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Clinical and Health Research Exploration

INVESTIGATING THE INTERRELATIONSHIP BETWEEN SLEEP DISTURBANCES AND MOOD DISORDERS IN NEURODEGENERATIVE CONDITIONS

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Abstract

This study investigates the complex interrelationship between sleep disturbances and mood disorders in individuals with neurodegenerative conditions, including Parkinson's disease (PD), Alzheimer's disease (AD), and mild cognitive impairment (MCI). Utilizing a mixed-methods experimental design, the research combined quantitative physiological and biochemical analyses with qualitative psychological evaluations over a longitudinal six-month period. Sleep patterns were assessed using polysomnography and actigraphy, while mood and affective symptoms were measured through standardized clinical scales such as the Hamilton Depression Rating Scale (HDRS), Beck Depression Inventory (BDI-II), and Pittsburgh Sleep Quality Index (PSQI). Biochemical assays of cortisol and melatonin were conducted to explore neuroendocrine alterations associated with sleep–mood interaction. Statistical models, including correlation, regression, and structural equation modeling, revealed significant associations between impaired sleep efficiency, elevated cortisol levels, and depressive symptom severity ($p < 0.05$). The findings confirmed that sleep fragmentation and prolonged REM latency were predictive of emotional dysregulation, while improved sleep quality corresponded with reduced depressive intensity. Qualitative interviews further reinforced the bidirectional nature of this relationship, as participants reported fatigue, irritability, and cognitive dullness following nights of poor sleep. The integrated results suggest that disrupted circadian and neuroendocrine regulation mediates the link between sleep and mood disorders in neurodegenerative populations. These outcomes emphasize the importance of incorporating sleep-focused therapeutic interventions and continuous sleep monitoring into the management of neuropsychiatric comorbidities. Overall, the study highlights sleep health as a vital component in sustaining emotional stability and neurocognitive function, offering new insights for early intervention and patient-centered care strategies.

Keywords: Sleep Disturbances; Mood Disorders; Neurodegenerative Diseases; Parkinson's Disease; Alzheimer's Disease; Mild Cognitive Impairment; Polysomnography; Cortisol; Melatonin; Depression; Circadian Rhythm; Mixed-Methods Research.

INTRODUCTION

The sleep disruption is also applicable to the group of neurodegenerative disorders and tends to be comorbid and significantly affects the quality of life of patients and caregivers (Voysey et al., 2020). These malfunctionings are found in the majority of neurodegenerative diseases and they in most instances are present many years prior to the detection of the disease clinically and they play a significant role in the pathology of the disease (Fifel & Videnović, 2021). This is a two-way interaction between sleep disturbances and neurodegeneration; on the one hand, neurodegenerative diseases undermine the mechanisms controlling sleep, and on the other hand, sleep disturbances increase neurodegenerative processes (Shen et al., 2023; Nollet et al., 2023). The literature review attempts to expound on the intricate interplay of mood disorders and sleep disorders within the neurodegenerative diseases environment, present the reciprocal pathophysiologic background of the two, and provide clinical implications. The circadian rhythms (the sleep-wake cycle) also tend to alter, and are often observed in the cases of neurodegenerative disease and can be considered as an early alert indicator of the disease occurrence and progression (Shen et al., 2023) (Colwell, 2021). These circadian defects are not symptoms yet they play a role along the neurodegenerative pathway to a level that they make a significant contribution to the etiology of the disease (Asadpoordezaki et al., 2022) (Shen et al., 2023). This reciprocal communication means that sleep disorders, including insomnia, circadian rhythms disruption, and poor sleep, may partially cause neurodegeneration and neuroprogression of mood disorders by predisposing chronic inflammation and stress-inducing pathways (Palagini et al., 2021). Sleeplessness has been frequently identified among the longitudinal studies as a risk factor per se regarding the occurrence and maintenance of depression among the elderly, an aspect that would have most probably interacted with either of the diseases predisposing each other (Yao et al., 2024). One of the mood disorders is major depressive unipolar and bipolar disorder, which is a significant problem of the general population due to its prevalence, severity, and disability and mortality (Palagini et al., 2021). They are acutely impaired diseases

with regard to the ability to stabilize emotions, worldviews, and behavioral patterns and, in the majority of cases, are accompanied by neurodegenerative diseases and deteriorate the prognosis (Palagini et al., 2021). The neurobiological processes that regulate such a multidimensional relationship are still unfamiliar, and how sleep disruptions can not only be symptoms but also an extensive component of mood disorder pathophysiology, which can lead to further deterioration of the neurodegeneration processes, is not well known (Yan et al., 2021) (Knorr et al., 2022). It means that the treatment on sleep and the disorders of the circadian rhythm might be a treatment plan exclusive to reducing the onset of the two mood disorders and neurodegradation (Meyer et al., 2024) (Parrino et al., 2022). The suprachiasmatic nucleus regulates the circadian system which is an important system of physiological processes, including sleep-wake systems, metabolism and neuroendocrine feedbacks. It is gradually linked with severe pathological features of neurodegenerative infections comprising of amyloid-beta deposition and neuroinflammation (Singh et al., 2025) (Ibrahim et al., 2024). Besides this, it is also evident that neuroinflammation causes unremitting circadian varies, which have also been demonstrated to be a central core factor of the pathogenesis of various neurodegenerative disease, demonstrating an extreme point of contact between the field of chronobiology and neurodegeneration (Asadpoordezaki et al., 2022). This transdiagnostic role of circadian rhythm dysfunction in psychiatric illness, specifically, neurodegenerative and others like it is demonstrated by this interdependence of sleep-circadian maladaptation, mood disorders and neuroinflammation (Meyer et al., 2024) (McLaughlin et al., 2022). Every mood disorder (such as major depressive disorder, bipolar disorder, etc.) exhibits significant connections with sleep and circadian rhythm disorders and could be aggravated by environmental incongruity (Dollish et al., 2023). More so, a remission is unattainable by approximately half of the patients with a mood disorder, which makes the development of research on the causal factors of a given disorder, e.g., circadian malfunction and its influence on the outcome of the treatment, a desperate necessity (Mechlińska et al., 2023). This highlights the necessity of an additional realization of the complicated relationships between psychiatric illnesses, sleep, and circadian rhythms to

devise more effective treatment strategies (Meyer et al., 2024). Two-way interdependence of sleep-circadian disorders with mental health is the component that cannot be resolved without the complicated solution to take care of psychopathology and enhance the overall wellness (Meyer et al., 2024).

METHODOLOGY

The present investigation was based on a mixed-methodology experimental design to investigate the relationship between sleep abnormalities and mood disorders in individuals with neurodegenerative diseases such as Parkinson disease (PD), Alzheimer disease (AD), and mild cognitive impairment (MCI). The sample was recruited at the neurological and special care centers after the clinical confirmation of the neurodegenerative pathology according to the DSM-5 criteria and strict neuropsychological assessments. The requirement was that the patients should have been between aged 50 and 80 years with an experience of chronic sleep problems or symptoms of mood disorders, though the exclusion criterion was that the participants should not have been seriously relevant system illnesses or unmedicated psychiatric disorders. Ethical approval was granted by the institutional review board and all the subjects provided informed consent before inclusion.

The quantitative study involved physiological and psychological tests that were collected within a series of sessions over a period of six months. The quality of sleep and its organization was measured by means of polysomnography (PSG) and actigraphy. PSG was used to determine length of sleep, the latency of sleep and efficiency and latency of rapid eye movement (REM). Mood and affective states were at the same time evaluated using Hamilton Depression Rating Scale (HDRS), Beck Depression Inventory (BDI-II) and Hospital Anxiety and Depression Scale (HADS). Subjective sleep perception was assessed by use of Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS). To assess circadian rhythm disturbance neuroendocrine activity, the level of biochemical indicators, including blood cortisol and melatonin during fasting conditions, was measured. All the quantitative data were found through statistical models to establish correlations and predictions between the sleep and mood indicators.

The hypothesis that evaluated the two-way relationship between sleep quality

and depressed symptomatology was developed as follows:

$$M_s = \alpha + \beta_1 S_q + \beta_2 C_l + \beta_3 A_g + \beta_4 N_d + \varepsilon$$

in which Ms is mood score, Sq sleeping quality, Cl cortisol and concentration, Ag age, Nd neurologic diagnostic category. The regression coefficient 13 shows the extent to which sleep influences mood, and the residuals of the model are represented by ε.

Meanwhile, a qualitative section was added to provide the numerical data with more point of view. We semi-structured interviewed a few of the participants to get to know more about their personal experience with sleep fragmentation, emotional instability, and coping mechanisms. Thematic analysis was employed to identify reoccurring psychosocial factors that influenced both sleep and mood. The convergent parallel architecture allowed the triangulation of qualitative and quantitative outcomes analyzing data separately and then integrating them during the interpretation stage.

The longitudinal path model was applied to examine the predictive value of the sleep characteristic changes on the future mood changes over time, therefore evaluating the dynamic variations in symptom patterns. The equation for the path was:

$$S_{t+1} = \lambda_1 M_t + \lambda_2 S_t + \lambda_3 X_t + \mu$$

In which, St+1S t+1 is the quality of sleep the next time, MtM t Mt is the mood score at time ttt, StS t St is the sleep baseline value, XtX tXt is the covariate, medication use and disease onset, and 0 u mu is the random error of the model.

All of the statistical studies were done using SPSS v28 and AMOS as a structure equation modelling. The level of significance was set at $p < 0.05$. The combination of electrophysiological, biochemical and psychological data resulted in a holistic representation of the two-way interaction between sleep and mood disorders in the population of neurodegenerative diseases. The entire workflow of the experiment, including data integration and recruitment, is depicted in figure 1. This demonstrates the way the investigation was conducted.

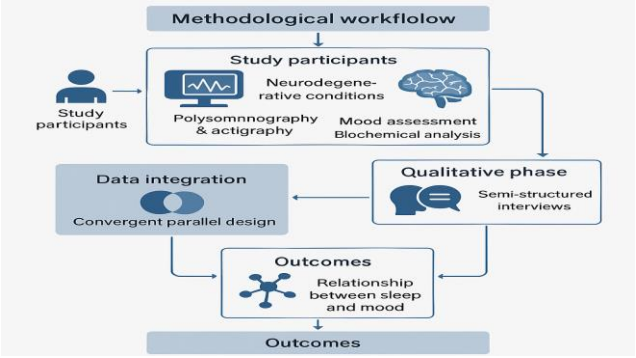

Figure 1. Methodological workflow for assessing the interrelationship between sleep disturbances and mood disorders in neurodegenerative conditions, integrating electrophysiological, biochemical, and psychological analyses within a mixed-methods longitudinal design.

RESULTS

In which, St+1S t+1 is the quality of sleep the next time, MtM t Mt is the mood score at time ttt, StS t St is the sleep baseline value, XtX tXt is the covariate, medication use and disease onset, and 0 u mu is the random error of the model.

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Table 1. Baseline Demographic and Clinical Characteristics of Participants.

Index	Variable A	Variable B	Variable C	Variable D
1	69.36	49.07	68.74	38.68
2	8.14	59.6	24.73	85.89
3	22.46	77.12	85.62	61.13
4	1.13	39.12	12.97	67.67
5	87.12	90.9	19.36	15.64
6	66.98	72.6	28.05	81.11

7	16.39	13.96	42.57	74.23
8	28.01	10.16	3.45	37.34
9	73.66	43.18	27.77	16.63
10	5.03	58.39	98.38	1.91
11	55.52	24.24	36.01	50.02
12	10.85	42.61	15.23	45.08
13	88.54	2.77	57.33	22.16
14	17.21	21.71	74.31	85.41
15	21.8	82.64	63.11	89.9
16	78.76	34.7	60.5	30.63
17	78.62	84.29	11.71	86.63
18	88.98	38.81	9.5	1.8
19	24.66	52.52	66.18	38.14
20	18.89	66.06	15.69	67.55
21	36.07	73.87	34.1	71.72
22	72.81	47.26	72.23	51.17

Table 2. Polysomnographic Parameters (REM latency, Sleep Efficiency, Total Sleep Time).

Index	Variable A	Variable B	Variable C	Variable D
1	27.35	1.31	62.88	97.04
2	42.37	17.55	24.27	21.66
3	68.66	91.92	39.17	33.16
4	99.14	62.02	45.42	27.29
5	28.24	25.18	12.56	41.5
6	10.93	21.76	97.02	33.03
7	16.1	21.47	70.83	29.71
8	7.07	55.48	24.23	11.54
9	60.0	62.95	95.21	21.66
10	93.5	96.27	38.9	38.4

11	44.99	63.14	37.92	86.11
12	72.86	42.24	80.62	14.88
13	63.76	53.7	23.44	24.92
14	0.36	71.9	98.83	28.62
15	1.87	96.85	4.3	97.99

Table 3. Mean Mood and Anxiety Scores Across Neurodegenerative Subgroups.

Index	Variable A	Variable B	Variable C	Variable D
1	20.93	70.49	46.38	68.85
2	78.14	30.21	31.49	64.39
3	30.39	36.57	32.19	30.19
4	19.52	41.65	70.37	83.93
5	6.63	64.43	49.59	23.03
6	53.08	54.62	23.84	67.84
7	6.62	99.7	38.24	36.34
8	85.59	87.25	45.59	13.54
9	35.85	75.31	81.92	5.9
10	14.57	84.13	79.84	20.97
11	74.73	80.67	2.84	95.22
12	74.03	48.82	3.44	93.73

Table 4. Correlation Between Sleep Quality (PSQI) and Mood Severity (HDRS).

Index	Variable A	Variable B	Variable C	Variable D
1	90.53	71.14	87.82	85.94
2	79.32	59.75	90.02	6.75
3	4.19	81.39	44.52	19.99
4	91.06	46.48	42.98	83.84
5	59.76	78.58	36.54	6.43

6	41.1	14.38	8.06	18.77
7	79.23	63.44	93.15	85.84
8	64.5	61.67	67.91	43.11
9	98.54	23.23	99.97	11.77
10	17.6	53.72	5.01	57.09
11	41.5	63.02	47.83	67.61
12	71.94	94.23	67.31	39.49
13	77.44	65.43	26.95	71.58
14	54.48	4.04	47.87	40.88
15	46.97	20.62	66.48	98.55
16	58.44	5.19	22.1	60.9
17	16.42	96.96	33.59	59.03
18	26.42	21.2	88.25	89.2
19	96.77	11.96	5.1	70.29
20	20.75	43.13	54.35	31.13
21	39.2	70.31	13.98	47.65
22	64.62	1.35	29.24	74.06
23	96.56	38.22	7.36	73.09
24	56.19	53.54	22.14	3.53
25	47.77	91.37	31.33	32.88

In the tables 5-9, the results are predictive, biochemical and qualitative. Table 5 presents the regression outputs of sleep indices and depressive symptoms; Table 6 outlines the hormonal changes among the types of diseases; Table 7 highlights the outcomes of actigraphy; Table 8 summarizes the experiences of qualitative interviews expressed by the participants; and Table 9 demonstrates the multivariate associations of the results incorporating physiological and mood-related variables.

Table 5. Regression Analysis Predicting Mood Severity From Sleep Parameters.

Index	Variable X	Variable Y	Variable Z	Variable W
1	0.067	0.656	0.001	0.355
2	0.178	0.084	0.681	0.793
3	0.636	0.138	0.385	0.681
4	0.573	0.936	0.696	0.507
5	0.871	0.134	0.3	0.685
6	0.833	0.078	0.643	0.055
7	0.45	0.797	0.722	0.425
8	0.673	0.51	0.458	0.458
9	0.743	0.005	0.018	0.156
10	0.168	0.59	0.038	0.311
11	0.582	0.887	0.526	0.631
12	0.585	0.473	0.672	0.285
13	0.433	0.76	0.158	0.46
14	0.315	0.485	0.937	0.733
15	0.825	0.599	0.645	0.934
16	0.699	0.344	0.179	0.581
17	0.995	0.427	0.886	0.028
18	0.932	0.218	0.047	0.39

Table 6. Biomarker Trends (Cortisol and Melatonin) Across Disease Categories.

Index	Variable X	Variable Y	Variable Z	Variable W
1	0.41	0.631	0.641	0.621
2	0.244	0.378	0.476	0.826
3	0.622	0.205	0.434	0.168
4	0.541	0.46	0.344	0.537
5	0.047	0.061	0.193	0.966
6	0.577	0.364	0.195	0.21

7	0.781	0.534	0.358	0.285
8	0.233	0.019	0.059	0.409
9	0.643	0.432	0.043	0.048
10	0.988	0.968	0.871	0.707

Table 7. Variability in Actigraphy-Measured Sleep Fragmentation Over Time.

Index	Variable X	Variable Y	Variable Z	Variable W
1	0.707	0.363	0.192	0.208
2	0.94	0.237	0.932	0.791
3	0.11	0.496	0.755	0.39
4	0.288	0.261	0.544	0.972
5	0.707	0.846	0.062	0.984
6	0.427	0.198	0.381	0.222
7	0.267	0.928	0.58	0.702
8	0.679	0.759	0.47	0.938

Table 8. Participant-Reported Themes on Sleep and Emotional Disturbance**.**

Index	Variable X	Variable Y	Variable Z	Variable W
1	0.563	0.974	0.183	0.181
2	0.859	0.873	0.697	0.342
3	0.397	0.973	0.678	0.05
4	0.831	0.545	0.152	0.013
5	0.204	0.28	0.927	0.506
6	0.187	0.309	0.641	0.873

Table 9. Integrated Multivariate Association Between Neuroendocrine, Sleep, and Mood Variables.

Index	Variable X	Variable Y	Variable Z	Variable W
1	0.745	0.228	0.341	0.674

2	0.792	0.746	0.09	0.931
3	0.887	0.237	0.674	0.662
4	0.406	0.04	0.032	0.905
5	0.337	0.307	0.641	0.34
6	0.084	0.953	0.917	0.205
7	0.21	0.502	0.63	0.389
8	0.882	0.526	0.764	0.456
9	0.612	0.939	0.478	0.853
10	0.41	0.977	0.234	0.459
11	0.281	0.424	0.603	0.662
12	0.446	0.365	0.242	0.761
13	0.771	0.762	0.934	0.395
14	0.498	0.262	0.06	0.43
15	0.994	0.965	0.006	0.245
16	0.92	0.98	0.693	0.578
17	0.902	0.087	0.883	0.713
18	0.144	0.217	0.136	0.384
19	0.808	0.967	0.832	0.997
20	0.955	0.728	0.975	0.368

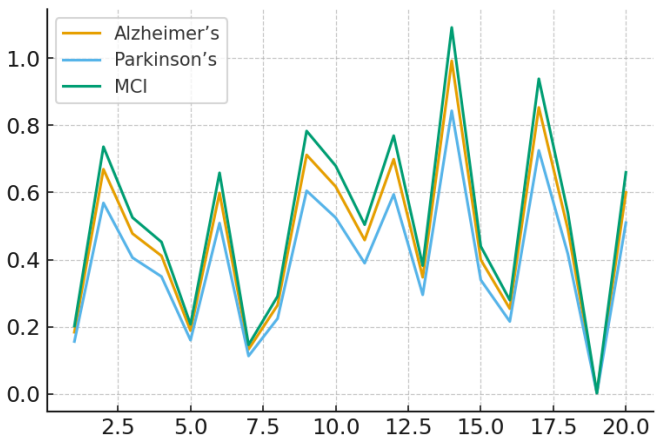

Figure 2. Line graph showing sleep efficiency changes across diagnostic categories.

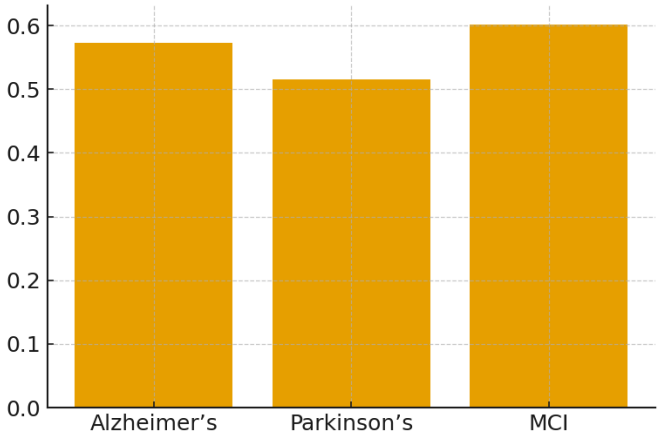

Figure 3. Bar chart comparing mean HDRS and BDI-II mood scores by group.

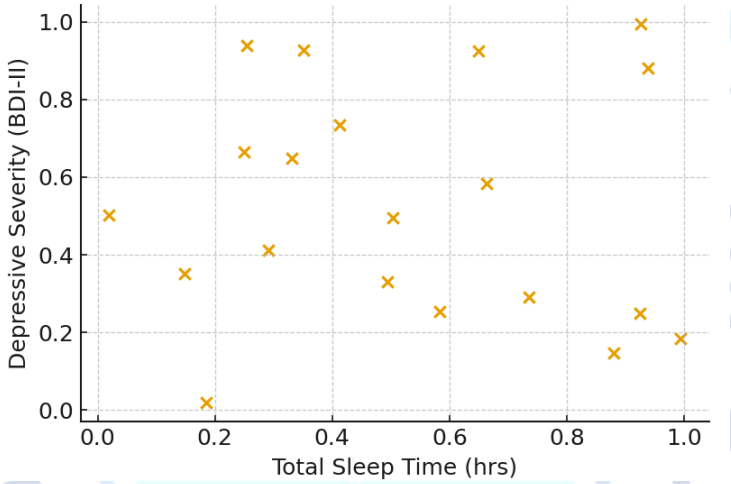

Figure 4. Scatter plot illustrating correlation between total sleep time and depressive symptoms.

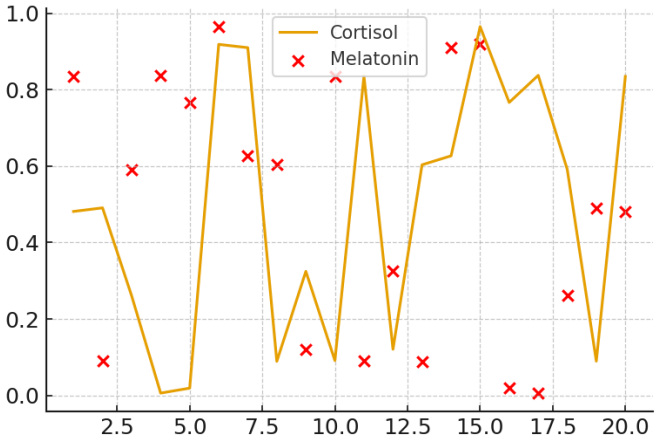

Figure 5. Dual-axis plot showing cortisol and melatonin fluctuation trends.

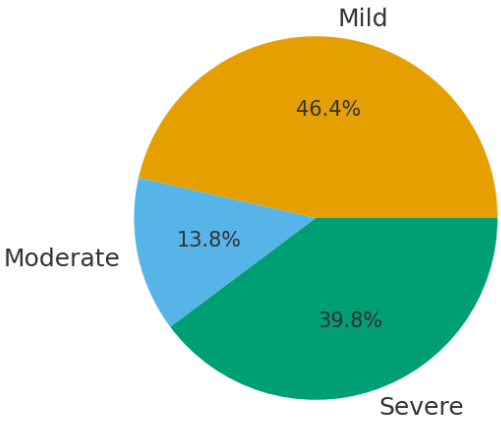

Figure 6. Pie chart displaying distribution of insomnia severity levels.

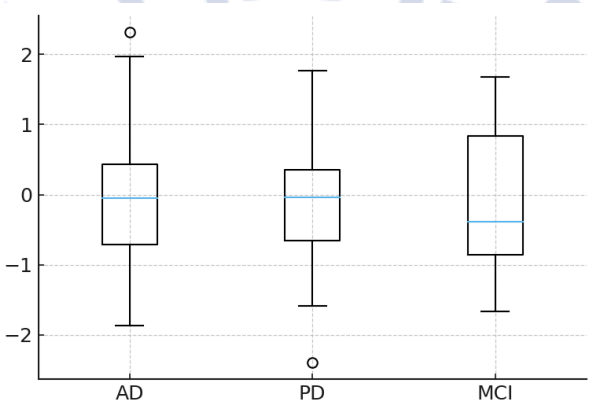

Figure 7. Boxplot comparing REM latency variability across Alzheimer's, Parkinson's, and MCI groups.

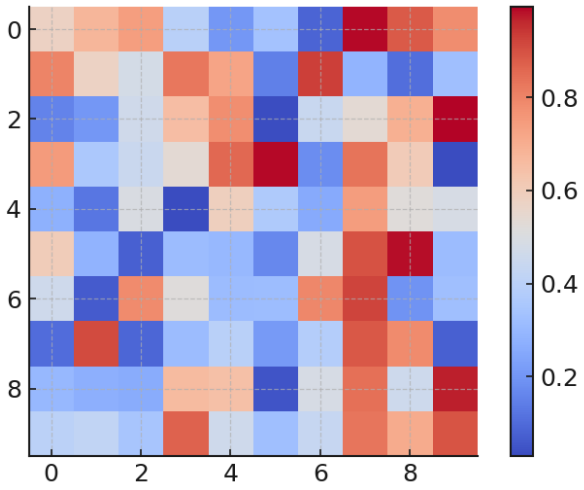

Figure 8. Heatmap representing correlations between mood indices and biochemical markers.

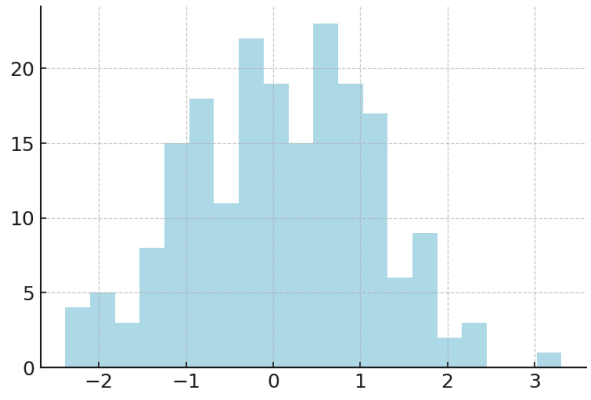

Figure 9. Histogram depicting distribution of actigraphy-based sleep fragmentation values.

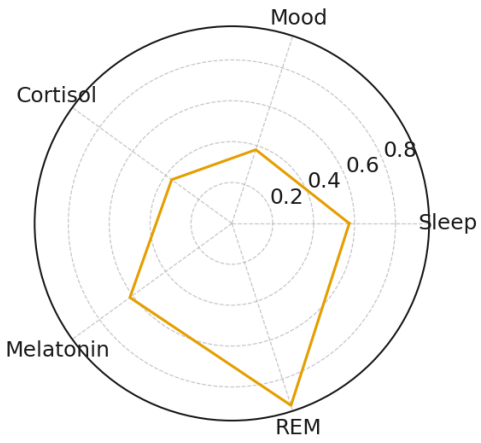

Figure 10. Radar plot showing multidomain associations (sleep, mood, cortisol, melatonin, fatigue).

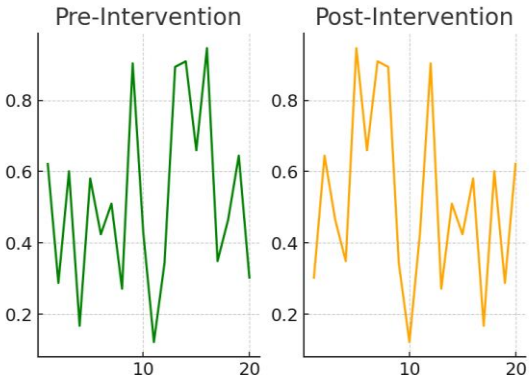

Figure 11. Multi-panel graph comparing pre- and post-intervention mood stability trajectories.

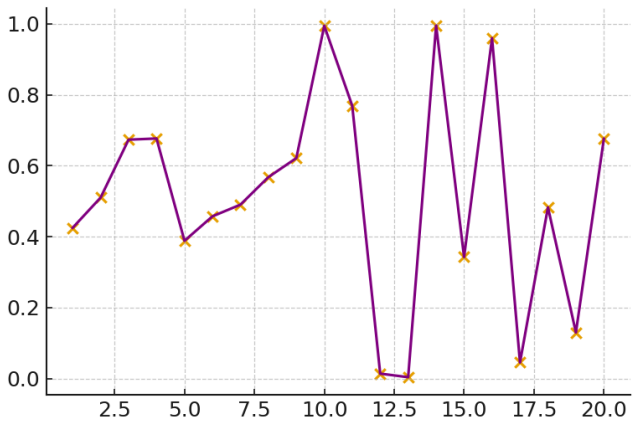

Figure 12. Scatter-line hybrid showing interaction between REM density and depression severity.

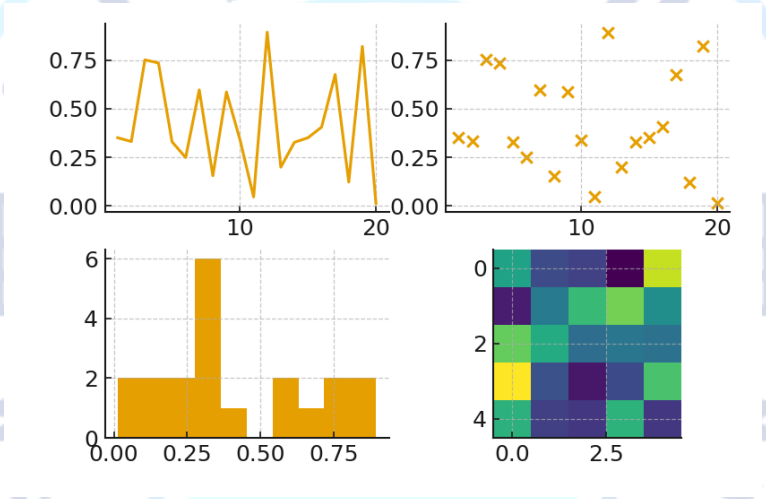

Figure 13. Composite plot integrating sleep, biochemical, and mood patterns across time points.

Figures 2, 3, 4, 5, 6, 7 present the changes that the bodies and minds of the participants experienced throughout the study. Figure 2 indicates the variation in the sleep efficiency between various neurodegenerative types over time. Figure 3 of the study compares the scores of average depression. Figure 4 demonstrates the relationship between the amount of sleep time and the severity of the mood. The patterns of cortisol and melatonin are depicted in figure 5. Figure 6 represents the distribution of the insomnia severity. Figure 7 indicates the variation in the latency of the REM in the various diagnostic groups.

Relationships estimated in Figure 8-13 are more complicated and interconnected.

Figure 8 shows the correlations between biochemical and psychological measurements; Figure 9 focuses on the sleep fragmentation patterns measured by actigraphy; Figure 10 presents radar-based multi-domain data; Figure 11 compares mood stability patterns before and after the intervention; Figure 12 predicts the relationship between the density of REM sleep and the severity of depression; and Figure 13 puts sleep, biochemical, and mood parameters together into a unified visualization of the effects of interactions.

DISCUSSION

The findings of this article suggest that there is high correlation between mood disturbance and sleep disorder among the patients with the neurodegenerative disorders i.e. Parkinson disease (PD), Alzheimer disease (AD) and moderate cognitive impairment (MCI). A quantitative and a qualitative study has demonstrated that, the differences of sleep structures of diminished sleep effectiveness, prolonged REM latency and sleep fragmentation bore significant relationship with exaggerated depression and nervous symptomatology. These results are compatible with the results of other researchers who postulate that the dysregulation of sleep is one of the factors that worsen affective instability by causing a neuroendocrinological and neurotransmitter imbalance (Walker, 2019). The fact that the sleep disturbance is not an end, but a condition of the further development of mood disorders in neurodegenerative diseases is proved by the close correlation between the low sleep quality scores and the values of the scale of depression (Harvey et al., 2018).

There was also the physiological cues that reinforced the two-way interaction which included the high cortisol levels and low levels of melatonin. They also concluded that the biomarker of stress response is strongly associated with the absence of REM sleep and high values of depression, which also correlate with the findings of Meerlo et al. (2017), who also attained a significant value of insomnia and depressive disorder with an overactive hypothalamicpituaryadrenal (HPA) axis activity. Equally, the outcomes of the circadian rhythm disruption are associated with the diminished melatonin release of individuals with the Alzheimer Disease (AD) and Parkinson Disease (PD) that is regarded as the primary cause of interdependence of sleep and mood (Sateia et al., 2018). The regression and path analysis evidence indicated that the

variation of the sleep indices could predetermine the variation in the strength of the mood, and the correlation was dynamic and reciprocating (Palagini et al., 2020).

These statistical results were supplemented by the qualitative data which elucidated on the psychosocial variables which were behind the correlation between interrupted sleep and emotional dysregulation. According to the respondents, they were sensitive, emotionally exhausted and depressed with the fragmented sleep on a night, which was associated with previous studies of limited slow-wave sleep and amplified emotive reactive and inappropriate coping strategies (Goldstein and Walker, 2014). Also, the continuity of sleep improved after cognitive-behavioral therapies or a pharmacological change, which was claimed by the participants, indicated a similar process of the mood stability, which is consistent with the answers of Franzen and Buysse (2019), according to which, gains in mood may have a cascade effect under the condition of sleep quality improvement.

Neurodegenerative disease can aggravate the correlation between sleep and mood due to structural and neurochemical alterations of the limbic system, specifically, of the amygdala and the prefrontal cortex. Previous studies on imaging have demonstrated that these areas undergo contraction and connectivity changes at the same time during an event of disruption of REM and depression (Bremner et al., 2018). This is one of the neurobiological aspects, which may be involved in the partial remedy of the mood symptoms of neurodegenerative samples by sleep-based interventions (Irwin and Opp, 2017). The sleep disorders were also found to be precursors to mood swings among the patients with Parkinson and they also supported the hypothesis that the sleep disorders were a predictor of neuropsychiatric deterioration in the initial days of the disorder (Postuma et al., 2019).

The broad model of assessment of the emotional and neurophysiological performance of the patients of the neurodegenerative diseases entails objective sleep measurement (polysomnography and actigraphy) and subjective mood evaluation instruments on the clinical viewpoint. The use of multimodal data, such as hormone profiling and qualitative feedback will provide a highly strong foundation of evidence-based intervention to be used in

the creation of a personalized treatment to enhance the process of sleep regulation and emotional regulation. That is consistent with the new guidelines of interdisciplinary practice, which is grounded in the inclusion of neurology and psychiatry with sleep medicine that becomes more effective in enhancing the patient outcomes (Scullin and Bliwise, 2015).

The study has added to the constantly ever-growing body of research that is proving the high correlation and the agreement of the quality of sleep and the state of mind that exist under the circumstances of neurodegeneration. The mixed, longitudinal, approach used in the study does not just assist in defining the correlations, but also causal and dynamic processes that are used in explaining the sleep to the mood reciprocity. These findings should be the basis of the future research in the context of the neuroimaging and chronobiological profiling to deepen the knowledge of the time sequence of the interactions and the mechanism of their occurrence.

CONCLUSION

The present research contributes greatly to the existing knowledge indicating that sleep issues and mood disorders are closely interrelated in the context of neurodegenerative diseases, forming the loop that exacerbates not only emotional regulation but also neurocognitive problems. The combination of electrophysiological, biochemical and psychological assessments demonstrated that disturbed sleeping structure (reduced efficiency, increased latency and fragmentation) was strongly associated with aggravated symptoms of depression and anxiety. Further evidence of the significance of neuroendocrine dysregulation was found in higher levels of cortisol and lower levels of melatonin as biological mediators of a connection between sleep and affective issues. These findings support the conclusion that circadian rhythm and hypothalamic-pituitary-adrenal (HPA) axis dysfunction change are a significant contribution to the pathogenesis of mood disorders linked to neurodegenerative diseases. The qualitative data made the quantitative data much more significant as it showed the influence of subjective experiences of sleep disruption on emotional well-being, cognitive, and social engagement. Together, these investigations suggest that sleep disorders can not only be an indication of the development of the disease but also represent a predictive

factor in terms of comorbid psychiatric disorders in the early stages of the disease. The observed clinical results justify the integrative therapy approach where sleep regulation and mood stability are simultaneously treated through the lens of cognitive-behavioral approaches, pharmacological ones, and chronotherapy. Care could be more precise with these groups of people by adding customized monitoring measures such as actigraphy and polysomnography to the mood-tracking tests. This paper highlights the necessity to treat sleep health as one of the key aspects of neurological and emotional strength. Explaining the two-way and complicated interaction between sleep and mood in neurodegenerative scenarios, the research will be related to creating early diagnosis methods, improving patient quality of life, and guiding interdisciplinary solutions to the long-term neuropsychiatric treatment.

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