Differences in Sperm Motility Based on Sleep Quality in Infertilized Men at RSKIA Sadewa

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ABSTRACT

Background : *Infertility is a significant reproductive health issue, with a global prevalence of 8–12% among couples of reproductive age. One contributing factor is decreased sperm quality, including sperm motility. Poor sleep quality is suspected to affect sperm motility through hormonal disruptions and oxidative stress; however, research in Indonesia remains limited.*
Objective : *This study aims to determine the differences in sperm motility based on sleep quality in infertile men at RSKIA Sadewa Yogyakarta.*
Methods : *This study employed an analytical observational design with a cross-sectional approach. The sample consisted of 60 infertile men aged 20–45 years who met the inclusion criteria. Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), while sperm motility data were obtained from medical records. Data were analyzed using the independent sample t-test and One-Way Anova.*
Results : *The results showed no significant difference in sperm motility between the good and poor sleep quality groups (p 0.374). The average sperm motility in the good sleep quality group was 48.43%, while in the poor sleep quality group, it was 43.90%. Confounding variables such as age, IMT, and smoking habits also did not show a significant effect on sperm motility.*
Conclusion : *Sperm motility in infertile men at RSKIA Sadewa who had good sleep quality was not significantly different compared to those with poor sleep quality.*

Keywords : *sleep quality, sperm motility, male infertility, PSQI, reproductive health*

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INTRODUCTION

Infertility is a reproductive health issue that is increasingly being encountered worldwide, with a global prevalence estimated at one in six individuals of reproductive age[1]. In Indonesia, the incidence of infertility reaches 15–25% among couples of reproductive age[2]. One of the main components in the causes of male infertility is sperm quality, which includes count, morphology, and motility. A decrease in sperm motility has been proven to significantly hinder fertilization ability and is a major contributor to couple infertility[3].

Sleep quality is an increasingly recognized lifestyle factor in the context of reproductive health. Optimal sleep is needed for physiological recovery, including hormone regulation that plays a role in spermatogenesis. However, due to the pressures of modern life, changes in work patterns, and psychological stress, the quality of sleep among the global population tends to decline[4]. Various studies show that poor sleep quality including sleep duration that is too short or too long, as well as sleep disturbances can disrupt the hypothalamic-pituitary-gonadal (HPG) axis and lead to a decrease in testosterone levels, which ultimately affects sperm motility[5].

Several international studies, such as those conducted by Du et al. and Chen et al. have shown a negative correlation between Pittsburgh Sleep Quality Index (PSQI) scores and semen parameters, particularly sperm motility and concentration[6,7]. However, most of these studies were conducted on healthy male populations or potential sperm donors, and there has been little research specifically examining infertile patient populations. In addition, similar research has not been found in Indonesia, so there is still a gap in local evidence that needs to be addressed through population-based studies in Indonesia.

This study aims to evaluate the differences in sperm motility based on sleep quality in infertile men at RSKIA Sadewa Yogyakarta. In addition to providing scientific information regarding the relationship between the two variables, this study also has practical value for clinicians in adopting an educational and preventive approach towards infertile patients. Unlike previous studies, this research focuses on a clinically diagnosed infertile population and employs an analytical approach based on primary data and medical records. Thus, the results of this study are expected to serve as a new scientific reference that strengthens the national literature on the contribution of lifestyle factors to the quality of sperm in infertile men.

METHOD

This research is an observational analytical study with a cross-sectional design using primary and secondary data from infertile male patients undergoing sperm analysis at the Sadewa Special Hospital for Mothers and Children (RSKIA), Sleman, Special Region of Yogyakarta. Data collection was conducted from December 2024 to March 2025.

The target population in this study is all infertile men undergoing fertility examination programs at RSKIA Sadewa. The sample was taken purposively with the inclusion criteria being infertile male patients aged 20–45 years who have just started a pregnancy program, are willing to participate in the study by signing the informed consent, and have complete data including age, body mass index (BMI), smoking habits, and sperm motility test results. Respondents are also required to fully complete the Pittsburgh Sleep Quality Index (PSQI) questionnaire which has been previously validated and shown to have good reliability and internal consistency in the Indonesian population[8]. Exclusion criteria include patients with genetic disorders such as Kartagener Syndrome or Primary Ciliary Dyskinesia, uncontrolled metabolic diseases such as diabetes mellitus, the presence of leukocytospermia in sperm analysis results, the use of hormonal drugs or chemotherapy, and the consumption of fertility supplements in the past month.

Based on calculations using the Lemeshow formula with a confidence level of 95%, a margin of error of 10%, and an estimated infertility proportion of 12%, a minimum sample size of 41 subjects was obtained. To anticipate potential invalid data or dropouts, the sample size was increased to 60 people. The independent variable in this study is sleep quality, measured using the PSQI and classified into two categories. The dependent variable is sperm motility, expressed as the percentage of progressive motility based on WHO guidelines (2021). The confounding variables analyzed include age, BMI, and smoking habits, which were taken from medical records. The analysis used includes the Independent Sample t-test and One-Way ANOVA test for analyzing confounding variables with more than two groups, while the t-test is used for variables with two groups. The normality test was conducted using the Kolmogorov-Smirnov test. All analyses were conducted using IBM SPSS version 27, with a p-value < 0.05 as the significance threshold.

RESULT

A total of 60 infertile male patients were included in this study, with the majority (48.3%) aged between 30–34 years (see **Table 1**). No participants were recorded in the 20–24 age group. The highest sperm motility was observed in the 35–39 years group (48.75%), while the lowest was in the 40–45 years group (35.50%). However, statistical analysis showed no significant difference in sperm motility across age categories ($p = 0.714$, **Table 1**).

Regarding body mass index (BMI), most participants fell into the Obesity I category (25–29.9), comprising 36.7% of the total sample. This group had the lowest mean sperm motility (36.7%), whereas the highest motility was found in the Obesity II group (≥30) at 49.15% (**Table 1**). Despite these differences, the association between BMI and sperm motility was not statistically significant (p = 0.878). A similar result was observed with smoking status; smokers had a lower sperm motility mean (41.88%) compared to non-smokers (49.44%), but the difference did not reach statistical significance (p = 0.139, **Table 1**).

Table 1. Characteristics of Patients in Oligospermia and Normozoospermia Groups

Characteristics	Details (n=60)		Sperm Motility Mean (%)	P Value
	N	%		
Age				
20-24 years	0	0	-	0.714
25-29 years	19	31.7	45.84	
30-34 years	29	48.3	47.14	
35-39 years	8	13.3	48.75	
40-45 years	4	6.7	35.50	
IMT				
<18,5 (Underweight)	0	0	-	0.878
18,5-22,9 (Normal)	10	16.7	42.30	
23-24,9 (Overweight)	15	25.0	45.73	
25-29,9 (Obesity)	22	36.7	46.45	
≥30 (Obesity II)	13	21.7	49.15	
Smoking Habit				
Yes	26	43.3	41.88	0.139
No	34	56.7	49.44	
Sperm Motility Interpretation				
≥32%	50	16.7	-	
<32%	10	83.3	-	

Table 2. PSQI Respondent Aspect

Variable	Details (n=60)		Sperm Motility Mean (%)	P Value
	N	%		
Subjective Sleep Quality				
Very good	11	18.3	40.36	0.193
Quite good	41	68.3	48.95	
Quite bad	6	10.0	45.67	
Very bad	2	3.3	22.50	
Sleep Latency				
≤15 minute	39	65.0	49.79	0.147
16-30 minute	15	25.0	39.20	
30-60 minute	6	10.0	40.00	
>60 minute	0	0	-	
Sleep Duration				
>7 hour	25	41.7	48.64	0.612
6-7 hour	23	38.3	46.43	
5-6 hour	10	16.7	38.80	
< 5 hour	2	3.3	49.00	
Sleep Efficiency				
>85%	55	91.7	46.95	0.580
75-84%	4	6.7	38.75	

65-74%	1	1.6	33.00	
<65%	0	0	46.95	
Sleep Disturbance				
None	2	3.3	63.00	0.469
Mild	46	76.7	45.74	
Medium	12	20.0	45.00	
Severe	0	0	-	
Use of Sleep Medication				
Never	58	96.7	46.83	0.305
Less than 1 time a week	1	1.6	36.00	
1 or 2 times a week	1	1.6	18.00	
≥3 x a week	0	0	-	
Daytime Dysfunction				
Never	28	46.7	45.36	0.976
Less than 1 time a week	26	43.3	47.08	
1 or 2 times a week	3	5.0	48.67	
≥3 x a week	3	5.0	43.33	
Sleep Quality Interpretation				
Good	30	50.0	48.43	0.374
Bad	30	50.0	43.90	

As detailed in **Table 2**, multiple dimensions of sleep quality were assessed using the Pittsburgh Sleep Quality Index (PSQI). The majority of participants rated their sleep as "quite good" (68.3%), with a corresponding sperm motility mean of 48.95%. The lowest mean motility (22.50%) was observed among those reporting "very bad" sleep quality. Nonetheless, no statistically significant association was found between subjective sleep quality and sperm motility ($p = 0.193$, **Table 2**).

Sleep latency analysis showed that 65% of subjects fell asleep within 15 minutes, and this group had the highest sperm motility (49.79%). Conversely, those with a sleep latency of 30–60 minutes had a lower motility mean (40.00%), though the result was not statistically significant ($p = 0.147$, **Table 2**). Similar patterns were observed for sleep duration, efficiency, disturbance, use of sleep medication, and daytime dysfunction, none of which showed significant correlations with sperm motility ($p > 0.05$ for all, **Table 2**).

When overall sleep quality was categorized into "good" and "poor" groups, both comprised 50% of the sample. The good sleep quality group had a slightly higher mean motility (48.43%) compared to the poor sleep group (43.90%), but again, the difference was not statistically significant ($p = 0.374$, **Table 2**).

In summary, while descriptive trends were noted, none of the examined variables from both demographic factors and sleep quality dimensions showed statistically significant differences in sperm motility (see **Table 1** and **Table 2**).

DISCUSSION

This study explored the association between various demographic, lifestyle, and sleep-related factors with sperm motility among infertile men attending RSKIA Sadewa Yogyakarta. Although no statistically significant associations were observed between the examined variables and sperm motility, several notable trends emerged that are worth highlighting, especially when contextualized within existing literature and biological frameworks.

Age and Sperm Motility

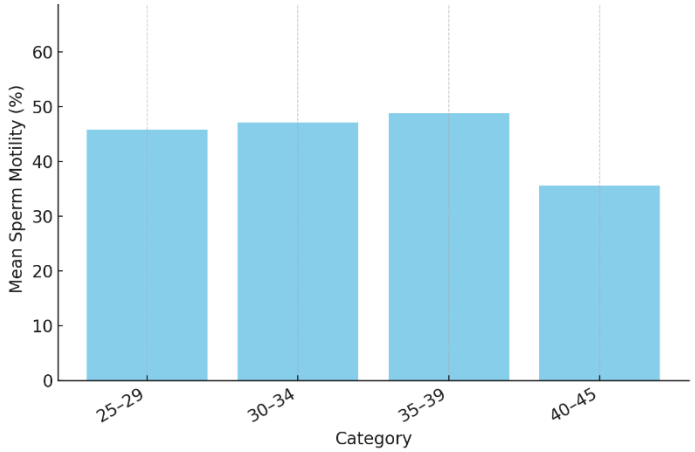

Figure 1. Age Group vs. Sperm Motility

The most common age group among infertile participants in this study was 30–34 years, which aligns with global data indicating that male infertility peaks in this age range [9,10]. Although age was not significantly associated with sperm motility ($p = 0.714$), a descriptive trend showed decreased motility with advancing age, with the lowest mean in the 40–45 age group (35.50%) (**Figure 1**). This aligns with literature indicating that aging may impair sperm quality through reduced testosterone levels, increased oxidative stress, and DNA fragmentation[11,12] .

Testosterone is essential for spermatogenesis and sperm motility via Sertoli cell regulation and mitochondrial support[13,14]. Increased age is also linked to reactive oxygen species (ROS) that damage sperm membranes and DNA, reducing motility[12,15].

Similar nonsignificant trends have been reported in other studies[7], although some found significant declines in motility after age 35 [16]. The lack of significance here may stem from small sample sizes in older age groups. Nonetheless, age remains an important consideration in male fertility assessments.

BMI and Sperm Motility

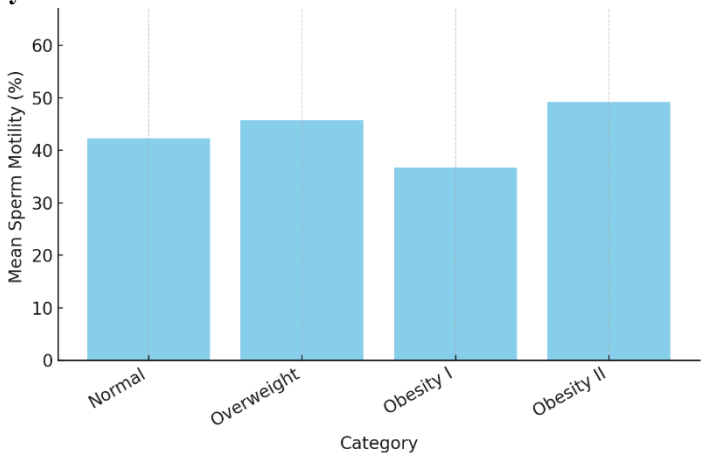

Figure 2. BMI Category vs. Sperm Motility

Most participants in this study were categorized as overweight or obese, reflecting Indonesia's rising obesity prevalence due to reduced physical activity and increased consumption of high-calorie diets[17,18]. As shown in **Figure 2**, sperm motility varied across BMI categories. Surprisingly, the highest motility was observed in the Obesity II group (BMI ≥30), while the lowest occurred in the Obesity I group. However, this variation was not statistically significant ($p = 0.878$).

This finding contrasts with prior studies, such as Putri & Nadhiroh (2024), which reported a negative association between BMI and sperm quality[18]. Obesity has been shown to impair male fertility by promoting

aromatization of androgens to estrogens in adipose tissue, thereby suppressing the hypothalamic–pituitary–gonadal (HPG) axis and reducing testosterone levels necessary for spermatogenesis[19].

The unexpected trend observed in this study may be due to behavioral compensations such as physical activity, healthier diets, or lower stress levels among some obese individuals. Additionally, inter-individual differences in fat distribution and metabolic health can influence reproductive function independently of BMI [20]. Thus, while BMI remains a relevant clinical factor, it may not fully capture the complexity of metabolic influences on sperm quality.

Smoking and Sperm Motility

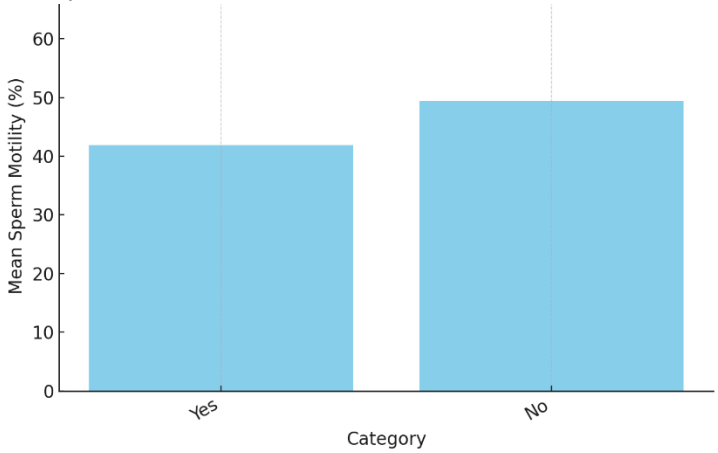

Figure 3. Smoking Habit vs. Sperm Motility

In this study, 43.3% of participants identified as smokers. As shown in **Figure 3**, the average sperm motility among smokers was 41.88%, notably lower than the 49.44% observed in non-smokers. Although this difference was not statistically significant ($p = 0.139$), the trend aligns with extensive literature reporting the detrimental effects of smoking on male fertility[15,21].

Tobacco smoke contains over 7,000 chemicals, many of which are cytotoxic, mutagenic, and endocrine-disrupting. Nicotine promotes vasoconstriction, reducing testicular blood flow and impairing Sertoli and Leydig cell function, which are essential for spermatogenesis and testosterone production[22]. Carbon monoxide binds to hemoglobin more avidly than oxygen, creating hypoxic testicular conditions that elevate reactive oxygen species (ROS) production[11,12]. Elevated ROS induce lipid peroxidation of sperm membranes, mitochondrial dysfunction, and DNA fragmentation, leading to impaired sperm motility and morphology[7].

Biomarkers such as 8-hydroxydeoxyguanosine (8-OHdG) and malondialdehyde (MDA) have been found at higher levels in the seminal plasma of smokers compared to non-smokers, supporting the oxidative stress mechanism[7]. This oxidative damage compromises not only spermatogenesis but also sperm function post-ejaculation.

Interestingly, a considerable number of non-smokers in this study also exhibited reduced sperm motility, underscoring the multifactorial nature of male infertility. Other factors such as chronic psychological stress, suboptimal sleep quality, obesity, sedentary lifestyle, environmental toxins, and genetic or endocrine disorders may contribute to impaired sperm quality[23]. For example, non-smokers with poor sleep or obesity may experience similarly reduced fertility as some smokers. Given these findings, smoking remains a critical modifiable risk factor for male infertility. Clinicians should prioritize smoking cessation interventions as part of comprehensive fertility management.

Subjective Sleep Quality and Sperm Motility

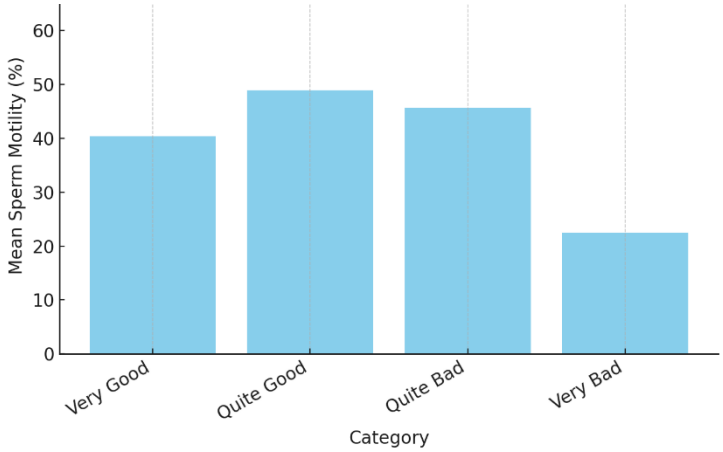

Figure 4. Subjective Sleep Quality vs. Sperm Motility

Most respondents rated their sleep quality as "quite good," which accounted for the largest group. As shown in **Figure 4**, participants reporting "very bad" sleep quality had the lowest mean sperm motility (22.50%), whereas those with "quite good" sleep quality exhibited the highest motility (48.95%). Although the difference was not statistically significant ($p = 0.193$), this trend suggests a possible association.

Subjective sleep quality reflects an individual's perception of their sleep, influenced by factors such as occupation, stress tolerance, and daily demands[24]. Chen et al. demonstrated that poor subjective sleep quality was significantly associated with reduced sperm motility, even after adjusting for confounders[7]. Sleep quality affects hormonal regulation, particularly the nocturnal secretion of testosterone, which is critical for spermatogenesis and sperm motility[25].

Testosterone levels peak during REM sleep stages; thus, fragmented or poor-quality sleep can blunt these peaks, impairing the hypothalamic–pituitary–gonadal axis and leading to diminished sperm quality[26,27]. Although our findings did not reach statistical significance, the observed pattern aligns with existing literature indicating that improving sleep quality may have beneficial effects on male reproductive health.

Sleep Latency and Sperm Motility

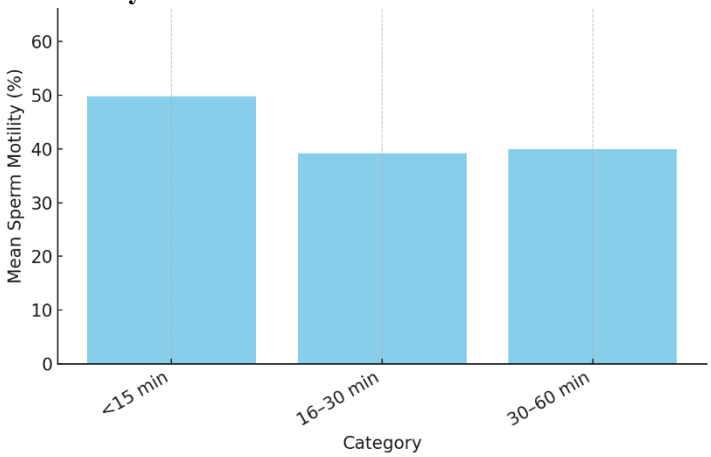

Figure 5. Sleep Latency vs. Sperm Motility

Most participants reported a sleep latency of ≤15 minutes, indicating that they could fall asleep relatively quickly. As illustrated in **Figure 5**, this group had the highest mean sperm motility (49.79%). Although the association between sleep latency and sperm motility was not statistically significant ($p = 0.147$), the observed trend aligns with previous studies. Chen et al. found that men with shorter sleep latency had higher circulating testosterone levels and more favorable semen parameters, including motility[7].

Sleep latency the time taken to transition from wakefulness to sleep is a key indicator of sleep quality and circadian alignment[28]. Shorter latency typically reflects healthy homeostatic sleep drive and well-regulated circadian rhythms. Both systems critically regulate the endocrine environment, particularly the pulsatile

secretion of luteinizing hormone (LH) and the nocturnal surge of testosterone, which are essential for maintaining spermatogenesis.

Testosterone secretion is highly sleep-dependent, with levels rising soon after sleep onset and peaking during the first REM sleep episode. Disruption or delay in sleep onset may blunt this testosterone peak, especially if early sleep stages are compromised. Individuals with prolonged sleep latency may experience reduced duration or quality of slow-wave sleep (SWS) and REM cycles, impairing neuroendocrine signaling necessary for reproductive hormone production[23,29].

Sleep Duration and Sperm Motility

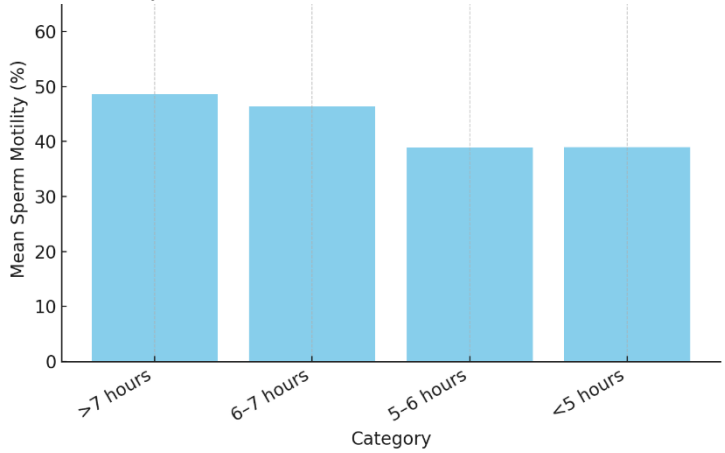

Figure 6. Sleep Duration vs. Sperm Motility

More than 40% of participants reported sleeping more than 7 hours per night, consistent with current recommendations for optimal adult sleep duration. Interestingly, **Figure 6** shows that the highest mean sperm motility was observed among those who slept less than 5 hours per night, a finding that contrasts with the majority of existing literature. Numerous studies have demonstrated that both insufficient (<6 hours) and excessive (>9 hours) sleep are associated with decreased semen quality, including reduced sperm motility, concentration, and abnormal morphology[7,30].

The regulation of spermatogenesis is intricately linked to the hypothalamic–pituitary–gonadal (HPG) axis, which is influenced by circadian rhythms governing the pulsatile secretion of luteinizing hormone (LH) and testosterone both essential for normal sperm production[3,31]. Disruption in sleep duration can impair this hormonal balance, adversely affecting sperm parameters.

The paradoxical observation of higher motility in short sleepers may be explained by several confounding factors. Participants with shorter sleep durations might engage in compensatory behaviors such as increased physical activity, healthier nutrition, or lower psychological stress, mitigating the negative effects of reduced sleep[32]. Additionally, genetic factors such as polymorphisms in circadian clock genes (e.g., PER3, CLOCK) could confer individual resilience to shortened sleep, allowing some men to maintain reproductive hormone homeostasis despite less sleep[23].

Moreover, the quality of sleep—encompassing sleep continuity, efficiency, and architecture—may be more critical than total sleep quantity. For example, short sleepers with high sleep efficiency and minimal nocturnal awakenings may preserve nocturnal testosterone peaks during REM sleep, which is vital for sperm motility[23]. Conversely, long sleepers with fragmented or low-quality sleep might experience impaired hormonal signaling despite adequate sleep duration.

Testosterone secretion is highly sleep-dependent, beginning shortly after sleep onset and peaking during the first REM cycle. Disruptions in sleep architecture especially reductions in slow-wave sleep (SWS) and REM can blunt this peak, impairing spermatogenesis and sperm function[23,29]. Thus, sleep duration must be considered alongside sleep quality to fully understand its impact on male fertility.

Sleep Efficiency and Sperm Motility

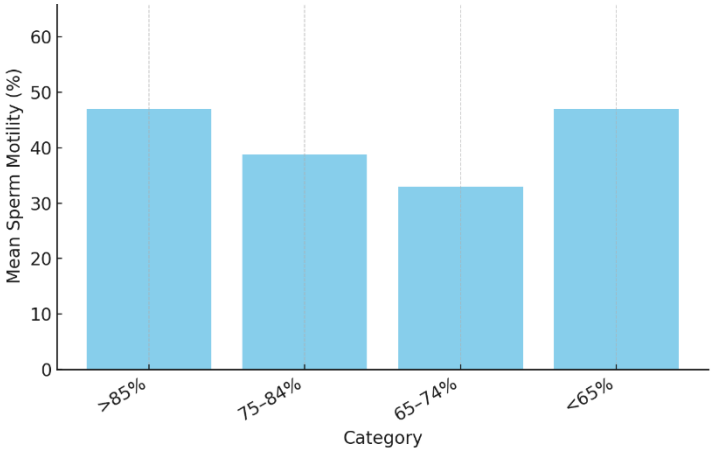

Figure 7. Sleep Efficiency vs. Sperm Motility

In this study, most participants exhibited high sleep efficiency, with over 90% categorized in the >85% range, which is generally considered indicative of healthy and restorative sleep. As shown in **Figure 7**, both the >85% and <65% sleep efficiency groups demonstrated the highest average sperm motility (46.95%), while the 65–74% group showed the lowest (33.00%). Although the association between sleep efficiency and sperm motility was not statistically significant ($p = 0.580$), the pattern observed suggests a potential non-linear or U-shaped relationship, where both very low and very high efficiency may be linked to better motility, albeit through different underlying mechanisms.

High sleep efficiency typically reflects uninterrupted sleep with minimal awakenings, supporting stable circadian rhythms and optimal hormonal regulation[33]. Testosterone, a key hormone for spermatogenesis, is secreted in a pulsatile manner during early sleep stages, particularly during slow-wave and REM sleep. These stages are more prominent in high-efficiency sleep patterns. Adequate testosterone levels are crucial for the function of Sertoli cells, which maintain the seminiferous tubule environment necessary for sperm maturation[14,29]. Therefore, it is plausible that participants with >85% sleep efficiency maintained optimal reproductive hormonal cycles, contributing to better sperm motility.

Interestingly, the <65% sleep efficiency group also showed relatively high motility. While this finding may appear contradictory, it could be influenced by sample size limitations or physiological compensations such as daytime napping, better physical fitness, or genetic resilience to sleep fragmentation. Some individuals may naturally require less sleep or recover more efficiently, maintaining reproductive function despite poor sleep patterns. Variations in circadian clock genes like *PER3* and *CLOCK* may underlie this resilience, influencing both sleep structure and hormonal stability[29].

On the other hand, the 65–74% group exhibited the lowest motility, possibly reflecting a chronic suboptimal sleep state. Unlike the <65% group, which may include more extreme but adaptive sleepers, this intermediate group may experience persistent but unrecognized sleep fragmentation. This can disrupt the balance of testosterone and cortisol—two hormones with opposing effects on spermatogenesis. While testosterone supports Sertoli cell function, cortisol, a catabolic stress hormone, can suppress the hypothalamic–pituitary–gonadal (HPG) axis and impair sperm production[3,14].

It is important to note that sleep efficiency, while useful as a general indicator, does not provide insights into sleep architecture, quality, or circadian timing. High efficiency does not always equate to optimal sleep, especially if it occurs in the context of low physical activity, depressive states, or hypersomnia[34]. Conversely, low efficiency may not always indicate dysfunction, particularly in individuals with adaptive physiology. Thus, sleep efficiency should be interpreted in context, ideally alongside objective sleep assessments such as polysomnography, hormonal profiling, and lifestyle factors to accurately evaluate its impact on male reproductive health.

Sleep Disturbance and Sperm Motility

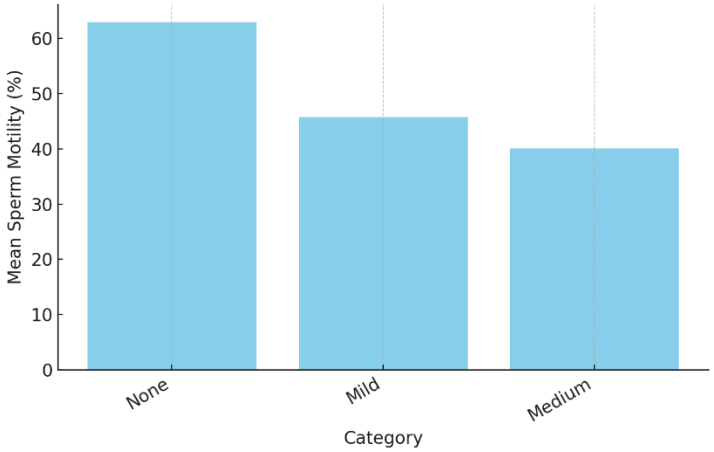

Figure 8. Sleep Disturbance vs. Sperm Motility

In this study, mild sleep disturbances were most commonly reported, indicating that many participants experienced some level of disrupted sleep. As shown in **Figure 8**, those with no reported disturbances had the highest sperm motility, while mild and medium disturbance groups showed lower values. Although the association was not statistically significant ($p = 0.469$), the trend suggests potential biological relevance.

Sleep disturbances, even when mild, can fragment sleep architecture especially slow-wave and REM sleep which are crucial for testosterone and luteinizing hormone (LH) secretion[12,26]. Disruption of these hormonal rhythms may impair Sertoli cell function and spermatogenesis, ultimately reducing sperm motility. Furthermore, fragmented sleep elevates oxidative stress markers like reactive oxygen species (ROS) and 8-OHdG, which can damage sperm membranes, DNA, and mitochondria—components essential for motility[7].

The absence of statistical significance may be due to the mild and possibly infrequent nature of the disturbances reported, or underreporting via subjective tools like the PSQI. Moreover, variations in individual stress response, sleep need, or genetic resilience could buffer the impact of sleep disturbance in some participants[29].

Thus, while the results were inconclusive statistically, the physiological pathways involved support the possibility that sleep disturbances even at low intensity can negatively influence sperm motility.

Use of Sleep Medication and Sperm Motility

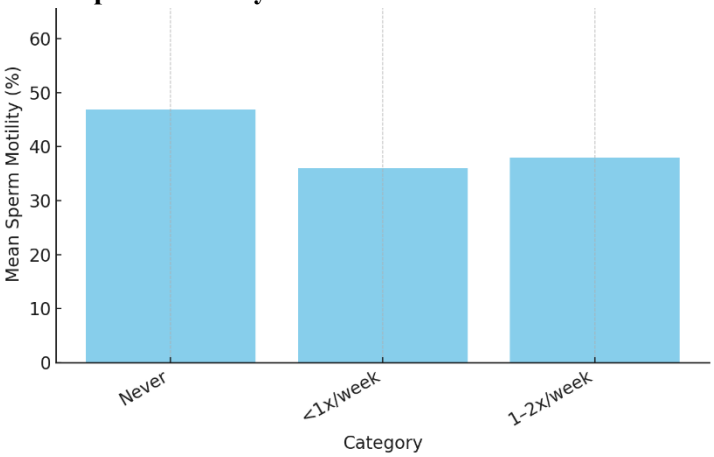

Figure 9. Use of Sleep Medication vs. Sperm Motility

In this study, nearly all participants (96.7%) reported never using sleep medications, resulting in limited sample size for statistical comparison. As shown in **Figure 9**, non-users had the highest mean sperm motility (46.83%), while those who used sleep medication regardless of frequency had lower motility. Although no statistical analysis was conducted due to the small number of users, the observed trend raises concerns regarding the potential reproductive effects of sleep medications.

Several classes of sleep medications, particularly benzodiazepines and non-benzodiazepine hypnotics, are known to interfere with the hypothalamic–pituitary–gonadal (HPG) axis. These agents can alter the neuroendocrine regulation of gonadotropin-releasing hormone (GnRH), thereby reducing the secretion of luteinizing hormone (LH) and testosterone both critical for spermatogenesis[12]. Chronic use of these medications may blunt nocturnal testosterone surges by disrupting normal sleep architecture, particularly REM sleep, which is essential for hormonal pulsatility.

Furthermore, some sleep medications have sedative and muscle-relaxing effects that may impair testicular thermoregulation or reduce sperm motility directly by affecting mitochondrial function[35]. Benzodiazepines, in particular, have been associated with increased oxidative stress and suppressed steroidogenesis in animal models, though evidence in humans remains limited.

Given the extremely small proportion of medicated subjects in this study, conclusions must be drawn with caution. Nonetheless, the lower motility among users aligns with mechanistic evidence that sleep medications may compromise reproductive function. Further research involving larger samples of sleep medication users preferably with stratification by drug class, dosage, and duration is necessary to clarify this relationship.

Daytime Dysfunction and Sperm Motility

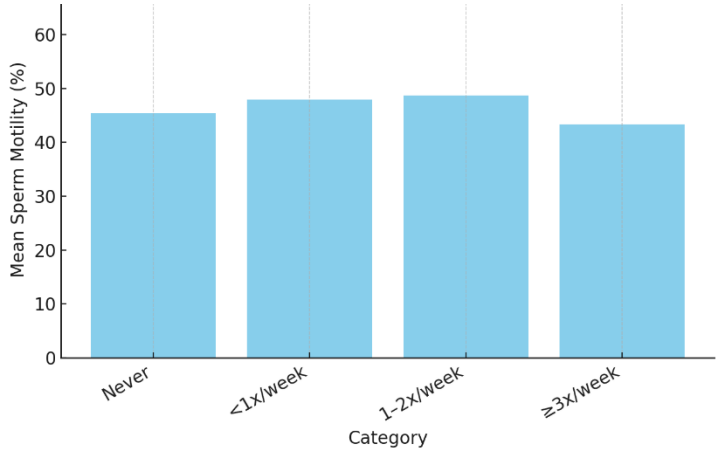

Figure 10. Daytime Dysfunction vs. Sperm Motility

In this study, nearly half of the participants reported no daytime dysfunction, indicating that most individuals maintained adequate levels of alertness and daily functioning despite variations in sleep quality. As shown in **Figure 10**, the highest average sperm motility was unexpectedly observed in participants who reported experiencing daytime dysfunction once or twice per week. However, this trend was not statistically significant ($p = 0.976$), suggesting a weak or inconsistent relationship between subjective daytime performance and sperm motility.

Daytime dysfunction, as measured by the Pittsburgh Sleep Quality Index (PSQI), reflects perceived impairments in alertness, concentration, and energy during waking hours. These impairments are often linked to poor sleep quality or quantity[36]. Previous studies, such as that by Ji et al., have associated good sleep quality with reduced daytime impairment, emphasizing that restful sleep contributes to optimal cognitive and physical performance during the day[12]. However, the direct connection between daytime dysfunction and male reproductive parameters remains unclear. To date, there is insufficient evidence to establish a consistent or biologically plausible link between daytime dysfunction and semen quality, including sperm motility[12].

One possible explanation for the lack of association is that daytime dysfunction is influenced by a wide array of non-reproductive factors. These include mental health conditions such as anxiety or depression, nutritional status, occupational workload, and psychological stress—all of which may impact daily functioning but not necessarily affect spermatogenesis or sperm quality. Additionally, the perception of dysfunction is subjective and may vary with mood or lifestyle factors, introducing potential bias and limiting its utility as a proxy for sleep-dependent hormonal regulation.

General Sleep Quality and Sperm Motility

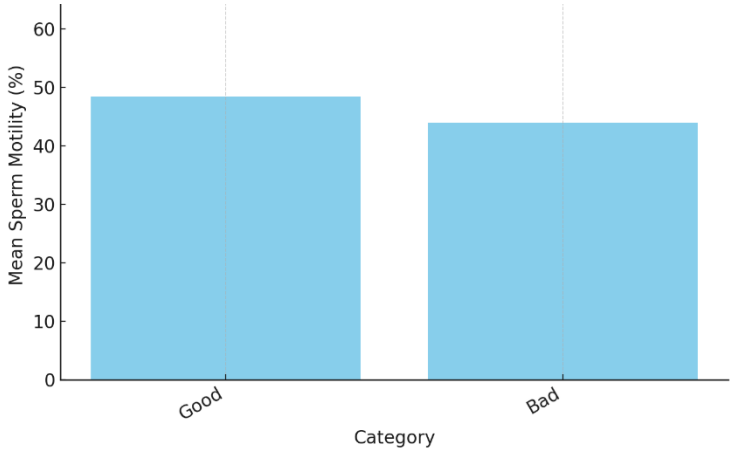

Figure 11. Sleep Quality vs. Sperm Motility

Our primary hypothesis proposed that poor sleep quality would be significantly associated with reduced sperm motility in infertile men. Although statistical analysis using the independent sample t-test did not show a significant difference between the groups ($p > 0.05$), the average sperm motility was lower in the poor sleep quality group (43.90%) compared to the good sleep quality group (48.43%) (**Figure 11**). While this difference may appear modest, the trend observed is biologically meaningful and is supported by a growing body of evidence linking sleep disturbances to impaired male reproductive function.

Sleep quality plays a vital role in maintaining the balance of the hypothalamic–pituitary–gonadal (HPG) axis, which governs the release of key reproductive hormones[37]. Testosterone secretion, in particular, is closely tied to sleep architecture. Under normal physiological conditions, testosterone levels peak during the night, particularly during rapid eye movement (REM) sleep, and decline progressively throughout the day[26,27]. Disruptions in REM or slow-wave sleep due to fragmented or poor-quality sleep can blunt this nocturnal testosterone surge, reducing its bioavailability and impairing spermatogenesis.

Furthermore, poor sleep quality may also suppress the pulsatile release of luteinizing hormone (LH), which is essential for stimulating Leydig cells in the testes to produce testosterone[38,39]. Inadequate LH stimulation and lowered testosterone levels disrupt multiple stages of spermatogenesis, including sperm cell differentiation, mitochondrial maturation, and motility acquisition. Testosterone also supports the epididymal environment, where sperm gain progressive motility and fertilizing capacity[40]. Thus, reduced hormonal support due to sleep disruption may impair both sperm quantity and function.

Beyond hormonal mechanisms, chronic poor sleep has been associated with increased oxidative stress, a key contributor to male infertility. Fragmented or insufficient sleep elevates reactive oxygen species (ROS) production and decreases antioxidant defense mechanisms, leading to cellular damage. In semen, excessive ROS can cause lipid peroxidation of the sperm membrane, mitochondrial dysfunction, and DNA fragmentation all of which adversely affect sperm motility and fertilization potential[7,41]. Elevated levels of oxidative biomarkers such as 8-hydroxy-2'-deoxyguanosine (8-OHdG) have been detected in sleep-deprived individuals and correlated with reduced semen quality.

In summary, while the statistical association between sleep quality and sperm motility in this study was not significant, the observed trend, as visualized in **Figure 11**, is consistent with the known physiological effects of sleep on the male reproductive axis. The multifaceted impact of poor sleep including hormonal disruption, oxidative damage, and mitochondrial impairment supports the hypothesis that sleep quality plays a clinically relevant role in male fertility outcomes. Future studies with larger samples and objective sleep measurements may further elucidate this relationship.

CONCLUSION

This study investigated the relationship between sleep quality, as measured by the Pittsburgh Sleep Quality Index (PSQI), and sperm motility among infertile men. While no statistically significant associations were found between overall sleep quality or its individual components and sperm motility, several trends suggest potential biological relevance. Participants with better subjective sleep quality, shorter sleep latency, and higher sleep efficiency consistently demonstrated higher mean sperm motility compared to their counterparts with poorer sleep metrics. Additionally, demographic and lifestyle variables such as age, BMI, and smoking habits did not show significant correlations, yet descriptive patterns aligned with existing literature on reproductive health.

The absence of statistical significance may be attributed to the limited sample size, the use of self-reported sleep assessments, and the cross-sectional design of the study. Despite these limitations, the findings underscore the complex and multifactorial nature of male infertility and support the hypothesis that sleep, through its influence on endocrine regulation and oxidative balance, plays an important role in maintaining sperm quality.

Future studies should incorporate objective sleep measurements (e.g., actigraphy or polysomnography), detailed hormonal and oxidative stress profiling, and larger, more diverse populations. Longitudinal designs are also needed to explore causal relationships and assess whether interventions aimed at improving sleep can lead to measurable improvements in semen parameters.

In conclusion, while the results of this study did not yield statistically significant outcomes, they provide valuable insights into potential links between sleep behavior and male reproductive health. Sleep quality should be considered a relevant lifestyle factor in the evaluation and management of male infertility.

REFERENCES

1. WHO World Health Organization. Infertility [Homepage on the Internet]. 2023 [cited 2024 Apr 8];Available from: https://www.who.int/news-room/fact-sheets/detail/infertility
2. Badan Penelitian dan Pengembangan Kesehatan Kementerian Kesehatan RI. Riset Kesehatan Dasar. Expert Opin Investig Drugs 2013;
3. Akbar A. Gambaran Faktor Penyebab Infertilitas Pria Di Indonesia : Meta Analisis. Jurnal Pandu Husada 2020;1(2):66.
4. Caetano G, Bozinovic I, Dupont C, Léger D, Lévy R, Sermondade N. Impact of sleep on female and male reproductive functions: a systematic review. Fertil Steril [homepage on the Internet] 2021;115(3):715–731. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0015028220321889
5. Qin X, Fang S, Cai Y. Sleep disorders and risk of infertility: A meta-analysis of observational studies. PLoS One [homepage on the Internet] 2023;18(10 October):1–10. Available from: http://dx.doi.org/10.1371/journal.pone.0293559
6. Du CQ, Yang YY, Chen J, Feng L, Lin WQ. Association between sleep quality and semen parameters and reproductive hormones: A cross-sectional study in Zhejiang, China. Nat Sci Sleep 2020;12:11–18.
7. Chen HG, Sun B, Chen YJ, et al. Sleep duration and quality in relation to semen quality in healthy men screened as potential sperm donors. Environ Int 2020;135:105368.

8. Setyowati A, Chung MH. Validity and reliability of the Indonesian version of the Pittsburgh Sleep Quality Index in adolescents. Int J Nurs Pract 2021;27(5):1–7.
9. Huang B, Wang Z, Kong Y, Jin M, Ma L. Global, regional and national burden of male infertility in 204 countries and territories between 1990 and 2019: an analysis of global burden of disease study. BMC Public Health 2023;23(1):1–12.
10. Williams VF, Atta I, Stahlman S. Brief Report: Male Infertility, Active Component, U.S. Armed Forces, 2013–2017. 2019;26(03):2013–2017.
11. Evans EPP, Scholten JTM, Mzyk A, et al. Male subfertility and oxidative stress. Redox Biol [homepage on the Internet] 2021;46:102071. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2213231721002305
12. Walke G, Gaurkar SS, Prasad R, Lohakare T, Wanjari M. The Impact of Oxidative Stress on Male Reproductive Function: Exploring the Role of Antioxidant Supplementation. Cureus 2023;15(7):1–13.
13. Lei T, Yang Y, Yang W-X. Luteinizing Hormone Regulates Testosterone Production, Leydig Cell Proliferation, Differentiation, and Circadian Rhythm During Spermatogenesis. Int J Mol Sci [homepage on the Internet] 2025;26(8):3548. Available from: https://www.mdpi.com/1422-0067/26/8/3548
14. Mattos K de, Pierre KJ, Tremblay JJ. Hormones and Signaling Pathways Involved in the Stimulation of Leydig Cell Steroidogenesis. Endocrines [homepage on the Internet] 2023;4(3):573–594. Available from: https://www.mdpi.com/2673-396X/4/3/41
15. Sengupta P, Pinggera GM, Calogero AE, Agarwal A. Oxidative stress affects sperm health and fertility—Time to apply facts learned at the bench to help the patient: Lessons for busy clinicians. Reprod Med Biol 2024;23(1):1–17.
16. Sharma A, Kumar M. Basics of Human Andrology: A Textbook 1st ed. 2017 Edition. 2017;
17. Unicef. Analisis Lanskap Kelebihan Berat Badan dan Obesitas di Indonesia. Unicef [homepage on the Internet] 2023;6. Available from: https://www.unicef.org/indonesia/media/16691/file/Ringkasan untuk Pemangku Kebijakan.pdf
18. Putri, Nadhiroh SR. Tinjauan Literatur: Hubungan Obesitas dan Infertilitas pada Pria Usia Produktif. Amerta Nutrition [homepage on the Internet] 2024;8(2):318–327. Available from: https://e-journal.unair.ac.id/AMNT/article/view/46752
19. Rochira V, Carani C. Estrogens, Male Reproduction and Beyond [Homepage on the Internet]. 2020; Available from: http://www.ncbi.nlm.nih.gov/pubmed/26438567
20. Venigalla G, Ila V, Dornbush J, et al. Male obesity: Associated effects on fertility and the outcomes of offspring. Andrology [homepage on the Internet] 2025;13(1):64–71. Available from: https://onlinelibrary.wiley.com/doi/10.1111/andr.13552
21. Zulfikar MA, Hestianah EP, Hermadi HA, et al. Effect of Exposure to E-Cigarette Vapor and Cigarette Smoke on Seminiferous Tubules Diameter and Spermatozoa Quality of Mice (Mus musculus). Ecology, Environment and Conservation 2022;28(3):1246–1251.
22. Akbar A. Pengaruh Paparan Asap Rokok Terhadap Sperma Pria: Systematic Review. J Chem Inf Model 2021;53(February).

23. Zhong O, Liao B, Wang J, Liu K, Lei X, Hu L. Effects of Sleep Disorders and Circadian Rhythm Changes on Male Reproductive Health: A Systematic Review and Meta-analysis. Front Physiol [homepage on the Internet] 2022;13. Available from: https://www.frontiersin.org/articles/10.3389/fphys.2022.913369/full
24. Fitriyani, Fathurrahman A, Mandala Z. Gambaran Kualitas Tidur Pada Mahasiswa Pendidikan Profesi Dokter Di Rumah Sakit Pertamina Bintang Amin. Jurnal Ilmu Kedokteran dan Kesehatan [homepage on the Internet] 2024;11(6):2549–4864. Available from: http://ejurnalmalahayati.ac.id/index.php/kesehatan
25. Fusco F, Longo N, Sio M De, et al. Impact of Circadian Desynchrony on Spermatogenesis: A Mini Review. Front Endocrinol (Lausanne) [homepage on the Internet] 2021;12:800693. Available from: http://www.ncbi.nlm.nih.gov/pubmed/34975770
26. Li L, Lin W, Wang Z, et al. Hormone Regulation in Testicular Development and Function. Int J Mol Sci [homepage on the Internet] 2024;25(11):5805. Available from: https://www.mdpi.com/1422-0067/25/11/5805
27. Yao Y, Silver R. Mutual Shaping of Circadian Body-Wide Synchronization by the Suprachiasmatic Nucleus and Circulating Steroids. Front Behav Neurosci [homepage on the Internet] 2022;16. Available from: https://www.frontiersin.org/articles/10.3389/fnbeh.2022.877256/full
28. Foster RG. Sleep, circadian rhythms and health. Interface Focus [homepage on the Internet] 2020;10(3):20190098. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32382406
29. Zhao X, Lan M, Li H, Yang J. Perceived stress and sleep quality among the non-diseased general public in China during the 2019 coronavirus disease: a moderated mediation model. Sleep Med [homepage on the Internet] 2021;77:339–345. Available from: https://doi.org/10.1016/j.sleep.2020.05.021
30. Yland JJ, Eisenberg ML, Willis SK, et al. a Prospective Study of Male Sleep and Semen Quality. Fertil Steril [homepage on the Internet] 2021;116(3):e333. Available from: https://doi.org/10.1016/j.fertnstert.2021.07.897
31. Jamil M. Deteksi Kelainan Organ Reproduksi Wanita yang Mempengaruhi Infertilitas dan Pemeriksaannya, serta Hak Pasien Berdasarkan Permenkes No. 04 Tahun 2018. Implementasi Pengabdian Mayarakat Kesehatan (JIPMK) 2024;6(2).
32. Theorell-Haglöw J, Lemming EW, Michaëlsson K, Elmståhl S, Lind L, Lindberg E. Sleep duration is associated with healthy diet scores and meal patterns: results from the population-based EpiHealth study. J Clin Sleep Med [homepage on the Internet] 2020;16(1):9–18. Available from: http://www.ncbi.nlm.nih.gov/pubmed/31957658
33. Desai D, Momin A, Hirpara P, Jha H, Thaker R, Patel J. Exploring the Role of Circadian Rhythms in Sleep and Recovery: A Review Article. Cureus [homepage on the Internet] 2024;Available from: https://www.cureus.com/articles/258897-exploring-the-role-of-circadian-rhythms-in-sleep-and-recovery-a-review-article

34. Alnawwar MA, Alraddadi MI, Algethmi RA, Salem GA, Salem MA, Alharbi AA. The Effect of Physical Activity on Sleep Quality and Sleep Disorder: A Systematic Review. Cureus [homepage on the Internet] 2023;15(8):e43595. Available from: http://www.ncbi.nlm.nih.gov/pubmed/37719583
35. Vahedi Raad M, Firouzabadi AM, Tofighi Niaki M, Henkel R, Fesahat F. The impact of mitochondrial impairments on sperm function and male fertility: a systematic review. Reprod Biol Endocrinol [homepage on the Internet] 2024;22(1):83. Available from: http://www.ncbi.nlm.nih.gov/pubmed/39020374
36. Pilz LK, Keller LK, Lenssen D, Roenneberg T. Time to rethink sleep quality: PSQI scores reflect sleep quality on workdays. Sleep [homepage on the Internet] 2022;41(5). Available from: https://academic.oup.com/sleep/article/doi/10.1093/sleep/zsy029/4840620
37. Marques P, Sousa Lages A De, Skorupskaite K, Rozario KS, Anderson RA, George JT. Physiology of GnRH and Gonadotrophin Secretion [Homepage on the Internet]. 2020; Available from: http://www.ncbi.nlm.nih.gov/pubmed/1359459
38. Shivalingaiah SB, Tuladhar S, Mahalakshmi AM, et al. Sleep and Gonadotrophin Hormones. Int J Nutr Pharmacol Neurol Dis [homepage on the Internet] 2021;11(1):17–26. Available from: https://journals.lww.com/10.4103/ijnpnd.ijnpnd_97_20
39. Cavalhas-Almeida C, Cristo MI, Cavadas C, Ramalho-Santos J, Álvaro AR, Amaral S. Sleep and male (In)Fertility: A comprehensive overview. Sleep Med Rev [homepage on the Internet] 2025;81:102080. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1087079225000334
40. Sullivan R, Mieusset R. The human epididymis: its function in sperm maturation. Hum Reprod Update [homepage on the Internet] 2020;22(5):574–587. Available from: https://academic.oup.com/humupd/article-lookup/doi/10.1093/humupd/dmw015
41. Marinaro JA, Schlegel PN. Sperm DNA Damage and Its Relevance in Fertility Treatment: A Review of Recent Literature and Current Practice Guidelines. Int J Mol Sci [homepage on the Internet] 2023;24(2). Available from: http://www.ncbi.nlm.nih.gov/pubmed/36674957