5-2024

Examining the Effects of Menstrual Cycle Phase and Hormonal Contraceptive Use on Women's Sleep

Charles Ethan Coombs
University of Arkansas, Fayetteville

Examining the Effects of Menstrual Cycle Phase and Hormonal Contraceptive Use on Women's Sleep

An Undergraduate Honors Thesis

in the

Department of Psychological Science
Fulbright College of Arts and Sciences
University of Arkansas
Fayetteville, AR

by

Charles Ethan Coombs

Abstract

Women overrepresent men for sub-optimal sleep, a consequence of hormone fluctuation in the menstrual cycle affecting sleep regulatory pathways. While research has examined the prevalence of sub-optimal sleep through cycle phases, little research has examined how hormonal contraceptives (HC's) could similarly affect women's sleep, while also neglecting to utilize subjective sleep measures. In this study, we examine subjective sleep quality among naturally cycling (NC) women, women using different HC types, and between active and inactive phase pill users by subjecting 463 women to a subjective sleep battery. We hypothesized that HC users would report more sub-optimal sleep than NC women. Our study yielded non-significant total findings between sleep and HC/NC women, but marginally significant trends between individual HC types and active versus inactive pill phases. These findings suggest a more nuanced relationship between hormones and sleep, motivating future research to further delineate this relationship to improve women's sleep-health outcomes.

Examining the Effects of Menstrual Cycle Phase and Hormonal Contraceptive Use on Women's Sleep

We all have a notion of what it means to have optimal and sub-optimal sleep. Whether it is the acute alertness and energy coming from a good night's sleep or the chronic grogginess and latency from a week of lacking sleep, it is non-trivial how directly sleep impacts our daily lives, functioning, and wellbeing. The past several decades of sleep research have made clear that the impact and necessity of optimal sleep goes far beyond our day-to-day experience with how sleep affects us. Optimal (or adequate) sleep has been continuously identified as an integral analeptic property for sickness, injury, and cognition, fostering and maintaining improvement in physiological and mental well-being, as well as longevity (Luyster et al., 2012; Scullin & Bliwise, 2015; Steptoe et al., 2008). In contrast, sub-optimal sleep, which encompasses a range of sleep issues such as a lack of (or insufficient) sleep, excessive sleep, irregular sleep, and disturbed sleep, has been associated with a variety of acute and chronic detrimental health outcomes. Insufficient sleep has been associated with an increased risk of cardiovascular disease, the development of certain cancers, and Alzheimer's disease (Chattu et al., 2019; Walker, 2008). For individuals consistently sleeping less than just six hours per night, there was a ten-fold greater risk of premature mortality than those who consistently get between seven and nine hours per night (Chattu et al., 2019). Other forms of sub-optimal sleep, such as excessive sleep, excessive sleepiness, and forms of sleep obstruction such as sleep apnea, have been shown to have similar health outcomes, both directly and indirectly (Chattu et al., 2019; Marshall et al., 2008; Ohayon et al., 2013). With research continuing to find such inimical health consequences, the need to investigate risk factors and treatments for sub-optimal sleep and sleep pathologies becomes immediately salient.

Although the pathogenesis of sleep disorders varies widely for each individual, with factors such as genetics, obesity, and or depression influencing the development of such pathologies, the largest and most well known health disparity in sleep issues exists between the sexes (Dzaja et al., 2005; Krystal, 2003). Women disproportionately suffer from a variety of sleep pathologies when compared to men, vastly outnumbering men for clinical sleep pathologies such as insomnia, being more likely to report daytime sleepiness, and commonly experience other disordered sleep throughout periods of their life, such as Restless Leg Syndrome and sleep apnea during adolescence and pregnancy (Mallampalli et al., 2014; Bezerra et al., 2020). Given such frequent overrepresentation, even when controlling for comorbidities, it is likely that what predisposes women to such sleep pathologies has a more biological than behavioral basis. Indeed, the relative complexity of women's hormones and their fluctuations over the course of the menstrual cycle may predicate such vulnerabilities and underlie the relative skew of their representation (Krystal, 2003).

The menstrual cycle constitutes the regular secretion of specific hormones in various amounts over a 21-30 day period, compartmentalized into three phases: the follicular, ovulatory, and luteal phase (Baker & Lee, 2018). Each cycle phase is characterized by its specific pattern of sex hormones, which help facilitate biological and behavioral adaptations in order to conceive and in anticipation of possible conception. Estradiol, progesterone, luteinizing hormone (LH), and follicular stimulating hormone (FSH) are the primary sex steroids that paint the hormonal patterns for each cycle phase and are the main drivers of events like menstruation and ovulation that are consequent of each cycle phase. The follicular phase is characterized by low levels of all sex steroid hormones, with estradiol beginning to increase toward the end of the phase. It is in this early follicular phase when menstruation happens and in the late follicular phase (often

distinguished as the pre-luteal phase) when the body begins to prepare for ovulation by regenerating the endometrial, creating the necessary habitat for a matured egg (Hawkins & Matzuk, 2008). The ovulatory phase, which captures the day of ovulation and the 4 preceding days, is characterized by high levels of estradiol and surges in luteinizing hormone (LH) and follicle stimulating hormone (FSH), allowing for ovarian follicles to mature into an egg for conception. In the luteal phase, the body is preparing for the possibility that conception occurred during ovulation. FSH and LH drop, estradiol regresses to moderate levels, and progesterone increases to the highest amounts over the entire cycle.

This biological rhythm is derivative of an intimate connection between the female reproductive organs and the brain. Ovarian hormone receptors exist extensively throughout the central nervous system as well as the brain (Baker & Lee, 2018). Given this, it is not surprising that the influence of ovarian steroid hormones extends beyond coordinating ovarian and uterine reproductive processes. Indeed, ovarian steroids are consequently thought to indirectly impact major systems of sleep regulation simply as a repercussion of shared sleep regulatory and ovarian steroid pathways (Baker & Lee, 2018; Mong & Cusmano, 2016). More directly, ovarian steroids are thought to modulate circadian rhythm by influencing the suprachiasmatic nucleus, a region in the hypothalamus thought to be responsible for the regulation of physiological circadian rhythms (Baker & Lee, 2018). This consequently allows for sleep sufficiency to change according to relative circulating hormone levels, creating regular periods of more and less optimal sleep in relationship to the cycle phase/hormonal profile at the time.

In the current literature investigating this relationship between ovarian steroids and sleep, researchers have often relied on a combination of objective and subjective measures of sleep. Objective measures often will deploy methods like polysomnography or actigraphy to record

brain activity and or body movement, allowing one to differentiate changes in sleep and wakefulness and sleep architecture, i.e. the time spent in either rapid eye movement sleep (REM sleep) and non-rapid eye movement sleep (NREM sleep), which are broken into 4 stages (Deatherage et al., 2009; Landry et al., 2015). In contrast, subjective measures rely on quantifying one's subjective experience of their sleep quality and daily functioning based on their sleep (Fabbri et al., 2021).

For women who have regular menstrual cycles (naturally cycling/NC women), the most profound and consistent patterns in sleep disturbance appear in the luteal phase (Dzaja et al., 2005). Compared to the follicular phase, women in the luteal phase were found to have modulated sleep architecture, resulting in less overall REM sleep and an increase in Stage 2 sleep in the early and late luteal phase (Baker et al., 2001; Driver et al., 1996). It has also been shown that, due to progesterone's inherent thermogenic properties, the progesterone surge during the luteal phase constitutes a rise in basal body temperature, impeding the requisite dramatic drop in body temperature for adequate sleep (Baker et al., 2001; Okamoto-Mizuno & Mizuno, 2012). Additionally, women with irregular cycles report worse subjective sleep quality, even when controlling for confounding variables, suggesting that variance in sleep quality could be dependent on both absolute and differential levels of ovarian steroids at sleep dependent thresholds (Baker & Lee, 2018). Though studying NC women gives insight into the dynamics and potential origins of pathologically sub-optimal sleep in its most organic state, very little research has been done to include an equally large subject group of women with a very different hormonal profile: women on hormonal contraceptives.

Hormonal contraceptives (HC's) primarily consist of a synthetic version of progesterone, called progestin, and sometimes a synthetic version of estradiol, called ethinyl estradiol (Mitchell

& Welling, 2020). Most hormonal contraceptives work by inducing and maintaining a level of progestin high enough to prevent the LH surge that is necessary to initiate ovulation, thus preventing conception (Mitchell & Welling, 2020). Because of this, HC users have a relatively static hormonal profile over time, only changing in oral contraceptive pill (OCP) users for one week out of the month during their inactive pill phase to allow for menstruation—very much in contrast to the dynamic shifts in hormones from naturally cycling women. The apparent hormonal flatlining of women on HC's becomes important because while maintaining high levels of progestin is the primary component to preventing pregnancy, it could also be effectively keeping women in a type of perennial luteal phase, possibly eliciting the very similar worsened sleep outcomes as if they were actually in the luteal phase. Moreover, while different HC methods, such as Nexplanon, hormonal IUD, or OCP's have the same fundamental functionality in their induced progestin surge, HC methods and OCP brands can vary in the hormonal formula they use to prevent conception. Progestin in particular varies in formula by progestin generation (either being 1st, 2nd, 3rd, or 4th generation formulas), with different OCP brands often being distinguished by which progestin generation they utilize (Mitchell & Welling, 2020). These different generations have been shown to have both overreaching biological and psychobehavioral effects, making any potential differences in effects by HC's immediately more nuanced than simply comparing women using HC's with NC women (Mitchell & Welling, 2020; Oslakovic & Zadro, 2014).

One study explored whether taking OCP's containing progestin raised basal body temperature, which would be consistent with naturally cycling effects of progesterone-driven increases in basal temperature observed in the luteal phase (Baker et al., 2001). When compared to NC women, those taking oral contraceptives had a basal body temperature most similar to NC

women in the luteal phase. However, basal body temperature was consistently higher among OCP users than all phases of NC women. Researchers also investigated what happened to core body temperature during the placebo phase of taking OCP's, hypothesizing that there would be little change due to exogenous hormones being more potent and lasting longer than endogenous hormones. Consistent with their hypothesis, basal body temperature was not found to significantly decrease at all during this phase. In this study, Baker and colleagues (2001) also explored how different components of sleep, such as sleep architecture, might compare between women who are naturally cycling and women using oral contraceptives. Hormonal contraceptives were found to induce an increase in Stage 2 sleep compared to the follicular and luteal phase, despite Stage 2 sleep already being upregulated in the luteal phase. Inconsistent with heightened slow wave sleep in the luteal phase, HC use elicited a decrease in slow wave sleep compared to both phases of NC women. Most surprisingly, these components of sleep seem to change during the inactive oral contraceptive phase, approaching more normal sleep patterns, while core body temperature remained high, suggesting that these components of sleep are influenced independent from core body temperature. Within this, it is evident that it's unclear as to what the consequential sleep quality effects are from the change in sleep architecture competing with heightened core body temperature.

As an addendum to the literature covered so far, it is important to point out that the vast majority of the research literature for both sleep in NC women and sleep in women using HC's has relied heavily on objective sleep measurements rather than subjective measurements. This is important because although changes in objective measures can be predictive of parallel changes in subjective measures (and vice versa), discrepancies in objective evaluations of one's sleep with their predicted subjective sleep outcomes often permeate the literature (Buysse et al., 2008;

Landry et al., 2015; Lockley et al., 1999). Although attempting to alleviate these discrepancies has been prominent in the literature involving NC women, the same attention has not been given to women on HC's. There consequently lacks sufficient research that relates how these objective sleep measures in women using HC's are realized among women's subjective experience. Along with this, previous research has often failed to distinguish their measured sleep effects from progestin generations in OCP users. It is still unclear as to what the range of sleep pathologies (both at clinical and subclinical levels) are most induced, nor has there been much research investigating wakefulness symptoms related to sleep, such as daytime sleepiness. Furthermore, little to no research has investigated how subjective sleep changes between the active and inactive phase in OCP users.

The purpose of this study was to examine the effect hormonal contraceptives play in women's sleep, compared to naturally cycling women. Specifically, I was interested in exploring this using subjective sleep measures, motivated by both attempting to relate the changes in objective sleep measures found in the research literature as a predictive cue, as well as the fact that subjective sleep quality in women using HC's has not been well researched. Further, I aimed to delineate whether different hormonal contraceptive types (i.e. Nexplanon, hormonal IUD, and OCPs) and progestin pill generation moderate such effects. Finally, within OCP users, I wanted to investigate the potential differences in subjective sleep quality between active and inactive pill phase users.

To measure a wider range of potential subjective sleep issues, assess potential underlying sleep pathologies that could occur, as well as to capture wakefulness symptoms like daytime sleepiness, I chose to use both the Insomnia Severity Index (ISI) and Hypersomnia Severity Index (HSI). Together, these indices capture the extremes of sleep pathologies on opposite ends

of one another; ISI evaluating problems with diminished sleep and augmented wakefulness, and HSI with augmented sleep and diminished wakefulness.

Female participants were subject to a one-time sleep inventory and a suite of questions delineating their cycle phase or, if they were on HC's, what type of HC, generation, and if they were in an active or inactive phase. I hypothesize that hormonal contraceptive users will have higher total HSI and ISI scores than those who are naturally cycling. Our study of active versus inactive phases users and its relationship to subjective sleep quality will be exploratory.

Method

Participants

Female participants were recruited broadly from the University of Arkansas community via digital and physical advertisements, as well as through the Psychology Department subject pool. In terms of compensation, participants recruited from the community were entered into a gift card drawing and those recruited from the subject pool received credit toward their participation requirement. In total, 463 women completed the measures for the present study. Participants were on average 19 years old (M = 19.86, SD = 3.76, range: 18-44). Over half of participants were Caucasian (87.10%), followed by participants who were Black/African American (3.10%), those with more than one race (3.10%), Native American/Alaskan Natives (3.10%), Asians (1.40%), those who did not wish to report/where of an unknown race (1.3%), those who identified as "Other" (0.50%), and Native Hawaiians/Pacific Islanders (0.40%); (12.60%) of participants identified their ethnicity as Hispanic or Latina.

Procedure and Materials

Participants were directed to an online questionnaire with the first page being the informed consent. The first part of the survey asked participants to answer questions about their

menstrual cycle and their current hormonal contraceptive use. Among participants, 197 were naturally cycling and 266 were on a hormonal contraceptive. To estimate what phase of the menstrual cycle naturally cycling women were in, they were asked "What phase of your ovulatory cycle are you currently in?" with options being "I currently have my period", "The first day of my period was ~1 week ago", "The first day of my period was ~2 weeks ago", "I start my period in ~2 weeks", "I start my period in ~1 week", and "other, please specify". Although the cycle phase measurement in these data was not precise, we nevertheless split naturally cycling women into their approximated cycle phases. We divided women into the "Luteal" and follicular phase, and further the follicular phase into "Menstruating" and "Pre-Luteal" to differentiate for confounding variables related to discomfort during menstruation that could impact sleep, which are not present in the late-follicular (Pre-Luteal) phase (Nowakowski et al., 2013). Women were classified as Menstruating if they said they were currently on their period (n = 35), Pre-Luteal if they said they had their period 1-2 weeks ago (n = 80), and Luteal if they said they expected their period in 1-2 weeks (n =82). Of the women using hormonal contraceptives, group sizes varied by hormonal contraceptive type – group sizes listed as follows; Nexplanon (n =33), hormonal IUD (n =30), and OCP's (n =203). To identify progestin generation, women on OCP's were asked to report brand information and progestin type of the pill they were currently taking; from this we were able to code for the progestin generation used in each brand. Of those taking OCP's, group sizes varied by progestin generation – group sizes listed as follows; 1st generation pill (n =82), 2nd generation pill (n =22), 3rd generation pill (n =65), and 4th generation pill (n =34). The vast majority of OC users were on one of their active pill weeks (n =164), with the remaining OC users on their inactive pill week (n =46), (i.e., they were taking the placebo place-holder pills).

Lastly, participants were administered two subjective sleep questionnaires: the Hypersomnia Severity Index (HSI) and the Insomnia Severity Index (ISI). Individually, each scale is important because they allow one to gauge the range in which one might be suffering from one of these disorders up to a clinical threshold. Good internal consistency and validity has been shown for both the HSI ($\alpha = 0.79$) and ISI ($\alpha = 0.74$) (Shahid et al., 2012; Kaplan et al., 2019). The HSI is a 9-item likert-type scale, ranging from 0-4, measuring hypersomnia symptoms over the course of the past month, with higher scores indicating greater severity in hypersomnia symptoms. The first four items and the last item in the scale ask about explicit symptoms of hypersomnia, e.g. "Sleep too much at night," from 0 ("Not at all") to 4 ("Very much"), and "Do you ever have 'sleep attacks,' defined as unintended sleep in inappropriate situations", from 0 ("Not at all") to 4 ("All the time"). The other four items asked generally about how such sleep patterns affect one's life, e.g. "How NOTICABLE do others think your sleeping problem is in terms of impairing the quality of your life?", from 0 ("Not noticeable at all") to 4 ("Very much noticeable") and "How WORRIED/DISTRESSED are you from your current sleep problem?", from 0 ("Not at all") to 4 ("Very much"). The ISI is a 7-item likert-type scale, ranging from 0-4, of a similar format to the HSI, measuring insomnia symptoms over the course of the past 2 weeks, with higher scores indicating greater severity in insomnia symptoms.

The ISI also differentiates the clinical significance of score ranges, from a score of 0-7 indicating no clinical significance of insomnia, to a score of 22-28 indicating severe clinical insomnia. The first three questions ask about the severity of insomnia symptoms, e.g. "Difficulty falling asleep" and "Difficulty staying asleep," from 0 ("None") to 4 ("Very"). The last four items ask generally about how such sleep patterns affect one's life, e.g. "How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern," from 0 ("Very

Satisfied") to 4 ("Very Dissatisfied") and "How WORRIED/DISTRESSED are you about your current sleep problem," from 0 ("Not at all worried") to 4 ('Very much worried").

Results

Table 1

Descriptive statistics for primary analyses.

	HSI			ISI		
Groups	*n*	*M*	*SD*	*n*	*M*	*SD*
Menstruating	35	22.60	5.42	34	9.26	5.60
Pre-luteal	80	21.36	5.55	80	8.59	5.33
Luteal	82	22.49	5.72	81	9.58	6.12
1st Gen Pill	82	22.67	5.77	82	9.05	6.19
2nd Gen Pill	22	20.91	5.17	23	8.83	6.79
3rd Gen Pill	65	21.32	5.37	66	9.83	5.90
4th Gen Pill	34	20.35	5.25	33	7.70	4.75
Hormonal IUD	30	23.00	6.19	33	10.30	5.42
Nexplanon	33	24.09	7.14	33	10.52	5.87

First, to test my hypothesis that HC's would decrease sleep quality, I conducted a one-way ANOVA to compare the HSI and the ISI between the target groups of women based on each contraceptive type, OCP generation, and menstrual cycle phase. For HSI, the omnibus test indicated that there were no significant differences between the groups, $F(8, 454) = 1.61$, $p = .120$. Nevertheless, I decided to explore whether there were any patterns of interest between the groups in the post-hoc analyses. I found that for women using OCPs containing a fourth 4th generation progestin, HSI was significantly lower (and thus had better subjective sleep scores) than in comparison to women using OCPs containing a 1st generation progestin ($p = .047$) women using the Nexplanon implant ($p = .008$), women using the hormonal IUD ($p = .065$), and marginally lower than NC women in the luteal phase ($p = .068$) but not any other group (all p's $\geq .103$). Further, HSI was significantly higher in Nexplanon using women compared to the NC women who were pre-luteal ($p = 0.022$) and OCPs containing 2nd ($p = .044$), 3rd ($p = .024$), and

4th (p = .008) progestin generations.

I conducted a parallel analysis for ISI and also found that there were no significant differences between groups, $F(8, 456) = 0.896$, p = .519. Post hoc analyses revealed that women using OCPs containing 4th generation progestins had marginally lower ISI scores than women using the Nexplanon implant (p = .050), the hormonal IUD (p = .069), and women using OCPs containing 3rd generation progestins (p = .086).

Finally, I examined the effect of active versus inactive phases of OCP's on subjective sleep quality. The active phase yielded a marginally higher ISI score across OCP users than the inactive phase, $F(1, 208) = 2.80$, p =.096. However, no significant differences were found for HSI between the active or inactive phase, $F(1, 207) = 0.115$, p = .735.

Discussion

The impetus for this study was to investigate the relationship between subjective sleep quality and hormonal contraceptive usage, compared to naturally cycling women. Specifically, I wanted to differentiate how subjective sleep quality relates to HC type, OCP generation, and whether one was in the active or inactive pill phase. We subjected women to a battery of questions ascertaining details about their HC usage in order to categorize HC user type, generation, and pill phase, and menstrual cycle information to calculate cycle phase for NC women; the sleep battery was then administered which measured insomnia and hypersomnia symptoms.

We hypothesized that there would be worse total subjective sleep outcomes for women on HC's versus NC women. Overall, the main hypothesis was not supported, as there were no significant differences between groups of women on the ISI or HSI. However, when investigating this, we did find suggestive trends. The most interesting and persistent patterns

between ISI and HSI emerged from the 4th generation pill, which seemed to yield both lower HSI and ISI when compared with Nexplanon, IUD, and 3rd generation pills. Moreover, Nexplanon was shown to have consistently worse subjective sleep outcomes, showing both higher ISI and HSI scores than all other groups. Overall, more trending differences between the groups appeared to be in relation to hypersomnia symptoms than insomnia symptoms. This is particularly interesting since the majority of the literature on sleep pathologies in women focuses on clinical insomnia rather than hypersomnia.

For the active versus inactive pill users, only a difference was detected in ISI, with users in the active pill phase reporting more ISI symptoms; however, this result was only marginally significant and thus not providing strong support for my hypothesis. Although the findings are preliminary, they could be an indication that it would be important to continue investigating how hormonal contraceptive use affects women's sleep.

Limitations and Future Directions

There was more than one caveat in my study that could have led to such findings, the most salient of that being the participant sample size related to HC users. There was a skewed sample both between HC type and within OCP users. Only roughly 24% of HC users were either on a hormonal IUD (30 participants) or Nexplanon (33 participants), compared to the 203 OCP users. This is particularly limiting considering some of the most interesting results between individual variables was with Nexplanon. Further, 2nd and 4th generation OCP users also had low sample sizes, with their combined representation only amounting to roughly 27% of our OCP user sample. Again, this is particularly limiting considering some of the most interesting interactions on an individual and comparison level occurred between 4th generation OCP users.

Despite hypersomnia symptoms being the prevalent trend in differences between groups,

HSI was not found to be a significant variable at all in the active versus inactive phase OCP users. It is highly likely that this is because of an inherent difference between the timeframes the HSI and ISI ask participants to reflect upon, in evaluating their own sleep. While the ISI asks for participants to answer based on their sleep within the past 2 weeks, the HSI asks participants to reflect on their sleep within the past month. Because the inactive pill phase only represents one week out of the month for OCP users, if there were any changes in HSI score within the week of their inactive phase it would be comparatively insignificant to whatever sleeping patterns were normally experienced during the rest of the month. This could be contributing to why there was not a significant difference in HSI scores for this part of the study.

While my study provides insight into trends among HC users' subjective sleep experience, going forward, it would be useful to replicate such measurements alongside objective measures, such as using actigraphy, polysomnography, and or recording changes in basal body temperature. The usage of sleep diaries could also be deployed for methodological plurality in subjective measures. This would allow one to more rigorously study differences in objective measures among HC factors and their relationship to subjective measures.

Recording basal body temperature, in particular, could be interesting in of itself, as the consistency in high basal body temperature among HC users was one of the core lines of research that drove my hypothesis (Baker et al., 2001). We also understood that basal body temperature persisted with the active versus inactive phase of OCP users, despite sleep architecture changing, driving my secondary research effort in sleep quality over these phases. Seeing that there was a marginally significant trend for ISI scores to be worse in the active versus inactive phases for OCP users, this could relate to the regression of sleep architecture to a more normal structure during the inactive pill phase. Furthermore, this could suggest a delineation

between the sleep quality impact of changes in sleep architecture versus the compounded heightened basal body temperature, persisting during the inactive phase. Moreover, it has not been studied yet how basal body temperature is affected based on progestin generation or how it compares across HC types. Investigating this relationship could be revealing about how and why exactly certain generations have the effects they do.

Women continue to be the primary subject group that suffers from pathological sleep, yet this, along with the effects of hormonal contraceptives, continues to be an under researched area, despite the very direct medical implications it has on women's livelihood. Although behavioral interventions to improve sleep hygiene can be a powerful remedy for disordered sleep, the full breadth of treatment outcomes for disordered sleep in women would be rendered incomplete without taking into consideration how much hormonal variance is of impact and can be treated. Further, as the pathology seems to succinctly be consequential of women's hormonal complexity, it is imperative that research continue in investigating this relationship and innovating both hormonal as well as behavioral treatment options.

References

Baker, F. C., & Lee, K. A. (2018). Menstrual cycle effects on sleep. *Sleep medicine clinics*, *13*(3), 283-294.

Baker, F. C., Mitchell, D., & Driver, H. S. (2001). Oral contraceptives alter sleep and raise body temperature in young women. *Pflugers Archiv : European journal of physiology*, *442*(5), 729–737.

Bezerra, A. G., Andersen, M. L., Pires, G. N., Banzoli, C. V., Polesel, D. N., Tufik, S., & Hachul, H. (2020). Hormonal contraceptive use and subjective sleep reports in women: An online survey. *Journal of sleep research*, *29*(6), e12983.

Buysse, D. J., Hall, M. L., Strollo, P. J., Kamarck, T. W., Owens, J., Lee, L., Reis, S. E., & Matthews, K. A. (2008). Relationships Between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and Clinical/Polysomnographic Measures in a Community Sample. *Journal of Clinical Sleep Medicine*, *04*(06), 563–571.

Chattu, V. K., Manzar, M. D., Kumary, S., Burman, D., Spence, D. W., & Pandi-Perumal, S. R. (2019, March). The global problem of insufficient sleep and its serious public health implications. In *Healthcare* (Vol. 7, No. 1, p. 1). Multidisciplinary Digital Publishing Institute.

Deatherage, J. R., Roden, R. D., & Zouhary, K. (2009). Normal Sleep Architecture. *Seminars in Orthodontics*, *15*(2), 86–87.

Driver, H. S., Dijk, D. J., Werth, E., Biedermann, K., & Borbély, A. A. (1996). Sleep and the sleep electroencephalogram across the menstrual cycle in young healthy women. *The Journal of Clinical Endocrinology & Metabolism*, *81*(2), 728-735.

Dzaja, A., Arber, S., Hislop, J., Kerkhofs, M., Kopp, C., Pollmächer, T., ... & Porkka-Heiskanen,

T. (2005). Women's sleep in health and disease. *Journal of psychiatric research*, *39*(1), 55-76.

Fabbri, M., Beracci, A., Martoni, M., Meneo, D., Tonetti, L., & Natale, V. (2021). Measuring Subjective Sleep Quality: A Review. *International Journal of Environmental Research and Public Health*, *18*(3), 1082.

Hawkins, S. M., & Matzuk, M. M. (2008). The Menstrual Cycle. *Annals of the New York Academy of Sciences*, *1135*(1), 10–18.

Krystal, A. D. (2003). Insomnia in women. *Clinical cornerstone*, *5*(3), 41-50.

Landry, G. J., Best, J. R., & Liu-Ambrose, T. (2015). Measuring sleep quality in older adults: a comparison using subjective and objective methods. *Frontiers in Aging Neuroscience*, *7*.

Lockley, S. W., Skene, D. J., & Arendt, J. (1999). Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *Journal of Sleep Research*, *8*(3), 175–183.

Luyster, F. S., Strollo, P. J., Zee, P. C., & Walsh, J. K. (2012). Sleep: A Health Imperative. *Sleep*, *35*(6), 727–734.

Mallampalli, M. P., & Carter, C. L. (2014). Exploring sex and gender differences in sleep health: a Society for Women's Health Research Report. *Journal of women's health*, *23*(7), 553-562.

Marshall, N. S., Wong, K. K., Liu, P. Y., Cullen, S. R., Knuiman, M. W., & Grunstein, R. R. (2008). Sleep apnea as an independent risk factor for all-cause mortality: the Busselton Health Study. *Sleep*, *31*(8), 1079–1085.

Mitchell, V. E., & Welling, L. L. (2020). Not all progestins are created equally: considering

unique progestins individually in psychobehavioral research. *Adaptive Human Behavior and Physiology*, *6*(3), 381-412.

Mong, J. A., & Cusmano, D. M. (2016). Sex differences in sleep: impact of biological sex and sex steroids. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *371*(1688), 20150110.

Nowakowski, S., Meers, J., & Heimbach, E. (2013). Sleep and Women's Health. *Sleep Medicine Research*, *4*(1), 1–22.

Ohayon, M. M., Reynolds, C. F., 3rd, & Dauvilliers, Y. (2013). Excessive sleep duration and quality of life. *Annals of neurology*, *73*(6), 785–794.

Okamoto-Mizuno, K., & Mizuno, K. (2012). Effects of thermal environment on sleep and circadian rhythm. *Journal of Physiological Anthropology*, *31*(1).

Oslakovic, S., & Zadro, R. (2014). Comparison of the impact of four generations of progestins on hemostatic variables. *Clinical and Applied Thrombosis/Hemostasis: Official Journal of the International Academy of Clinical and Applied Thrombosis/Hemostasis*, *20*(4), 448–455.

Scullin, M. K., & Bliwise, D. L. (2015). Sleep, Cognition, and Normal Aging. *Perspectives on Psychological Science*, *10*(1), 97–137.

Steptoe, A., O'Donnell, K., Marmot, M., & Wardle, J. (2008). Positive affect, psychological well-being, and good sleep. *Journal of Psychosomatic Research*, *64*(4), 409–415.

Walker, M. P. (2008). Cognitive consequences of sleep and sleep loss. *Sleep Medicine*, *9*(1), S29–S34.