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# Differences in sexual desire between women with clinical versus biochemical signs of hyperandrogenism in polycystic ovarian syndrome

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ABSTRACT

The role androgens play in female sexual desire remains unclear. We investigated whether androgen sensitivity or elevated androgen levels contributed to sexual desire using a motivational model of sexual desire. Eighty-five women diagnosed with polycystic ovary syndrome (PCOS) were categorized depending on whether they exhibited clinical symptoms of androgen sensitivity or high biochemical androgen levels. Additionally, instead of looking at desire as a uniform construct, we divided desire based on the reasons why women experienced desire, thus distinguishing desire to have sex for relational purposes from the desire to have sex for mating selection or physical pleasure. Findings confirmed that clinical signs suggesting sensitivity to androgen levels, but not biological levels of androgens per se predicted levels of sexual desire. Moreover, in agreement with our hypothesis, we found support for a relationship between androgen sensitivity and some, but not other aspects of sexual desire. Cues that are most closely related to mating selection were significantly associated with androgen sensitivity, but not cues associated with desiring sex to feel emotionally close or create a love bonding with a partner. This study presents a new way to investigate desire and shows some preliminary findings on the importance to consider androgen sensitivity when investigating the relationship between sexual desire and hormones.



## Introduction

The relationship between androgens and sexual desire continues to be unclear potentially due to error bias introduced by individual differences in androgen sensitivity between women, and by an oversimplistic conceptualization of sexual desire as a single cohesive phenomenon (e.g., Bancroft, 2003; Basson, 1999). The present study aimed to untangle the link between androgens and desire by including an analysis of androgen sensitivity and by introducing a more complex conceptualization of desire more in line with clinical observations (Levine, 2002). To this end, we studied women with polycystic ovary syndrome (PCOS) who exhibited clinical symptoms of hyperandrogenism (e.g., acne, hirsutism and alopecia) and/or had high biochemical levels of androgens (hyperandrogenemia). Individuals with high sensitivity to androgens may show hyperandrogenic symptoms despite relatively normal androgen levels. Conversely, some women may have less sensitivity to androgens and may not show clear signs of hypearandrogenism despite elevated androgen levels. The main question addressed by this study is whether differences in androgen sensitivity or biological androgen levels are more relevant for understanding the link between androgens and sexual desire. The second aim was to explore sexual desire utilizing a more sophisticated conceptualization that takes into consideration the co-existence of alternative biological pathways that ignite different motivations for sexual activity.

When it comes to understanding the relationship between androgens and female sexual behavior and function, the evidence is inconsistent. Numerous studies illustrate that testosterone administration enhances sexual desire in postmenopausal women (for a review, see Flöter, 2009). However, some studies also show a significant improvement in desire when estrogen alone is administered to menopausal women and adding testosterone does not add to the effects of estrogen (Davis et al., 1995) and other studies show that testosterone increases the effects of estrogen on desire (Flöter et al., 2002). Also, although findings point to a significant association between androgens and sexual function, the nature of this relationship remains unclear.

Scholars have argued that abnormally low levels of free testosterone (FreeT), are associated with very low sexual desire, a condition called Female Androgen Insufficiency (Bachmann et al., 2002). Although such condition has been criticized (Rivera-Woll et al., 2004) on the basis that the relationship between free testosterone and sexual desire is not clearly understood in the normal population, that measures of testosterone in women are not very sensitive and therefore include a large error variance, and that large, well-controlled studies have failed

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to find a relationship between androgens and desire in the normal population (Davis et al., 2005; Santoro et al., 2005).

A plausible explanation for the conflicting findings concerning the influence of androgens on sexual desire may lay in the different sensitivities women show to androgens. Graham et al. (2007) proposed that individual differences in sensitivity to androgen may explain why some women, more than others, experience greater sexual side effects to hormonal contraceptives. Common signs of heightened sensitivity to androgen levels include hirsutism, alopecia, and acne (Dunaif et al., 2008).

The operationalization of sexual desire could also provide an explanation for the unclear relationship between androgens and sexual desire. Currently, desire is considered the motivation for sexual behavior (for a review, see Pfaus, 2009). Measures of sexual desire focus on "how much sexual desire" the individual has despite the function of the behavior. However, women may desire sex for a number of different reasons. For example, some women may experience sexual desire because they are craving an emotional connection with a partner, while, at other times, they may be craving the physical pleasure derived from arousal and orgasm, and yet at other times it is desire activated by a general feeling of romance and passion. Despite these different types of scenarios and reasons that may lead a woman to desire sex, the current operationalization of desire remains focused on the interest to engage in sexual behaviors. This limited conceptualization has been criticized by clinicians who have observed different types of sexual desire (Levine, 2002, 2003) and by researchers that have identified a variety of reasons why people engage in sexual activities (Cooper et al., 1998; Meston and Buss, 2007). A useful quantification of different types of sexual desire is provided by a recently validated measure, the Cues of Sexual Desire Scale (McCall and Meston, 2006), a scale that distinguishes between four internally-consistent types of desire that can be grouped into two main categories: (1) desire in response to sexual arousal or external erotic cues, e.g., feeling sexual arousal, watching erotic material, or seeing someone attractive (Erotic/Explicit and Visual/Proximity cues), and (2) desire response to relationally-relevant cues, e.g., feeling connected to a partner, having a romantic dinner, wanting to experience greater intimacy (Romantic/Implicit and Love/Emotional cues). Given the proposed differentiation in motivations for sexual activities, it is feasible that different hormonal patterns control the different patterns and thus androgens may be involved in only some of sexual desire experienced by women.

Women with PCOS can be a useful population to use to study sexual desire and androgens for a number of reasons. Previous studies have either focused on natural changes in androgens that occur during menopause, the menstrual cycle, or changes induced by hormone replacement or hormonal contraception (for a review, see Basson, 2007; Bullivant et al., 2004; Davis and Tran, 2001; Wierman et al., 2010). Testosterone levels are small in women and hard to detect, potentially preventing the accurate and reliable observation of their influence on sexual desire. Moreover, it is unknown whether artificial levels of androgens can compare to endogenous levels in their effects on desire. Women with PCOS provide an alternative way to overcome these limitations because several of them experience naturally high levels of androgens and this facilitates the distinction between low and high androgens (Rotterdam ESHRE/ASRM-Sponsored PCOS ConsensusWorkshop Group, 2004). In this population, it is also possible to study sensitivity to androgens, which is characterized by clinical symptoms such as alopecia, acne, and hirsutism, symptoms that in the general population may be harder to detect or identify. Moreover the differences in androgens between women with PCOS are due to natural androgens and not artificially elevated androgens, thus allowing for a specific study of natural androgens. There are, however, some limitations in utilizing this sample, such as the fact that these women have a dysfunction and therefore may be essentially different from healthy women. Overall, given the lack of information on women with PCOS and sexual desire, as well as the limited knowledge on the relationship between desire and androgens, we felt this study would be able to provide useful insight into androgen mechanisms of sexual desire despite these limitations.

It is important to clarify that among women with PCOS, some show clinical signs of hyperandrogenemia, such as hirsutism, acne and alopecia, and have relatively high levels of androgens, while others exhibit these clinical features but show androgen levels within the normal range (Norman et al., 2007), thus allowing us to distinguish between the effects of high androgens and high sensitivity on sexual desire. In this study we differentiated between women showing and not showing at least one clinical sign of hyperandrogenism (i.e., hirsutism, alopecia, and acne), and between women with and without biochemical markers of hyperandrogenism (i.e., high biological levels of androgens = hyperandrogenemia). Based on the hypotheses proposed by Graham et al. (2007) on the importance of androgen sensitivity for the understanding of sexual desire, we expected that clinical hyperandrogenism (Clinical group vs. NonClinical groups), more than biological hyperandrogenemia (Biochemical vs. NonBiochemical), would positively predict desire. A second aim of this study was to determine whether some aspects of sexual desire are more closely connected to androgens than others. In particular, we expected that desire in response to Erotic/Explicit and Visual/Proximity cues, as identified in the Cues of Sexual Desire Scale (McCall and Meston, 2006), would be more closely associated with androgen sensitivity because these are aspects of desire that tend to be associated with the physiological and/or mating selection functions of sex. Conversely, we did not expect androgens to be associated to relationally-bound desire, such as Emotional/Bonding and Romantic/Implicit cues, a function we would expect to be under the control of processes linked with bonding and forming a long-term relationship.

## Material and methods

### *Participants*

A total of 85 women, aged 18 to 40 years ($M = 25.4$, $SD = 5.77$), on average 12.8 years post menarche ($SD = 5.43$) participated in the study. Participants were referred to the Outpatient Clinic of Gynecological Endocrinology of the University of Pavia between 2008 and 2009 because of oligomenorrhea (eight or fewer menstrual periods annually). According to NIH criteria and to Rotterdam criteria (Rotterdam, 2004), women were diagnosed with PCOS if at least one of the following was present in addition to oligomenorrhea: a clinical sign of hyperandrogenism, in particular hirsutism (defined as a Ferriman–Gallway score > 8), a biochemical sign of hyperandrogenism (hyperandrogenemia defined as FreeT > 3.6 pg/mL, and/or dehydroepiandrosterone sulphate [DHEAS] > 3.6 μg/mL, and/or androstenedione [A] > 3.1 ng/mL), or the presence of polycystic ovaries on ultrasound. Polycystic ovaries were determined by the presence of increased ovarian size and/or at least 12 follicular cysts measuring 2 to 9 mm (Norman et al., 2007). FreeT, A and DHEAS were chosen as markers of hyperandrogenism because, as previously reported in Italian PCOS samples (Battaglia et al., 2008; Fruzzetti et al., 2009), they significantly segregate patients from controls in routinely practice. Subjects with hyperprolactinemia, hypo- or hyperthyroidism, congenital adrenal hyperplasia, Cushing's syndrome, or androgen-secreting tumors were excluded from this study. No subject was using medication known to influence endocrine and lipid profiles, including hormonal contraceptives.

Within this study, women were categorized as either Clinical or NonClinical, depending on whether they showed clinical signs of sensitivity to androgens (hyperandrogenism), defined as signs of hirsutism, acne or alopecia (for a description of the assessment of these symptoms see below). Participants were also categorized as Biochemical or NonBiochemical, depending on whether they had naturally high levels of androgens (hyperandrogenemia), defined as FreeT > 3.6 pg/mL, DHEAS > 3.6 mg/mL, and/or A > 3.1 ng/mL. These groups were not