American Journal of Multidisciplinary Research & Development (AJMRD)
*Volume 08, Issue 01 (**January - 2026**), PP 35-37*
ISSN: 2360-821X
www.ajmrd.com

Research Paper **Open Access**

CHANGES IN SERUM ELECTROLYTES, UREA, AND CREATININE LEVELS FOLLOWING SLEEP DEPRIVATION IN ALBINO RATS.

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ABSTRACT: Sleep deprivation is a major physiological stressor that disrupts endocrine and metabolic homeostasis, potentially altering electrolyte balance and renal function. This cross sectional study evaluated the effects of sleep deprivation on serum electrolytes, urea, and creatinine levels in albino rats. Twenty four albino rats (150–200 g) were randomly divided into control and sleep-deprived groups. Sleep deprivation was induced using the random noise method for 72 hours. Blood samples were collected from lateral tail vein. Serum sodium, potassium, chloride, bicarbonate, urea, and creatinine were measured using ISE, urease Berthelot and Jaffe slot biochemical methods respectively. Sleep-deprived rats showed significantly higher mean serum sodium (143.67 ± 1.118 mmol/L verses 141.00 ± 1.225 mmol/L) ($P < 0.05$), chloride (101.00 ± 1.000 mmol/L versus 98.22 ± 0.441 mmol/L; ($P < 0.05$), urea (8.011 ± 1.789 µmol/L versus 5.722 ± 0.908 µmol/L) ($P < 0.05$), and creatinine (91.311 ± 9.203 µmol/L versus 50.489 ± 18.857 µmol/L) ($P < 0.05$) compared to controls. Potassium and bicarbonate showed no significant differences ($P > 0.05$). These findings indicate that sleep deprivation induces marked alterations in electrolyte and renal parameters, suggesting renal stress and impaired homeostasis as physiological consequences of sleep loss even when it is not too prolonged.

Keywords: Sleep deprivation, serum electrolytes, urea, creatinine, renal function, albino rats

I. INTRODUCTION

Sleep is a natural, recurring state of rest for the body and mind characterized by reduced consciousness, limited movement, and decreased responsiveness to external stimuli. It is essential for physical and mental health, allowing the body to repair, grow and consolidate memories. Sleep is typically divided into several stages, including REM (Rapid Eye Movement) and non-REM, each serving different functions such as tissue repair, energy restoration and cognitive processing, sleep is crucial for overall well-being and optimal functioning [1].

Sleep deprivation is consistently getting less sleep than needed and can have profound effects on various physiological and biochemical system [3], disruption of the sleep impair essential functions which can lead to range of health issues. In the mammalian brain, the sleep-wake cycle is orchestrated by a complex network of discrete neuronal populations that induce sleep or wakefulness via promoting or suppressing effect [2].

Sleep plays a crucial role in maintaining physiological balance, particularly in metabolic and renal functions. Chronic or acute sleep deprivation triggers sympathetic activation, oxidative stress, and endocrine disruption, all of which can influence renal homeostasis and electrolyte regulation [1–3]. Previous studies have demonstrated that insufficient sleep is associated with increased serum sodium, urea, and creatinine, indicating stress-induced renal impairment [4, 5].

Electrolytes, urea and creatinine are critical biochemical markers, often used to assess kidney function, electrolyte balance and overall metabolic health. Electrolyte balance is vital for nerve conduction, muscle function, and acid-base regulation. Disturbance in these parameters can reflect compromised kidney function or metabolic dysregulation [6]. In sleep deprived state, significant physiological alteration occurs, and the implications of changes in these markers are profound, especially in animal model such as albino rats [4]. Sleep deprivation affects the hypothalamic pituitary-adrenal (HPA) axis, leading to elevated cortisol level, which in turn cause electrolyte

imbalance, altered secretion of antidiuretic hormone (ADH) due to change in circadian rhythms that affect fluid retention, thereby altering sodium and potassium levels [5]. This imbalance in electrolytes due to change in ADH secretion can also lead to hypertension and cardiovascular complications in these rats. Sleep deprivation can impair renal perfusion, reducing the glomerular filtration rate (GFR), which eventually lead to increase in blood creatinine levels [6]. A study on albino rats demonstrated that prolong sleep deprivation leads to a significant increase in serum urea (due to increase protein catabolism) and creatinine levels, indication of reduced kidney function and possible progression to renal injury [7]. Despite growing evidence linking sleep loss to cardiovascular and metabolic diseases, limited data exist on how acute sleep deprivation affects renal biomarkers in animal models within the Nigerian setting.

This study investigated the effects of sleep deprivation on serum electrolytes, urea, and creatinine levels in albino rats and understanding these biochemical responses provides insight into how sleep loss influences renal physiology and systemic homeostasis.

II. MATERIALS AND METHODS

Experimental Animals

Twenty four healthy albino rats weighing 150–200 g were obtained from the Animal House of Enugu State University Teaching Hospital, Enugu, Nigeria. The rats were acclimatized for one week under standard laboratory conditions (temperature 25 ± 2 °C, 12 h light/dark cycle) with free access to feed and water.

Experimental design

The animals were divided randomly into two groups (n = 12 each):

Group 1 (Control): Normal sleep cycle.

Group 2 (Sleep-deprived): Subjected to sleep deprivation for 72 hours using the Random/Environmental Noise Method, which exposed rats to intermittent auditory disturbances preventing sustained sleep [8].

Sample collection and biochemical analysis

At the end of the experiment, blood samples were collected via tail vein and saphenous vein puncture into plain tubes. Serum was separated and analyzed for:

Electrolytes (Na^+, K^+, Cl^-, HCO_3^-) using potentiometric methods (ISE, Amemiya, 2007) [9] .

Urea determined by urease Berthelot method.

Creatinine measured by the Jaffe Slot alkaline reaction

Statistical analysis

Data were expressed as mean ± SEM. Differences between groups were analyzed using ANOVA, and independent Student's t-test, with $p < 0.05$ considered statistically significant.

III. RESULTS

Table 1 show the mean level of serum electrolytes between sleep deprived rats and control

Variables (mmol/L)	Sleep deprived	Control group	T-value	P
Sodium	143.67 1.118	141.0 1.225	9.238	< 0.05
Potassium	4.778 0.778	5.00 0.487	-0.794	> 0.05
Chloride	101.0 1.000	98.22 0.441	2.800	< 0.023
Bicarbonate	26.44 1.333	26.33 0.500	0.217	> 0.05

Table 1 indicating significant higher sodium and chloride level compared with the control group

Table 2 shows mean level of serum urea and creatinine between sleep deprived rats and control

Variables (mmol/L)	Sleep deprived	Control group	T-value	P
Urea	8.011 1.789	5.722 0.908	2.968	< 0.05
Creatinine	91.311 9.203	50.489 18.857	8.809	< 0.05

Table 2 indicating significant markedly elevated urea and creatinine level compared to the control group.

IV. DISCUSSION

Sleep deprivation in albino rats is an experimental model for studying the physiological and biochemical effects of stress, cognitive impairment, and metabolic disturbances. Prolonged loss of sleep leads to oxidative stress, hormonal imbalance, glucose dysregulation, and behavioral changes. It affects neurotransmitter systems and can impair memory, immune function, and learning. In this experiment, sleep deprivation in albino rats caused significant alterations in specific biochemical parameters. Mean serum sodium (143.67 ± 1.118 mmol/L) was significantly higher in sleep-deprived rats compared to controls (141.00 ± 1.225 mmol/L; $P < 0.05$). Chloride also

increased significantly ($P < 0.05$). However, potassium (4.778 ± 0.778 mmol/L vs 5.000 ± 0.487 mmol/L; $P > 0.5$) and bicarbonate (26.44 ± 1.333 mmol/L vs 26.33 ± 0.500 mmol/L; $P > 0.05$) showed no significant differences.

Urea and creatinine levels were markedly elevated in sleep-deprived rats (8.011 ± 1.789 µmol/L and 91.311 ± 9.203 µmol/L, respectively) compared to controls (5.722 ± 0.908 µmol/L and 50.489 ± 18.857 µmol/L), with $p < 0.05$. These results indicate that sleep deprivation induces renal stress and electrolyte imbalance even when it is too prolonged.

This study demonstrated that sleep deprivation significantly elevates serum sodium, chloride, urea, and creatinine levels, suggesting disturbances in electrolyte balance and renal function. The observed hypernatremia and hyperchloremia are consistent with previous findings indicating that sympathetic activation and stress-induced antidiuretic hormone release during sleep loss can impair water and sodium regulation [7, 10].

The increase in serum urea and creatinine among sleep-deprived rats reflects reduced renal clearance and possible glomerular filtration impairment [11, 12]. These alterations may result from hemodynamic changes, oxidative stress, or altered hypothalamic–pituitary signaling linked to prolonged wakefulness [13].

The non-significant changes in potassium and bicarbonate align with studies reporting that short-term sleep deprivation may not markedly affect acid–base status (14). Overall, these findings highlight that even brief sleep loss can provoke renal and metabolic dysregulation, reinforcing the importance of adequate sleep for maintaining physiological balance.

V. CONCLUSION

Sleep deprivation significantly alters serum electrolytes and renal biomarkers, indicating possible renal stress and impaired homeostatic control. Elevated sodium, chloride, urea, and creatinine levels suggest that inadequate sleep can trigger biochemical changes characteristic of early renal dysfunction. Further research is warranted to elucidate the mechanisms linking sleep loss and renal physiology in humans.

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