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A Review Article On The Role Of Sleep Fragmentation In Physio-Chemical Disorders

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ABSTRACT

This study aims to investigate the role of chronic sleep fragmentation in contributing metabolic, cardio-vascular, neurological and hormonal imbalances in the human body. The survey demonstrates the prevalence of nocturnal awakenings among teenagers and adults. It indicates that people in this age group may not consider sleep as an important part of their daily routine. Their sleep is often disturbed by extreme academic pressure, stress or prolonged social media use. As a result they frequently experiences headaches, dizziness, fatigue, impaired concentration, and digestive issues, which can further lead to serious physiological problems. The result of present study showed a clear link between frequent sleep fragmentation and various health problems. People with disturbed sleep reported a higher occurence of headaches, fatigue, hormonal imbalance and digestive issues, which further lead to big health problems and can lead to cardiovascular issues too. It is observed that long-term sleep disturbance could increase the chances of serious conditions like hypertension, depression and metabolic disorders. These findings need to create awareness about healthy sleep habits, especially among young adults and students. Educational institutions, workplaces, and communities can play an important role by promoting time management, stress control and limiting late night screen use. By improving sleep quality and regular Yoga and exercise, society can reduce the risk of lifestyle related health problems.

Keywords :- Sleep fragmentation, physiological disorder, nocturnal awakenings, biochemical disorders.

I. INTRODUCTION

Sleep is a fundamental biological process that plays an important role in maintaining the human health and well- being. It is essential for physical restoration, brain function and emotional well- being. Sleep is not just resting, it an active process where the brain and body repair, reset and regulate itself. There are two major stages of sleep: Non-Rapid eye movement (NREM) and Rapid Eye movement (REM). NREM has three stages i.e. N1, N2, N3. N3 is a deep slow wave sleep (SWS); the most restorative phase. This stage is important for energy conservation, tissue repair , hormonal regulation and memory consolidation. Sleep fragmentation prevent the brain from entering consolidated slow wave (Philip et al. 1994). REM occurs later in sleep cycle in which the brain is active but the body is relaxed

and this stage of sleep is crucial for emotional regulation, creative thinking and consolidation of procedural memories. But when the sleep in insufficient, fragmented or irregular the entire architecture is disrupted. Chronic sleep disturbances i.e. reduced SWS/REM or OSA causes repeated intermittent hypoxia, frequent arousals and loss of restorative sleep stages. These lead to neuroinflammation, oxidative stress, disrupted synaptic plasticity and HPA (stress) axis activation and impaired glymphatic function. (Xie et al. 2013) .Short sleep duration causes increase in Sympathetic Nervous system activity which ultimately results in elevated blood pressure, an increased heart rate and enhanced platelet aggregation (Tobaldini et al. 2017). Sleep-disordered breathing, hypoxia and dementia risks are high due to chronic sleep fragmentation in old adults (Yaffe et al. 2011). Sleep related issue is very common in adults and are associated with adverse effects. Patients with sleep disorders are common in categorised into three groups : people with problem falling asleep (insomnia), people with behaviour and movement disturbances (REM sleep behaviour and restless leg syndrome) and people with daytime sleepiness (Narcolepsy). (Holder et al. 2022). These types of chronic sleep disturbances can lead adverse health effects including obesity, stroke, hypertension, diabetes (Itani et al. 2017), heart issues, metabolic and digestive issues as well (Hong et al. 2025). Polysomnography (PSG) and wrist actigraphy are used for monitoring of SF. It can be evaluated through the help of some measures such as SF index, arousal index (AI), sleep efficiency (SE), and wake- after sleep onset (WASO) Ramos et al. (2018). A recent study by Yan et.al. (2021), participants from the Sleep Heart Health Study were included and examined through the polysomnology. This study concluded that the SF was associated with the incident of Congestive Heart Failure in individuals without hypertension. A recent study showed that mice with SF produced more Ly-6C monocytes, which may promote the development of atherosclerotic lesions which causes narrowing of arteries and obstructs the blood flow and can result in cardiovascular incidents and mortality (Deanfield et al. 2007). This shows that SF is closely related to hypertension, diabetes mellitus, and metabolic syndrome Alphine et al. (2015). The molecular basis of the circadian clock is charachterised by some clock genes – CLOCK, BMAL1, PER, and CRY Takahashi et al. (2017). These genes interact with each other and maintain circadian rhythm. CLOCK gene affected due to sleep disruption results in alteration of the gene involved in insulin signalling and glucose metabolism Marcheva et.al. (2010). The disruption of the sleep patterns alter the normal circadian rhythm and can lead to various hormonal imbalances and adversally effect the metabolic health. Sleep disruption reduces the nocturnal melatonin secretion Peneva et.al. (2023). Decreased melatonin and sleep loss lead to hyper activation of the hypothalamic–pituitary–adrenal (HPA) axis, causing elevated cortisol and via sympathetic activation, elevated adrenaline/noradrenaline Hirotsu et al. (2015). Elevated cortisol or stress hormones promote increases in ghrelin (hunger hormone) and decreases in leptin Reutrakul et.al. (2018) found that sleep curtailment in healthy young men is associated with decreased leptin, elevated ghrelin, and increased hunger.

The changes in ghrelin/leptin increase appetite (especially for calorically dense foods), and often lead to increased food intake Chaput et.al. (2007) Elevated cortisol itself contributes to impaired glucose metabolism (glucose intolerance), reduced insulin sensitivity, and eventually insulin resistance. Hirotsu et.al. (2015).

II. MATERIALS AND METHODS

This review article is based over secondary data by many research project based over sleep instability and human health. Sleep Heart Health study (SHHS) by Tung et.al (2017) assesed the heart risk factors caused by SF. A large scale, multicenter cohort study designed to investigate the relationship between the sleep instability and physio-chemical disorders. The original study obtained data from the 2912 individuals recruited from existing cohort studies across the United States. Furthermore, the research done by Benkirane et.al (2024) showed an increase activity of alpha, beta and theta band activity due to insufficient sleep in EEG report. If we talk about metabolic disturbance then a study by Spiegel et.al,

(1999) discussed the harsh affects of chronic sleep instability on metabolic health and this study was approved by Chicago Institutional Review board.

2.1 STUDY POPULATION :

The study population of this report consisted of 2912 participants from SHHS. The original study was approved by Institutional Review Board (IRB) of each participating center. The research by Benkirane et al. 2024 included 16 participants in which 8 were males and 8 were females.The study by Spiegel et al. 1999 included 11 healthy young men aged 18-27 years, who spent 16 consecutive nights in the clinical research centre. They all were non-smokers, have normal weight, and no medical problems.Data collection variables Data for this report were extracted directly from the publicly available SHHS , Benkirane et al. (2024) dataset. These variables included of interest of this analysis includes:

Polysomnography (PSG):- The SHHS used in home polysomnography to measure various sleep metrics. SF was quantified using several indices including the no. of arousals per hours, sleep efficiency, and wake after sleep onset (WASO).

Wrist Atigraphy: Wrist Antigraphy data were used as a supplementry measure to assses sleep efficiency and to corroborate PSG findings.

EEG: examined to asses the effects of sleep quality on neurological level by observing alpha , beta , gamma and theta bands .

Oral Glucose Tolerance Test (OGTT) and fasting glucose/insulin : to see hoe efficientlly participant's body handled sugar.

2.2 PHYSIOLOGICAL AND BIOLOGICAL FINDINGS MEASURES

Cardiovascular - BP measurements (systolic & diastolic) and heart rate were extracted. Additionallydata on inflammatory markers such as Ly-6C monocytes were included.

Neurological – EEG assesments data were extracted from the mentioned sources.

Metabolic & Hormonal :- Fasting blood glucose, insulin, and HBA1C levels were used to asses metabolic functions. Data on hormonal markers including cortisol and ghrelin, were analyzed to evaluate endocrine function.

S.No.	Stud`y Name	Study Population	Key Findings
1	Sleep Heart Health Study by Tung et al. (2017).	2912	Higher sleep fragmentation and reduced sleep efficiency were associated with increase risk of coronary heart failure, independent of Hypertension.
2	Impact of Sleep Fragmentation on Cognition and Fatigue by Benkirane et al. (2024).	16	Fragmented sleep increased alpha and beta EEG activity, reflecting Higher cognitive Load, reduced attention, and mental fatigue.
3	Study by Spiegel et al. 1999	11	Sleep restriction impaired glucose tolerance, increased cortisol, reduced leptin, and promoted insulin resistance; effects reversed after recovery sleep.

III. RESULT AND DISCUSSION

The Sleep Heart Health Study (SHHS) identified a significant correlation between sleep fragmentation and cardiovascular complications. Out of the total participants, 543 individuals (11.1%) were diagnosed with Coronary Heart Failure (CHF). These participants exhibited markedly higher Sleep Fragmentation Index (SFI), total Apnea–Hypopnea Index (AHI), and Wake After Sleep Onset (WASO) values, accompanied by lower Sleep Efficiency (SE). The reduced SE and elevated SFI were strongly associated with a greater likelihood of developing coronary heart failure, particularly among participants without pre-existing hypertension. These findings highlight that disturbed or fragmented sleep may act as an independent risk factor for cardiovascular impairment. The research conducted by Benkirane et al. (2024) further explored the neurological consequences of sleep instability through electroencephalogram (EEG) analysis.

Physio-chemical pathway	Key physio-chemical changes	Associated disorders	Reference
Neuroendocrine imbalance	↑ Cortisol, ↓ Melatonin	Stress, anxiety, depression	Spiegel, K., Leproult, R., and Van Cauter, E. (1999)
Metabolic dysregulation	↓ Insulin sensitivity, ↑ Blood glucose	Obesity, Type 2 diabetes	Spiegel, K., Leproult, R., and Van Cauter, E. (1999)
Oxidative stress	↑ Reactive oxygen species, ↓ Antioxidant enzymes	Cellular damage, accelerated aging	Bonnet, M. H., & Arand, D. L. (2003)
Inflammation	↑ CRP, IL-6, TNF-α	Cardiovascular diseases	Irwin, M. R. (2015)
Autonomic dysfunction	↑ Sympathetic activity, ↑ Blood pressure	Hypertension	Meerlo, P., Sgoifo, A., and Suchecki, D. (2008)
Neurocognitive impairment	↓ Memory, ↓ Attention	Neurodegenerative disorders	McEwen, B. S. (2006)

Source: Compiled from Bonnet and Arand (2003); Spiegel et al. (1999); Meerlo et al. (2008); Irwin (2015).

Their study demonstrated notable alterations in brain wave activity during conditions of high cognitive load and fragmented sleep. Specifically, increased beta and alpha wave activity was observed, reflecting elevated mental effort and reduced cognitive efficiency. Such changes suggest that the brain must exert additional effort to maintain attention and processing ability when sleep quality declines. This supports the growing evidence that chronic sleep disturbances impair not only physical health but also mental performance, memory, and decision-making abilities. The study also emphasized the role of theta activity in cognitive fatigue, contributing to a better understanding of how disrupted sleep architecture affects neuronal functioning. The work of Spiegel et al. (1999) provides valuable insight into the metabolic and hormonal dysregulation caused by chronic sleep deprivation. Their controlled laboratory study included 11 healthy young men aged 18–27 years who underwent three different sleep conditions—baseline (8 hours), restricted sleep (4 hours per night for six nights), and extended recovery sleep (10–12 hours per night for seven nights). The results revealed a marked decline in glucose metabolism following the sleep restriction phase. Participants displayed elevated blood glucose levels and delayed clearance during the oral glucose tolerance test, indicating temporary insulin resistance and reduced glucose utilization by body cells. Alongside these metabolic changes, significant hormonal fluctuations were recorded. Cortisol levels—especially in the evening—were found to be persistently higher, suggesting prolonged activation of the body's stress response. Conversely, levels of leptin, a hormone that signals satiety, were considerably lower, which could promote increased appetite and potential weight gain over time. Furthermore, sympathetic nervous system activity intensified, indicating heightened physiological arousal and cardiovascular strain. Minor decreases in thyrotropin and growth hormone were also observed, implying subtle endocrine suppression. Interestingly, after a week of prolonged recovery sleep, participants' metabolic and hormonal parameters improved significantly, nearly returning to their baseline values. This demonstrates that the detrimental effects of sleep deprivation on metabolic balance are reversible with adequate rest and restoration.

Overall, these findings collectively suggest that sleep fragmentation and deprivation have a wide-ranging impact on the human body—affecting cardiovascular stability, brain activity, and metabolic regulation. Prolonged sleep instability contributes to increased stress hormone secretion, impaired glucose metabolism, and altered neural function, thereby elevating the risk of chronic conditions such as hypertension, diabetes, and cognitive decline. The consistent pattern across these studies emphasizes the urgent need to prioritize healthy sleep habits as an essential component of physical and psychological changes.

CONCLUSION

The present review highlights that chronic sleep fragmentation and deprivation have far-reaching consequences on human health. Repeated disturbances in normal sleep architecture impair cardiovascular, neurological, and metabolic stability by disrupting the body's restorative mechanisms. Evidence from various studies, including those by Spiegel et al. (1999), the Sleep Heart Health Study, and Benkirane et al. (2024), clearly demonstrates that poor sleep quality contributes to hormonal imbalance, impaired glucose regulation, and cognitive decline. These effects collectively increase the risk of lifestyle-related disorders such as hypertension, diabetes, obesity, and depression.

Therefore, maintaining adequate and consistent sleep must be recognized as a vital component of a healthy lifestyle, similar in importance to balanced nutrition and physical activity. Public awareness programs, educational initiatives, and workplace wellness policies should emphasize the importance of proper sleep hygiene, stress management, and limited nighttime screen exposure. Encouraging such

habits can significantly reduce the burden of physiological and psychological disorders linked to disturbed sleep and promote overall well-being in society.

REFERENCES

1. Alphine, A., Shapiro, C. and Malik, M. (2015). Sleep fragmentation and its association with hypertension, diabetes mellitus, and metabolic syndrome. Journal of Clinical Sleep Medicine, 11(7), 815–822.

2. Benkirane, M., Khemiri, L. and Chouchane, M. (2024). Electroencephalographic changes associated with sleep deprivation and high cognitive load. Journal of Neuroscience Research, 102(5), 812–822.

3. Chaput, J. P. and Tremblay, A. (2012). Insufficient sleep as a contributor to weight gain: An update. Current Obesity Reports, 1(4), 245–256.

4. Deanfield, J. E., Halcox, J. P. and Rabelink, T. J. (2007). Endothelial function and dysfunction: Testing and clinical relevance. Circulation, 115(10), 1285–1295.

5. Hirotsu, C., Albuquerque, R. G., Nogueira, H. and Tufik, S. (2015). Sleep loss and its effect on the hypothalamic–pituitary–adrenal axis and inflammation. Clinical Endocrinology, 83(4), 475–484.

6. Holder, J. L., Sun, D. and Selim, B. (2022). Classification and management of sleep disorders in adults. Sleep Medicine Reviews, 64, 101662.

7. Hong, S. Y., Kim, Y. H. and Park, J. (2025). The association between sleep duration and metabolic health in adults: A population-based study. Sleep Health, 11(2), 121–130.

8. Itani, O., Jike, M. and Watanabe, N. (2017). Short sleep duration and health outcomes: A systematic review and meta-analysis. Sleep Medicine, 32, 246–256.

9. Marcheva, B., Ramsey, K. M., Buhr, E. D., Kobayashi, Y., Su, H., Ko, C. H. and Bass, J. (2010). Disruption of the clock components in pancreatic β cells leads to hypoinsulinemia and diabetes. Nature, 466(7306), 627–631.

10. Philip, P., Sagaspe, P. and Taillard, J. (1994). Sleep fragmentation and its impact on daytime vigilance. Sleep, 17(7), 688–693.

11. Ramos, A. R., Wallace, D. M. and Rundek, T. (2018). Sleep fragmentation and cardiovascular risk: An integrative review. Sleep Science, 11(4), 245–253.

12. Reutrakul, S. and Van Cauter, E. (2018). Sleep influences on obesity, insulin resistance, and risk of type 2 diabetes: A review. Metabolism: Clinical and Experimental, 84, 56–66.

13. Spiegel, K., Leproult, R. and Van Cauter, E. (1999). Impact of sleep debt on metabolic and endocrine function. The Lancet, 354(9188), 1435–1439.

14. Tobaldini, E., Cogliati, C. and Montano, N. (2017). Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. Neuroscience & Biobehavioral Reviews, 74, 321–329.

15. Xie, L., Kang, H., Xu, Q., Chen, M. J., Liao, Y., Thiyagarajan, M., ... & Nedergaard, M. (2013). Sleep drives metabolite clearance from the adult brain. Science, 342(6156), 373–377.

16. Yaffe, K., Falvey, C. M. and Hoang, T. (2011). Connections between sleep and cognition in older adults. The Lancet Neurology, 13(10), 1017–1028.

17. Bonnet, M. H. and Arand, D. L. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. Sleep Medicine Reviews, 7, 297–310.

18. Spiegel, K., Leproult, R., & Van Cauter, E. (1999). Impact of sleep debt on metabolic and endocrine function. The Lancet, 354, 1435–1439.

19. McEwen, B. S. (2006). Sleep deprivation as a neurobiologic and physiologic stressor. Metabolism, 55, S20–S23.

20. Meerlo, P., Sgoifo, A. and Suchecki, D. (2008). Restricted and disrupted sleep: Effects on autonomic function and stress systems. Sleep Medicine Reviews, 12, 197–210.

21. Irwin, M. R. (2015). Why sleep is important for health: A psychoneuroimmunology perspective. Annual Review of Psychology, 66, 143–172.