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BRIEF REPORT

Salivary testosterone levels are associated with Compulsive Sexual Behavior (CSB) in men but not in women in a community sample

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ABSTRACT

Background and aims: Despite the inclusion of Compulsive Sexual Behavior (CSB) as a diagnostic entity in the ICD-11 and the increasing number of studies addressing psychological factors leading to its onset and maintenance, little is known about the role of hormonal factors when accounting for this clinical condition (especially in women). This study aimed to provide insights into the association between testosterone levels (i.e., the androgen more intimately linked to sexual desire and arousability) and CSB in both men and women. *Methods:* A total of 80 participants (40 men [*M*age = 22.31; *SD* = 2.93] and 40 women [*M*age = 21.79; *SD* = 2.06]) provided a saliva sample for the estimation of the level of free testosterone and completed a battery of measures assessing CSB and other related sexual domains (sexual sensation seeking and online/offline sexual behavior). *Results:* In men, salivary testosterone had a positive and significant correlation with three scales assessing CSB (*r* between 0.316 and 0.334). In women, these correlations were small and non-significant (*r* between 0.011 and 0.079). In both men and women, the level of salivary testosterone had small non-significant correlations with the other domains of sexual behavior assessed. *Discussion and conclusions:* Individuals' level of testosterone may contribute to the etiopathogenesis of CSB, but only in men. In women, alternative psychological –i.e., motivational, behavioral, or cognitive– processes may be playing a more central role in the expression of this condition.

KEYWORDS

Compulsive Sexual Behavior, sex hormones, testosterone, biological sex

INTRODUCTION

For some individuals, excessive and uncontrolled involvement in different sexual activities may become problematic and associated with severe problems in various aspects of daily living (Walton, Cantor, Bhullar, & Lykins, 2017), in many cases requiring the provision of mental healthcare to gain control over their sexual impulses (Antons et al., 2022). After a long discussion around the precise clinical formulation and categorization of this clinical presentation, the World Health Organization officially included the new category of "compulsive sexual behavior disorder" (CSBD) as an impulse-control disorder in the International Classification of Diseases (ICD-11) (Kraus et al., 2018). However, the dispute around its nosology and etiopathogenesis remains (Castro-Calvo et al., 2022).

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The recognition of Compulsive Sexual Behavior (CSB)[1] as an impulse-control disorder in the ICD-11 has been long debated since a competitive view is that this condition should rather be classified as a sexual disorder (Reed et al., 2022). According to this view, CSB represents the upper end of a continuum of sexual drive (i.e., a goal-oriented motivational state of wanting to have a sexual experience that leans individuals toward or pushes them away from sexual behavior [Levine, 2003; White & Acevedo, 2023]), in which hypoactive sexual desire constitutes the polar opposite. This was the approach behind the failed attempt to include CSB –operationalized as "Hypersexual Disorder"– as a sexual disorder in the DSM-5 (Kafka, 2010), or behind the outdated category of "excessive sexual drive" from the ICD-10 (unlike the current ICD approach, also categorizing CSB in the chapter on sexual dysfunctions) (Briken, 2020). At a theoretical level, three models capture this view: the «Dual Control Model» (Bancroft & Vukadinovic, 2004), the «Sexhavior Cycle» (Walton et al., 2017), and the more recent «Integrated Model of CSBD» (Briken, 2020). The Dual Control Model proposes that CSB is the result of an increased sexual excitation together with a decreased sexual inhibition. Similarly, the Sexhavior Cycle suggests that a biologically heightened sexual urge constitutes the substrate triggering subsequent stages in the cycle of out-of-control sexual behavior. Finally, the Integrated Model of CSBD argues that individuals with CSB experience an imbalance between sexual inhibition and excitation attributable to multiple pathways (among which testosterone is considered as a prerequisite for CSB) (Briken, 2020). Recent attempts to identify cohesive subcomponents of CSB have concluded that this condition comprises two correlated but distinguishable dimensions: problematic sexuality (including essential features of CSB such as the lack of control over sexual behavior or the use of sex as a coping mechanism) and sexual drive (related to high sexual excitement and more erotophilic tendencies) (Knight & Du, 2021). Even when this approach recognizes the presence of different pathways to CSB, an increased expression of sexual arousal and desire is still considered a primary component of CSB.

If these theoretical models were correct and sexual drive is playing a central role in the etiopathogenesis of CSB, then factors affecting the expression of an increased sexual excitation would be of relevance for the understanding of this condition. Among these factors, sex hormones –in particular, free testosterone (or the level of circulating bioavailable testosterone [Shea, Wong, & Chen, 2014])– may be playing a central role (Chatzittofis et al., 2022). Testosterone is an essential biomarker of sexual development and differentiation (particularly in men), but also one of the most important sex hormones during adulthood (Shea et al., 2014). The impact of testosterone in the expression of both normal and pathological sexual desire and arousability is well known (Bancroft, 2005). As CSB is characterized by an increased sexual drive that goes beyond individuals self-control (Knight & Du, 2021), thus testosterone would constitute a putative contributor to CSB (Briken, 2020). According to Chatzittofis et al. (2022), the level of free testosterone may influence on the etiopathogenesis of CSB through their role in the hypothalamus-pituitary–gonadal (HPG) axis: the HPG axis is crucial in the regulation of sexual desire, with testosterone acting directly and indirectly (through the conversion to estradiol) on its regulation. An imbalance in the level of free testosterone may result in a dysregulation of sexual desire, potentially leading to the symptoms of hypersexual desire and out-of-control sexual behavior that characterize CSB (Chatzittofis et al., 2022). However, to date, only two studies have explored the association between testosterone and CSB. Rodríguez-Nieto, Dewitte, Sack, and Schuhmann (2021) found correlations ranging from 0.34 to 0.51 between salivary testosterone and CSB in a sample of 69 healthy young men. On the other hand, Chatzittofis et al. (2020) did not find significant differences in the level of plasma testosterone after comparing 69 men with CSB and 39 healthy controls. However, correlations between plasma testosterone and CSB were near significance in the total sample ($r = 0.24$) and significant in the subsample of men with CSB ($r = 0.28$). A third study indirectly addressed the role of testosterone in CSB by testing the effect of testosterone suppression on its manifestation in a sample of men with pedophilic interests (Landgren, Olsson, Briken, & Rahm, 2022). These authors found that testosterone withdrawal had a significant impact on the reduction of CSB symptoms at ten weeks of initiating the treatment. However, this treatment also reduced sexual desire, rising questions about its specificity.

All the studies conducted so far providing insights into the association between the level of testosterone and symptoms of CSB comprised male samples, meaning that the association between testosterone and CSB in women remains unknown. Given this gender gap and the preliminary nature of available results, the aim of this research was to explore the association between testosterone levels and CSB in both men and women. This study may contribute to the development of a recent and promising area of research related to the neurochemical and hormonal contributors to CSB (Chatzittofis et al., 2022).

METHODS

Participants and procedure

Data acquisition for this study took place over 2015–2016. Participants were recruited from a database of subjects that one year ago had taken part in a larger data collection effort

[1]Note that the clinical diagnosis of CSBD represents the upper end of a continuum of severity and impairment, but taxometric studies have demonstrated that individual differences in out-of-control sexual behavior are dimensional (i.e., a matter of degree instead of a matter of kind) (Graham et al., 2016). This finding suggests that data derived from self-report scales of CSB, particularly when administered in community samples (as in this research), are better treated from a dimensional approach and may be understood as an indicator of the proneness to display out-of-control sexual behaviors. Therefore, in this study, we use the term CSB instead of its pathological counterpart to refer to this dimensional approach to the study of out-of-control sexual behavior in a community sample.

examining the sociodemographic, sexual, and clinical profile of adults with and without CSB (Castro-Calvo, Gil-Llario, Giménez-García, Gil-Juliá, & Ballester-Arnal, 2020). A random sample of participants in this previous research was selected and received an invitation to participate in the present study. Those who expressed their interest and met the inclusion criteria (age between 18 and 30 years old and without medical conditions or pharmacological treatments that could affect testosterone levels) completed an in-lab assessment. A total of 40 men between 18 and 29 years old (M = 22.31) and 40 women between 18 and 28 years old (M = 21.79) participated in the study (see Table 1 for a description of sociodemographic and sexual characteristics).

To avoid variations due to natural daily oscillations of testosterone, participants were assessed between 09:00–11:00 am. At arrival, participants signed consent form, completed a paper-and-pencil assessment battery, and provided a saliva sample. To ensure the reliability and validity of testosterone measurements, participants were asked to refrain from using alcohol or coffee for 12 h before the collection of saliva sample, as well as from eating, smoking, drinking, chewing gum, or brushing their teeth for 60 min before. Average time

Table 1. Participants' characteristics

	Men (n = 40) % (n) or M (SD)	Women (n = 40) % (n) or M (SD)	Inferential statistic	Size effect
Sociodemographic data				
Age (range 18–29)	22.31 (2.93)	21.79 (2.06)	t = −0.89	d = 0.20
Steady partner (yes)				
Single	38.5% (15)	7.7% (3)	X^2 = 12.26**	V = 0.39
Steady partner	48.7% (19)	84.6% (33)		
Casual relationships	12.8% (5)	7.7% (3)		
Sexual orientation				
Straight	84.6% (33)	89.7% (35)	X^2 = 10.05**	V = 0.36
Bisexual	0% (0)	10.3% (4)		
Gay/lesbian	15.4% (6)	0% (0)		
Testosterone				
Salivary testosterone (pg mL^{-1})	63.59 (27.09)	38.68 (17.81)	t = 4.79***	d = 1.08
Compulsive Sexual Behavior				
HBI Total score (range 19–95)	37.38 (15.17)	28.26 (11.01)	t = 3.04**	d = 0.68
CSB diagnosis according to the HBI cut-off score[a]	17.1% (7)	5.1% (2)	X^2 = 3.14	V = 0.20
SCS Total score (range 10–40)	16.28 (6.52)	12.67 (4.56)	t = 2.83**	d = 0.64
CSB diagnosis according to the SCS cut-off score[b]	15.4% (6)	5.1% (2)	X^2 = 2.22	V = 0.17
SAST Total score (range 0–25)	6.33 (4.91)	2.49 (3.50)	t = 3.97***	d = 0.90
CSB diagnosis according to the SAST cut-off score[c]	15.4% (6)	5.1% (2)	X^2 = 2.22	V = 0.17
ISST Total score (range 0–25)	9.49 (4.32)	4.44 (3.11)	t = 5.91***	d = 1.34
Sexual dispositional traits				
SSSS Total Score (range 11–44)	28.62 (6.86)	24.28 (6.64)	t = 2.83**	d = 0.64
Offline and online sexual behavior				
Lifetime number of sexual partners	7.54 (7.96)	4.82 (4.65)	t = 1.82	d = 0.41
Frequency of sexual activity (including masturbation)				
<6 times per year	2.6% (1)	0% (0)	X^2 = 19.03**	V = 0.464
Around 1 time per month	0% (0)	2.6% (1)		
2 or 3 times per week	0% (0)	2.6% (1)		
1 time per week	2.6% (1)	23.1% (9)		
2 or 3 times per week	17.9% (7)	38.5% (15)		
>3 times per week	76.9% (30)	33.3% (13)		
Average frequency among those reporting >3 times per week[d]	7.70 (4.85)	6.46 (2.66)	t = 0.86	d = 0.31
Minutes per week devoted to online sexual activities	133.07 (113.30)	26.15 (37.17)	t = 5.59***	d = 1.26

Note: Some of the variables included missing data, thus explaining that the sum of participants' responses in some variables do not add up to 40. HBI: Hypersexual Behavior Inventory; SCS: Sexual Compulsivity Scale; SAST: Sexual Addiction Screening Test; ISST: Internet Sex Screening Test; SSSS: Sexual Sensation Seeking Scale; [a] = HBI score ≥53 (Reid et al., 2011); [b] = SCS score ≥24 (Parsons, Bimbi, & Halkitis, 2001); [c] = SAST score >13 (Carnes, 1989); [d] = participants reporting a sexual frequency >3 times per week were asked to report the exact number of orgasms achieved per week (total sexual outlet); *p < 0.05; **p < 0.01; ***p < 0.001.

to complete the study was around one hour and participants received 10€ as compensation.

Measures

Salivary testosterone. To measure endogenous levels of testosterone, participants provided a saliva sample (approximately 1 mL) via passive drool into a DiaMetra saliva collection device. Saliva samples were frozen immediately after collection and stored at −20 ºC until the end of the data collection period. After approximately one month of storage at −20 ºC, samples were packed on dry ice and shipped to an independent laboratory for the analysis of salivary free testosterone. Samples were assayed using a commercially available enzyme immunoassay technique (DiaMetra ELISA kit). The DiaMetra ELISA kit allows for the determination of testosterone in a range from 10 pg mL^{-1} to 1,000 pg mL^{-1}. Intra- and inter-assay coefficients of variation were ≤8% and ≤13.2% respectively. As for its analytical sensitivity, the lowest detectable concentration of testosterone distinguishable from zero was 2.96 pg mL^{-1}. The validity of this method was further supported by its correlation with the results from another commercially available ELISA method (the DRG ELISA kit [r = 0.983]). All the assays passed the laboratory control and provided reliable results.

Questionnaires. Compulsive sexual behavior was assessed through the Spanish version of the Hypersexual Behavior Inventory (HBI, Ballester-Arnal, Castro-Calvo, Gil-Juliá, Giménez-García, & Gil-Llario, 2019, α = 0.95 [Spanish version]; Reid, Garos, & Carpenter, 2011 [English version]), the Sexual Compulsivity Scale (SCS, Ballester-Arnal, Gómez-Martínez, Gil-Llario, & Salmerón-Sánchez, 2013, α = 0.91 [Spanish version]; Kalichman & Rompa, 1995 [English version]), and the Sexual Addiction Screening Test (SAST, Carnes, 1983 [English version]; Castro-Calvo, Ballester-Arnal, Billieux, Gil-Juliá, & Gil-Llario, 2018, α = 0.88 [Spanish version]). Participants also completed the Internet Sex Screening Test (ISST, Ballester-Arnal, Gil-Llario, Gómez-Martínez, & Gil-Juliá, 2010, α = 0.84 [Spanish version]; Delmonico, Miller, & Miller, 2003 [English version]), a scale measuring out-of-control online sexual behavior. Participants' tendency towards sexual excitement and novel sexual experiences was measured by the Spanish adaptation of the Sexual Sensation Seeking Scale (SSS, Ballester-Arnal, Ruiz-Palomino, Espada, Morell-Mengual, & Gil-Llario, 2018, α = 0.87 [Spanish version]; Kalichman & Rompa, 1995 [English version]). Finally, participants were asked to report: (a) frequency of solo and partnered sexual activity (7-point Likert scale ranging from 1 ["*Less than 6 times per year*"] to 7 ["*More than three times per week*"]); (b) exact frequency among those who answered "*more than three times per week*" in previous question; and (c) time online for sexual activities (minutes per week).

Data analysis

All analyses were performed using SPSS (version 26.0) and G*Power (version 3.1). Descriptive analyses were first conducted to profile participants according to sociodemographic data, level of salivary testosterone, CSB, sexual sensation seeking, and online/offline sexual behavior. Student t-tests and chi-square tests were then used to investigate differences according to the biological sex in continuous and categorical variables. Cohen's d and Cramer's V were computed to estimate effect size of these differences. Correlational analyses (Pearson's r) were finally used to determine the association between the measures of sexual behavior collected and the level of salivary testosterone. Confidence Intervals (CI) for r' estimates were based on 1,000 bootstrapped samples.

Ethics

This study was approved by the ethics committee of the Jaume I University. Participants were informed about the study aim and provided informed consent.

RESULTS

Figure 1 shows correlations between salivary testosterone level and the different aspects of sexual behavior assessed, disaggregated by the biological sex. In men, scores on three measures of CSB had a positive and significant correlation with salivary testosterone: HBI (r = 0.322; p = 0.046), SCS (r = 0.334; p = 0.038), and SAST (r = 0.316; p = 0.050). As the CI for r intersected with zero in the correlations with the HBI (95% CI: −0.046, 0.610) and SAST (95% CI: −0.067, 0.630), these results should be interpreted with caution: although normal theory estimated p value was significant, bootstrapped CI suggests that the correlation between testosterone and these two measures of CSB may not be significant. The level of salivary testosterone had small non-significant correlations with the other domains of sexual behavior assessed (r between −0.016 and 0.192).

In women, the level of salivary testosterone did not significantly correlate with any of the aspects of sexual behavior assessed (r between −0.150 and 0.079).

DISCUSSION

This research aimed to explore the association between testosterone and CSB, an aspect that may contribute to the characterization of the factors leading to this condition. In line with previous observations, men scoring higher on CSB showed increased levels of salivary testosterone (r between 0.316 and 0.334). These figures resonate with those obtained by Rodríguez-Nieto et al. (2021) in a sample of healthy young men (r between 34 and 0.51) and by Chatzittofis et al. (2020) in a sample of men with CSB (r = 0.28), meaning that both the proneness to experience out-of-control sexual behaviors in men without CSB and the severity of the condition in clinical patients may be heightened by the presence of this hormonal disposition. As the levels of salivary testosterone did not correlate with other domains of sexual

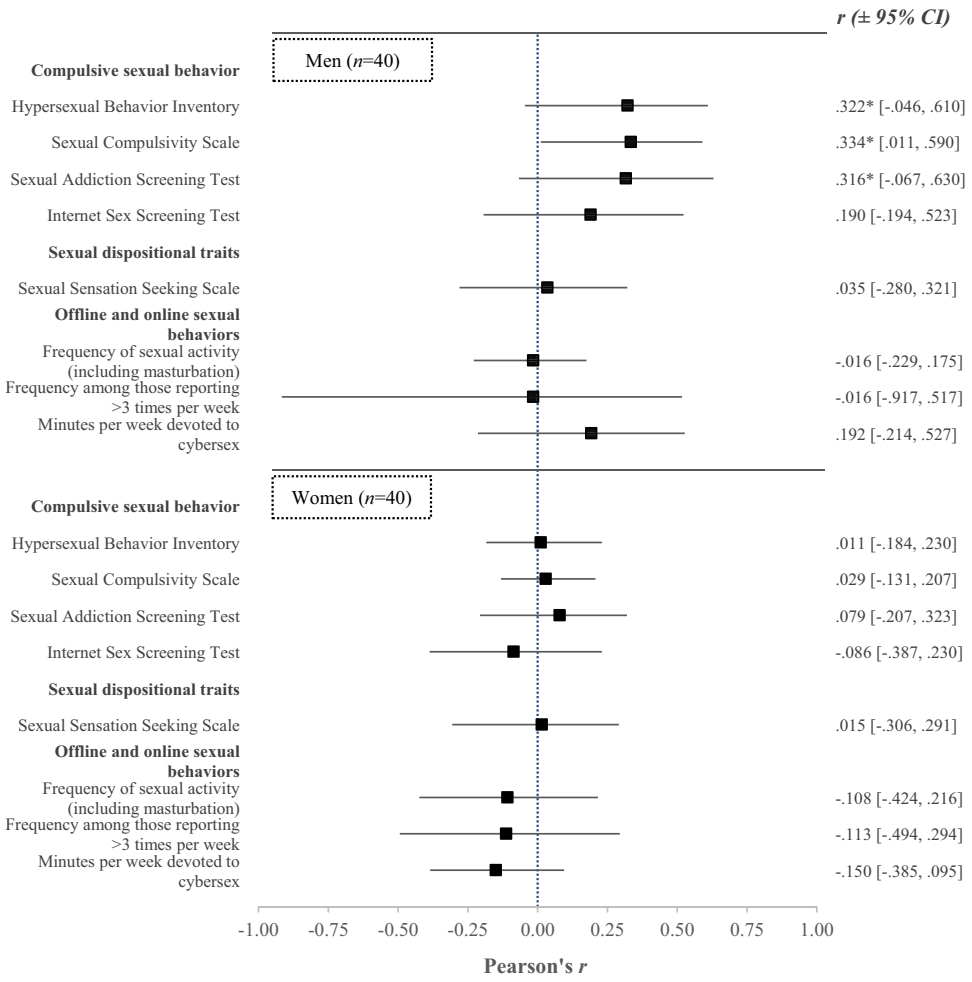

Fig. 1. Correlations between levels of salivary testosterone and the different aspects of sexual behavior assessed

behavior intimately linked to CSB (e.g., sexual sensation seeking or sexual frequency), we may infer that the contribution of this sex hormone when accounting for out-of-control sexual behavior is specific (i.e., our results support the unique contribution of testosterone to CSB proneness without the intervention in other sexual domains). This specificity resonates with contemporary approaches to the characterization of CSB, distinguishing two relatively independent subcomponents comprising this condition (Knight & Du, 2021): a subcomponent of "problematic sexuality" (as measured by most available screening scales, such as the SCS, HBI, and SAST) related to the level of salivary testosterone and a dimension of "sexual drive" (as measured by aspects such as sexual frequency or sexual sensation seeking) unrelated to the level of testosterone. This finding may seem paradoxical, as someone may expect that testosterone (a sex hormone intimately linked to sexual desire and arousability [Bancroft, 2005]) should be associated with sexual drive rather than with symptoms of lack control over sexual behavior. However, as pointed out by Chatzittofis et al. (2022), the HPG axis may interact with other endocrine systems (such as the hypothalamus-pituitary adrenal axis [HPA], highly influenced by the effect of cortisol), thus explaining why testosterone –a priori linked to high sexual desire– may play a significant role in the expression of symptoms of sexual disinhibition. Supporting this point, previous studies have found that testosterone is associated with CSB symptoms in the presence of low levels of cortisol (Chatzittofis et al., 2016, 2020).

That said, it is important to note that these results should be interpreted with caution: confidence intervals of the association between CSB and testosterone were wide, and in some cases (in particular, the association between testosterone and HBI and SAST scores), intersected with zero. This means that despite the significant association between testosterone and CSB, the influence of the former on the etiopathogenesis of CSB would be limited and unstable. Thus, in the best scenario (i.e., assuming that testosterone has an unquestionable positive association with CSB), testosterone should be considered one of the risk factors that, in conjunction with multiple other biopsychosocial factors, leads to the onset and maintenance of this condition (Briken, 2020). This observation warrants the exploration of the interaction between testosterone and other risk factors when it comes to explaining the clinical expression of CSB.

At a theoretical level, these findings support models proposing that one of the core features explaining CSB is the presence of an increased sexual drive

(Bancroft & Vukadinovic, 2004; Briken, 2020; Walton et al., 2017). However, as this study is correlational (and therefore, causality cannot be stablished), we cannot discard that heightened levels of testosterone are the result –and not the cause– of CSB. Supporting this alternative explanation, some studies have found that previous exposition to sexual stimuli may alter subsequent testosterone levels (Redouté et al., 2000). At a clinical level, our results should not be interpreted as a foundation for the use of testosterone suppression in CSB: whereas the level of testosterone has a direct and specific effect on CSB, the impact of testosterone suppression on reducing symptoms of CSB is mediated by an even more pronounced reduction of sexual interest in general (Landgren et al., 2022). As the therapeutic aim of treatments for CSB should be the recovery of normal sexual functioning rather than its suppression, antiandrogen therapy do not seem to constitute an advised therapeutic approach in patients without concomitant paraphilic or sexual-offending tendencies (Chatzittofis et al., 2022).

In women, salivary testosterone level did not correlate with CSB (r between 0.011 and 0.079). This finding dismisses the hypothesis of this sex hormone as a precursor of CSB in women, meaning that alternative psychological –i.e., motivational, behavioral, or cognitive– processes may be playing a more central role in the expression of this condition. This conclusion goes in line with the findings from recent systematic reviews showing that certain predisposing factors increasing the likelihood of CSB (e.g., the level of impulsivity or the presence of a previous sexual dysfunction) play a more relevant role in men (Kowalewska, Gola, Kraus, & Lew-Starowicz, 2020). Unfortunately, little is known about neurobiological and neurocognitive mechanisms leading to CSB in women (Kowalewska et al., 2018), but it may be also the case of testosterone. More generally, this conclusion resonates with alternative conceptualizations of female sexuality advocating for a limited role of biological underpinnings on females' sexual desire (Basson, 2000). Supporting this limited influence of sex hormones in women's sexual behavior, literature exploring the influence of testosterone in women's seems to be inconsistent and pointing out a limited impact (Bancroft, 2005).

This study is not without limitations. First, free testosterone level was measured using saliva samples. While research suggest that salivary testosterone is correlated with serum testosterone, this method is limited in several ways (Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004). Also related to testosterone measurement, female participants were not asked to report the phase of the menstrual cycle during the testosterone measurement. In women, salivary testosterone may vary depending on the menstrual cycle (with higher levels observed during mid-cycle) (Bui et al., 2013). While this aspect has a significant impact on the level of free testosterone, the size of its influence is limited (in the range between $\pm$ 0.3 pg mL^{-1}) (Salonia et al., 2008), leading some experts to conclude that "*the elevation of mid-cycle testosterone concentrations is statistically significant, although not clinically relevant*" (Bui et al., 2013, p. 96). This means that research replication controlling for the menstrual phase is warranted, yet major changes should not be expected in the study findings. Furthermore, the limited sample size prevents us to provide definitive conclusions on the relationship between CSB and testosterone, meaning that further research is required (especially in women). Finally, the low number of participants meeting the threshold for a CSBD diagnosis (between 5.1% and 17.1%) calls into question the generalizability of the results in clinical samples. As taxometric studies conclude that differences in out-of-control sexual behavior are dimensional (Graham, Walters, Harris, & Knight, 2016), we may expect that the same pathways –and variables– leading to higher levels of CSB would be involved in its pathological expression. However, studies with larger clinical samples are warranted to confirm this point.

CONCLUSION

Individuals' level of testosterone may contribute to the etiopathogenesis of CSB, but only in men. In women, alternative psychological –i.e., motivational, behavioral, or cognitive– processes may be playing a more central role in the expression of this condition.

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