
12. Task-to- Recovery Change in Oxytocin, Stress Assessment	.28*	.11	.09.	.15	−.24	−.13	.01	−.34	−.03	−.13	.14

* $p < .05$.
** $p < .01$.
*** $p < .001$.

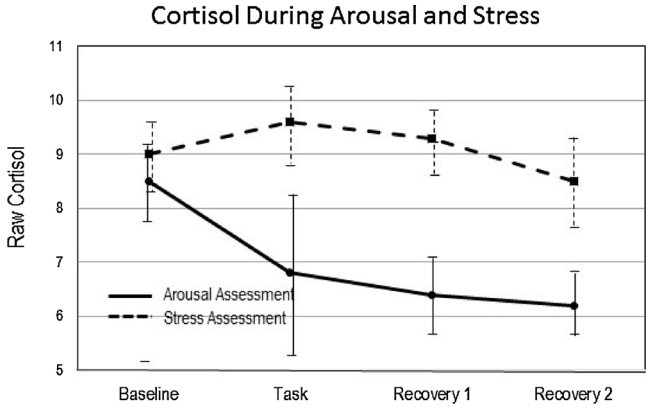

Fig. 2. Raw Cortisol Levels Throughout the Psychological Stress Assessment and the Sexual Arousal Assessment.

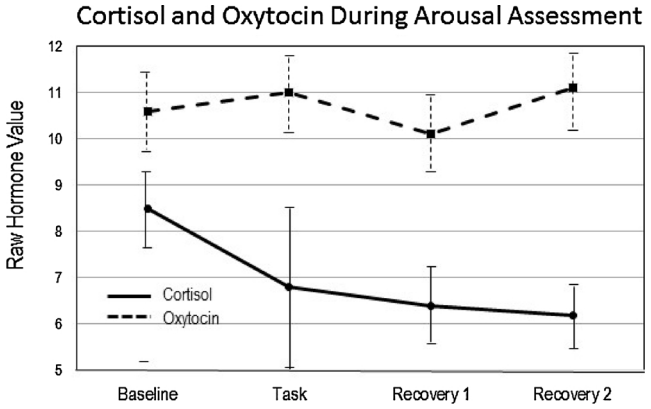

Fig. 4. Raw Cortisol and Raw Oxytocin Throughout the Sexual Arousal Assessment.

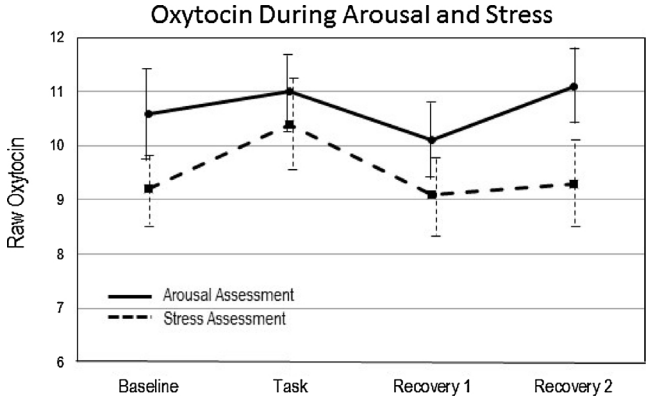

Fig. 3. Raw Oxytocin Levels Throughout the Psychological Stress Assessment and the Sexual Arousal Assessment.

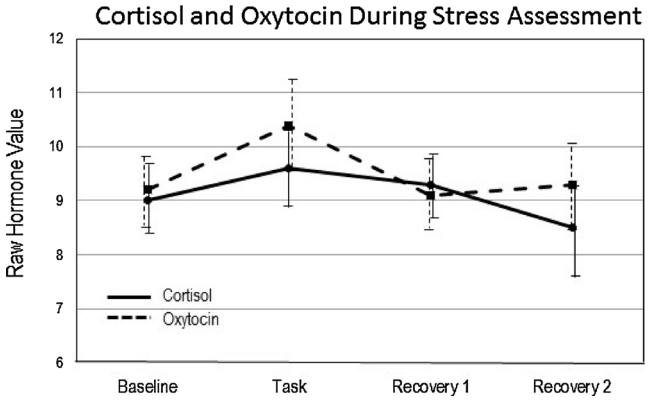

Fig. 5. Raw Cortisol and Raw Oxytocin Throughout the Psychological Stress Assessment.

5.3. Exploratory questions

We repeated the analytical models used to test Hypothesis 2 and 3, but for the *arousal* assessment rather than the stress assessment (Exploratory Question 1). These results are presented in the "Arousal" column of Table 3. Because this amounts to two exploratory tests for each hormone, we used a Bonferroni correction factor of 2. As shown in Table 3, participants who showed higher levels of oxytocin during the arousal task showed significantly higher levels of cortisol during the second recovery, $b = .37$, corrected $p = .01$. There were no significant associations between task levels of cortisol during the arousal task and oxytocin recovery from the arousal task. To ensure that we fully characterized the oxytocin/cortisol relationship, we followed-up with ancillary tests for *concurrent* associations between cortisol and oxytocin arousal reactivity and recovery, and found no such associations, $b_{\text{reactivity}} = -.04$, $p = .69$, $b_{\text{first recovery}} = -.09$, $p = .48$, $b_{\text{second recovery}} = .07$, $p = .47$.

We also conducted exploratory tests of *task differences* for each hormone (Exploratory Question 2). Specifically, we planned to test whether the magnitude and direction of task reactivity and recovery differed significantly for the arousal assessment versus the stress assessment (within each hormone, separately) and whether the

Table 3
Results of Multilevel Model Predicting Oxytocin During the Sexual Arousal Assessment and the Stress Assessment.

Parameter	Arousal	Stress	χ^2 (df = 1) for Arousal/Stress Contrast
DV: Cortisol			
Change from Baseline to Task	−.23***	.08	14.5*
Change from Task to First Recovery	−.38*	.36 [t]	8.1*
Moderator: Baseline Oxytocin	−.11	−.09	
Moderator: Task Oxytocin	.25	−.07	2.1
Change from Task to Second Recovery	−.43**	.26	6.9*
Moderator: Baseline Oxytocin	−.25	.16	
Moderator: Task Oxytocin	.37*	−.31[t]	9.5*
DV: Oxytocin			
Change from Baseline to Task	.11*	.10*	.03
Change from Task to First Recovery	.02	−.22	1.7
Moderator: Baseline Cortisol	−.22	−.13	
Moderator: Task Cortisol	.16	.18*	.01
Change from Task to Second Recovery	.10	−.30*	4.4
Moderator: Baseline Cortisol	.06	.13	
Moderator: Task Cortisol	−.12	−.05	.27

moderating effects outlined in Hypothesis 2 and 3 differed significantly for the arousal assessment versus the stress assessment. The results of these single degree of freedom chi-square tests are displayed in the final column of Table 3. Because there are four exploratory tests for each hormone, we used a Bonferroni correction factor of 5. As show in Table 3, there were significant differences between the arousal assessment and the stress assessment for all but one of the relevant cortisol parameters: baseline to task, χ^2 (1 *df*) = 14.5, corrected p = .02, task to first recovery, χ^2 (1 *df*) = 1, corrected p = .02, task to second recovery, χ^2 (1 *df*) = 6.9, corrected p = .04, moderating effect of oxytocin reactivity on first cortisol recovery, χ^2 (1 *df*) = 2.1, corrected p = .62, moderating effect of oxytocin reactivity on second cortisol recovery, χ^2 (1 *df*) = 9,5 corrected p = .01. There were no significant differences between the arousal assessment and the stress assessment for any of the oxytocin parameters, as shown in Table 3.

Lastly, we tested whether women's cortisol reactivity to the stress task was predicted by their cortisol reactivity to the arousal task, and whether women's oxytocin reactivity to the stress task was predicted by their oxytocin reactivity to the arousal task (Exploratory Question 3). We found no such associations: Oxytocin reactivity to the stress task was unrelated to oxytocin reactivity to the arousal task (b = -.03, p = .68), and cortisol reactivity to the stress task was unrelated to cortisol reactivity to the arousal task (b = -.01, p = .91).

6. Discussion

The present research advances our understanding of the dynamic relationship between salivary cortisol and oxytocin in response to different emotional states. A unique aspect of our research is that we measured both of these hormones in response to two different tasks (one inducing sexual arousal and one inducing psychological stress), conducted two weeks apart with the same participants. This approach allowed us to test whether levels of one hormone at specified points in time related to concurrent or subsequent levels in the other hormone. Our design also allowed us to investigate whether a woman's pattern of hormonal response to sexual arousal predicted her pattern of hormonal response to psychological stress.

Consistent with previous research (Brown et al., 2016), we found that baseline levels of oxytocin were significantly correlated with baseline levels of cortisol, for both the arousal assessment and the stress assessment. Yet this was the only episode for which we found concurrent associations across hormones. Across both tasks, we did not find associations between baseline-to-task changes in cortisol and baseline-to-task changes in oxytocin; nor did we find correlations between task-to-recovery changes in cortisol and oxytocin. Given that rigorous testing of associations between *simultaneous* patterns of hormonal change requires accurate measurement of the precise timing of hormonal release, we are reluctant to strongly interpret our findings on this point (which were not the focus of our primary hypotheses). We maintain that future research should continue investigating this question using assessment techniques that allow for more precise assessment of the timing of hormonal release, such as serum assays.

Our main hypotheses concerned the relationship between oxytocin and cortisol during and after psychological stress. Previous research suggests an inverse relationship between oxytocin and cortisol during stress, suggestive of a cortisol-regulatory function of oxytocin (Ditzen et al., 2009; Heinrichs et al., 2003; Pierrehumbert et al., 2010), but findings have been conflicting (Altemus et al., 2001; McQuaid et al., 2016; Taylor et al., 2006), with some studies finding *positive* associations between oxytocin and cortisol during stress (de Jong et al., 2015; Engert et al., 2016; Yuen et al., 2014). Our research sought to clarify the interrelationship of cortisol and oxytocin during stress by examining whether (1) women with higher levels of cortisol stress reactivity would show elevated post-stress levels of oxytocin, and (2) women with higher levels of oxytocin during or immediately after stress would show lower levels of post-stress cortisol. As predicted, we found that the women who showed greater oxytocin reactivity to the stress task had greater declines in cortisol from the stress task to the first recovery period. This finding is in line with previous research indicating that the stress-regulatory functions of oxytocin are associated with the post-stress recovery period in cortisol (Engert et al., 2016). This supports a growing body of research suggesting the importance of attending to post-stress recovery in cortisol and not simply reactivity levels (Bendezú and Wadsworth, 2017; Janson and Rohleder, 2017; Maeda et al., 2017). Yet contrary to our prediction, we did not find that higher levels of oxytocin stress reactivity predicted significantly lower post-stress levels of cortisol. Hence, our results provide stronger support for the potential regulating effect of oxytocin on cortisol than the regulating effect of cortisol on oxytocin. Further research is required to replicate these effects, and ideally to include more frequent assessments in order to more accurately capture the timing of such effects. Similarly, research combining more frequent assessments with larger samples would permit the use of structural equation modeling, which would permit more robust conclusions about the ordering of coregulatory effects. Also, it bears noting that although our stress task elicited a statistically significant increase in oxytocin, it did not elicit a statistically significant increase in cortisol (although it succeeded in eliciting statistically significant increases in state anxiety). It is unclear whether low levels of cortisol stress reactivity affected our hypothesis tests, and future research should address these questions with designs that provoke larger stress-related increases in cortisol.

Future research should also examine different phases of the menstrual cycle. All of our participants were in the follicular phase of their cycle during the arousal task and the luteal phase of their cycle during

the stressor. Some research has found that cortisol responses to experimental stress are greater during the luteal than the follicular phase (Kirschbaum et al., 1999; Montero-López et al., 2018), whereas others have observed the opposite pattern (Bouma et al., 2009; Walder et al., 2012) or have found no cycle differences – and no significant stress-induced cortisol increases overall, as in the present study (Childs et al., 2010). Other studies have found that the association between cortisol and *subjective* stress is greater during the luteal than the follicular phase (Duchesne and Pruessner, 2013). Given these mixed findings, it is not clear whether cycle phase contributed to our findings (and, in particular, the low levels of cortisol reactivity to stress observed in our sample). On this topic, cycle-related variation in oxytocin is also important to consider. A recent meta-analysis found that although basal oxytocin levels tend to increase with ovulation, there is little difference between the early follicular and mid-luteal phases (Engel et al., 2018). Yet no research has systematically compared oxytocin *reactivity* across different cycle phases, and future research should test for such difference and investigate whether cycle-related variation in oxytocin stress reactivity may play a role in cycle-related changes in cortisol stress reactivity.

Our analyses of cortisol and oxytocin responses to the experimental induction of sexual arousal task yielded a notably different patterns of results than that observed for the stress task: Specifically, oxytocin arousal reactivity was unrelated to cortisol arousal recovery, and cortisol arousal reactivity was unrelated to oxytocin arousal recovery. These findings are consistent with previous research suggesting that the relationship between these hormones may be context-specific (Brown et al., 2016; Cardoso et al., 2014). We further explored the issue of task-specific effects by directly comparing responses to the arousal versus the stress task, separately for each hormone. These comparisons revealed divergent patterns for oxytocin versus cortisol. For oxytocin, increases and decreases across the experimental session (baseline, task, first recovery, second recovery) largely paralleled one another, as can be seen in Fig. 3. Yet for cortisol, the pattern that emerged across the arousal assessment was significantly different from the pattern that emerged across the stress assessment. Cortisol declined significantly in response to the arousal task, whereas it increased (although not significantly) in response to stress, and the difference in reactivity across tasks was significant (notably, because these tests are conducted using multilevel parallel process models, they control for within-subject dependency). Similarly, cortisol showed continuing declines from the arousal task to the first and second recovery, whereas oxytocin showed the opposite pattern. Again, these task differences were statistically significant. Finally, the relationship between oxytocin task reactivity and post-task cortisol recovery was significantly different across tasks. Whereas higher oxytocin during stress was associated with lower cortisol during post-stress recovery, higher oxytocin during sexual arousal was associated with higher cortisol during post-arousal recovery. Our study design is the first to permit direct testing of such within-person, cross-task differences, given that we assessed the same participants across both tasks, and the results raise interesting questions about the role of psychological context in shaping coordinated patterns of oxytocin versus cortisol response. A useful direction for future research is to assess the interplay between these and other hormones before, during, and after a range of psychological states in order to fully characterize their relationship, and to understand their respective mechanisms of action. In particular, it will be valuable to focus on psychological states that are known to relate to oxytocin levels, such as trust, empathy, caregiving, and social affiliation (Campbell, 2010; Heinrichs et al., 2013; Kosfeld et al., 2005; Ross and Young, 2009).

A final contribution of our research is that we tested whether interindividual differences in oxytocin or cortisol reactivity for one type of psychological state extended to other psychological states. In other words, do women with the highest levels of oxytocin reactivity to sexual arousal *also* show the highest oxytocin reactivity to psychological stress? Although we found that women's baseline levels of cortisol and oxytocin before the stress task were significantly associated with their baseline levels prior to the arousal task, we did not find that a woman's reactivity to the arousal task was associated with her arousal reactivity to the stress task, for either hormone. Hence, it appears that although *tonic* levels of oxytocin and cortisol show significant within-person consistency, task responses involving different psychological states do not. This finding further underscores the context-specificity of oxytocin and cortisol responses. Given the relevance of menstrual cycle phase for both oxytocin and cortisol release, future research should reexamine this question in women who undergo the two tasks during the same menstrual cycle phase. Future research should also reexamine this question using tasks that elicit more similar psychological states: For example, although a woman's degree of oxytocin reactivity to sexual arousal may not predict her degree of oxytocin reactivity to stress, might it predict her oxytocin reactivity to stimuli eliciting other positive psychological states, such as trust or empathy? Such research may help to clarify whether there are larger categories of psychological states (for example, positive versus negative) which elicit within-person consistency in hormonal response, or whether a person's pattern of hormonal responses is highly differentiated across a wide range of psychological states.

Our study has several important limitations. As noted earlier, our stress task did not elicit a statistically significant increase in cortisol, and it is not clear how this may have affected our findings, and whether menstrual cycle phase may have played a role. Also, although we collected our salivary samples of oxytocin and salivary samples of cortisol at the same time, this does not imply that they represent exactly synchronous endocrinological responses. Hence, although the timed intervals of assessment allow us to establish the basic ordering of responses, they do not allow us to specify the time scale over which oxytocin responses may influence subsequent changes in cortisol. Future research with more frequent measurements, perhaps using serum assessments in concert with salivary assessments, may contribute to future specificity on this question. Second, our research used exclusively female participants, given previous research on gender differences in oxytocin responsivity. It will be valuable for future research to directly compare the dynamic relationship between oxytocin and cortisol among both women and men. Greater ethnic and educational diversity is also an important priority for future research. It is possible that individuals of different ethnic and educational backgrounds make have different appraisals of experimental tasks such as those used in the present study, and it is important for future research to account for such possibilities. The use of a sample containing a sizeable percentage of sexual-minority women is another unique feature of our study which may limit its generalizability, but which simultaneously makes a notable contribution to the existing literature on these topics, given that most previous research uses exclusively heterosexual participants (or does not assess the sexual orientation of participants). Although there is no theoretical basis to expect sexual-minority women to show different patterns of hormonal response to stress or sexual arousal than heterosexual women, it will be valuable for future research to continue collecting more sexually diverse samples in order to comprehensively address this question.

In summary, our study makes an original contribution to our emerging understanding of the complex interrelationship between cortisol and oxytocin across different psychological states. Specifically, it provides the first direct comparison of a single group of women's oxytocin and cortisol responses to two markedly different psychological contexts: sexual arousal versus psychological stress. Hence, it provides novel evidence that the coordinated functioning of these two hormones under stress *differs* from their coordinated functioning under other psychological states. Our emerging understanding of the coregulatory relationship between oxytocin and cortisol will benefit from future such studies examining how their association varies across different psychological contexts.

Conflict of interest

None.

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