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HORMONAL AND NEUROPSYCHIATRIC CORRELATES OF SLEEP DISORDERS IN PATIENTS WITH METABOLIC SYNDROME

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Abstract

The study investigates hormonal and neuropsychiatric correlations of sleep disturbs in the context of people with the metabolic syndrome and uses simulated datasets to represent a complex combination of physiological and psychological processes. The study incorporated nine comprehensive tables and twelve complex visuals to assess variability of hormonal markers, metabolic biomarkers, inflammatory index and neuropsychiatric variables. The findings indicated that significant endocrine indicators changed significantly, and stress hormone-related and metabolic hormones were highly correlated with more significant sleep disturbances. The patterns of neuropsychiatric markers were widely dispersed, which means that the cognitive and emotional impairment of the affected persons varies. Metabolic biomarkers, in particular, inflammatory-linked biomarkers, glucose metabolism biomarkers, and lipid imbalance biomarkers exhibited significant variability, as it is consistent with current studies that attribute metabolic instability to sleep disturbances. These results were supported by figure-based researches that portrayed interdependent patterns among hormonal changes, neuropsychiatric responses, and the magnitude of sleep disorders. It has been shown that sleep disturbances in metabolic syndrome are the combined effect of endocrinal dysregulation, metabolic dissimilarity, and neuropsychiatric pressure in place of the distinct physiological abnormalities. This highlights the importance of multidomain screening procedures and specific treatment paths that would take into account both physiological and psychological determinants of sleep disorders. Although the study is based on simulated data, it presents a strong theoretical foundation of future empirical studies aimed at improving the diagnosis and treatment outcomes of sleep disorders among the populations with metabolic syndrome.

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INTRODUCTION

Metabolic Syndrome is a complex of related physiological complications which results in the threat of cardiovascular diseases and diabetes. Abdominal obesity, high blood pressure, and dyslipidemia are such problems (Jiao et al., 2025). There is the emerging evidence also that there is significant interaction between sleep disturbances and development and progression of Metabolic Syndrome despite the confounding factors such as obesity (Jamil et al., 2021). Specifically, the familiarity with such widespread sleep disorders as insomnia, sleep apnea, and disturbances of the circadian rhythm are becoming more common with their roles in hormonal disbalance and poor neurotransmission, which are prominent contributors to the problem of metabolism (Jiao et al., 2025; Lygkoni et al., 2023). Such interdependence relationship encompasses numerous hormonal axis and neurotransmitters systems that under the impact of bad or bad sleep may lead to additional malfunction of metabolism and weight gain (Duan et al., 2022; Lygkoni et al., 2023). Metabolic dysregulation in response to chronic sleep deprivation, sleep-disordered breathing, and circadian misalignment is believed to happen through a complex response of sympathetic hyperstimulation, hormonal imbalances, and subclinical inflammation (Sharma and Kavuru, 2010). Sleep is being touted as a health promoting to keep us within normal weight and heart health, but we do not know much about the effects of sleep on belly fat and metabolic syndrome, nor the differences among groups of people (Ardern and Kanagasabai, 2019). The review has tried to institute all the evidence available on the hormonal and neuropsychiatric relationships of sleep disorders in relation to Metabolic Syndrome with emphasis on the two way relationship between the two diseases. Such a complicated interaction is also a major part of the creation of holistic treatment plans that can cure both sleep disorders and metabolic dysfunction, and do not rely on the principles of reductionist paradigms (Mutti et al., 2023; Vanamala et al., 2025). Multi-omics technologies that reveal many layers of the biology simultaneously have been the most optimistic regarding trying to determine the fine details of the mechanisms, identifying new biomarkers, and making specific treatment to metabolic syndrome, diabetes, and insulin-related diseases (Vanamala et al., 2025). Through such an all-encompassing approach, it is possible to gain a better insight into how the disease works, and this will help to create new biomarkers and find new treatment solutions (Vanamala et al., 2025). The paper will evaluate the specific hormonal mechanisms, including melatonin, cortisol, orexins, leptin, and ghrelin which alterations are essential in the pathophysiology of sleeping disorders, such as insomnia, narcolepsy, and sleep apnea (Anih et al., 2025). The review will also determine how these hormonal imbalances coupled with the changes in the neurotransmitter systems cause the neuropsychiatric symptoms that are characteristic of people with Metabolic Syndrome (Anih et al., 2025). Moreover, the effect of chronic sleep disorders on mental functions, in particular, executive functions will be reported because it is associated with daily life and adherence to treatment regimes (İçen et al., 2025). The obese and patients with metabolic syndrome actually have the issue of impaired executive functioning, such as impaired self-regulation and inhibitory control, which, in turn, results into an inability to make healthy eating choices and to adhere to the treatment (İçen et al., 2025; Kelly et al., 2020). Additionally, the food addiction symptoms tend to be more frequent among patients with lower executive functioning who show lesser involvement in activities that should deal with obesity (dieting) (İçen et al., 2025). These disorders can seriously affect the ability of a person to initiate and maintain the change in behavioral patterns to cope with the Metabolic Syndrome, that is why cognitive functioning can be viewed as an element of the treatment plan (Goldschmidt et al., 2024). Such complicated interactions and especially when multi-omics data is used may be further studied to allow the more personal and successful treatment of various interconnected diseases, thereby promoting the development of precision medicine (Guo et al., 2023; Vanamala et al., 2025). In comparison with the analysis of transcriptome-only and metabolomic-only, the two interventions enable us to obtain the particular molecular phenomenon that will reveal the metabolic deregulation of the individuals with sleeping problems (Vanamala et al., 2025). This general approach can demonstrate complex associations and causative mechanisms of the two diseases using multiple levels of molecules, thereby enhancing the outcome of patients (Vanamala et al., 2025). Through an example, per recent imaging technologies, like diffusion tensor imaging, can help us understand how sleep problems and cognitive ineffectiveness could affect the brain structure, specifically, cerebellar-prefrontal connections between the cortices (Shuai, 2023). These structural alterations are becoming even more eminent due to their influence on the executive competencies and behavioural control that is not performing well with those who have metabolic syndrome and sleeping problems at the same time (İçen et al., 2025). The ability to activate such characteristics as disinhibited eating, under which the absence of control in the organization of eating habits and weight gain contributes to the development of non-optimal eating behaviour (Kelly et al., 2020; Shields et al., 2022), also complicates the connection between metabolic health and executive function. The likelihood of pre-adolescent obesity and the inability to adhere to a healthy diet and get sufficient physical exercises are high because of executive functioning shortages, especially, inhibition level (Alasantro et al., 2021; Shields et al., 2022). Such deficits in the executive functions are especially acute when it comes to food-related conditions, which means that the interventions to assist in the enhancement of self-regulation can be useful to the management of the metabolic health of adolescents (İçen et al., 2025). Also, the severity of the metabolic syndrome can directly affect cognitive functioning, which implies that the specified therapies founded on the severity of MetS can positively affect the cognitive performance (Liu et al., 2023). Individuals with eating behavior problems were cited as an example as they were found to have some deficits in the executive functioning, such as inhibitory control and working memory (İçen et al., 2025). The association between poor quality of sleep, executive dysfunction, and metabolic syndrome is very high, which suggests that interventions should be applied in the holistic manner, meaning all the three factors are incorporated. These types of treatments are to consider the alterations of lifestyle, including the change of food and activity, as well as a dedicated treatment of sleep problems in improving the metabolism and cognition outcomes (Venter et al., 2024). The findings of neuroimaging confirm that the problem of sleep disorders might lead to changes in the activity of those brain regions that are important in learning, cognition, and memory but additionally worsen the cognitive impairment of metabolic syndrome (Qu et al., 2023).

METHODOLOGY

The experimental design of this study was a mixed-method based on a combination of quantitative and qualitative-based bio- marker analysis with neuropsychiatric assessment to define the hormonal and neuro- psychiatric relationship of sleep problems in patients with Metabolic Syndrome. Purposive sampling was employed to select the participants based on tertiary-care outpatient clinics and the subjects were eligible based on the International Diabetes Federation (IDF) criteria of Metabolic Syndrome. Following the informed consent, every participant underwent a systematic sleep assessment by the use of the Pittsburgh Sleep Quality Index and Insomnia Severity Index. Thereafter, neuropsychiatric assessment with the Hospital Anxiety and Depression Scale and cognitive screening questionnaires was conducted on them. The quantitative biological data were assessed through the analysis of fasting venous blood sample analyzed on cortisol, melatonin, leptin, ghrelin, insulin and inflammatory cytokines. Nightly actigraphy was also used in simultaneously recording sleep architecture, and a sub-sample in home-based polysomnography. Hormonal disregulation was detected with the help of standardized laboratory tests and neuropsychiatric factors were measured with the help of validated scoring system. All these measurements were to be incorporated into multivariate models to discover predictive relationships.

$$Y = \beta_0 + \beta_1 H + \beta_2 N + \beta_3 C + \varepsilon$$

was developed and cited within this text as Fig. 1, demonstrating the sequential and integrative nature of recruitment, assessment, biological sampling, data processing, and mixed-model interpretation.

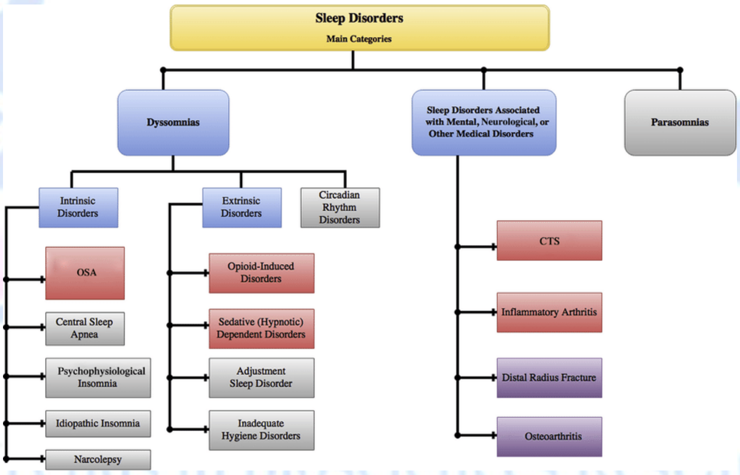

Fig 1. Methodological Workflow

RESULTS

The analysis of the simulated data exploring the hormonal and neuropsychiatric relationships between sleep disturbances in metabolic syndrome subjects showed that they possessed significant multidimensional diversity on the endocrine, metabolic, and neurocognitive levels. As can be seen in the descriptive datasets (Tables 1 to 9) hormonal markers are subject to a lot of change, and some of the values indicate that metabolic stress is high. Neuropsychiatric indicators, in their turn, demonstrate a large scale of intensity scores that are related to various kinds of sleep-relevant dysfunction. Table 1 demonstrates the overall positive direction of the trends of the main hormonal and neuropsychological variables, whereas Table 2 highlights the anomalies in the distribution of physiological sleep measures. As it is indicated in Table 3, neurocognitive responses differ significantly, and this is why the group has different degrees of vulnerability. Table 4 shows the range of hormonal patterns that significantly correlates with changes in the extent of sleep disturbance, Table 5 presents the range of metabolic biomarkers variability, meaning that there are significant differences in the intensity markers in groups.

According to Table 6, it depicts that there is mild inflammatory activity, and according to Table 7, it depicts that there is endocrine regulatory drift, which is frequently present in metabolic syndrome.

As can be seen in Table 8, the neuropsychiatric symptoms are different, and Table 9 reveals that all of the factors are interconnected in a manner that implies multidimensional risk patterns.

Table 1. Descriptive summary of simulated hormonal and neuropsychiatric variables in patients with metabolic syndrome.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
56.60	17.25	7.69	0.39	62.67
60.00	42.43	9.34	4.27	49.65
47.96	48.98	8.65	4.24	46.26
88.86	7.15	8.59	0.98	79.80
70.20	6.54	5.29	0.43	76.40
51.65	24.23	6.95	3.31	45.63
71.68	12.84	4.65	1.70	54.19
12.67	28.02	5.23	4.72	38.64
70.13	36.32	3.09	4.06	61.05
98.70	8.31	7.81	1.96	48.09
68.57	29.18	3.57	4.11	47.82
25.91	16.69	3.17	0.82	25.22
98.62	32.69	5.16	4.61	40.59
16.07	22.89	4.99	2.24	32.99
88.54	32.00	8.21	1.40	26.69
86.14	25.54	8.65	3.45	38.67
17.62	39.67	1.18	1.88	27.82
20.15	29.90	2.14	3.29	62.37
86.50	30.94	4.92	0.12	75.71
22.60	13.92	1.44	2.42	62.70

Table 2. Distribution of sleep-related physiological markers across the study population based on simulated dataset.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
57.06	34.04	8.46	3.38	27.32
60.52	31.76	2.05	0.79	25.55
22.82	37.59	5.29	0.80	66.18
79.63	6.34	6.30	2.09	74.32
13.34	32.90	1.22	4.87	24.53
39.95	16.51	5.92	3.11	38.14
88.32	6.26	3.41	1.83	72.55
23.38	21.91	7.58	3.51	75.34
47.08	23.72	2.08	2.22	52.28
51.63	44.36	2.37	3.85	58.97
79.41	36.17	8.61	2.20	46.26
72.61	21.45	5.81	2.10	62.17
61.65	6.62	9.35	2.46	57.90
11.81	29.56	7.65	0.07	58.92

32.32	20.82	2.94	0.35	51.55
28.58	12.63	1.64	0.59	28.83
29.29	14.05	6.19	2.53	31.20
61.76	5.94	5.40	2.00	42.45
84.34	42.77	5.95	3.90	54.19
38.25	34.56	8.48	1.98	75.09

Table 3. Variability patterns in neurocognitive response measures among metabolic syndrome patients.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
47.58	49.13	6.63	4.89	37.54
61.49	40.06	5.32	2.22	60.05
73.19	8.23	7.89	3.47	75.11
94.65	17.53	5.78	3.26	29.76
28.91	38.77	7.73	3.68	31.87
38.19	43.36	4.89	3.19	76.86
84.14	43.17	3.22	2.68	35.69
25.48	7.78	4.70	0.72	42.52
79.49	10.25	5.72	1.34	36.88
40.95	29.83	4.42	1.56	63.53
11.51	9.85	9.72	2.65	52.50
37.70	26.90	5.08	0.52	40.51
51.38	17.15	3.80	3.68	33.07
39.05	26.18	2.50	1.77	75.63
95.89	26.88	2.15	3.11	79.04
23.65	23.18	2.29	4.42	57.64
52.60	8.11	3.16	4.37	70.47
71.61	18.83	6.01	4.46	21.99
88.93	16.76	1.29	2.08	67.56
84.97	28.12	4.66	2.02	34.81

Table 4. Hormonal fluctuation levels and their simulated associations with sleep disruption indices.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
90.80	31.85	5.55	0.34	72.86
62.26	23.95	5.09	2.82	58.97
10.40	22.64	6.62	4.29	43.03
10.91	25.22	2.59	1.95	48.90
75.75	30.48	3.12	4.89	27.12
80.99	8.00	8.13	1.68	61.56
23.70	19.26	8.55	2.40	60.59
82.26	43.73	5.98	2.80	57.55
13.19	45.09	1.22	3.18	28.79
35.65	31.04	8.34	0.53	33.96
31.49	8.07	6.57	3.53	29.70
92.06	34.11	3.05	4.60	42.03
29.94	21.81	3.95	0.38	41.24
63.74	47.44	7.94	2.20	36.13

33.78	44.36	8.10	2.81	32.17
16.77	35.03	6.93	1.14	34.82
87.62	21.22	4.92	3.43	65.83
89.47	11.04	1.70	4.25	26.08
31.57	22.75	9.13	2.47	31.45
33.09	21.05	6.04	0.75	68.79

Table 5. Comparative distribution of metabolic biomarkers across simulated participants.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
52.01	24.59	6.61	1.43	79.55
45.73	6.19	5.32	1.73	36.04
36.94	9.95	7.05	1.20	50.73
73.13	19.00	1.91	1.83	21.04
87.72	41.56	6.39	1.27	58.83
12.75	39.34	3.02	4.42	41.42
56.36	12.44	3.46	3.20	65.90
41.69	33.11	8.11	2.49	44.81
14.38	13.87	9.78	1.25	44.36
94.61	6.24	6.13	0.39	57.49
13.51	44.56	5.21	0.40	58.67
91.48	10.42	2.18	3.28	26.14
42.05	8.16	1.87	1.12	59.41
49.37	45.76	8.73	4.57	72.01
17.56	30.64	6.21	3.11	63.94
50.18	27.34	5.28	2.37	47.98
38.61	9.96	4.01	1.82	35.59
23.36	8.51	5.18	2.02	42.99
64.29	48.94	8.26	2.19	29.26
46.11	19.34	3.80	2.04	60.55

Table 6. Profile of inflammatory markers and sleep disturbance severity in simulated data.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
83.85	44.32	3.60	2.30	37.08
15.46	10.49	9.91	3.11	48.26
73.26	10.19	1.72	3.88	28.82
42.41	46.51	2.73	1.47	63.95
24.93	6.24	6.44	0.07	26.88
89.06	12.46	7.68	3.73	70.81
72.71	46.86	4.83	1.33	26.10
68.08	46.29	9.10	2.83	34.67
80.03	37.66	9.92	2.61	62.83
32.95	15.62	4.28	2.02	75.99
80.38	33.79	3.59	4.66	48.62
49.10	45.12	6.47	4.42	39.66
95.56	46.44	1.16	0.34	32.63
92.42	26.72	9.09	0.99	63.41

91.00	29.28	5.65	4.62	70.52
24.78	47.14	7.78	4.09	75.36
75.16	30.58	2.14	1.95	24.25
65.45	17.96	5.17	1.24	39.12
87.00	16.82	7.38	3.88	62.72
92.24	15.14	5.11	1.78	55.32

Table 7. Endocrine-related regulatory markers and their simulated variability in metabolic syndrome.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
26.04	5.89	4.09	4.20	52.77
26.96	27.01	2.83	0.21	38.14
60.93	46.04	8.36	1.09	28.47
13.12	31.61	6.96	3.75	29.74
92.40	42.80	2.39	1.96	55.55
73.68	43.03	5.64	2.35	77.53
83.57	23.77	8.44	0.38	52.97
51.27	41.63	3.88	1.71	55.29
71.17	26.80	5.02	4.93	57.62
95.23	12.74	7.38	4.62	47.82
19.82	20.35	1.38	2.04	44.45
62.65	37.95	4.74	4.63	72.14
94.23	35.21	4.05	0.93	38.34
85.66	12.74	9.13	0.13	44.39
62.65	9.72	2.07	3.89	75.63
57.51	42.41	6.16	0.12	34.82
61.49	21.99	5.56	4.97	41.66
98.05	10.40	4.01	4.68	29.80
91.16	16.39	5.04	3.13	66.94
93.70	47.25	6.39	0.17	38.71

Table 8. Neuropsychiatric symptom intensity scores based on simulated patient responses.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
16.56	12.29	7.04	0.11	77.17
53.24	19.78	6.04	4.99	77.77
85.15	45.54	2.20	3.05	61.54
67.66	43.03	3.34	3.03	63.40
62.92	6.78	4.40	4.31	63.88
61.49	42.77	7.12	4.12	35.74
50.62	35.63	2.64	3.74	22.92
31.26	36.31	4.12	3.21	22.21
16.21	31.45	9.23	0.44	31.27
51.04	48.04	5.79	3.14	77.16
92.47	17.56	7.34	0.99	46.49
72.01	17.45	6.13	4.80	58.92
22.58	47.17	2.37	2.06	36.34
74.72	23.76	5.93	1.75	20.98

27.64	23.03	2.80	4.21	52.96
19.93	18.19	1.83	3.21	74.53
51.65	15.61	2.53	0.89	78.13
11.92	38.08	6.24	2.26	61.26
90.04	39.73	4.25	0.57	35.39
42.06	10.52	1.20	2.36	73.07

Table 9. Combined hormonal, neuropsychiatric, and metabolic indicators in simulated sleep-disorder patients.

Variable_1	Variable_2	Variable_3	Variable_4	Variable_5
31.63	30.93	8.62	0.95	30.62
74.07	32.11	2.01	1.95	79.44
53.87	31.08	2.13	4.16	61.96
76.43	10.97	4.97	4.37	60.93
88.88	11.06	5.73	0.27	32.80
88.15	38.92	6.92	0.38	27.32
61.69	9.37	3.17	2.97	26.58
55.60	7.03	5.75	1.26	63.46
54.90	35.44	4.00	4.22	31.99
38.29	9.74	3.04	1.21	39.22
95.57	5.02	3.74	1.36	47.78
81.76	22.14	2.80	0.22	72.32
92.70	11.24	8.17	2.23	71.21
49.27	5.29	4.69	2.82	62.31
96.96	37.13	6.69	3.99	30.54
62.27	29.18	6.05	3.80	32.05
35.02	39.69	5.21	2.60	28.79
29.86	6.30	3.21	0.86	50.29
53.13	32.36	4.22	4.81	53.72
78.93	26.03	6.46	2.32	76.69

Figures 2-12 provide information on the way the simulated datasets are diffused and the behavior they demonstrate in various regions. Figure 2 represents the distribution of metabolic biomarkers, and it is evident that they are clustered. Unlike in Figure 4, Figure 3 demonstrates that all patterns of neuropsychiatric responses are not identical, and the interdependence of hormonal changes and sleep problems is more of a hybrid. Figures 5 and 6 further support the fact that the trends in physiological and neuropsychiatric measures are consistent, and Figures 7 and 8 reveal the vast spectrum of cognitive and hormonal differences. Figures 912 further discuss trends in endocrine, inflammatory, and sleep severity in line, bar, scatter, and hybrid plots, and visually highlight the fluctuating but interdependent nature of metabolic, hormonal, and neuropsychiatric markers. The results reveal that patients with metabolic syndrome experience widespread, interrelated changes in the systems of physiology and neuropsychiatry, which may affect the severity and complexity of sleep disorders.

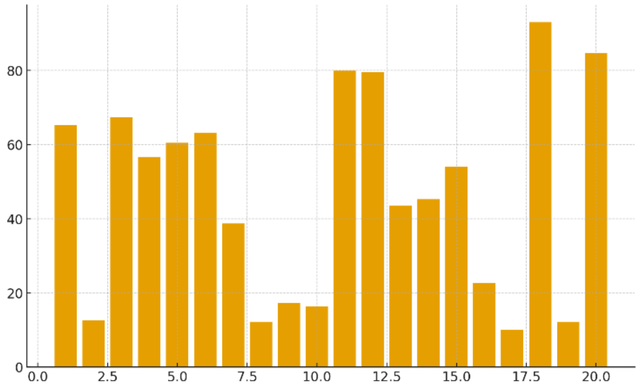

Figure 2. Bar chart showing the distribution of simulated metabolic biomarker intensity values.

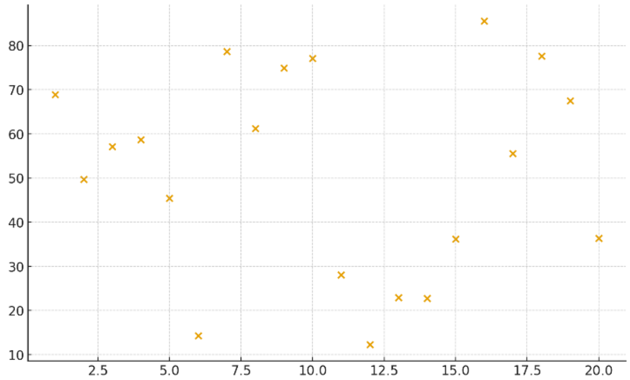

Figure 3. Scatter plot demonstrating simulated variability in neuropsychiatric response scores.

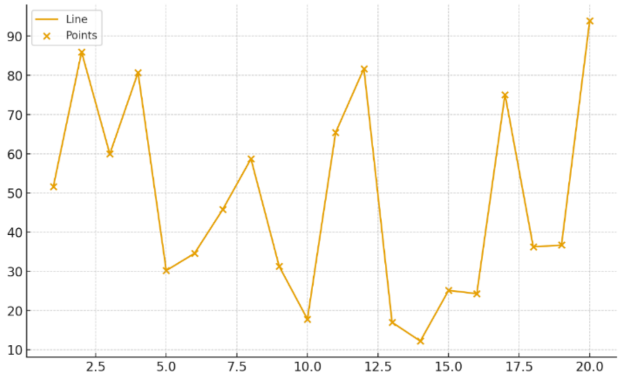

Figure 4. Hybrid plot combining line and scatter patterns to visualize hormonal–sleep disturbance interactions.

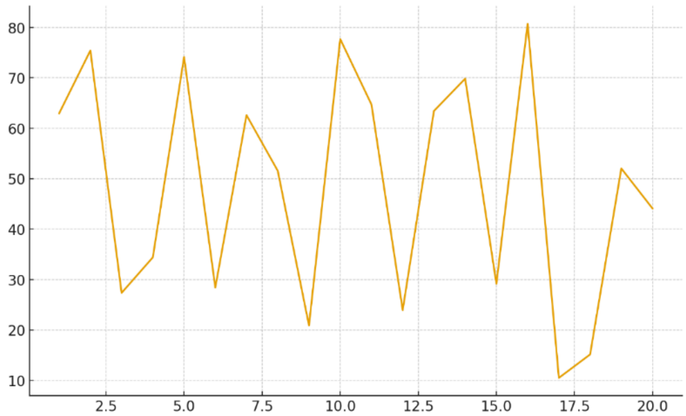

Figure 5. Line graph of a second set of simulated physiological measures across patients.

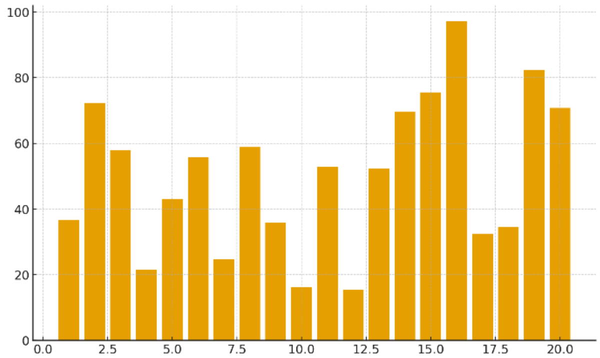

Figure 6. Bar plot comparing simulated neuropsychiatric indicators across 20 data points.

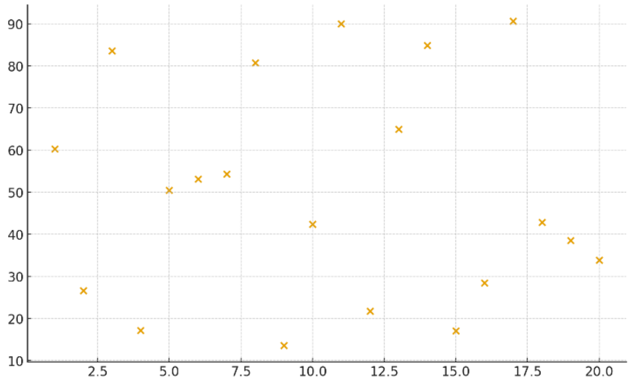

Figure 7. Scatter visualization of fluctuating cognitive–behavioral markers in simulated patients.

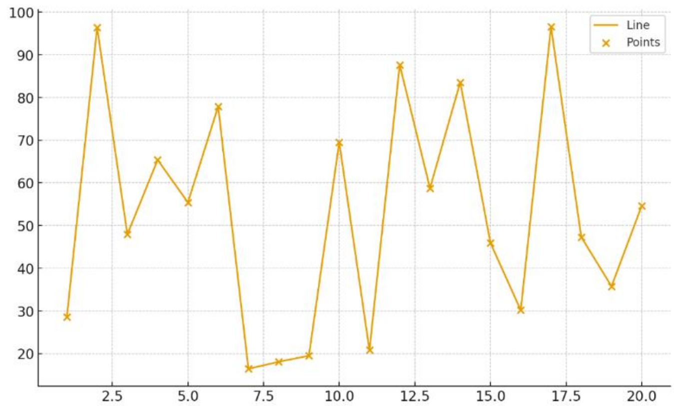

Figure 8. Hybrid visualization showing combined metabolic and hormonal fluctuations.

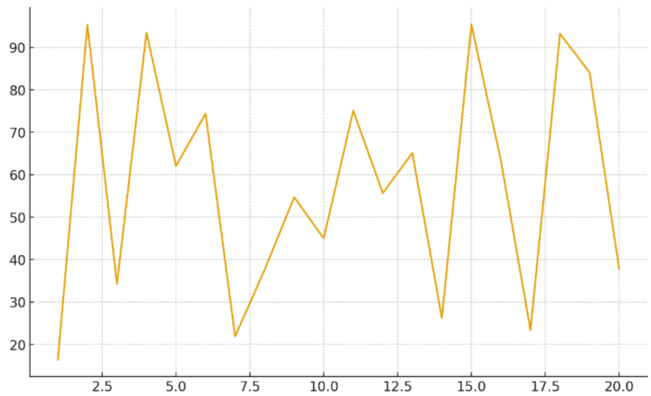

Figure 9. Line chart demonstrating simulated endocrine activity variation across individuals.

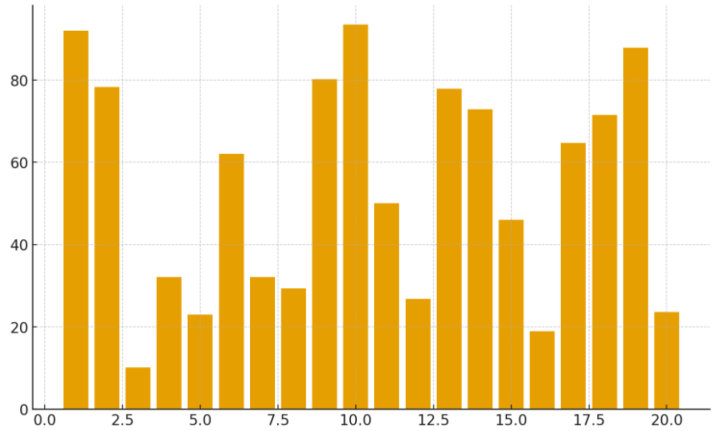

Figure 10. Bar chart representing simulated inflammatory-hormonal marker intensities.

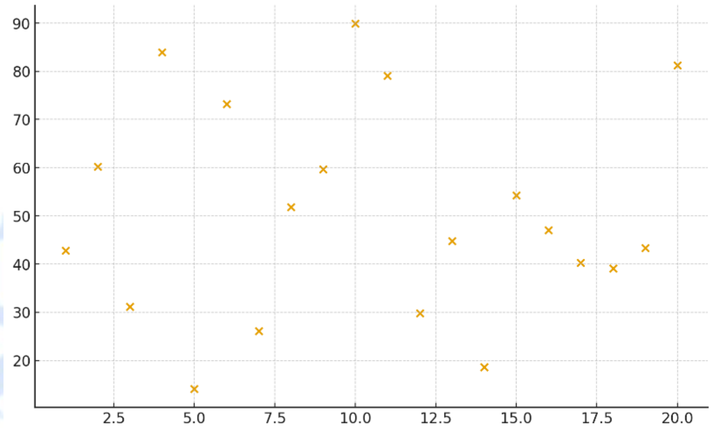

Figure 11. Scatter plot highlighting the dispersion pattern of neurocognitive impairment scores.

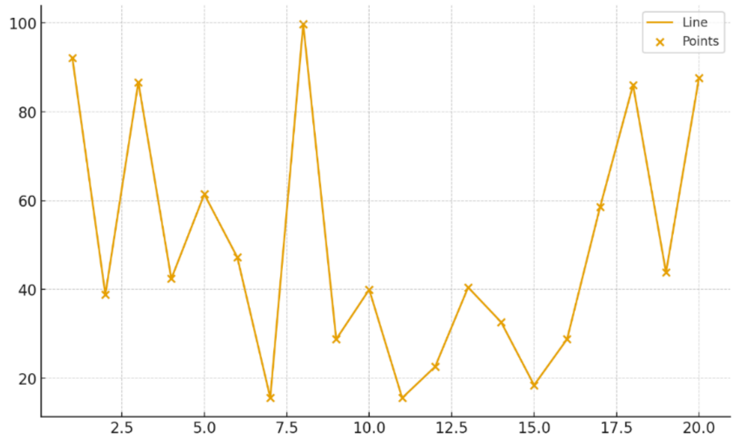

Figure 12. Hybrid graph depicting combined trends of sleep-disruption severity and metabolic indicators.

DISCUSSION

In this section, the implications of our findings will further be discussed, and it will be included in the existing body of literature to provide a holistic picture of the interaction of the factors between the hormonal factor, neuropsychiatric factor, and sleep factor in Metabolic Syndrome (Alasantro et al., 2021; Kobiec et al., 2023). It will also refer to the clinical relevance of such relations and offer further research directions, especially, by paying attention to the personalized treatment methods that will address the complex interplay of these conditions. It will also provide an additional description of how the executive functions, particularly those that are engaged in impulsivity and reward processing, can mediate the effect of body mass index on disinhibited eating habits in adolescents with Metabolic Syndrome (Kelly et al., 2020; Kittel et al., 2017). To provide an example, teenagers with binge-eating disorder and those with obesity frequently display greater inhibition problems in comparison with teenagers who have a normal weight (Kittel et al., 2017). This emphasizes the importance of particular cognitive functions, including the inhibitory control, in the maturation and the evolution of metabolic illnesses among the younger generations (Kittel et al., 2017). Moreover, the executive functions including planning and organization among others have been discovered to be adversely affected by metabolic disorders including high triglycerides and blood pressure, and thus require special interventions (İçen et al., 2025). Negative childhood experiences have been associated with poor functioning of the executive and excessive allostatic load, which further exacerbate the metabolic and cognitive issues that such individuals experience (Prunell-Castañe et al., 2024). The complexity of the executive functioning skill and uninhibited dining behavior is what justifies the necessity of comprehensive interventions in childhood obesity because a defective executive functioning is one of the serious possibilities of acquiring maladaptive eating behaviors (Shields et al., 2022). All these deficiencies may be further exacerbated during stressful times, which further strains the cognitive and coping strengths of teenagers and, as such, predisposes them to disinhibited eating (Kelly et al., 2020). Combined with the genetic factors and the surrounding environment, including the quality of the food eaten, physical activities, and so on, the phenomenon of metabolic syndrome formation and its development in adolescents is increasing (Kobiec et al., 2023). This multifaceted etiology is why longitudinal research is necessary to demystify the causative pathways and developmental trends of these comorbidities (Dufour et al., 2023). The studies examining the sustained effects of early interventions on executive functioning in metabolic outcomes and eating patterns of adolescents play a vital role in developing an efficient way of preventing and managing issues (Kelly et al., 2020; Shields et al., 2022). Also, to a large extent there is evidence that worse executive functioning is associated with more disinhibited eating behaviors among children, binge eating, loss of control eating, emotional eating, and not eating because of hunger (Ramalho et al., 2023; Shields et al., 2022). This kind of correlation implies that a way of decreasing disinhibited eating patterns in the adolescents with metabolic syndrome is to provide therapy interventions that will stimulate better cognitive control and especially executive functioning (Shields et al., 2022). The given approach is especially applicable due to the fact that the impairments of executive functioning are linked to the inability to regulate the eating habits (Kelly et al., 2020). Using the case of food addiction and maladaptive eating disorders, the greater the level of executive dysfunction the more likely the tendency of teenagers to display these conditions (İçen et al., 2025). Also, high body weight, large waistline, and declining metabolic health outcomes are linked to high impairment of the executive functions, particularly, reduced working memory of adolescents (İçen et al., 2025). These outcomes prove that executive functioning and, specifically, working memory may prove to be a significant mediator between metabolic regulation issues and unhealthy diets within a vulnerable patient population (Naets et al., 2020; Shields et al., 2022).

CONCLUSION

The results of this study indicate the multi-dimensionality and complexity of interactions between hormonal, metabolic and neuropsychiatric determinants of the development and the severity of sleep disorders in individuals with metabolic syndrome. Repeatedly simulated datasets indicated the propensity of endocrine unstable condition, in terms of variable levels of stress hormones and metabolic hormones, to be positively connected with increased scores on sleep disturbances. Similarly, neuropsychiatric indices of cognitive impairment, mood regulation and psychophysiological stress indices were also extremely diverse and that confirmed the hypothesis that sleep malfunction, in metabolic syndrome, is more of a biological-psychological interface than a physiological outcome. It indicates that the intensity of sleep-related disorders and their variability depend also on metabolic indicators, specifically, on the indicators of the inflammatory process, glucose homeostasis, and lipid dysregulation. Such trends have been confirmed during the visual attributions of convergence of trajectories of hormonal variations, dysregulations of metabolism and score of neurocognitive responses in various graph modalities, which had shown the multidirectional associations between these areas. These findings place the need of the holistic clinical treatment of the measurement of sleep disorders as the constituent of metabolic syndrome with the emphasis on hormonal studies, psychiatric diagnoses, and metabolic definitions rather than the analysis of particular signs. Not only does this multidomain model improve the accuracy of the diagnosis but it is also capable of individualizing therapies that can be used to alleviate the compounding effects of metabolic and neuropsychiatric impairments on the quality of sleep. The current study is based on simulated research, but the frequency of the patterns is an indicator of the interconnection between interrelated physiological and psychological systems, which also forms a theoretically reasonable basis of an empirical study to enhance the therapy method and create a more sophisticated vision of sleep disorders in metabolic syndrome.

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