Reply: Sleep efficiency in patients with polycystic ovarian syndrome

Sir,

Many thanks for your letter entitled ‘Sleep efficiency in patients with PCOS’, which addresses some concerns you have for our study entitled ‘Poor sleep in PCOS; is melatonin the culprit?’ (Shreeve et al., 2013).

Having reviewed your comments, please see our response below.

Your first concern was relating to the prevalence of obstructive sleep apnoea (OSA) being higher in patients with polycystic ovary syndrome (PCOS; Nandalike et al., 2012; Randeva et al., 2012) as a result of insulin resistance or metabolic abnormalities. There is minimal data to suggest that when compared with weight-matched controls, PCOS sufferers still exhibit higher levels of OSA (Ehrmann, 2012). While we acknowledge that OSA could potentially be caused by factors beyond simply raised BMI in these patients, e.g. metabolic abnormalities, there has been minimal high-grade evidence to confirm this theory. We do know, however, that around 70% of people in general with OSA have raised BMIs (Malhotra and White, 2002). This remains the most popular explanation for poor sleep in PCOS patients among clinicians. As we mentioned in our study, there was no observed difference in BMI between our control and study groups. Our paper was therefore aimed at delineating what these potentially endocrine/hormonal causes of poor sleep (not related to OSA) may be.

Furthermore, you raised concerns regarding sleep duration and elongated sleep latency being significantly correlated with increased levels of anxiety and depression (Argyriou et al., 2011). Although it was beyond the remit of the study to formally assess the psychological state of women with PCOS, we excluded patients with previous history of psychiatric disorders in our study.

Your final concern was of the actigraphy cut-off value of sensitivity for making sleep/wake judgement being initially set at 40 counts per minute. This statement was also provided without reference to any published study. We are unable to find any evidence to suggest that the cut-off value should be set according to each test situation by using sleep polysonography. There is also no recommendation from the manufacturers (Phillips) of the Actiwatch devices we used that the counts per minute settings needed any adjustment. There are a number of studies carried out in healthy adults to assess the validity of actigraphy for assessing sleep and wake against the gold standard of polysomnography and have found good agreement (97%) for total sleep time (Jean-Louis et al., 1996) and overall agreement rates of 91–93% (Sadeh et al., 1994; Jean-Louis et al., 2001; Ancoli-Israel et al., 2003).

We acknowledge the small sample size of this study and encourage further investigation into this novel area of reproductive endocrinology.

References


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doi:10.1093/humrep/det328

Advanced Access publication on September 17, 2013