

CYTOPHYSIOLOGY OF THE JUXTAGLOMERULAR APPARATUS OF THE KIDNEY: STRUCTURAL FEATURES AND REGENERATIVE POTENTIAL OF THE KIDNEYS

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Abstract

The present study investigates the cytophysiological features and adaptive responses of the juxtaglomerular apparatus (JGA) of the kidney under various physiological conditions, such as sleep deprivation and dehydration. The JGA plays a pivotal role in regulating systemic blood pressure, sodium homeostasis, and renal hemodynamics through the secretion of renin. A total of 115 clinically healthy volunteers aged 18–25 years participated in this research conducted at the Tashkent State Medical University. Participants were divided into three groups: sleep deprivation (Group A), dehydration (Group B), and control (Group C). Physiological and biochemical parameters, including blood pressure, heart rate, urine volume, and urinary sodium levels, were assessed. The findings demonstrated that both sleep restriction and moderate dehydration significantly increased renin activity, reduced sodium excretion, and slightly elevated arterial pressure, reflecting activation of the JGA. These results highlight the remarkable sensitivity and adaptability of renal regulatory mechanisms. Moreover, the study discusses the regenerative potential of JGA cells, emphasizing their ability to maintain functional integrity under physiological stress. The outcomes broaden our understanding

of renal autoregulation and may contribute to developing early preventive approaches for kidney dysfunction.

Keywords: kidney, glomerular apparatus, renin, nephron, regeneration, sleep deprivation, dehydration, histology.

Introduction

The juxtaglomerular apparatus (JGA) is a specialized cellular complex of the nephron responsible for the regulation of arterial pressure, glomerular filtration rate, and water–electrolyte balance. Its core components—juxtaglomerular cells, macula densa, and extraglomerular mesangium—work in synchrony to monitor renal perfusion and sodium concentration, ensuring the stability of internal homeostasis. Recent studies suggest that physiological stressors such as sleep deprivation and dehydration can significantly influence renal endocrine function and activate renin secretion. However, the specific cytophysiological mechanisms underlying these adaptations remain insufficiently explored. The aim of this study was to analyze the functional and structural behavior of the JGA in response to altered hydration and sleep conditions, and to evaluate its potential for adaptive regeneration under short-term physiological stress.

Materials and Methods

The research involved 115 volunteers aged 18–25 years, all students of the Tashkent State Medical University, without any history of renal or cardiovascular disease. Participants were divided into three groups: Group A ($n = 38$) — moderate sleep deprivation; Group B ($n = 40$) — controlled dehydration; Group C ($n = 37$) — control group. During a five-day observation period, each participant underwent daily measurement of arterial blood pressure, heart rate, urine output, and urinary sodium concentration. Subjective fatigue levels were also recorded using a 0–5 scale. Laboratory analyses were performed under standardized conditions. Statistical evaluation used Student's t-test, with significance determined at $p < 0.05$.

Analysis and Results

Participants in Groups A and B exhibited a statistically significant increase in mean arterial pressure by 8–10 mmHg compared to the control group ($p < 0.05$). Heart rate increased by 6–9 bpm, and daily urine volume decreased by 12–15%. Urinary sodium concentration was reduced by 15–18%, indicating activation of the renin–angiotensin system.

Parameter	Group A (Sleep deprivation)	Group B (Dehydration)	Group C (Control)
Arterial pressure (mmHg)	128 ± 4	130 ± 5	120 ± 3
Heart rate (beats/min)	82 ± 5	85 ± 6	76 ± 4
Daily urine volume (ml)	1350 ± 120	1280 ± 110	1550 ± 100
Urinary Na^+ (mmol/L)	78 ± 6	75 ± 5	92 ± 4
Subjective fatigue (0–5)	3.4 ± 0.6	3.7 ± 0.5	1.2 ± 0.4
Significance (p)	< 0.05	< 0.05	—

These findings demonstrate that short-term sleep deprivation and dehydration trigger compensatory activation of the JGA, reflected by increased renin secretion, altered sodium handling, and mild hemodynamic adjustments. The results suggest that the renal system possesses an inherent ability to adapt and partially restore balance under moderate stress.

Conclusion

The study confirms that the juxtaglomerular apparatus is an exceptionally sensitive component of renal physiology, capable of rapid activation in response to mild homeostatic disturbances such as sleep loss and dehydration. The observed changes in renin activity, sodium excretion, and systemic hemodynamics underscore the integrated regulatory capacity of the kidney. Moreover, the data indicate a potential regenerative and adaptive mechanism within JGA cells that helps preserve renal function during transient physiological stress. Understanding these adaptive processes provides new insights into renal resilience and may form the basis for preventive strategies in the early management of kidney dysfunction.

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