Archives of Sexual Behavior
https://doi.org/10.1007/s10508-018-1231-6

ORIGINAL PAPER

Average Associations Between Sexual Desire, Testosterone, and Stress in Women and Men Over Time

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Received: 1 May 2017 / Revised: 1 November 2017 / Accepted: 8 May 2018

Abstract

Sexual desire and testosterone are widely assumed to be directly and positively linked to each other despite the lack of supporting empirical evidence. The literature that does exist is mixed, which may result from a conflation of solitary and dyadic desire, and the exclusion of contextual variables, like stress, known to be relevant. Here, we use the Steroid/Peptide Theory of Social Bonds as a framework for examining how testosterone, solitary and partnered desire, and stress are linked over time. To do so, we collected saliva samples (for testosterone and cortisol) and measured desire as well as other variables via questionnaires over nine monthly sessions in 78 women and 79 men. Linear mixed models showed that testosterone negatively predicted partnered desire in women but not men. Stress moderated associations between testosterone and solitary desire in both women and men, but differently: At lower levels of stress, higher average testosterone corresponded to higher average solitary desire for men, but lower solitary desire on average for women. Similarly, for partnered desire, higher perceived stress predicted lower desire for women, but higher desire for men. We conclude by discussing the ways that these results both counter presumptions about testosterone and desire but fit with the existing literature and theory, and highlight the empirical importance of stress and gender norms.

Keywords Sexuality · Sexual desire · Testosterone · Stress · Cortisol · Gender

Introduction

Sexual desire—its causes, correlates, antecedents, and outcomes—continues to make its way into research scholarship and lay discussions. More specifically, discourse suggests that there is a clear, direct, causal, and positive link between testosterone and desire, despite the lack of supporting evidence (van Anders, 2012; van Anders, Goldey, & Bell, 2014). These assumptions about desire–testosterone links have the potential to lead scientific research about sexuality in empirically unsupported directions, misinform medical perspectives surrounding sexual health, and muddy conceptualizations of sexuality in the general population.

While research has demonstrated that testosterone can be linked to various forms of sexuality, it points to a range of directions, mediators, and moderators that are important for these nuanced associations. Unfortunately, in the case of testosterone and desire, these links have been understudied and reflect mixed findings. While several factors likely contribute to mixed findings about sexual desire and testosterone, there are three issues that might be particularly implicated: (1) sexual desire is conceptualized and measured in various ways that might be differentially related to testosterone; (2) despite empirical evidence of the importance of stress, experimental approaches infrequently take it into account as a potential influence on desire–testosterone links; and (3) the use of cross-sectional methods (i.e., examining links between

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s10508-018-1231-6) contains supplementary material, which is available to authorized users.

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Published online: 29 May 2018

individuals) limits understanding of how these links might function within individuals.

Considering the Multifaceted Nature of Desire

Mixed findings from research about links between testosterone and sexual desire may reflect that desire is typically studied as a unitary phenomenon, even as empirical research increasingly demonstrates its multifaceted nature (Brotto, 2010a, b; Chadwick, Burke, Goldey, Bell, & van Anders, 2017a, Chadwick, Burke, Goldey, & van Anders, 2017b; Edelstein, Chopik, & Kean, 2011; Goldey, Posh, Bell, & van Anders, 2016; Goldhammer & McCabe, 2011; Mark, Fortenberry, Herbenick, Sanders, & Reece, 2014; Meana, 2010). One framework—the Steroid/Peptide Theory of Social Bonds (S/P theory) (van Anders, Goldey, & Kuo, 2011)—is useful for theorizing how testosterone and multiple facets of desire might be differentially linked. According to S/P theory, sexuality can be constituted by nurturance and/or eroticism (an aspect of "competition," a category which can also include power; see van Anders, 2013), but these are linked to testosterone in opposite ways. Eroticism, which could be defined as aspects of sexuality tied to bodily pleasure (van Anders, 2015), is positively linked to testosterone, whereas nurturance, defined as feelings of warm loving closeness (van Anders, 2015; van Anders et al., 2011), is negatively linked to testosterone (van Anders et al., 2011). Like sexuality in general, sexual desire itself could reflect a combination of nurturance and eroticism in differing proportions. In this case, negative *or* positive associations between testosterone and desire would be expected, depending on the form desire takes. This theoretical framework has been supported more recently, as testosterone has been shown to be linked with some facets of sexual desire but not others (Chadwick et al., 2017b).

Research has repeatedly demonstrated (at least) two different forms of desire along other axes that are only moderately related: solitary desire, the desire to engage in sexual activity alone (e.g., with masturbation), and partnered desire, the desire to engage in sexual activity with another person. Solitary and partnered desire are correlated with each other at only moderate levels and may be associated with testosterone in different ways (van Anders, 2012; van Anders & Dunn, 2009; van Anders et al., 2011; van Anders, Hamilton, & Watson, 2007a, b; van Anders & Hampson, 2005). See Table 1 in van Anders (2012) for a review of studies exploring associations, with effect sizes, between sexual desire and T in healthy women and men.

Testosterone and Partnered Desire

Despite assumptions that testosterone should be positively linked to sexual desire, there are reasons to predict the opposite association. Following the S/P Theory, partnered desire could be positively linked to testosterone because of its erotic nature or negatively linked because of its nurturant nature (Goldey et al., 2016; van Anders et al., 2011). However, data linking testosterone and partnered desire are mixed and, at times, contradictory. These links are also different depending on gender/sex.

Some research has shown that women who have hypoactive sexual desire disorder (a diagnosis now outdated) have lower partnered desire *and* higher free testosterone than women who experience no sexual problems (Heiman et al., 2011), suggesting that partnered desire and testosterone may be negatively associated. Conversely, other research suggests that women with desire disorders and healthy women do not

Table 1 McDonald's omega and Cronbach's alphas for each scale at each session

Measure	Session	ω_h	α
Dyadic sexual desire (8 items)	0	.77	.89
	1	.70	.92
	2	.79	.91
	3	.77	.92
	4	.81	.91
	5	.82	.90
	6	.69	.89
	7	.74	.88
	11	.67	.91
Solitary sexual desire (3 items)	0	–	.91
	1	–	.90
	2	–	.92
	3	–	.92
	4	–	.92
	5	–	.86
	6	–	.90
	7	–	.78
	11	–	.82
Perceived stress scale (10 items)	0	.75	.91
	1	.64	.85
	2	.60	.86
	3	.65	.88
	4	.65	.90
	5	.76	.90
	6	.75	.88
	7	.41	.88
	11	.74	.88

ω_h = Omega hierarchical, ω_h, (Zinbarg, Revelle, Yovel, & Li, 2005), reflects how any general factor accounts for a proportion of variance in the scale score. Omega hierarchical could not be computed for the solitary sexual desire because the procedure requires at least 4 items. α = Coefficient (i.e., Cronbach's) alpha. All values were calculated using the psych package (Revelle, 2017). See data supplement for details

differ significantly in testosterone (Basson, Brotto, Petkau, & Labrie, 2010; Schreiner-Engel, Schiavi, White, & Ghizzani, 1989). Additionally, while some previous research in non-clinical populations suggests that testosterone is not correlated with partnered desire (Cappelletti & Wallen, 2016; Motta-Mena & Puts, 2017; Roney & Simmons, 2013; van Anders & Dunn, 2009; van Anders et al., 2007a, b), when cortisol and perceived stress are controlled for in analyses, a negative correlation between testosterone and partnered desire in women has been documented (van Anders, 2012).

In contrast to research with women, much of the research examining the link between partnered desire and testosterone in men suggests that they are not related (Mazur, Mueller, Krause, & Booth, 2002; Sadowsky, Antonovsky, Sobel, & Maoz, 1993; van Anders & Dunn, 2009), even when stress is controlled for (van Anders, 2012). But why might testosterone be negatively linked with partnered desire in women, but unlinked with partnered desire in men? One possibility is that women's partnered desire is more strongly characterized by nurturance, which is associated with lower testosterone according to S/P theory. This makes sense considering that women's sexual desire is expected to be oriented toward a relationship partner and characterized by romance and a desire for intimacy (Wiederman, 2005), and that women often cite closeness and intimacy as important reasons for engaging in sex (Basson, 2000; Carroll, Volk, & Hyde, 1985; Denney, Field, & Quadagno, 1984; Leigh, 1989). Men's sexual desire, on the other hand, is expected to be more erotic; it is characterized as insatiable and oriented toward pleasure and orgasm (Wiederman, 2005). Moreover, men tend to expect that they will orgasm during most sexual encounters with a partner (Salisbury & Fisher, 2014). Thus, it is possible that men's sexual desire for a relationship partner may be characterized strongly by both nurturance and eroticism (Chadwick et al., 2017a), negating associations with testosterone. This is perhaps supported by research showing that partnered men's hormonal associations may depend on the target of their desire, which may be differentially erotic (Goldey, Avery, & van Anders, 2014); for example, partnered men's testosterone has been shown to have a positive association with desire for uncommitted sex outside of their relationship (Puts et al., 2015).

Testosterone and Solitary Desire

Relative to research on partnered desire and testosterone, there is less literature discussing potential links between testosterone and solitary desire in women and men. Following expectations from the S/P theory, solitary desire is more consistently positively correlated with testosterone because it is more likely to map onto eroticism (Goldey et al., 2016; van Anders et al., 2011). However, findings are still mixed. In women, some studies have suggested that they are positively linked (van Anders, 2012; van Anders, Brotto, Farrell, & Yule, 2009; van Anders et al., 2007a, b) while, in others, no association was observed (van Anders & Dunn, 2009; van Anders & Hampson, 2005). Similarly, with men, solitary desire has occasionally been shown to be correlated with testosterone (van Anders & Dunn, 2009), but other literature fails to support this association (van Anders, 2012). Thus, focusing solely on partnered desire when examining desire–testosterone links, as is common for the current literature, is problematic for understanding desire in general. Partnered and solitary desire differ and overlap in divergent ways with eroticism and nurturance (showing some weak and some moderate correlations; Chadwick et al., 2017a) and, for this reason, scientific research that examines the association between hormones and solitary and partnered desire separately may present a clearer picture of the association between endocrine measures and sexuality.

Stress as a Potential Moderator of Desire–Testosterone Links

Taking solitary and partnered desire into account, and considering how gender may influence the characterization of sexual desire, allows for a more nuanced understanding of desire–testosterone links, but even so, the mixed pattern of results makes it tempting to interpret the findings as null or inconclusive. One possibility for explaining these mixed findings is that associations between testosterone and desire may not be direct, i.e., connections between testosterone and desire may be more complex than typically presumed, and may involve mediators or moderators.

Stress—physiological and psychological—is one parameter generally assumed to be associated with sexual desire, but rarely studied in conjunction with testosterone. In contrast, studies on other social variables *have* focused on stress in relation to links with testosterone (e.g., sexual motivation, status seeking, marital quality) (Booth, Granger, Mazur, & Kivlighan, 2006; Lemaire, Taylor, & Mormède, 1997; Mehta & Prasad, 2015). Specifically, the dual hormone hypothesis suggests that testosterone is positively associated with status-seeking tendencies (e.g., aggression and successful competitive negotiations), but perhaps only in individuals with low levels of cortisol though findings here, too, are mixed (Dabbs, Jurkovic, & Frady, 1991; Geniole, Carre, & McCornick, 2011; Hamilton, Carre, Mehta, Olmstead, & Whitaker, 2015; Harden, Kretsch, Tackett, & Tucker-Drob, 2014; Mehta & Josephs, 2010; Pfattheicher, Landhäußer, & Keller, 2013; Popma et al., 2007; Zilioli, Caldbick, & Watson, 2014). Given that previous research has found associations between sexual desire and testosterone and sexual desire and stress, this leads us to question: how might stress, cortisol, and testosterone be related to sexual desire?

Stress Measurement

Stress researchers typically expect that, during stressful conditions, individuals will shift physical resources (e.g., energy) away from sexual motivation and behavior toward behaviors required for immediate survival (Laugero & Moberg, 2000; Moberg & Mench, 2000). This leads to the prediction that stress and sexuality should be negatively correlated. However, empirical findings about the association between sexual variables and measures of stress (e.g., self-reports, cortisol levels) are mixed, with some studies showing that stress is positively associated with sexuality (Goldey & van Anders, 2012; Hamilton, Rellini, & Meston, 2008; Lopez, Hay, & Conklin, 2009; Roney, Lukaszewski, & Simmons, 2007) and others showing that stress is negatively associated with sexual activity or functioning (Bodenmann, Atkins, Schar, & Poffet, 2010; Hamilton et al., 2008; Hamilton & Julian, 2013; Hamilton & Meston, 2011; Hou, Xiong, Wang, Chen, & Yuan, 2014; Meston & Lorenz, 2013; Minnen & Kampman, 2000). These mixed directions of findings between desire and stress may reflect differences in how stress is operationalized (Goldey & van Anders, 2012), as there is evidence that measures of self-reported stress and cortisol diverge (Rosal, King, Ma, & Reed, 2004). The operationalization of stress may have implications for its impact on desire–testosterone links, which highlights the importance of exploring both physiological and self-reported stress.

For example, testosterone has been shown to inhibit cortisol responses in men (Rubinow et al., 2005). And, some research suggests that testosterone is negatively correlated with partnered desire in women, but this association is specific to women with high cortisol levels (van Anders, 2012). Since a large portion of testosterone is released via activation of the HPA axis in women relative to men (Abraham, Chakmakjian, Buster, & Marshall, 1975; van Anders, 2013; Wajchenberg et al., 1986), stress could actually lead to increases of both testosterone and cortisol in parallel in women (van Anders, 2013). Cortisol also may be a predictor of sexual desire on its own, as some research has found positive associations between the two (van Anders, 2012; van Anders & Dunn, 2009) though this has not always been replicated and may differ by gender/sex and by partnered and solitary desire (van Anders, 2012; van Anders & Dunn, 2009). Perceived stress also seems to matter in different ways (van Anders, 2012). For example, participants have reported that partnered sexual desire is negatively correlated with stress (Carvalheira & Traeen, 2014; Janssen, Macapagal, & Mustanski, 2013). While fewer studies have examined solitary desire and psychological stress, previous research also suggests that desire to masturbate may increase when stressed or in need of a release (Bowman, 2014; Graham, Sanders, Milhausen, & McBride, 2004). And some research indicates that lower desire for sexual activity with a partner but higher desire to engage in solitary sexual activity may be associated with stressful conditions (Graham et al., 2004). The inclusion of self-report measures of stress alongside endocrine measures may further facilitate understanding of desire–testosterone links as well as the mechanisms of the dual hormone hypothesis (Mehta & Josephs, 2010).

Can Longitudinal Methods Clarify Desire–Testosterone Links?

A statistical design that explores longitudinal associations could be potentially useful in resolving the many inconsistencies in the literature on desire–testosterone links. While there are several studies that consider the change in testosterone levels before and after sexual interactions (Goldey & van Anders, 2011, 2012; Lopez et al., 2009; Roney et al., 2007), these have generally not focused on sexual desire specifically. Longitudinal approaches have yet to be employed in the study of testosterone and desire, but collecting information on individuals' testosterone and desire across multiple time points would allow for repeated sampling in a way that provides insight into what desire–testosterone links look like on average over time.

Present Study

In the present study, we used longitudinal methods to model the average association between sexual desire, testosterone, and stress in healthy women and men over time. We controlled for body mass index (BMI) (Cupisti et al., 2007; Nackers et al., 2015; Shamim, Khan, & Arshad, 2015), time since waking (Dabbs, 1990; Gettler, McDade, Agustin, Feranil, & Kuzawa, 2014), relationship status (Dibble, Goldey, & van Anders, 2017; Gray, Ellison, & Campbell, 2007; van Anders & Goldey, 2010), illness (Boonekamp, Ros, & Verhulst, 2008; Lassek & Gaulin, 2009) and hormonal contraceptive use (in women only) (Boozalis, Tutlam, Chrisman Robbins, & Peipert, 2016; Winkler & Sudik, 2009). To do this, we used linear mixed modeling to examine associations between desire, testosterone, and stress.

Given previous findings and theoretical foundations, we developed the following hypotheses: (1) Higher testosterone would be associated with lower partnered desire in women but not men; (2) stress would moderate the association between testosterone and partnered desire in women in the following way: Women with higher cortisol levels would have a stronger negative association between partnered desire and testosterone than women with low cortisol levels; (3) cortisol and perceived stress would both be negatively linked with partnered sexual desire in women and men; (4) higher testosterone would be positively associated with higher solitary desire in women, but not men; and (5) cortisol and perceived

Table 2 Number of women, men, and total number of participants included in the analyses at each session (measured in months from baseline session)

Session number	0	1	2	3	4	5	6	7	11
Women	61	51	44	39	36	35	29	19	27
Men	60	43	33	31	25	24	23	15	20

stress would be positively linked with solitary sexual desire in women and men.

Method

Participants

Participants were 157 first-year university students (79 men, 78 women) recruited for the Implications of Partnerships Around the College Transition (ImPACT) Study (first described in van Anders, Goldey, Conley, Snipes, & Patel, 2012a, van Anders, Tolman, & Volling, 2012b), a study examining longitudinal associations between hormonal, health, social, and sexual variables during the first year of college. Participants self-identified their race/ethnicity by choosing from a preset list of options: 37 identified as Asian, four as Black/African American, three as Hispanic/Latino, 90 as White, 12 as Multiracial, one as Hawaiian/Pacific Islander, and 10 as other or non-responsive participants. A majority of the students self-identified their sexual orientation as heterosexual (91.7%, $n = 144$), and we coded a small number as sexual minorities (4.5%, $n = 7$); the rest did not answer (3.8%, $n = 6$). Participants identified their relationship status at the study's onset: 45.9% ($n = 72$) were single, 19.7% ($n = 31$) were casually partnered, 33.1% ($n = 52$) were in a committed relationship, and 1.3% ($n = 2$) did not respond. Most participants were 18 or 19 years old at baseline (95.5%, $n = 150$), 1.9% ($n = 3$) were between the ages of 20 and 22, and four did not respond.

Participants completed up to nine study sessions over the course of 12 months. At each session and in line with recommended guidelines for conducting hormone studies with humans (van Anders, 2012), we excluded participants who reported the presence of medical conditions or current use of medications affecting cortisol, testosterone, or sexual drive/functions from analyses. We also excluded nicotine users in line with these guidelines (van Anders, 2012); this choice is supported by a meta-analysis study showing that smoking cigarettes affects men's testosterone and may affect women's testosterone (Zhao, Leung, Lin, & Schooling, 2016) as well as women's and men's cortisol levels (Steptoe & Ussher, 2006). The number of participants who were excluded from analyses at each session varied depending on the participants' specific responses at each session. For example, if a participant reported that they used nicotine at one of the sessions but not others, then they were only excluded from the session when they reported the nicotine use.

At baseline, there were a total of 121 participants (60 men and 61 women) who were not excluded due to medication/nicotine use. On average, there were approximately 68 individuals who were eligible for analyses at each session. Over time, the number of participants completing each session generally decreased for both men and women, with the lowest number of participants ($n = 34$) at 7 months from baseline (see Table 2 for *n*s by session).

Materials

Questionnaires

Health and Demographics

There were two versions of the health and demographics questionnaire: a longer one at the study's baseline and completion sessions, and a shorter one repeated at each follow-up study session. The shorter version, used at all sessions, asked about medication use (including hormonal contraceptive use), nicotine use, and current illnesses. Additionally, at all sessions, the participants self-identified their relationship status and sexual orientation/identity. The longer version contained the same items as the shorter version with the addition of questions regarding participants' demographics such as their race/ethnicity, gender/sex, parental income, and parental education. Participants were also asked to report weight and height at baseline, which were used to calculate BMI, and to report any medical conditions affecting hormones.

Sexual Desire Inventory (SDI) (Spector, Carey, & Steinberg, 1996)

This questionnaire assesses participants' level of sexual desire, defined as interest in or wish for sexual activity. Our version included one additional item, "During the last month, *how often* have you had sexual thoughts?" The SDI includes two

subscales, one representing solitary sexual desire and one representing dyadic (partnered) sexual desire, as well as a total score. For both solitary and partnered sexuality, items ask about topics such as desired frequency of sexual activity, importance of fulfilling sexual desire, and strength of desire. Our adapted SDI contains 15 questions, and participants indicate their strength and frequency of desire over the past month on 8-point scales. For strength, the response options range from *1 = "no desire"* to *7 = "strong desire";* for frequency, the scale ranges from *0 = "not at all"* to *7 = "many times a day"*. The range of possible values for the solitary desire subscore is 2–23, and the range of possible values for the partnered desire subscore is 6–62.[1] See Table 1 for dyadic and solitary reliability calculations for each session.

Perceived Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983)

This scale measures the extent to which participants perceived their life events as stressful over the past month. The validated 10-item version uses a Likert scale with responses ranging from *0 = Never* to *4 = Very often*. Example items include "In the last month, how often have you felt that you were unable to control the important things in your life?" and "In the past month, how often have you found that you could not cope with all the things that you had to do?" Responses were coded such that higher scores corresponded to higher perceived stress. See Table 1 for PSS reliability calculations for each session.

Hormone Samples

We measured cortisol and testosterone using saliva samples, which are minimally invasive. They are also commonly used in psychological research and are less likely to produce an undesired stress response in comparison with blood draws. Salivary testosterone and cortisol assays are well validated. Salivary cortisol correlates well with serum (Lippi et al., 2009). Additionally, salivary testosterone shows high correlations with free serum testosterone (Khan-Dawood, Choe, & Dawood, 1984; Magrini, Chiodoni, Rey, & Felber, 1986) and total serum testosterone in men (Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004; Shirtcliff, Granger, & Likos, 2002). Salivary testosterone is considered to reflect the fraction of testosterone that is not bound or weakly bound to binding proteins; therefore, it is more available to bind with receptors and potentially more relevant to behavior and/or desire (Quissell, 1993).

The participants provided saliva samples into 17-mL polystyrene tubes by passive drool (for a review, see van Anders et al., 2014). When samples were provided in the laboratory (e.g., the baseline sessions), samples were immediately frozen at − 20 degree Celsius. When samples were provided at home (i.e., most follow-up and final sessions), participants stored the sample in their own freezer to freeze the sample until they could arrange a pickup or drop-off at the laboratory, where they were frozen at − 20 degree Celsius until assay. Samples were assayed at the Core Assay Facility at the University of Michigan. Samples were radioimmunoassayed for cortisol and testosterone in multiple batches using commercially available kits from Siemens (Washington, DC, USA) following previously validated protocols (Campbell, Schultheiss, & McClelland, 1999; Wirth, Welsh, & Schultheiss, 2006). The inter-assay coefficients of variation (CV) for cortisol were 8.2% for high levels and 13.80% for low levels, and the intra-assay CVs for cortisol were 2.86 and 7.84% for high and low levels, respectively. The inter-assay CVs for testosterone were 8.6, 8.2, and 25.90% for high, medium, and low, respectively, and the intra-assay CVs for testosterone were 3.16 and 12.83%, at high and low levels, respectively. We note that intra- and inter-assay CVs are somewhat high at low levels of T; however, other studies measuring salivary T have reported similar CV values (Edelstein, van Anders, Chopik, Goldey, & Wardecker, 2014; Liening, Stanton, Saini, & Schultheiss, 2010; Stanton, Liening, & Schultheiss, 2011). Analytical sensitivity for the Siemens T assay in our laboratory was 1.14 pg/mL.

Procedure

Participants completed the baseline session between August and October of their first year at the university (all testing occurred in the years 2009 and 2010). All baseline sessions were scheduled between 12 and 7 pm, in order to maximize time available for testing while avoiding the high fluctuating levels of hormones around the time of waking (van Anders et al., 2014). We instructed participants to refrain from eating, drinking (besides water), smoking, chewing gum, or brushing teeth for one hour prior to the scheduled session. The individuals were given consent forms upon arrival to read and sign. A research assistant then provided instructions about completing the online questionnaire and providing the saliva samples before leaving the participant alone in a private room to complete the study. The baseline questionnaire included the health and demographics questionnaire, relationship and sexuality questionnaires, PSS, SDI, and additional measures

[1] On the final session survey, one item of the dyadic desire subscale (i.e., "When you have sexual thoughts, how strong is your desire to engage in sexual behavior with a partner?") was missing two response options (4 and 6 on the 1–8 scale) due to a survey error. Responses were calculated as participants entered them (i.e., 1 = 1, 2 = 2, 3 = 3, 5 = 5, 7 = 7, 8 = 8), but we note that participants did not have the option to input 4 or 6.

not used in current analyses.[2] At the completion of the baseline session, participants were compensated with $15.

Participants were then sent home with saliva tubes for the follow-up sessions. These follow-up sessions took place approximately once per month for 7 months during the academic year and then once again at the beginning of the following school year. We asked participants to collect the saliva sample between 2 pm and 6 pm on the same day while completing the online follow-up questionnaire. There was some variation in compliance with these instructions, but 95% (n = 535) of the 565 observations with recorded dates for both saliva and survey collections provided the saliva sample between 5 days before and 5 days after taking the survey; furthermore, we controlled for time of day (specifically time since waking) in our analyses. Because research has repeatedly shown that menstrual phase need not be controlled for in T analyses unless it is of special interest (for a review, see van Anders et al., 2014), women were tested during any phase of their menstrual cycle. We also reminded participants not to eat, drink, smoke, chew gum, or brush teeth for one hour prior to saliva collection. Participants stored the sample in their own freezer until a research assistant could pick it up or until they dropped the sample off at the laboratory. Follow-up questionnaires included a subset of the baseline measures (including the PSS and SDI), with the exception of the final questionnaire which was identical to baseline. The participants were compensated with $10 for completion of each follow-up. If the participants completed at least eight of the nine total sessions, they were awarded a bonus of $25.

Statistical Analyses

We analyzed data with R version 3.4.1 (R Core Team, 2016). We replaced missing values for individual questionnaire items with participant's average item score for the relevant subscale at that session, as long as no more than three items from the subscale were missing (Tinsley & Brown, 2000).

For our main analyses, we used version 1.1–13 of the lme4 package (Bates, Maechler, & Walker, 2015) to fit linear mixed models (LMM) and generalized mixed models (GMM) to predict solitary and partnered sexual desire from testosterone, PSS, cortisol, and control variables. With linear mixed models, analysts can model how data cluster within groups (within participants in our case). And, rather than estimating an average value for some variable and predicting that one value for each participant—a fixed effect—it is possible to choose to estimate a unique value for each participant; in this way, each participant's estimate acts like a random deviation from a hypothetical distribution with a common mean—a random effect. Another strength of linear mixed models is that they use data from participants who do not complete all sessions (West, 2009; West, Welch, & Galecki, 2014). For reporting purposes, n in the analyses below refers to the total number of observations included in the analysis and may include multiple measures from the same individual at different sessions.

We analyzed data separately for women and men, given differences in distributions of testosterone by gender/sex (van Anders, 2012). Thus, we ran four different models, i.e., to predict (1) partnered desire in women, (2) partnered desire in men, (3) solitary desire in women, and (4) solitary desire in men.

In all models, partnered or solitary desire was treated as the dependent variable, the predictors of interest were testosterone, PSS, and cortisol. Results from Little's chi-square test of MCAR (Little, 1988) suggested that men's testosterone levels were missing completely at random, $\chi^2(128) = 148.82$, $p = .100$, but men's cortisol levels were not, $\chi^2(129) = 157.03$, $p = .047$; the same test suggested that women's testosterone levels, $\chi^2(130) = 143.49$, $p = .198$, and women's cortisol levels, $\chi^2(156) = 162.91$, $p = .336$, were missing completely at random. Because we found suggestive evidence ($p < .05$) that data were not always missing completely at random, we used the *corFiml()* function from the psych package in R (Revelle, 2017) to conduct full information maximum likelihood zero-order correlations. According to the package documentation, "The basic FIML algorithm is to find the pairwise ML solution for covariances and means for every pattern of missingness and then to weight the solution by the size of every unique pattern of missingness."

We were then able to test whether potential variables that sometimes act as confounds (van Anders et al., 2014) such as body mass index (BMI) (Cupisti et al., 2007; Nackers et al., 2015; Shamim et al., 2015), time since waking (Dabbs, 1990; Gettler et al., 2014), relationship status (Dibble et al., 2017; Gray et al., 2007; van Anders & Goldey, 2010), illness (Boonekamp et al., 2008; Lassek & Gaulin, 2009) and hormonal contraceptive use (in women only) (Boozalis et al., 2016; Winkler & Sudik, 2009) correlated with either men's or women's hormone levels at each time point. Because each of these covariates correlated with testosterone or cortisol at least one time point, and because we included each time point in all our models, we included all these covariates in all models we present.

[2] In the interest of full reporting, we note that these measures included the Investment Model Scale (Rusbult, Martz, & Agnew, 1998), Quality Marriage Index (Norton, 1983), UCLA Multidimensional Condom Attitudes Scale (Helweg-Larsen & Collins, 1994), Index of Sexual Satisfaction (Hudson, Harrison, & Crosscup, 1981), Experiences in Close Relationships Scale (Brennan, Clark, & Shaver, 1998), General Well-Being Schedule (Dupuy, 1973), Klein Sexuality Grid (Klein, Sepekoff, & Wolf, 1985), Multidimensional Scale of Perceived Social Support (Zimet, Dahlem, Zimet, & Farley, 1988), Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988), Rosenberg Self-Esteem Scale (Rosenberg, 1965), Sex-Role Traditionalism Scale (Peplau, Hill, & Rubin, 1993), and UCLA Loneliness Scale (Russell, Peplau, & Ferguson, 1978).

Session number was recorded in months since baseline. For example, the baseline session was coded as Time = 0, the second session was coded as Time = 1, and the final session was coded at time = 11, because of the summer vacation. All control variables were time variant with the exception of BMI, which was only measured at baseline. The continuous independent variables were mean-centered (separately by gender/sex) and then standardized so that they shared a common scale. Widely different variances can make it difficult to estimate model parameters. Finally, there is reason to believe that testosterone interacts with both cortisol and perceived stress (van Anders, 2012). Therefore, we explored potential interactions.

Following standard practice (e.g., van Anders et al., 2012), we removed testosterone and cortisol outliers (±3 SD) within each time point (see Table 2 for number of participants included at each time point, after exclusions). For women, we observed 15 cortisol outliers across 10 participants out of the entire sample. For men, we observed six cortisol outliers across five participants out of the entire sample. For women, we observed 12 testosterone outliers across five participants (we had already excluded four of these because they were also cortisol outliers). In men, we observed seven testosterone outliers across seven participants (none of these were also cortisol outliers).

Before our main analyses, we tested whether testosterone, cortisol, perceived stress, partnered sexual desire, and solitary sexual desire changed over time, on average (see data supplement for details). For these models, we specified random slopes for time and random intercepts for subjects. For these and for the models we present in our main analysis, degrees of freedom were calculated based on the Satterthwaite approximation. As indicated by the fixed coefficient for time in each model, we found no evidence that men's testosterone, cortisol, perceived stress, partnered sexual desire, or solitary sexual desire significantly changed over time. We also did not find evidence that women's cortisol, perceived stress, partnered sexual desire, or solitary sexual desire changed over time. However, we found evidence that women's testosterone significantly increased over time, $\beta = 0.25$, 95% CI [0.07, 0.43], $t(24.18) = 2.88$, $p = .008$ (see data supplement for model details).

Overall, because there was no evidence that multiple variables of interest changed over time, we assessed how testosterone, cortisol, perceived stress, partnered sexual desire, and solitary sexual desire were associated on average across time points for women and men. Accordingly, we examined how levels of these variables were associated over time but, given that they were largely static, we did not examine how changes in them were associated.

Results

Descriptive Analyses

Testosterone Values

We tested whether men and women differed in average testosterone levels, as would be expected. For these models, we regressed testosterone values onto the categorical gender/sex variable and the time variable, and we specified random slopes for time and random intercepts for subjects (see data supplement for details). As indicated by the fixed coefficients for gender/sex, men had significantly higher testosterone values than women, on average, $\beta = 55.78$, 95% CI [51.26, 60.30], $t(115.21) = 24.43$, $p < .001$. However, readers might interpret this effect with caution; the statistical model we used assumes that testosterone levels have a normal distribution in both samples and that the variances in both samples are equal, but the testosterone distributions for men and women in our sample were different (see data supplement for testosterone distributions and model residual plots).

Stress Measurements

We also tested whether women and men differed in cortisol levels and perceived stress. For these models, we regressed cortisol values or perceived stress scores onto the categorical gender/sex variable and the time variable, and we specified random slopes for time and random intercepts for subjects (see data supplement for details). As indicated by the fixed coefficients for gender/sex, women had significantly higher cortisol values than men, on average, $\beta = 0.32$, 95% CI [0.05, 0.59], $t(124.51) = 2.37$, $p = .02$. Women also scored higher on perceived stress, on average, than men, $\beta = 2.21$, 95% CI [0.30, 4.12], $t(122) = 2.30$, $p = .02$.

Solitary and Partnered Sexual Desire

For descriptive purposes, we tested whether men and women differed in partnered and solitary sexual desire. For these models, we regressed sexual desire subscale scores onto the categorical gender variable and the time variable, and we specified random slopes for time and random intercepts for subjects (see data supplement for details). As indicated by the fixed coefficients for gender, men reported significantly higher partnered sexual desire, $\beta = 5.65$, 95% CI [1.73, 9.57], $t(132.69) = 2.85$, $p = .005$, and solitary sexual desire (natural log-transformed due to skew), $\beta = 0.57$, 95%

CI [0.31, 0.82], $t(132.75) = 4.43$, $p < .001$, than women. For women, solitary and partnered desire scores were positively correlated, $r(327) = .45$, $p < .001$. This was also true for men, $r(254) = .28$, $p < .001$. Additionally, the difference between the correlations for women and men was statistically significant, $z = 2.35$, $p = .019$, such that solitary and partnered desire were more weakly correlated in men than in women.

We then fit a series of mixed effects models predicting partnered and solitary sexual desire separately for men and women. We specified all models with and without random slopes for time (see data supplement for details). In all but one of the models in which we specified random slopes for time, the variance partitioning coefficient (VPC; West et al., 2014) for the random slope accounted for less than one percent of the random variance in the sexual desire subscale scores. Moreover, when we inspected plots of these models' random effects estimates for time and subject, nearly all their 95% prediction intervals captured zero. This indicated that including random slopes for time was reasonable because time was a meaningful variable in the study's design (Barr, Levy, Scheepers, & Tily, 2013); however, it also could be reasonable to exclude random slopes for time because the random effect for time accounted for so little observed variance in sexual desire; it was possible that time contributes close to zero variance. Neither of these approaches is better justified in general so, in the spirit of parsimony (i.e., estimating fewer parameters to increase power; Bates, Kliegl, Vasishth, & Baayen, 2015), we report models that excluded random slopes for time. The interested reader can examine models that include these random slopes in our data supplement.

Main Analyses

Given the high proportion of observed random variance due to subjects (see percentage of variance in Table 3), it would be inappropriate to analyze our data using a fixed-effects-only model (i.e., assuming every participant has the same average). For the remaining analyses, we used linear mixed models to account for random variance due to subjects.

Partnered Sexual Desire

Model 1

Women with higher perceived stress reported significantly lower partnered sexual desire, on average, $p = .009$ (Table 3). There was no significant association between cortisol and partnered sexual desire, $p = .341$. We also found that women with higher testosterone reported significantly lower partnered sexual desire, on average, $p = .003$, replicating previous findings (van Anders, 2012). The association between testosterone and partnered sexual desire did not significantly depend on cortisol or perceived stress, and including both these interaction terms with testosterone did not significantly improve model fit (AIC = 1802.0 vs. 1804.2; BIC = 1852.2 vs. 1861.6; logLik = − 887.00 vs. -8887.09), $\chi^2(2) = 1.80$, $p = .406$, so we excluded them.

Model 2

In contrast to the findings with women, men with higher perceived stress reported significantly higher partnered sexual desire, on average, $p = .039$ (Table 3). However, like with women, there was no significant association between cortisol and partnered sexual desire, $p = .578$. These relationships did not significantly depend on testosterone, and including both interaction terms with testosterone did not significantly improve model fit (AIC = 1518.5 vs. 1522.0; BIC = 1562.7 vs. 1573.0; logLik = − 746.22 vs. − 746.00), $\chi^2(2) = 0.4483$, $p = .799$, so we excluded them. The lack of a significant association between testosterone and partnered desire in men replicated previous findings (van Anders, 2012).

Solitary Sexual Desire

Model 3

Across the study, a large number of women (55.8%) reported the lowest amount of solitary sexual desire possible (which replicates previous findings, van Anders, 2012); no transformation produced values that would satisfy linear regression assumptions. Thus, to assess women's solitary sexual desire, we binned their values into two categories: some desire (solitary sexual desire score > 2) and the lowest possible desire (solitary sexual desire score = 2) as has been done previously (van Anders, 2012). We modeled this outcome in a mixed effects logistic regression. We did not find a significant association between women's solitary sexual desire and perceived stress, $p = .11$, or cortisol, $p = .34$ (Table 3). However, a statistical trend showed an interaction between perceived stress, testosterone, and the probability of reporting some solitary sexual desire, $p = .063$. Specifically, women with higher levels of testosterone who also reported more stress were more likely to report some solitary sexual desire rather than practically none (see Fig. 1 and Table 4 for simple slopes). We found no significant interactions with cortisol, and including this interaction term with cortisol did not significantly improve model fit (AIC = 222.53 vs. 223.89; BIC = 273.01 vs. 277.97; logLik = − 97.263 vs. − 96.943), $\chi^2(2) = 0.639$, $p = .424$, so we excluded it.

Model 4

For men, we addressed skewness in solitary sexual desire by log-transforming their levels. Main effects showed that

Table 3 Fixed effect and random effect estimates predicting desire from perceived stress, cortisol, and testosterone in women and men

Fixed effect	Coefficient (SE)	*t* value	*df*	*p* value
Model 1: Partnered desire predictors in women				
Intercept	20.69 (3.07)	6.74	250.24	<.001*
BMI	2.23 (1.21)	1.85	58.78	.069†
Time since waking	0.22 (0.37)	0.59	202.15	.559
Single relationship status	6.97 (1.41)	4.95	247.37	<.001*
Casual relationship status	2.27 (1.26)	1.81	223.37	.072†
Minor illness	− 4.84 (2.88)	− 1.68	199.10	.094†
Major illness	0.34 (1.01)	0.34	213.98	.737
Contraceptive use	2.00 (2.01)	0.99	238.01	.321
Session number	0.05 (0.12)	0.43	205.95	.665
Cortisol	0.47 (0.49)	0.96	210.73	.341
Perceived stress	− 1.40 (0.53)	− 2.64	227.23	.009*
Testosterone	− 10.29 (3.42)	− 3.01	222.51	.003*
Random effect	σ	% of variance		
Subject				
Intercept	10.28	80.80%		
Residual	5.01	19.20%		
Model 2: Partnered desire predictors in men				
Intercept	32.96 (1.61)	20.49	142.02	<.001*
BMI	0.4 (1.11)	0.36	59.60	.721
Time since waking	− 0.15 (0.45)	− 0.34	171.61	.732
Single relationship status	10.47 (1.57)	6.66	203.47	<.001*
Casual relationship status	5.58 (1.67)	3.33	199.15	.001*
Minor illness	− 7.87 (6.18)	− 1.27	158.96	.205
Major illness	− 0.19 (1.22)	− 0.15	168.08	.877
Session number	− 0.14 (0.14)	− 0.98	173.29	.330
Cortisol	0.33 (0.60)	0.56	180.24	.578
Perceived stress	1.21 (0.58)	2.08	208.63	.039*
Testosterone	0.41 (0.73)	0.56	185.71	.578
Random effect	σ	% of variance		
Subject				
Intercept	7.65	65.40%		
Residual	5.56	34.60%		
Fixed effect	Coefficient (SE)	*z* value	–	*p* value
Model 3: Log-odds of solitary desire predictors in women				
Intercept	1.79 (2.81)	0.64	–	.522
BMI	2.58 (1.36)	1.9	–	.057†
Time since waking	0.51 (0.35)	1.48	–	.139
Single relationship status	0.19 (0.96)	0.2	–	.841
Casual relationship status	− 0.67 (1.05)	− 0.64	–	.522
Minor illness	− 15.55 (9533.07)	0	–	<.001*
Major illness	− 0.43 (0.8)	− 0.54	–	.589
Contraceptive use	− 0.64 (1.26)	− 0.51	–	.610
Session number	− 0.04 (0.11)	− 0.36	–	.719
Cortisol	0.42 (0.44)	0.95	–	.342
Perceived stress	3.47 (2.17)	1.6	–	.110
Testosterone	1.16 (3.17)	0.36	–	.719
Perceived stress × testosterone	5.02 (2.7)	1.86	–	.063†
Random effect	σ	% of variance		
Subject				
Intercept	5.04	100%		

Table 3 (continued)

Fixed effect	Coefficient (SE)	*t* value	*df*	*p* value
Model 4: Solitary desire predictors in men				
Intercept	1.92 (0.11)	17.79	136.35	<.001*
BMI	0.11 (0.08)	1.44	59.42	.154
Time since waking	0.04 (0.03)	1.34	161.04	.182
Single relationship status	0.01 (0.1)	0.12	205.15	.908
Casual relationship status	0.07 (0.1)	0.69	186.81	.491
Minor illness	− 0.52 (0.37)	− 1.41	150.82	.160
Major illness	0.15 (0.07)	2.01	157.54	.046*
Session number	0.02 (0.01)	1.81	161.74	.071†
Cortisol	0.15 (0.06)	2.47	178.39	.014*
Testosterone	0.01 (0.05)	0.13	177.14	.899
Perceived stress	0.11 (0.05)	2.13	181.47	.034*
Cortisol × testosterone	− 0.08 (0.03)	− 2.58	167.01	.011*
Perceived stress × testosterone	− 0.08 (0.04)	− 2.39	171.78	.018*
Random effect	σ	% of variance		
Subject				
Intercept	0.56	74.10%		
Residual	0.33	25.90%		

BMI, time since waking, cortisol, perceived stress, and testosterone are gender-mean-centered and standardized. For Model 3, coefficients represent associations between their predictors and the log-odds of experiencing at least some solitary sexual desire. Committed relationship was a reference category in all models

*$p < .05$.

†Statistical trend at $p < .10$

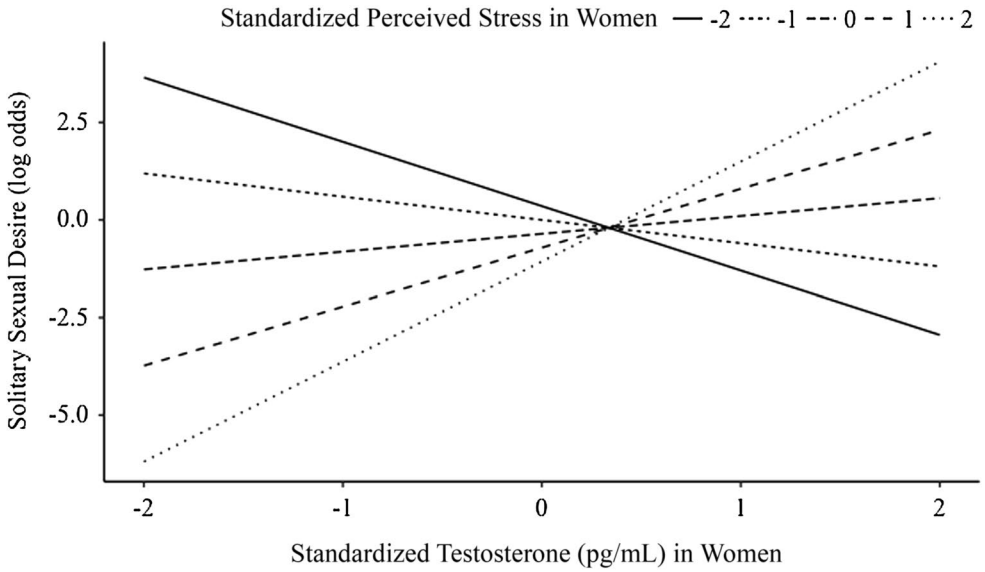

Fig. 1 In women, the association between testosterone (standardized with respect to the mean for women) and solitary sexual desire (log odds) moderated by perceived stress (standardized with respect to the mean for women). Lines represent linear relations at different values of perceived stress (PSS)

men with higher cortisol reported significantly more solitary sexual desire, on average, $p = .014$; and men with higher perceived stress reported significant more solitary desire, on average, $p = .034$ (Table 3). Both cortisol and perceived stress had significant interactions with testosterone. That is, men with higher cortisol and testosterone had significantly less solitary sexual desire, $p = .011$; and men with higher perceived stress and testosterone had significantly less solitary sexual desire, $p = .018$ (see Figs. 2 and 3 and Table 5 for simple slopes).

Table 4 Women: simple slope estimates at levels of perceived stress for the association between testosterone and solitary sexual desire

Perceived stress level	Coefficient (SE)	*z* value	*p* value
− 3.0	− 11.6 (5.99)	− 1.94	.052
− 2.5	− 9.08 (4.65)	− 1.96	.050
− 2.0	− 6.57 (3.31)	− 1.99	.047
− 1.5	− 4.06 (1.98)	− 2.05	.040
− 1.0	− 1.55 (0.75)	− 2.08	.038
− 0.5	0.96 (0.91)	1.06	.289
0.0	3.47 (2.17)	1.6	.110
0.5	5.99 (3.50)	1.71	.087
1.0	8.50 (4.84)	1.76	.078
1.5	11.01 (6.18)	1.78	.075
2.0	13.52 (7.53)	1.8	.072
2.5	16.03 (8.87)	1.81	.070
3.0	18.54 (10.22)	1.81	.070

Perceived stress is gender-mean-centered and standardized. Coefficients represent relationships between testosterone and the log-odds of experiencing at least some solitary sexual desire at different standardized values of perceived stress

Discussion

In the present study, we examined how testosterone, cortisol, and perceived stress were linked with partnered and solitary sexual desire in women and men on average over multiple time points. Our results supported other previous one-time findings and theory that challenged common assumptions about desire–testosterone links. Specifically, we found that testosterone was negatively associated with partnered desire in women and that there was no evidence of an association in men. We also found that stress, especially psychological stress, was relevant to partnered desire on its own in women and men. While testosterone was not a sole predictor of probability of solitary desire in women or solitary desire in men on its own, it did interact with measures of stress to predict solitary desire. Ultimately, our findings largely supported expectations based on the S/P theory (van Anders et al., 2011) and may justify modifications to the dual hormone hypothesis (Mehta & Josephs, 2010).

Partnered Sexual Desire, Testosterone, and Stress

Hypothesis 1 that higher testosterone would be associated with lower partnered desire in women but not men was supported. Hypothesis 2, however, was not supported; neither perceived stress nor cortisol moderated associations between partnered desire and testosterone in women or men. This was unexpected given previous evidence that suggests a negative correlation between testosterone and partnered desire but only in women who have higher levels of stress (van Anders, 2012). Still, our results reinforce the basic finding that testosterone and partnered desire are negatively correlated, and in women only. The negative association we found between partnered sexual desire and testosterone clearly contradicts popular discourse and non-empirical scientific assumptions, but fits with existing empirical data (Mazur et al., 2002; Sadowsky et al., 1993; van Anders, 2012; van Anders & Dunn, 2009).

Hypothesis 3 posited that cortisol and perceived stress would be negatively linked with partnered desire in women and men. This was partially supported in women and partially reversed in men: In women, perceived stress was negatively associated with partnered desire. In men, surprisingly, perceived stress was positively associated with partnered desire

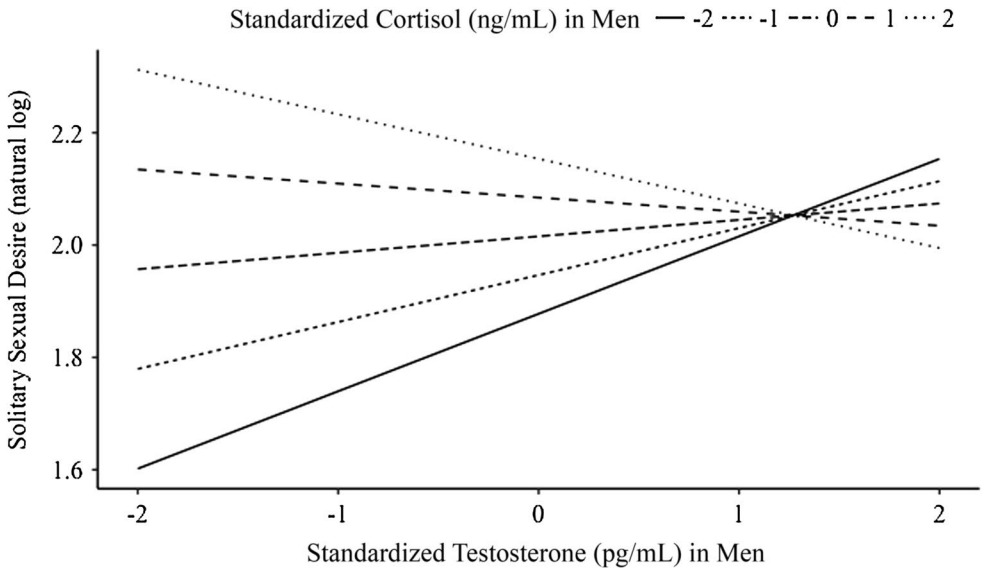

Fig. 2 In men, association between testosterone (standardized with respect to the mean for men) and solitary sexual desire (natural log) moderated by cortisol (standardized with respect to the mean for men). Lines represent linear relations at different values of cortisol

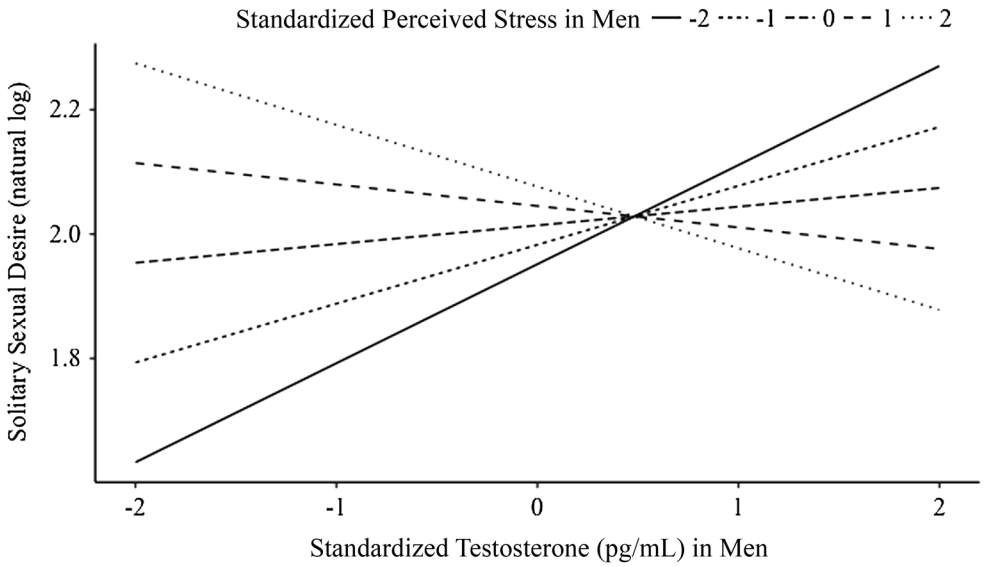

Fig. 3 In men, the association between testosterone (standardized with respect to the mean for men) and solitary sexual desire moderated by perceived stress (standardized with respect to the mean for men). Lines represent linear relations at different values of perceived stress (PSS)

(though this fits with some research on negative mood facilitating sexuality in some men) (Carvalheira & Traeen, 2014; Janssen et al., 2013). These findings suggest that gender/sex is implicated in the links between stress and partnered desire, as the associations differed by gender/sex in ways yet to be understood. One possibility is that gender/sex divergences in the ways that partnered desire and psychological stress are linked reflect different kinds of stressors. Regardless of origin, this gender/sex divergence in the effects of stress on partnered desire could mean that stressful times may have opposing effects on sexual desire within heterosexual couples, which may have ramifications for relationship satisfaction and quality.

Our hypothesis that cortisol would be negatively linked with partnered desire was not supported in women or men. The lack of links between partnered desire and cortisol is perhaps surprising, given the presence of significant associations between partnered desire and *psychological* stress in women and men. This suggests that self-report or psychological stress may be a more useful measure of stress when it comes to sexual desire. However, researchers have often turned to physiological measures as more objective, or reflective of actual experiences, than self-report measures. When it comes to stress and desire, self-reported stress may be more critical than cortisol, however, challenging the primacy of endocrine over psychological methods.

Solitary Sexual Desire, Testosterone, and Stress

In women, we hypothesized that testosterone and solitary sexual desire would be positively linked (Hypothesis 4) and that stress, both psychological and endocrine (cortisol), would be positively associated with solitary desire in women and men (Hypothesis 5). Our hypotheses were partially supported. A statistical trend showed that testosterone and solitary desire were positively linked in women, but only in those with high levels of perceived stress. For women with lower levels of perceived stress, testosterone negatively impacted the probability of solitary desire. In men, we observed that, at levels of stress at/below the mean for both cortisol and perceived stress, the association between testosterone and solitary desire was more positive than the association between testosterone and solitary desire at higher levels of stress. In other words, testosterone and solitary desire were more likely to be positively linked in men with low stress. Thus, both for women and men, the link between testosterone and solitary desire was moderated psychological stress, but the direction of these links differed by gender/sex. Similarly, gender/sex mattered for cortisol, which moderated the association between testosterone and solitary desire in men but not women.

While stress was not linked directly with solitary sexual desire in women or men, the interaction effect observed speaks to its importance for desire. However, cortisol seems to be less relevant to desire in women than in men, given the nonsignificance of the interaction term in women. It is also important to note that, while we interpret our results as stress moderating the solitary desire–testosterone associations, this interaction could also be described as testosterone levels moderating the desire–stress associations. Together, these variables interact when predicting solitary desire in women and men.

Table 5 Men: simple slope estimates at levels of perceived stress and cortisol for the association between testosterone and solitary sexual desire

	Coefficient (SE)	*t* value	*p* value
Perceived stress level			
−3.0	0.26 (0.1)	2.51	.013
−2.5	0.22 (0.09)	2.48	.014
−2.0	0.18 (0.07)	2.4	.017
−1.5	0.13 (0.06)	2.21	.028
−1.0	0.09 (0.05)	1.81	.072
−0.5	0.05 (0.04)	1.08	.282
0.0	0.01 (0.05)	0.13	.897
0.5	− 0.04 (0.05)	− 0.67	.504
1.0	− 0.08 (0.07)	− 1.2	.232
1.5	− 0.12 (0.08)	− 1.52	.130
2.0	− 0.16 (0.1)	− 1.72	.087
2.5	− 0.21 (0.11)	− 1.86	.065
3.0	− 0.25 (0.13)	− 1.95	.053
Cortisol level			
− 3.0	0.24 (0.1)	2.36	.019
− 2.5	0.2 (0.09)	2.27	.024
− 2.0	0.16 (0.08)	2.13	.035
− 1.5	0.12 (0.06)	1.89	.060
− 1.0	0.08 (0.06)	1.51	.133
− 0.5	0.04 (0.05)	0.92	.359
0.0	0.01 (0.05)	0.13	.897
0.5	− 0.03 (0.05)	− 0.68	.497
1.0	− 0.07 (0.06)	− 1.3	.195
1.5	− 0.11 (0.07)	− 1.71	.089
2.0	− 0.15 (0.08)	− 1.96	.052
2.5	− 0.19 (0.09)	− 2.13	.035

Perceived stress and cortisol are gender-mean-centered and standardized. The outcome variable, solitary sexual desire, is natural log-transformed. We computed *p* values based on the degrees of freedom for the perceived stress × testosterone interaction term, $df = 171.78$, and the cortisol × testosterone interaction term, $df = 167.01$

The S/P Theory and Partnered Desire

Findings for partnered desire supported previous findings (van Anders, 2012) and, although we did not test nurturance and eroticism as components of our participants' partnered sexual desire, the results aligned with expectations and predictions based on the S/P theory (van Anders et al., 2011). For example, the negative association between partnered desire and testosterone was replicated in women (van Anders, 2012) and the absence of an association between partnered desire and T was replicated in men (Mazur et al., 2002; Sadowsky et al., 1993; van Anders, 2012; van Anders & Dunn, 2009). In the context of S/P theory, the negative association between partnered desire and T in women makes sense given the evidence that women's partnered sexual desire likely reflects more "nurturant" desires (not because women's sexual desire is more naturally nurturant, but for pragmatic reasons: Closeness is a more likely outcome of sex with men than orgasm is for most women) (Richters, de Visser, Rissel, & Smith, 2006) and nurturance is linked with lower T (Das, 2017; van Anders et al., 2011). But, why is there a lack of a positive association between testosterone and partnered desire in men? It is possible that, for men, partnered desire is more equally erotic and nurturant than commonly thought. Research supports this idea: For example, men's sexual fantasies show high proportions of nurturance (Goldey et al., 2014) and factors like partner focus and intimacy as well as eroticism are major components of sexual desire in men (Chadwick et al., 2017a, b). Clearly, assumptions about desire as solely erotic at all, and especially so in men, are countered by empirical evidence in ways that have broad implications for potential associations with testosterone, as per the S/P theory (van Anders et al., 2011), and for conceptualization of, and predictions about, sexual desire.

Solitary Desire, the S/P Theory, and the Dual Hormone Hypothesis

While the results for partnered desire follow patterns that are exhibited in other findings and supported by the S/P theory (van Anders et al., 2011), the findings for solitary desire were novel, new, and unexpected. One way to address them is to consider the findings in light of both the S/P theory and the dual hormone hypothesis. The findings about associations between solitary desire, testosterone, and stress in men follow patterns that mimic the dual hormone hypothesis (Mehta & Josephs, 2010). Yet, in women, the associations often follow patterns that show a *reversal* of the dual hormone hypothesis (Mehta & Josephs, 2010). Yet this "reversal" is potentially a misnomer, since these "reverse" associations have actually been shown in the previous literature (Denson, Ronay, von Hippel, & Schira, 2013; Welker, Lozoya, Campbell, Neumann, & Carre, 2014). Despite this other pattern failing to support the dual hormone hypothesis and instead opposing predictions, this mismatch between data and theory has not been considered as a theoretical challenge to the dual hormone hypothesis. Attending to and accounting for gender/sex may be crucial for understanding when and how the dual hormone hypothesis might apply to women (who are half the population that the theory is supposed to apply to), and the S/P theory provides ways to do so.

Limitations

While the longitudinal nature of our research design, inclusion of both psychological and physiological measures of stress, and consideration of sexual desire as multifaceted were key strengths of our research, there were multiple contextual variables that we did not measure that might be critical. For example, research has indicated that factors such as relationship satisfaction, mood, self-esteem, body image, and age have robust effects on partnered and solitary sexual desire in women and men (Goldey et al., 2016; Janssen, McBride, Yarber, Hill, & Butler, 2008; see Mark & Lasslo, 2018 for an overview; Regan & Dreyer, 1999); and, although we controlled for more variables than many hormone studies, we were not able to control for all of the potentially relevant contributors to sexual desire. Future research could provide deeper insight into the associations between hormones and sexual desire over time by also assessing these factors. Additionally, research suggests that masturbation might be important for associations between testosterone and desire (van Anders, 2012), but the impact of masturbation frequency was not examined in these analyses. We also did not measure multifaceted sexual desire apart from solitary and partnered facets, but research from our laboratory and others is demonstrating how critical this can be to understanding desire (Chadwick et al., 2017a). Our sample was also relatively young; thus, the length of time in a committed relationship (if participants were in one) was likely shorter than that of older individuals, which could be meaningful for experiences of sexual desire. Future research should include a broader age range to assess the effects of differences in relationship experiences. Furthermore, we only had a limited number of sexual minority participants (5.0%, $n = 6$) and no gender minority participants, which precluded exploring these experiences. Our measures of gender/sex were limited to identity, and so issues of femininity and masculinity were not addressed but are likely to be informative, especially in relation to issues of gender norms. We also note that we assessed participants' BMI at the baseline session only; because it was correlated with testosterone and cortisol at all time points, we included this baseline measurement as a covariate in all models; however, it is possible that participants' BMI may have changed throughout the study in ways that affected testosterone and cortisol data. Finally, though it is complex to estimate power for a statistical design like this, post hoc power analyses suggested that our effects were likely underpowered (at estimates of 20% power; see data supplement for details); thus, our findings should be interpreted with this in mind. Including a larger sample size and increasing participant retention at each time point would increase confidence in the power of our results.

Conclusions

In the present study, we found that testosterone and partnered desire were linked in women but not men, replicating past findings (van Anders, 2012). Our research challenges, again, assumptions about testosterone that equate it to masculinity, men, and/or sexuality (van Anders, 2013). Moreover, it also provides a challenge to beliefs that testosterone equates to more desire, since women had lower testosterone at times of higher partnered desire.

Our results also highlight the importance of considering stress in research about testosterone and sexual desire. Here, we found that stress was a *predictor* of partnered desire and a *moderator* of solitary desire–testosterone associations. And, we observed that both endocrine and self-report measures influenced desire, with one sometimes more meaningful than the other. Self-report measures are often sidelined or seen as somehow less real than endocrine measures, but our research showed that self-report measures can be at least as, if not more, meaningful than hormones depending on the question.

The importance of gender/sex in understanding links between testosterone and desire has often been limited to culturally based assumptions that men have desire and women don't because men have testosterone and women's don't (or have low testosterone), despite research showing that gender/sex differences in testosterone do not account for gender/sex differences in desire (van Anders, 2012). Yet, desire might actually be experienced with some variability by gender/sex in ways that are relevant for hormonal associations. Assumptions that any differences in how women and men experience desire necessarily reflect innate, predetermined, or hormonally induced patterns is empirically problematic, as it assumes the direction of effect without testing it. Our research is suggestive that directional associations between testosterone and desire can only be understood within social context, since all desires are situated.

Acknowledgements These analyses used previously collected data from the Implication of Partnership Around the College Transition (ImPACT) in collaboration with Dr. Terri Conley and Dr. Divya Patel. Salivary assays were conducted at the Core Assay Facility, University of Michigan. Jessica C. Raisanen, M.S.P.H. is now affiliated with the Johns Hopkins Berman Institute of Bioethics in Baltimore, MD. Sari van Anders is now the Canada 150 Research Chair in Social Neuroendocrinology, Sexuality, and Gender/Sex, and Professor of Psychology, Gender Studies, and Neuroscience, at Queen's University, Kingston, ON, Canada.

Funding This study was funded by faculty discretionary funds.

Compliance with Ethical Standards

Conflicts of interest The authors declare that they have no conflict of interest.

Research Involving Human Participants All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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