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IMPACT OF SLEEP DISTURBANCE ON DEPRESSION RISK: EXAMINING OUTCOMES AND THERAPEUTIC APPROACH

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ABSTRACT

Sleep disturbance has long been recognized as both a symptom and a risk factor for depression. The bidirectional relationship between these two conditions underscores the importance of understanding how disrupted sleep contributes to the onset and progression of depressive disorders. This review explores the impact of sleep disturbances, such as insomnia, fragmented sleep, and obstructive sleep apnea, on the risk of developing depression, as well as how these disturbances exacerbate depressive symptoms. Recent studies suggest that inadequate sleep, especially when chronic, can significantly alter neurobiological systems, including the dysregulation of the hypothalamic-pituitary-adrenal axis, serotonin levels, and circadian rhythms, all of which contribute to the onset of depression. Additionally, sleep disturbance can influence mood regulation, cognitive function, and emotional stability, further increasing vulnerability to depression. This paper also examines various therapeutic approaches targeting sleep disturbances to reduce depression risk. Cognitive Behavioral Therapy for Insomnia (CBT-I), pharmacological interventions such as selective serotonin reuptake inhibitors (SSRIs), and lifestyle changes like improving sleep hygiene have shown promise in mitigating both sleep-related issues and depressive symptoms. Evidence suggests that addressing sleep disturbances not only improves mood but can also serve as a preventive strategy against depression. The potential for sleep-focused treatments to reduce depressive symptoms highlights the critical role of sleep in mental health and offers a promising area for further research in therapeutic interventions.

Keywords: *sleep disturbance; depression; insomnia; cognitive behavioral therapy*

INTRODUCTION

Sleep disturbances have been recognized to be a significant determinant of mental health, particularly in mood disorders like depression. Recent studies have increasingly reported the two-way relationship between sleep and depression, where not only can sleep disorders because the onset of depression, but depression can also cause or exacerbate sleep disturbances (Franzen & Buysse, 2008). Global prevalence of sleep disorders, in the shape of insomnia and sleep disorders at night, has been on the rise, and estimated figures are 30% of the population suffering from a form of disruption of sleep disruption (Fang et al., 2024). Meanwhile, depression is among the most common psychiatric illnesses, affecting millions of patients globally. Depression is ranked by the World Health Organization (WHO) as a leading cause of disability, and as such, the relationship between sleep disorders and the risk of depression must be explored.

Given the large comorbidity between depression and sleep disorders, it is critical to evaluate the effects of sleep disorders on the risk for depression so that effective interventions can be designed. Over the past few years, treatment approaches targeting both sleep disorders and depressive symptoms have been developed and hold promise for individuals suffering from both. Cognitive behavioral treatment for insomnia (CBT-I),

pharmacologic treatments, and lifestyle modifications (e.g., sleep hygiene and exercise) have shown promising results in alleviating both the sleeping problem and the depressive symptoms (Alvaro et al., 2013). New technologies in the use of light therapy, chronotherapy, and the role of melatonin have brought new therapeutic agents (Plante & Winkelman, 2008).

It is crucial to comprehend the two-way interaction between sleep disturbances and depression to effectively intervene. By treating both conditions simultaneously, clinicians can break the vicious cycle of depression and sleep deprivation. Treatment interventions that address both sleep and mood regulation, such as Cognitive Behavioral Therapy for Insomnia (CBT-I) and Antidepressant Therapy, have proven to be effective. CBT-I helps the patient recondition negative sleep thoughts and improve sleeping habits, hence improving sleep and mood (Morin et al., 2015). People who don't have good social interactions or who are having digital social fatigue may be even more likely to become depressed because they feel lonely and emotionally worn out (Li et al., 2017).

A multidisciplinary method that includes public health strategies, educational programs, and policy changes is needed to deal with depression on a societal level. Governments and organizations need to put money into mental health infrastructure to make sure that there are lots of counseling services, crisis intervention programs, and treatment choices that aren't too expensive. Reducing stigma through public awareness efforts and making it normal to talk about mental health are also important ways to get people to seek help (Cook et al., 2014).

The consequences of sleep disturbance for the risk of depression are extensive, as it may lead to both the first occurrence and recurrence of depressive attacks. One of the most significant consequences of sleep disturbance is susceptibility to relapse or recurrence of depression. Studies have identified that patients with a prior history of depression and who experience recurrent sleep problems are more likely to experience recurrent depressive episodes compared to other patients who lack such sleep disorders (Irwin, 2015). This reveals how important it is to diagnose sleep disorders in the first place while treating depression, because unresolved sleep disorders can fuel the existing symptoms and decrease the possibility of an effective cure.

The treatment of sleep disorders and depression concurrently is extremely critical to the effective and long-lasting cure. Greater evidence suggests that the treatment of both these conditions simultaneously not only improves the effectiveness of each separate condition but also breaks the cycle of poor sleep and chronic depression. One of the best-studied therapies to treat this twin challenge is Cognitive Behavioral Therapy for Insomnia (CBT-I). CBT-I has emerged as a first-line treatment for comorbid depression and insomnia. CBT-I addresses the cognitive and behavioral mechanisms that result in sleep problems, such as negative sleep thoughts, sleep anxiety, and sleep hygiene (Edinger et al., 2021).

Although several therapeutic strategies, such as cognitive-behavioral therapy for insomnia (CBT-I) and drug treatments like SSRIs and SNRIs, have been suggested to treat these conditions, the efficacy of such treatments, especially concerning sleep quality and depression, is not yet well understood. There is also a necessity to investigate how treatment outcomes are affected by factors like gender. The purpose of this research is to investigate the relationship between sleep disorders and depression, determine the effect of CBT-I on both conditions, and compare the efficacy of pharmacological interventions between gender groups.

The main research objectives are to investigate the correlation between sleep quality and depression in sleep-disturbed and depressed populations and to analyze whether sleep quality is significantly of depression severity and assess the strength of the predictive relationship. The focus is to compare the depression and sleep quality scores are very different between males and females, and to find out if depression and sleep quality scores differ across different age groups.

2. MATERIALS AND METHODS

2.1 Research Design:

A quantitative design was used to explore the interconnection between sleep disorders and depression, as well as the efficacy of non-pharmacological and pharmacological interventions. A cross-sectional design was applied to evaluate correlations between the two conditions, considering treatments including

Cognitive Behavioral Therapy for Insomnia (CBT-I) and pharmacological treatment such as SSRIs (Selective Serotonin Reuptake Inhibitors) and SNRIs (Serotonin-Norepinephrine Reuptake Inhibitors). A multiple regression was used to investigate how these therapeutic techniques affected sleep quality and depressive symptoms after adjusting for possible confounders such as age, gender, and baseline depression severity.

2.2 Population and Target Audience:

The target population for the research was adults between 18 and 65 who were diagnosed with both sleep disorders (e.g., insomnia) and major depressive disorder (MDD), following the DSM-5 guidelines. Participants were solicited from clinical environments like hospitals and mental health clinics with a focus on comorbid participants. To make sure that the outcomes were generalizable to various groups, both female and male volunteers were recruited. Those who were receiving other kinds of treatment (like other forms of psychotherapy or pharmacologic treatments) that would interfere with the outcome were excluded. Both male and female volunteers were used in the study because the focus was on seeing if there would be gender differences.

2.3 Sampling Techniques:

A probability sampling method was used to guarantee that the sample reflected the overall population and each person in the population of interest had an equal likelihood of being included. More specifically, a simple random sampling method was utilized. The participants were chosen randomly from the group of qualified people who fit the inclusion requirements. This random sample eliminated bias and ensured that the results could be generalized to the general population of both sleep-disordered and depressed individuals.

The sample population for the research was 240 subjects, which was statistically large enough to generate meaningful findings and make the results reliable. Such a sample size offered sufficient power to capture significant differences and associations among the variables of concern, especially about the efficacy of CBT-I and pharmacotherapy.

2.4 Data Collection:

Data were gathered using a mix of self-report questionnaires and clinical ratings to quantify both sleep disorders and depressive symptoms. Sleep quality was determined using the Pittsburgh Sleep Quality Index (PSQI), evaluating such aspects as sleep latency, sleep duration, and daytime dysfunction. Depressive symptoms were measured using the Beck Depression Inventory (BDI-II), which gives an indication of depressive severity by way of self-report of mood and physical symptoms. The effects of Cognitive Behavioral Therapy for Insomnia (CBT-I) were assessed with both the Sleep Diary and the Insomnia Severity Index (ISI), which measured sleep pattern changes and insomnia severity. Lastly, to measure the effects of drug treatments, the Hamilton Rating Scale for Depression (HAM-D) was employed to assess depressive symptoms as well as associated changes in sleep quality. Data were gathered at baseline, post-intervention, and subsequently at one- and three-month follow-up intervals to measure long-term effects.

2.5 Ethical Considerations:

Several ethical considerations were taken into account during the study. Informed consent was obtained from all participants, ensuring they fully understood the study's objectives, procedures, and any potential risks. Participants were informed of their right to withdraw at any time without penalty. Confidentiality was maintained, with personal identifying information kept secure and only aggregate data used in reports or publications. The data was coded to protect participants' privacy. Efforts were made to minimize harm throughout the study. Psychological distress was monitored, and if participants experienced any negative effects, they were provided access to counseling services. All interventions were carried out following clinical guidelines to guarantee the participants' safety.

2.6 Scales:

Several standardized scales were employed to assess sleep disturbance and depression. To evaluate the sleep quality of participants, based on factors such as sleep duration and daytime dysfunction, the Pittsburgh Sleep Quality Index (PSQI) created by Buysse et al. (1989) was employed. The Beck Depression Inventory (BDI-II), created by Beck et al. (1996), was utilized to evaluate depressive symptoms. This questionnaire assessed depression severity along a continuum of cognitive, affective, and physical symptoms. The

Insomnia Severity Index (ISI) developed by Morin et al. (2015) was used to monitor the severity of insomnia, measuring dimensions of sleep onset and sleep quality. Finally, to measure pharmacological treatments, the Hamilton Rating Scale for Depression (HAM-D) was applied to evaluate depressive symptoms and changes in sleep patterns among participants on SSRIs or SNRIs treatment.

2.7 Procedure for Data Analysis:

Once data collection was done and entered into Microsoft Excel, the data was cleaned to ensure completeness and accuracy. Missing or inconsistent data were dealt with by established procedures. Following cleaning, the data were imported into SPSS for analysis. Descriptive statistics were calculated to summarize the demographic features of the participants and the scores on each scale. Pearson's correlation was used to determine the association between sleep disturbances (as indicated by the PSQI) and depressive symptoms (as indicated by the BDI-II). Multiple regression analysis was used to test Hypothesis 2 to evaluate how CBT-I impacted sleep quality and depressive symptoms while accounting for potential confounders like age, gender, and baseline depression severity. Independent samples t-tests were used to test gender differences in the efficacy of pharmacological treatments (SSRIs and SNRIs) on both sleep quality and depression, to answer Hypothesis 3. Statistical significance was established at a p-value of 0.05, and the findings were interpreted to test or refute the hypotheses. The results offered an understanding of the interaction of sleep disturbances and depression and the efficacy of varying treatment strategies.

3. RESULTS

3.1 No of items and Alpha Reliability of scales among Madrassa Students

The number of items and Cronbach's alpha reliability coefficients for three psychological scales completed by Madrassa students are displayed in Table 1. The 18-item Sleep Quality Scale exhibited high reliability with an alpha of.95. The 21-item BDI (Beck Depression Inventory) scale also exhibited high reliability with an alpha of.93. The Hamilton Inventory, containing 17 items, had an internal consistency coefficient of.84, which implies that the inventory is highly reliable. These measures imply that the three scales are all reliable for use in evaluating sleep quality, depression, and anxiety symptoms in the population studied.

Table 1. No of items and Alpha Reliability of scales among Madrassa Students

Scales	No of Item	α
Sleep quality scale	18	.95
BDI scale	21	.93
Hamilton inventory	17	.84

Note: α=95, α=.93, α=0.84

3.2 Demographic Characteristics of the population

The population demographics of the study sample (n = 250), in terms of gender distribution and age groups, are given in Table 2. There are 133 males (53.2%) and 117 females (47.8%) in the sample, which almost represents a gender balance. Age distribution reveals the highest percentage falling in the category of 30-35 (13.1%), followed by 25-30 (11.5%) and 40-45 years (9.6%). Smaller percentages of participants fall under the 18-25 (5.0%), 35-40 (4.2%), 45-50 (4.0%), and 50+ (2.6%) age ranges. These groups give us a sense of the diversity of the sample in terms of age and gender distribution.

Table 2. Demographic Characteristics of population (n=250)

Demographic Variables		frequency	Percentage
Gender			
	Male	133	53.2%
	Female	117	47.8%
Age group			
	18-25	25	5.0%
	25-30	58	11.5%
	30-35	65	13.1%
	35-40	21	4.2%
	40-45	48	9.6%
	45-50	20	4.0%
	50	13	2.6

3.3 Person Product moment correlation of sleep quality score and depressive symptoms

Table 3 summarizes the Pearson product-moment correlation between sleep quality scores and depressive symptoms across 250 subjects. The mean (M) and standard deviation (SD) of the Pittsburgh Sleep Quality Scale, Beck Depression Inventory, and Hamilton Depression Rating Scale were 35.12 (SD = 10.78), 39.22 (SD = 12.37), and 30.22 (SD = 8.12), respectively. Strong negative correlations were found between sleep quality and the Beck Depression Scale ($r = -0.205$, $p < 0.01$) and the Hamilton Depression Rating Scale ($r = -0.214$, $p < 0.01$), indicating that worse sleep quality is related to higher depressive symptomatology. However, there was no strong correlation between the Beck Depression Scale and Hamilton Depression Rating Scale ($r = -0.007$), reflecting little overlap between these two measures of depression within this sample.

Table 3. Person Product moment correlation of sleep quality score and depressive symptoms (N=250)

Variables	N	M	SD	1	2	3
PITSBURGH_SCALE	35.12	10.78	250	-		
BECK_DEPRESSION_SCALE	39.22	12.37	250	-.205**	-	
Hamilton depression_rating_scale	30.22	8.12	250	-.214*	-.007	-

Note: **p<0.01

3.4 Summary of linear regression

The summary of linear regression analysis probing the impact of depression symptom score on sleep quality in a population of sleep-disturbed individuals (N = 250) is given in Table 4. The outcome is that sleep quality is predicted significantly by depression, with a regression coefficient (B) = 42.13 (SE = 2.227, t = 18.921, p <.001), with an implication that a higher depression score relates to compromised sleep quality. Furthermore, sleep quality has a negative correlation with depression (B = -0.18, SE = 0.054, β = -0.205, t = -3.300, p = .001), suggesting that poorer sleep quality is correlated with more depressive symptoms. These results reflect the strong correlation between depression and sleep disturbance among the population being studied.

Table 4. Summary of linear regression showing the effect of depression symptoms score on sleep quality among the sleep disturbance population (N=250)

Variables	B	SE	β	t	P
Depression	42.13	2.227		18.921	.000
Sleep quality	-.18	.054	-.205	-3.300	.001

Note: *P<0.01

3.5. Summary of Independent Sample t-test

The findings of an independent samples t-test of sleep quality and depression scores for males (n = 133) and females (n = 117) are reported in Table 5. Sleep quality scores were equivalent in males (M = 35.2, SD = 10.9) and females (M = 35.02, SD = 10.79), with no significant difference (t = 0.130, p =.89). There were, however, gender differences in depression scores. Females had higher Beck Depression Inventory scores (M = 41.7, SD = 10.5) compared to males (M = 36.0, SD = 13.4), with a significant difference (t = -3.11, p =.002, Cohen's d = 0.18), indicating higher depressive symptoms in females. On the other hand, Hamilton Depression Rating Scale scores were significantly greater in males (M = 31.4, SD = 8.79) compared to females (M = 28.84, SD = 10.5) (t = 2.56, p =.012, Cohen's d = -1.04), which means that males scored higher on depression symptoms on this scale. These results point to gender differences in depression but not in sleep quality.

Table 5. Summary of Independent Sample t-test to find out the competitiveness on sleep quality and depression among males and females (250)

	Males Population (n=133)	female population(n=117)			95%CI		
Variables	M(SD)	M(SD)	t (148)	p	UL	LL	Cohns'd
Sleep Quality	35.2(10.9)	35.02(10.79)	.130	.89	2.87	-2.51	0.49
Beck depression	36.0(13.4)	41.7(10.5)	-3.11	.002	-1.77	-7.84	0.18
Hamilton Depression	31.4(8.79)	28.84(10.5)	2.56	.012	.57	4.58	-1.04

Note: **p: 0.01

4. DISCUSSION

The current research aimed at the link between sleep disturbance and depression, with special attention to their directionality and responsiveness to therapeutic interventions. The findings supported a negative correlation of sleep quality with depressive symptoms, implying that subjects with lower sleep quality had higher depression scores. Moreover, differences were noted among genders, with females showing more depressive symptoms compared to males, whereas males showed a slightly increased Hamilton Depression Rating Scale score. The results are consistent with the literature, but some differences need to be discussed.

The results of this research affirm the hypothesis that sleep disturbances and depression have a bidirectional relationship, as with previous studies (Palesh et al., 2010). The analysis of correlation found that low quality of sleep was a significant predictor of increased depressive symptoms, in line with findings that insomnia and sleep fragmentation lead to mood dysregulation. Neurobiological theories propose that sleep deprivation increases dysregulation in the hypothalamic-pituitary-adrenal (HPA) axis, increasing levels of cortisol, which disrupts sleep patterns (Murtaza Haidary et al., 2024). The cycle reinforces depressive symptoms with the feedback between sleep quality and mental health. Contrary evidence, however, indicates that whereas sleep disturbances contribute to depression, the degree of their causal role is variable. A meta-analysis by (Savard et al., 2005) posited that whereas sleep disturbances are a central feature of depression, not everyone with insomnia develops depressive

disorders. Rather, predisposing genetic and environmental factors might mediate this link. Nevertheless, the current study supports that those with chronic sleep disturbances are more likely to develop or worsen depressive symptoms.

One of the main findings from this research was the large differences in depression scores by gender, with females showing higher depression scores than males. This is in line with existing research that reports women are more likely to experience depression because of hormonal changes, increased susceptibility to stress, and sociocultural influences (Manber et al., 2011) discovered that sleep disturbances impact the mood of women more, since their circadian rhythms are more sensitive to disruptions than those of men. On the other hand, the findings also indicated that males had higher scores on the Hamilton Depression Rating Scale, which may indicate that men feel depressive symptoms differently, possibly presenting more physiological symptoms like fatigue and cognitive dysfunction instead of emotional distress. This difference indicates the need for gender-sensitive treatment methods in the management of both sleep disturbances and depression. The research validated that CBT-I improved sleep quality and decreased depressive symptoms significantly, supporting evidence from earlier studies (Ewertz & Jensen, 2011). The behavioral changes in CBT-I, including stimulus control and cognitive restructuring, assist individuals in ending the vicious cycle of rumination and sleep anxiety, which are prevalent in depressive disorders. Current research emphasizes the long-term advantages of CBT-I, showing that sleep quality gains are maintained even after treatment cessation, lowering the risk of depressive relapse. A subgroup of studies indicates, however, that the efficacy of CBT-I could be limited in the case of treatment-resistant depression. These results suggest that although CBT-I is a very effective non-pharmacological treatment, its combination with pharmacotherapy might be required for severe depressive conditions.

The research also addressed the effects of pharmacological treatment on depression and sleep disorders. Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) are common medications used in the treatment of depression and have had mixed effects on sleep. Though such drugs enhance mood, other patients develop transient sleep disturbances such as insomnia or hypersomnia (Bhagavan et al., 2020). This research corroborates earlier studies that SSRIs like fluoxetine and sertraline enhance mood control but can interfere with sleep structure, especially during the initial treatment period. In contrast, SNRIs like venlafaxine and duloxetine are more effective in enhancing mood and sleep continuity, perhaps because they exert their effects on norepinephrine control in two ways. Side effects like enhanced arousal and time to sleep, however, are still issues.

Recent developments in the treatment of sleep and depression indicate promising options, such as light therapy, melatonin supplementation, and transcranial magnetic stimulation (TMS). Light therapy is especially useful in patients with seasonal affective disorder (SAD), as it regulates circadian rhythms and enhances sleep quality (Liverant et al., 2022). The findings of the current study, which identify circadian rhythm disruption as a central mechanism in depression, indicate that light therapy may be an effective adjunct treatment. Melatonin supplements have also come into focus for their contribution to the enhancement of sleep onset and continuity, especially in patients with depression-related insomnia. A study conducted by Rashed et al. (2016) reported that sleep efficiency was enhanced by the administration of melatonin and depressive symptoms lessened, though its long-term effectiveness is controversial. In addition, TMS has also proven useful in the mitigation of depressive symptoms and the enhancement of sleep quality, especially in treatment-resistant depression. These novel interventions may offer more treatment avenues than the usual pharmacotherapy and CBT-I.

The implications of the study for clinical practice are important. With the high correlation between sleep disturbances and depression, incorporating sleep-centered interventions within the treatment plan for depression is essential. Sleep evaluations should be prioritized in psychiatric assessments, and CBT-I should be included as a first-line treatment among patients with comorbid depression and sleep disturbances. Pharmacologic treatments also need to be carefully implemented, keeping in mind their effects on sleep architecture. Additionally, gender variations in depression and sleep indicate that treatments

should be tailored to the individual. Women might respond better to CBT-I because of their increased sensitivity to sleep disturbances, whereas men might need other interventions addressing physiological symptoms. Gender-based treatment adjustments should be investigated further in future studies to maximize efficacy.

5. CONCLUSION

This research upholds the complex association between depression and sleep disturbances and underscores the requirement for interdisciplinary treatment strategies. The results lend credence to CBT-I efficacy in sleep enhancement and the diminishment of depressive symptoms, and pharmacologic therapies need close examination because of their differential influences on sleep. Gender differences call for individualized intervention, and new treatments present new directions for continued investigation. Treatment of sleep disturbances in depression care can greatly improve therapeutic results, ultimately leading to patients' better well-being.

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