AMERICAN JOURNAL OF BIOSCIENCE AND CLINICAL INTEGRITY
https://biojournals.us/index.php/AJBCI
ISSN: 2997-7347

Article

Morphological and Morphometric Characteristics of the Kidneys and Testes of 6-Month-Old Rats Under Combined Deficiency Conditions

R. R. Baymuradov[1], Sh. J. Teshayev[2]

1,2. Bukhara State Medical Institute named after Abu Ali ibn Sino

Citation: Baymuradov R. R., Teshayev Sh. J. Morphological and Morphometric Characteristics of the Kidneys and Testes of 6-Month-Old Rats Under Combined Deficiency Conditions. American Journal Of Bioscience And Clinical Integrity 2026, 3(5), 1-5.

Received: 10th Feb 2026
Revised: 11th Mar 2026
Accepted: 24th Apr 2026
Published: 07th May 2026

Abstract: This study investigated the morphological and morphometric changes in the kidneys and testes of 6-month-old rats under conditions of combined deficiency of Zn, Se, Mg, and Fe trace elements. The results demonstrated pronounced dystrophic changes in the glomerular and tubular apparatus of the kidneys, structural remodeling of the filtration barrier, and interstitial remodeling processes. In the testes, marked thinning of the spermatogenic epithelium, reduction in germ cell population, decreased number of Leydig cells, and development of interstitial fibrosis were observed. The Johnsen scoring system confirmed a significant suppression of spermatogenesis. The obtained data indicate that combined trace element deficiency leads to severe structural and functional remodeling of both reproductive and excretory systems through oxidative stress and trophic disturbances.

Keywords: Combined micronutrient deficiency, zinc, selenium, iron, magnesium, kidney, testis, morphometry, spermatogenesis, oxidative stress, tubular apparatus, glomerulus, Leydig cells, interstitial tissue, Johnsen score.

Introduction

Trace elements are essential biological regulators involved in enzymatic reactions, antioxidant defense, and cellular proliferation. Their deficiency leads to structural and functional disturbances in multiple organs and systems. Zinc, selenium, iron, and magnesium are key components of antioxidant defense systems, and their deficiency enhances cellular membrane damage and mitochondrial dysfunction [1].

The kidney is highly sensitive to trace element deficiency due to its high metabolic activity, which results in impaired glomerular filtration and tubular reabsorption [2]. Similarly, the male

reproductive system is extremely vulnerable, and disturbances affect all stages of spermatogenesis, particularly meiosis and post-meiotic phases [3].

However, the complex morphological alterations of the kidneys and testes under combined micronutrient deficiency remain insufficiently studied. Therefore, this study aimed to investigate the systemic effects of combined trace element deficiency.[4]

Aim of the Study

To comprehensively investigate morphological and morphometric changes in the kidneys and testes of 6-month-old rats under combined Zn, Se, Mg, and Fe deficiency, to identify oxidative-dystrophic processes and evaluate their functional significance.[5]

Materials and Methods

The study was conducted on 6-month-old laboratory rats divided into two groups: a control group maintained on a standard diet and an experimental group subjected to a micronutrient-deficient diet for 3 months to induce Zn, Se, Mg, and Fe deficiency. At the end of the experiment, animals were sacrificed according to bioethical standards, and kidney and testicular tissues were collected. Samples were fixed in 10% neutral formalin, embedded in paraffin, and sectioned at 5–7 μm thickness.[6] Histological slides were stained with hematoxylin and eosin. Morphometric analysis was performed using a microscope with a digital image analysis system. In the kidneys, measurements included arteriole diameter, glomerular and Bowman's capsule dimensions, tubular diameter, and epithelial height. In the testes, seminiferous tubule diameter, epithelial thickness, Sertoli and Leydig cell counts, and germ cell population were assessed. Spermatogenesis was evaluated using the Johnsen scoring system. Statistical analysis was performed using Student's t-test, with significance set at $p < 0.05$.[7]

Results

Under conditions of combined micronutrient deficiency, pronounced vascular, glomerular, and tubular alterations were observed compared to the control group. The diameter of the afferent arteriole decreased to 14.7 ± 0.23 μm, while the efferent arteriole decreased to 11.4 ± 0.26 μm, indicating reduced glomerular perfusion and impaired intrarenal hemodynamics.[8] The glomerular diameter increased to 117.9 ± 1.20 μm, and the renal corpuscle diameter reached 131.2 ± 1.49 μm. The volume of Bowman's space significantly increased to 0.73 ± 0.03 $\times 10^6$ μm^3, indicating marked expansion of the filtration space.[9] The basement membrane was thickened, reflecting structural remodeling of the filtration barrier.In the tubular apparatus, the outer diameter of proximal convoluted tubules increased to 56.3 ± 0.74 μm, and that of distal tubules to 41.1 ± 0.39 μm. However, epithelial height significantly decreased: 14.1 ± 0.24 μm in proximal tubules and 8.6 ± 0.17 μm in distal tubules, indicating pronounced epithelial atrophy and reduced reabsorptive activity.[10] The total number of glomeruli remained relatively unchanged (30.2 ± 0.63 thousand vs. 30.6 ± 0.36 thousand in control), but their density significantly decreased: 134.8 ± 1.89/mm^3 in the cortical layer and 86.8 ± 0.89/mm^3 in the whole kidney. This reflects a relative increase in interstitial components. The cortical thickness decreased to 2.9 ± 0.04 mm, while the medullary thickness increased to 4.1 ± 0.05 mm, indicating redistribution of structural components within the renal parenchyma.[11] Thus Figure 1. combined micronutrient deficiency in 6-month-old rats led to marked narrowing of renal arterioles, glomerular hypertrophy with Bowman's space expansion, thickening of the basement membrane, tubular epithelial atrophy, reduced glomerular density, and significant remodeling of cortical and medullary structures.[12]

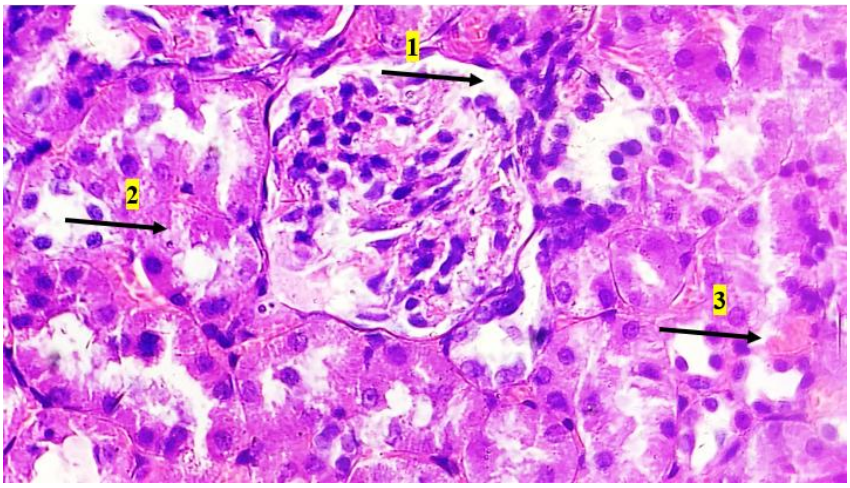

Figure 1. Microscopic image of the kidney of a 6-month-old rat under combined deficiency conditions. Hematoxylin and eosin staining. Obj. 20×, Oc. 40×. 1 — Expansion of Bowman's space and thickening of the basement membrane; 2 — Dystrophic changes in the epithelium of proximal tubules;

3 — Interstitial changes: focal edema and diapedetic hemorrhages.

The Figure 2. most sensitive structures were the proximal tubules (epithelial height decreased by approximately 25%) and glomerular density (reduced by 16%), indicating a deeper level of morphological decompensation compared to isolated micronutrient deficiencies.

Morphometric analysis of the testes demonstrated that combined micronutrient deficiency induces pronounced and complex remodeling affecting both the spermatogenic epithelium and the interstitial component. The thickness of the tunica albuginea increased to 138.4 ± 4.17 μm, indicating marked fibrotic remodeling. The number of interstitial areas increased to 17.5 ± 0.48, while the interstitial tissue area expanded by 21.6% to 5.6 ± 0.20 $\times 10^5$ μm^2, reflecting a significant expansion of the stromal component and a relative reduction of the parenchymal compartment.[13]

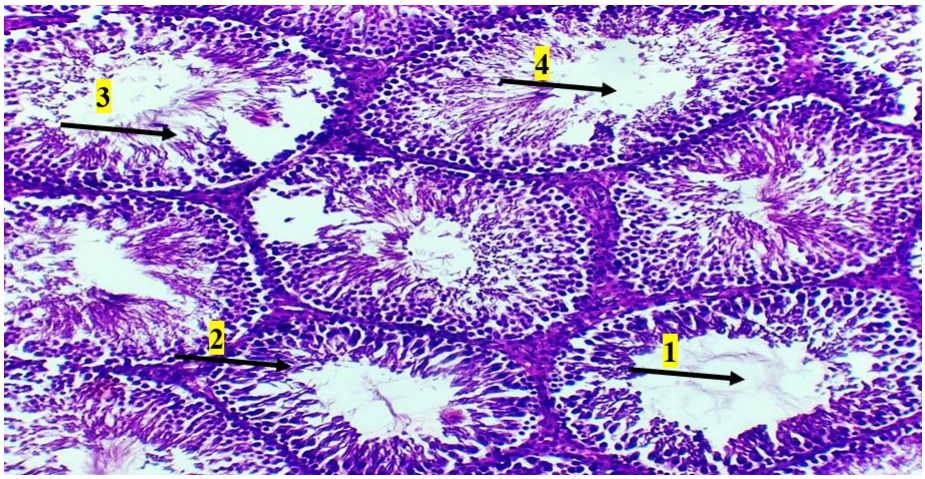

Figure 2. Microscopic image of the testis of a 6-month-old albino rat from the experimental group under combined deficiency conditions. Hematoxylin and eosin staining. Obj. 20×, Oc. 4×. 1 – Pronounced dilatation of seminiferous tubules accompanied by widening of their lumens; 2 – Thinning of the spermatogenic epithelium and reduction of cellular density; 3 – Decrease in the number of germinal elements and disruption of their layered organization; 4 – Marked reduction of mature spermatozoa within the tubular lumen.

The number of seminiferous tubules per field of view decreased to 9.1 ± 0.36 (−11.3%), which is associated with structural reorganization. The cross-sectional area of the tubules remained relatively unchanged (5.9 ± 0.18), but their internal architecture underwent pronounced alterations. The tubular

lumen area increased by 25.2% to 10.5 ± 0.33, accompanied by marked thinning of the spermatogenic epithelium. The area of the spermatogenic epithelium decreased by 14.1% to 3.9 ± 0.11, while its thickness declined to 58.6 ± 2.04 μm (−34.0%), representing one of the most pronounced indicators of degenerative changes.

The number of Sertoli cells moderately decreased to 9.2 ± 0.29 (−5.9%), indicating partial impairment of supporting function. The number of spermatogonia decreased by 19.4% to 10.5 ± 0.32, and their area decreased by 5.4%, indicating suppression of the proliferative compartment. The number of spermatocytes decreased by 28.7% to 33.9 ± 1.21, reflecting significant disruption of meiotic processes. The most pronounced changes were observed in the post-meiotic stages: the number of round spermatids decreased by 40.4% to 141.6 ± 3.88. The number of mature spermatozoa decreased by 34.4% to 261.4 ± 9.87, indicating a marked reduction in the final efficiency of spermatogenesis.[14]

The Table 1. number of Leydig cells decreased to 20.9 ± 0.37 (−24.3%), indicating a significant decline in steroidogenic function.

Johnsen score for the combined deficiency group (final evaluation):

Age	Experimental group	Morphological description of spermatogenesis	Balls
6 months	Combined deficiency (3 months)	Spermatogenesis is markedly suppressed; late stages are still partially preserved, and spermatozoa are present in the lumen. However, there is pronounced thinning of the spermatogenic epithelium, a significant reduction in spermatocytes, round spermatids, and mature spermatozoa, along with evident interstitial remodeling.	6,9–7,8 (7,4)

Combined micronutrient deficiency is characterized by pronounced thinning of the spermatogenic epithelium, a substantial reduction in meiotic and post-meiotic germ cell populations, a sharp decrease in mature spermatozoa, expansion of the interstitial tissue, a reduction in Leydig cell numbers, and marked fibrotic remodeling of the tunica albuginea. The observed alterations indicate severe suppression of spermatogenesis affecting all differentiation stages, particularly the meiotic and post-meiotic phases.[15]

Conclusion

Thus, the combined deficiency of Zn, Se, Mg, and Fe trace elements exerted a synergistic damaging effect on rat testes, manifested by pronounced dystrophic changes of the spermatogenic epithelium, reduced function of androgen-producing Leydig cells, and expansion of the interstitial stromal component. The observed morphological and morphometric alterations were significantly more severe compared to isolated deficiencies of individual elements, indicating a multifactorial mechanism of damage in reproductive tissue

REFERENCES

[1] A. S. Prasad, "Zinc: An overview," *Nutrition*, vol. 24, pp. 961–965, 2013.

[2] World Health Organization, *Trace Elements in Human Nutrition and Health*. Geneva, Switzerland: WHO, 2020.

[3] P. I. Oteiza, "Zinc deficiency and oxidative stress," *Free Radical Biology & Medicine*, vol. 53, pp. 1748–1754, 2012.

[4] M. Valko, H. Morris, and M. T. Cronin, "Metals, toxicity and oxidative stress," *Current Medicinal Chemistry*, vol. 12, pp. 1161–1208, 2016.

[5] A. C. Guyton and J. E. Hall, *Textbook of Medical Physiology*. Philadelphia, PA, USA: Elsevier, 2016.

[6] W. Maret, "Zinc biochemistry: From a single zinc enzyme to a key element of life," *Advances in Nutrition*, vol. 4, pp. 82–91, 2017.

[7] R. J. Aitken and S. D. Roman, "Antioxidant systems and oxidative stress in the testes," *Molecular Reproduction and Development*, vol. 75, pp. 169–180, 2008.

[8] A. Fallah, A. Mohammad-Hasani, and A. H. Colagar, "Zinc is an essential element for male fertility," *International Journal of Reproductive BioMedicine*, vol. 16, pp. 385–392, 2018.

[9] K. M. Hambidge and N. F. Krebs, "Zinc deficiency: A special challenge," *Journal of Nutrition*, vol. 137, pp. 1101–1105, 2007.

[10] N. F. Krebs, "Update on zinc deficiency and excess in clinical pediatric practice," *Annals of Nutrition and Metabolism*, vol. 64, pp. 1–10, 2014.

[11] J. King, "Zinc: An essential but elusive nutrient," *American Journal of Clinical Nutrition*, vol. 94, no. 2, pp. 679–684, 2011.

[12] R. S. Gibson, "Zinc deficiency and human health: Etiology, health consequences, and future solutions," *Plant and Soil*, vol. 361, pp. 291–299, 2012.

[13] S. J. Fairweather-Tait et al., "Zinc in human health and disease," *Nutrition Research Reviews*, vol. 24, no. 2, pp. 176–188, 2011.

[14] A. H. Colagar and E. T. Marzony, "Zinc levels in seminal plasma are associated with sperm quality in fertile and infertile men," *Nutrition Research*, vol. 29, pp. 82–88, 2009.

[15] International Zinc Association, *The Importance of Zinc in Human Health*. Brussels, Belgium: IZA, 2019.