Authors’ Reply to the Letter to the Editor: “Sleep and sexual arousal: a complex issue”

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We would like to thank the esteemed colleagues for their interest in our paper published in the Journal of Sexual Medicine in January 20191. The results of our analysis on a large nationally representative sample of older adults in England provided epidemiological evidence to a well-known fact that sleep and sexual function, and their respective complexities, are intricate. This is something we were sure to highlight in our discussion section (p 431), as the regarded colleagues note in their letter to the editor (*insert reference after peer-review*).

The colleagues call for more attention to the associations between sleep deprivation and increased sexual arousal, the underlying mechanism of which could, at least in part, be explained by the interactions between dopaminergic and noradrenergic pathways2 3. However, this phenomenon is observed in a handful of animal model studies and to the best of our knowledge, only two studies on human participants have reported the same observations4 5. These studies were done on a relatively small number of participants (*n*=36 and *n*=275) and have several methodological issues. In the study by Ferini-Strambi et al.5, the authors observed a disinhibitory effect of sleep deprivation for men undergoing an audiovisual stimulation penogram (ASP) as means of diagnosing erectile-dysfunction. The authors concluded that effects of sleep deprivation prior to the ASP diagnostic procedure could lead to imprecise diagnoses in men reporting psychogenic impotence. The more recent, larger study by Costa et al.4, based on hypotheses from animal models, aimed to investigate associations of subjective sleep quality and self-reported unstimulated sexual arousal in young men and women. The authors evaluated a number of self-reported questionnaires on anxiety, sleep quality, sexual desire, and sexual practices, and measured salivary testosterone levels. They concluded that “…*consistently with other studies in humans and animals, the findings are congruent with the notion that lack of sleep can increase sexual arousal, but not frequency. Testosterone might play a role in the sexual arousal caused by a lack of appropriate sleep*”. Although this is a worthy effort to elucidate some of the complexities of the neuro-endocrine mechanisms in observational studies in humans, this study has several methodological limitations. The sample was not representative of the general population, consisting predominantly of university students (mean age reported as 23.08 and 24.06 years for women and men, respectively), where more than 60% of participants had a regular sexual partner for more than 3 years, and excluding people with same sex-preferences. Unfortunately, the authors supply no descriptive results for their measurements of anxiety, sleep quality or testosterone levels, which makes it difficult to understand the sample or the reasoning behind some of the analytic choices. Coincidentally, the authors offer no description of the statistical analyses done, but it appears there was no adjustment for potential confounding by variables known to be related to both sleep and sexuality (e.g. illness, alcohol use, levels of physical activity). The authors go on to report that unstimulated sexual arousal correlated with sleep problems among men and women with higher testosterone values. There were no significant correlations reported for sexual frequency or desire. Based on these limited analyses, the authors draw unsubstantiated conclusions on the connections between sleep problems, testosterone and sexual arousal. We therefore suggest these results are interpreted with caution and an understanding of the study’s limitations.

Consistent evidence shows that testosterone plays an important role in sexual arousal of adult men, with somewhat less clear evidence in older men, while in women the role of testosterone is more complex6. On the other hand, sleep problems and sleep deprivation have been shown to decrease levels of circulating testosterone in rat models, possibly due to increased serotonin that reduces the human chorionic gonadotropin expression of steroid acute regulatory protein in Leydig cells7. Due to the fact that most of the daily testosterone in men is released during sleep, sleep issues could plausibly lead to reduced testosterone levels8. A study by Leproult & Van Cauter9 reported that sleep restriction to 5h per night for one week in a small convenience sample of young healthy men led to a decrease of daytime testosterone levels by 10-15%, which may lead to signs of androgen deficiency. In older men, testosterone levels could partly be predicted by total sleep time10. Moreover, due to its effects on frontal lobe impairment, sleep deprivation may negatively influence decision making: one study reported that sleep deprivation leads to an increase in men´s perceptions of women´s interest and intent to have sex11. Although the paucity of research on the influence of sleep problems on testosterone levels in women prevents firm conclusions, a pilot study by Kalmbach et al.12 reported that women with longer than average sleep duration reported better genital arousal and greater sexual desire than those with shorter than average sleep duration.

In conclusion, as sleep deprivation influences sex steroid hormone production, unravelling the mechanisms of this interplay could have clinical implications – especially as sleep issues are becoming increasingly prevalent. Despite evidence of effects of circadian activity on androgens, research is still lacking. Therefore, we join our esteemed colleagues in their call for more research, both epidemiological and laboratory-based, into the complex interactions between the internal hormonal milieus, sleep quality and sexual outcomes.

**References**

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