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IMPACT OF SLEEP DISORDERS ON BIOCHEMICAL PARAMETERS: A CROSS-SECTIONAL STUDY AMONG THE MEDICAL STUDENTS

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Abstract

Background: Sleep plays a crucial role in regulating metabolic processes. Inadequate or irregular sleep has been linked to disruption of metabolic homeostasis. This study investigates the association between sleep disorders and various biochemical parameters among healthy young adults.

Methods: A cross-sectional observational study was conducted on 100 university students aged 18–30 years. Participants were categorized into normal sleep and disordered sleep groups based on self-reported sleep quality and duration over the past week. Biochemical markers analyzed were fasting blood glucose (FBG), insulin, lipid profile, cortisol, and inflammatory markers.

Results: Participants with disordered sleep showed significantly higher levels of FBG (102.6 ± 10.1 mg/dL vs. 90.2 ± 8.4 mg/dL, $p < 0.001$), insulin (11.1 ± 3.0 µU/mL vs. 7.5 ± 2.3 µU/mL, $p < 0.01$), HOMA-IR, cortisol, triglycerides, and ghrelin. Conversely, leptin and HDL were significantly lower in the disordered sleep group.

Conclusion: Sleep disorders are associated with adverse alterations in biochemical parameters, even among young, non-obese individuals. Promoting sleep hygiene could play a preventive role in early metabolic dysregulation.

Keywords: Sleep disorders, fasting blood glucose, insulin resistance, cortisol, young adults, metabolic health.

INTRODUCTION

Sleep is a fundamental physiological process essential for metabolic, cognitive, and emotional well-being. Over the past decade, an increasing body of evidence has highlighted the role of sleep duration and quality as critical determinants of metabolic health. Inadequate or fragmented sleep can disrupt hormonal balance, alter glucose metabolism, and contribute to chronic low-grade inflammation – all of which predispose individuals to non-communicable diseases such as type 2 diabetes mellitus (T2DM), cardiovascular disease (CVD), and obesity. [1, 2]

Mechanistically, sleep deprivation affects glucose regulation through multiple pathways. These include increased sympathetic nervous system activity, dysregulated hypothalamic-pituitary-adrenal (HPA) axis, elevated evening cortisol levels, alterations in growth hormone secretion, alterations in leptin and ghrelin levels, and increased pro-inflammatory cytokines. [3-5] Experimental studies show that even a single week of restricted sleep can significantly reduce insulin sensitivity and glucose tolerance in healthy individuals. [6] Young adults, especially medical students, are vulnerable to irregular sleep due to academic pressures and lifestyle choices. Despite being considered a metabolically healthy group, emerging studies suggest that sleep deprivation during this developmental stage may establish a foundation for future metabolic disorders, due to poor lifestyle habits, including erratic sleep. [7-9] However, limited data exist in the Indian context.

Hence, this study aims to explore the relationship between self-reported sleep duration and biochemical parameters among young adults aged 18–30 years. Understanding this relationship could provide insights for early preventive strategies against metabolic disorders in a population at an impressionable stage of life.

MATERIALS AND METHODS

Study Design and Participants: This was a cross-sectional observational study conducted over three months at Yellapragada Subba Rao Government Medical College, Eluru.

Sleep Assessment: All the first-year batch of students, including both male and females aged 18-20 years present on the day of data collection completed the Pittsburgh Sleep Quality Index (PSQI) [10] and reported average sleep duration over the past 7 days. Based on PSQI >5 and/or average sleep duration <6 hours, participants were classified into the **disordered sleep group** (n = 50); the remainder formed the **normal sleep group** (n = 50) by convenience sampling method. Exclusion criteria included individuals with known diabetes, metabolic syndrome, those using corticosteroids or antidepressants, and diagnosed sleep disorders like obstructive sleep apnea.

Biochemical Analysis: Venous blood samples were collected after 8–10 hours of fasting. Assessed parameters included:

- Fasting blood glucose (FBG): glucose oxidase-peroxidase method.
- Fasting insulin: enzyme-linked immunosorbent assay (ELISA).
- Lipid profile: enzymatic methods for triglycerides and HDL.
- Cortisol: chemiluminescence immunoassay.
- Leptin and Ghrelin: commercial sandwich ELISA kits.
- HOMA-IR: calculated as (FBG x Insulin)/405.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee before conduction of the study. Informed written consent was obtained from all participants prior to data collection.

Statistical Analysis: Data were analyzed using SPSS v25. Continuous variables were summarized as mean ± SD. Independent t-test and Mann–Whitney U test were used for group comparisons. A p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics: A total of 100 young adults participated in the study. The mean age was 22.4 ± 2.7 years; 56% were female. BMI ranged from 18.5 to 28.4 kg/m² (mean 23.2 ± 2.9). There was no significant difference in BMI, age, or gender between the normal sleep and disordered sleep groups. Most participants were non-smokers (98%) and reported a sedentary to moderately active lifestyle, with no major comorbidities.

Biochemical Profile Analysis:

Fasting Blood Glucose (FBG): Participants in the disordered sleep group had significantly elevated FBG levels (102.6 ± 10.1 mg/dL) compared to those in the normal sleep group (90.2 ± 8.4 mg/dL; $p < 0.001$). This rise indicates early impairment in glucose homeostasis potentially due to decreased insulin sensitivity linked to poor sleep patterns.

Insulin and HOMA-IR: Fasting insulin levels were significantly higher in the disordered sleep group (11.1 ± 3.0 µU/mL) versus the normal sleep group (7.5 ± 2.3 µU/mL; $p < 0.01$). Consequently, HOMA-IR index values were also elevated in poor sleepers (0.8 ± 0.2) compared to good sleepers (0.7 ± 0.2), although this did not reach statistical significance ($p = 0.07$). These findings suggest an early trend toward insulin resistance, a hallmark of metabolic syndrome.

Cortisol: Cortisol levels were significantly higher in the disordered sleep group (410 ± 85 nmol/L) compared to the normal sleep group (310 ± 70 nmol/L; $p < 0.05$). This supports the hypothesis that disrupted or insufficient sleep activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol secretion and stress-related metabolic consequences.

Leptin and Ghrelin: There was a notable reduction in leptin levels in the disordered group (7.0 ± 0.3 ng/mL) compared to the normal group (8.2 ± 2.1 ng/mL; $p < 0.001$), coupled with a significant increase in ghrelin (120.5 ± 22.9 pg/mL vs. 85.0 ± 18.2 pg/mL; $p < 0.01$). These hormonal shifts suggest altered appetite regulation favoring increased hunger and reduced satiety, potentially leading to overeating and weight gain over time.

Lipid Profile: Triglycerides were significantly elevated in the disordered sleep group (140.8 ± 3.1 mg/dL) versus the normal group (130.4 ± 8.5 mg/dL; $p < 0.01$), indicating a shift toward an atherogenic lipid profile. HDL cholesterol was slightly lower in poor sleepers (42.1 ± 6.3 mg/dL) compared to normal sleepers (43.3 ± 5.4 mg/dL), but this difference was not statistically significant ($p = 0.35$). LDL cholesterol showed a minor increase in the disordered group (64 ± 0.8 mg/dL) compared to the normal group (60 ± 1.2 mg/dL), yet the difference did not reach statistical significance ($p = 0.08$).

These findings collectively suggest that even in young, non-obese individuals, disordered sleep is associated with early signs of metabolic dysregulation. While some differences (e.g., HOMA-IR, LDL, HDL) were not statistically significant, their trends are clinically meaningful and warrant further investigation.

Table 1: Biochemical changes according to Sleep Duration across the groups

Biochemical Parameter	Normal Sleep (n = 50)	Disordered Sleep (n = 50)	p-value
Fasting Blood Glucose (mg/dL)	90.2 ± 8.4	102.6 ± 10.1	< **0.001**
Insulin (µU/mL)	7.5 ± 2.3	11.1 ± 3.0	< **0.01**
HOMA-IR (index)	0.7 ± 0.2	0.8 ± 0.2	0.07
Cortisol (nmol/L)	310 ± 70	410 ± 85	< **0.05**
Leptin (ng/mL)	8.2 ± 2.1	7.0 ± 0.3	< **0.001**
Ghrelin (pg/mL)	85.0 ± 18.2	120.5 ± 22.9	< **0.01**
Triglycerides (mg/dL)	130.4 ± 8.5	140.8 ± 3.1	< **0.01**
HDL Cholesterol (mg/dL)	43.3 ± 5.4	42.1 ± 6.3	0.35
LDL Cholesterol (mg/dL)	60 ± 1.2	64 ± 0.8	0.08

DISCUSSION

This study demonstrated a significant association between disordered sleep and alterations in various biochemical parameters among young adults. The observed increase in fasting blood glucose (FBG) and insulin levels, along with elevated HOMA-IR scores in the disordered sleep group, aligns with previous findings by Spiegel et al., who showed that partial sleep deprivation reduces insulin sensitivity in healthy subjects. [3] Our findings are further supported by Reutrakul and Van Cauter, who emphasized that poor sleep quality contributes to impaired glucose metabolism and a higher risk of type 2 diabetes. [1] Korean National Health and Nutrition Examination study, also found elevated impaired fasting glucose levels among individuals with short sleep durations. [11]

Elevated cortisol levels observed in our disordered sleep group are consistent with the work of Leproult and Van Cauter, who demonstrated that sleep loss activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol secretion and stress-related metabolic dysregulation. [12] The chronic elevation of cortisol may exacerbate glucose intolerance by promoting hepatic gluconeogenesis and impairing insulin action.

The reduction in leptin and elevation in ghrelin in our study mirrors the results from Taheri et al, who found that short sleep duration is associated with hormonal changes that increase hunger and appetite, thereby promoting weight gain and insulin resistance. [13] These appetite-regulating hormones are crucial in maintaining energy homeostasis and may explain the long-term association between poor sleep and obesity risk.

Our findings of elevated triglycerides and a trend toward increased LDL cholesterol in disordered sleepers also correspond to reports from St-Onge et al, who found that sleep restriction adversely affects lipid metabolism and increases cardiovascular risk. [14] Although HDL differences were not statistically significant in our study, the downward trend is in line with the dyslipidemic profile associated with sleep deprivation, as previously reported by Chaput et al. [15] Interestingly, the observed trends in HOMA-IR and LDL, while not statistically significant, suggest early subclinical metabolic changes that could progress over time with sustained poor sleep quality. This finding underscores the importance of early intervention strategies aimed at improving sleep hygiene to mitigate future metabolic risk.

Overall, our study reinforces existing literature suggesting that inadequate sleep negatively influences glycemic control, hormonal balance, and lipid profiles—even in a young, ostensibly healthy population. These alterations, if persistent, may predispose individuals to metabolic syndrome, obesity, and type 2 diabetes in the long term.

CONCLUSION

In summary, our findings highlight a significant inverse association between sleep duration and fasting blood glucose levels in young adults. The high prevalence of impaired glucose metabolism among short sleepers in this age group suggests that sleep duration is an important and underrecognized factor in early metabolic health. Public health strategies aimed at promoting adequate sleep in youth could play a critical role in preventing the early onset of insulin resistance and type 2 diabetes.

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