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E-CADHERIN EXPRESSION IN KIDNEY TISSUE IN DIFFERENT TYPES OF ALIMENTARY MICROELEMENTOSIS

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Abstract.

Alimentary microelementosis – a condition arising from dietary deficiency of essential trace elements – is prevalent worldwide and has been associated with impaired cellular adhesion, compromised structural integrity of renal tissue, and progressive nephropathy. E-cadherin, the principal adhesion glycoprotein encoded by CDH1, plays a pivotal role in preserving the integrity of the renal tubular epithelium.

Objective. *To evaluate changes in E-cadherin expression in renal tissue of rats with various forms of dietary trace element deficiency (magnesium, iron, zinc, selenium, and their combination) using immunohistochemistry*

Materials and methods. *Ninety male outbred white rats (age 12-24 weeks; body weight 120 ± 11.3 to 215 ± 8.4 g) were divided into six groups: a control group maintained on a standard diet and five experimental groups fed ALTROMIN (Germany) diets with defined single or combined deficiencies of magnesium, iron, zinc, and selenium for 12 weeks. Immunohistochemical analysis was performed using a Ventana Benchmark XT system (Roche) with an anti-E-cadherin antibody, and expression was quantified using QuPath 0.5.1 software (H-score, percentage of positive cells). All experiments were conducted in accordance with the International Guidelines for Biomedical Research Involving Animals (EEC, Strasbourg, 1985), the European Convention for the Protection of Vertebrate Animals (ETS No. 123, Strasbourg, 1986), the Guide for the Care and Use of Laboratory Animals (ILAR, DELS), and Directive 2010/63/EU. Statistical analysis was performed using Microsoft Excel and IBM SPSS Statistics v.23, employing both parametric and non-parametric methods. Statistical analysis was performed using Microsoft Excel and IBM SPSS Statistics v.23, employing both parametric and non-parametric methods. Between-group comparisons were conducted using the Mann–Whitney U test and Student's t-test. Statistical significance was defined as $p < 0.05$. This study was conducted as part of the institutional research plan of Bukhara State Medical Institute entitled «Development of new approaches to early diagnosis, treatment, and prevention of pathological conditions affecting the health of the population of the Bukhara region after COVID-19 (2022-2026).»*

Results. *In the control group, renal tubular epithelial cells exhibited stable, high-level E-cadherin expression. Each form of trace element deficiency resulted in significantly decreased E-cadherin expression, epithelial disorganization, and weakened intercellular adhesion. The most pronounced reduction was observed in the combined deficiency group, where the percentage of E-cadherin–positive cells decreased from 28.1% in the control group to 14.6% in the combined deficiency group.*

Conclusions. *Alimentary microelementosis induces quantifiable alterations in E-cadherin expression in rat kidney tissue, exhibiting a cumulative detrimental effect in combined deficiency. These alterations represent early immunomorphological markers of nephron epithelial instability and may hold diagnostic and preventive significance for nutrition-associated renal diseases.*

Keywords: *E-cadherin; Kidneys; Micronutrient Deficiency; Magnesium; Iron; Selenium; Zinc; Immunohistochemistry; Alimentary Microelementosis; Epithelial-Mesenchymal Transition.*

Introduction

Micronutrient deficiency remains a highly prevalent nutritional problem worldwide, affecting an estimated one-third of the global population [1]. Data from the Global Burden of Disease 2019 study indicate that while the age-standardised prevalence of iron, vitamin A, and iodine deficiencies has declined since 1990, zinc, selenium, and magnesium inadequacies remain substantial, particularly in low- and middle-income countries [2]. Estimates from a pooled analysis of population-representative surveys show that 56% of preschool-aged children and 69% of non-pregnant women of reproductive age globally exhibit a deficiency in at least one core micronutrient (iron, zinc, vitamin A, or folate) [3]. The kidneys are highly susceptible to trace element imbalance, serving as the principal site of micronutrient homeostasis and a primary target organ for deficiency-related damage [4].

Micronutrient inadequacies, defined as intake below the Estimated Average Requirement, remain common in the United States despite high national income levels. National NHANES data indicate that magnesium is among the most under-consumed nutrients across all age groups, with inadequate intake reported in over 45% of Americans [5]. Iron deficiency affects approximately 17-18% of pregnant women in the third trimester and remains a leading contributor to anaemia in chronic kidney disease (CKD), exhibiting an estimated prevalence of 24-85% among patients, varying by CKD stage [6, 7]. Surveys of zinc and selenium status consistently reveal suboptimal intake in elderly and low-income populations, while zinc deficiency has been identified as a potential driver of the detrimental cycle linking CKD and hypertension [8].

European epidemiological data highlight significant north-to-south and east-to-west gradients in micronutrient status. Selenium levels in many parts of Europe are notably lower than those in the United States, with Eastern Europe demonstrating a lower average selenium intake than Western Europe [9]. Kidney cancer mortality, which is closely linked to trace element status, exceeds 50,000 deaths annually in Europe, while Mendelian randomisation analyses have confirmed a protective causal effect of serum zinc against malignant kidney neoplasms in European populations (OR 0.86; 95% CI 0.78-0.94; $p = 0.0016$) [10]. A prospective European cohort study of 261 patients with CKD

demonstrated that serum zinc and selenium levels exhibited independent positive correlations with renal function (β = 24.3 ± 8.6 and 60.3 ± 21.9, respectively; $p < 0.01$), whereas their deficiency was associated with progression to end-stage renal disease [11]. Hypomagnesaemia has been identified in approximately 15% of non-dialysis patients with CKD, even in stages G4–G5, with tubular dysfunction and interstitial fibrosis implicated as the primary causes of impaired magnesium reabsorption [12].

In Ukraine, iron deficiency anaemia and CKD-associated micronutrient depletion constitute a significant clinical burden. As the most prevalent nutritional deficit, iron deficiency contributes substantially to anaemia in CKD, a condition associated with increased mortality and reduced quality of life [13]. Nutritional surveys indicate that zinc intake in the Ukrainian adult population frequently falls below recommended levels, while selenium status mirrors the regional Central and Eastern European trend of moderate-to-severe dietary inadequacy. Inadequate magnesium intake has been linked to the progression of renal fibrosis through inflammatory and pro-oxidant pathways [14]. Such findings underscore the relevance of investigating alimentary microelementosis as a modifiable risk factor for kidney disease in this region.

Micronutrient deficiency represents a major public health challenge across Central Asian countries. Studies from Kazakhstan indicate a high prevalence of vitamin D, iron, and zinc deficiencies among children in urban areas, accompanied by marked regional and social inequalities in access to micronutrient-rich foods [15]. A nationally representative nutrition survey conducted in Uzbekistan in 2017 reported that nearly 50% of non-pregnant women exhibited iron depletion, with the prevalence of anaemia reaching 41% – figures that have remained largely unchanged despite a national wheat flour fortification programme [16]. Zinc deficiency has been identified as a widespread complication of iron-deficiency states, whereas selenium inadequacy, characteristic of the region's selenium-poor soils, contributes to impaired antioxidant defence in renal tubular epithelial cells [16, 17, 18]. Such regional data highlight the necessity of investigating the morphological consequences of alimentary microelementosis in experimental models.

Pathophysiological role of trace elements in renal tissue integrity. The kidneys play a central role in maintaining trace element homeostasis through regulated tubular reabsorption, yet remain vulnerable to the consequences of micronutrient imbalance [4]. Magnesium serves as a cofactor in over 300 enzymatic reactions; consequently, its deficiency impairs mitochondrial function, promotes reactive oxygen species (ROS) generation, and activates pro-inflammatory and pro-fibrotic pathways, including TGF-β and NF-κB signalling, ultimately leading to the disorganisation of renal tubular epithelial architecture [19, 20]. Iron is essential for erythropoiesis, hypoxia signalling, and mitochondrial function in renal cells; thus, the disruption of iron homeostasis is associated with the activation of HIF-1α-dependent pathways that promote epithelial-mesenchymal transition (EMT) in renal tubules [21]. Zinc deficiency has been directly associated with glomerulosclerosis, renal interstitial fibrosis, downregulation of E-cadherin, and upregulation of EMT markers (N-cadherin, vimentin, and SNAIL) in rat kidneys, a process mediated through Wnt/β-catenin and HIF-1α/PI3K signalling [22, 23]. Selenium exerts its function through selenoproteins, primarily glutathione peroxidase (GPX) and thioredoxin reductase (TrxR), to protect renal tubular cells from oxidative damage; consequently, selenium deficiency in experimental models induces significant proteinuria, glucosuria, proximal tubular injury, and decreased GPX activity [24, 25].

E-cadherin as a marker of renal tubular integrity. E-cadherin, a transmembrane glycoprotein of the cadherin superfamily encoded by the *CDH1* gene, serves as the principal mediator of calcium-dependent intercellular adhesion. Renal E-cadherin is abundantly expressed in the distal tubules, collecting ducts, and medullary segments, maintaining epithelial polarity and intercellular cohesion [26 The loss or dysfunction of E-cadherin represents a hallmark early event in tubular EMT, directly triggering this transition via matrix metalloproteinase-mediated ectodomain shedding, nuclear translocation of β-catenin, and transcriptional repression by Slug/Snail [27, 28]. E-cadherin suppression has been documented in models of renal fibrosis, hypoxia-induced EMT, zinc and iron deficiencies, and combined micronutrient depletion [22, 23, 29, 30]. Homeostasis of essential trace elements, encompassing absorption, storage, and renal excretion, is closely coupled to the structural and functional stability of tubular epithelial cells [31].

These data demonstrate the critical importance of investigating the morphological consequences of alimentary microelementosis in experimental models, alongside the relevance of E-cadherin as a quantitative immunomorphological marker of nephron epithelial integrity.

Objective

To evaluate the renal morphological structure across various forms of alimentary microelementosis in an experimental model using immunohistochemistry.

Materials and Methods

Ninety male outbred white rats (aged 12-24 weeks) with body weights ranging from 120 ± 11.3 g at baseline to 215 ± 8.4 g at study completion were included in this experimental study. The animals were housed in the vivarium of Bukhara State Medical Institute.

All experiments were conducted in accordance with the International Guidelines for Biomedical Research Involving Animals (EEC, Strasbourg, 1985), the European Convention for the Protection of Vertebrate Animals (ETS No. 123, Strasbourg, 1986), the Guide for the Care and Use of Laboratory Animals (ILAR, DELS), and Directive 2010/63/EU. The study was conducted with the approval of the Ethics Committee of Bukhara State Medical Institute (No. 12031).

A specialized certified diet (ALTROMIN Spezialfutter GmbH & Co., Germany; Certificate No. 36/2024) was utilized for the experiment. Each group was administered 10-mm pellets dispensed in 5-kg buckets (Table 1).

Table 1

Diet Types Used in the Experiment

No	Name	Purpose
1	C1000	Standard Diet
2	C1035	Magnesium Deficiency Diet
3	C1038	Iron Deficiency Diet
4	C1040	Zinc Deficiency Diet
5	C1045	Selenium Deficiency Diet
6	C1035 (Modified)	Combined (Magnesium, Iron, Zinc, Selenium) Deficiency Diet

The experimental groups were established concurrently and were comparable regarding age, body weight, housing, and dietary regimens Experimental models of nutritional deficiency were established according to the protocols of Qiao et al. [32] and Liu et al. [33], wherein a 12-week duration was sufficient to induce microelementosis.

The experiment comprised six groups: a control group (n=15) receiving a standard diet for 12 weeks; experimental group 1 (n=15) receiving a magnesium-deficient diet; group 2 (n=15) receiving an iron-deficient diet; group 3 (n=15) receiving a zinc-deficient diet; group 4 (n=15) receiving a selenium-deficient diet; and group 5 (n=15) receiving a combined (Mg+Fe+Zn+Se) deficient diet; all dietary interventions were maintained for 12 weeks. All animals were provided ad libitum access to their assigned diets and distilled water.

Upon completion of the experimental period, the animals were fasted prior to morning euthanasia. All procedures during euthanasia and necropsy were performed in strict adherence to biosafety regulations and ethical principles.

For immunohistochemical analysis, serial 3-µm-thick sections were deparaffinized, rehydrated, and immunostained using an automated Ventana Benchmark XT system (Roche, Switzerland). E-cadherin (the *CDH1* gene product, a membrane glycoprotein of the cadherin superfamily, and an epithelial adhesion protein) was utilized as the primary marker. Protein expression was quantified using QuPath 0.5.1 software, with the H-score and the percentage of positive cells automatically calculated for each tissue section.

The study was conducted in accordance with the principles of the Declaration of Helsinki (WMA, 2013 revision) and Directive 2010/63/EU. The protocol was approved by the Ethics Committee of the Bukhara State Medical Institute (Protocol No. 12031).

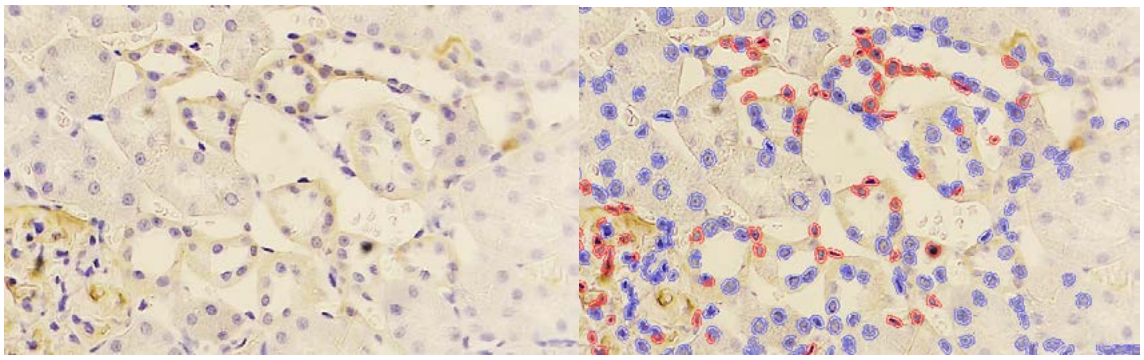
Data were analyzed using both parametric and non-parametric methods. Primary data were collected, verified, and systematized using Microsoft Office Excel 2010. Statistical analyses were performed using IBM SPSS Statistics v.23.

This study was conducted as part of the research plan of the Bukhara State Medical Institute (05.2024 DSc.217) entitled «Development of new approaches to early diagnosis, treatment, and prevention of pathological conditions affecting the health of the population of the Bukhara region after COVID-19 (2022-2026)». This work was executed as part of the approved research plan of the Bukhara State Medical Institute (05.2024 DSc.217).

Results and Discussion

E-cadherin expression in the renal tissue of 6-month-old white outbred rats was evaluated by immunohistochemistry in animals maintained on a standard diet and in those subjected to various dietary micronutrient deficiencies.

Control group. In animals maintained on a standard diet, renal tubular epithelial cells exhibited stable, high-level E-cadherin expression (total cells: 267; positive cells: 75; positive expression: 28.1%; total area: 707,518 px²) (Figure 1).

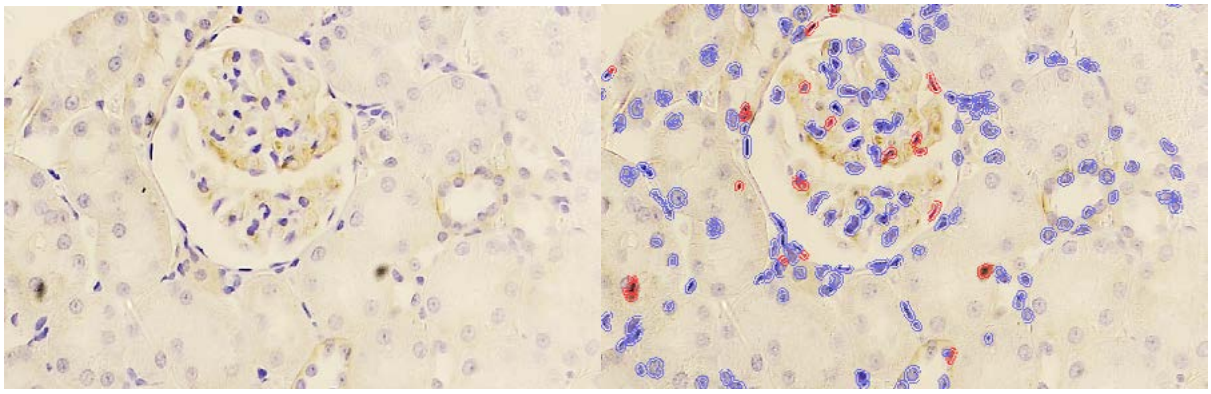


Total number of cells detected	267
Positive cells	75
Negative cells	192
Positive expression	28.1%
Total area	707518 px^2

Figure 1. Immunohistochemical evaluation of E-cadherin expression in the renal tissue of 6-month-old white outbred rats (control group). DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

Micronutrient deficiency was associated with decreased E-cadherin expression, epithelial disorganisation, and weakened intercellular adhesion, indicating the development of morphological alterations in the renal tissue.

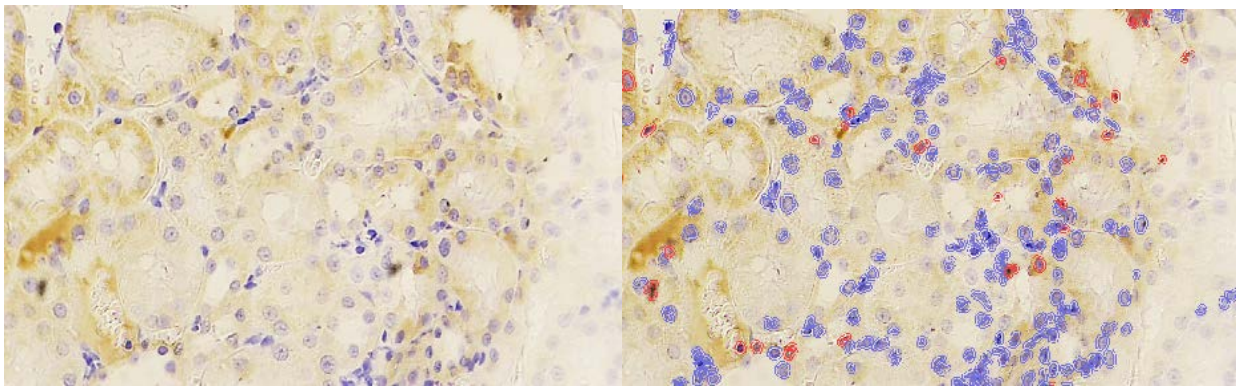
Magnesium deficiency. The magnesium-deficient group exhibited a significant reduction in E-cadherin expression within the renal tubular epithelium. Immunohistochemical analysis revealed diminished membranous staining with an uneven, fragmented distribution across certain areas, reflecting impaired intercellular adhesion, epithelial disorganisation, and dystrophic alterations (total cells: 185; positive cells: 39; positive expression: 21.1%; total area: 709,632 px²) (Figure 2).



Total number of cells detected	185
Positive cells	39
Negative cells	148
Positive expression	21.1%
Total area	709632 px^2

Figure 2. Immunohistochemical evaluation of E-cadherin expression in the renal tissue following magnesium deficiency. DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

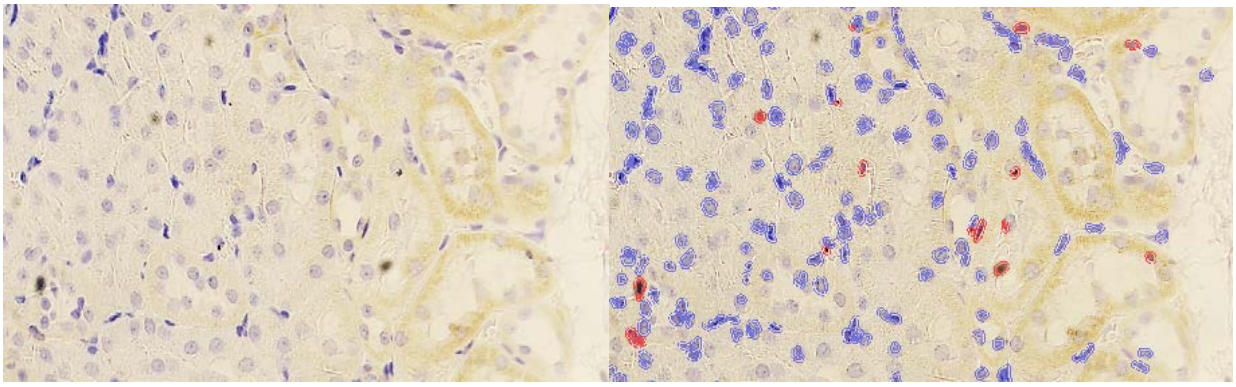
Iron deficiency. Iron deficiency was characterized by decreased E-cadherin expression in renal tubular epithelial cells, manifesting as reduced membranous staining intensity, heterogeneous distribution, and focal fragmentation. These morphological alterations reflected impaired intercellular adhesion, epithelial disorganisation, and dystrophic changes (total cells: 318; positive cells: 77; positive expression: 24.2%; total area: 872,544 px²) (Figure 3).



Total number of cells detected	318
Positive cells	77
Negative cells	243
Positive expression	24.2%
Total area	872544 px^2

Figure 3. Immunohistochemical evaluation of E-cadherin expression in the renal tissue following iron deficiency. DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

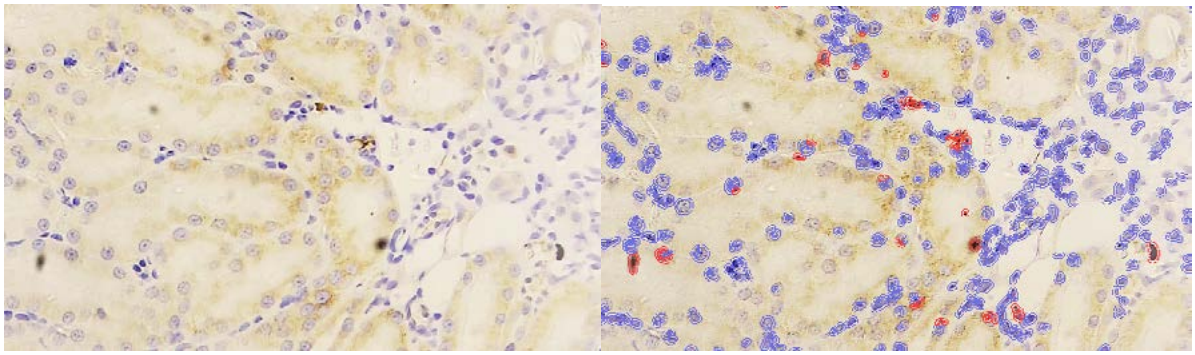
Selenium deficiency. The selenium-deficient group exhibited a significant reduction in E-cadherin expression within the renal tubular epithelium. Immunohistochemical analysis revealed diminished membranous staining with a discrete, heterogeneous pattern and focal loss of expression, reflecting weakened intercellular adhesion and epithelial disorganisation (total cells: 406; positive cells: 93; positive expression: 22.9%; total area: 858,711 px²) (Figure 4).



Total number of cells detected	406
Positive cells	93
Negative cells	333
Positive expression	22.9%
Total area	858711 px^2

Figure 4. Immunohistochemical evaluation of E-cadherin expression in the renal tissue following selenium deficiency. DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

Zinc deficiency. The zinc-deficient group exhibited a significant reduction in E-cadherin expression within the renal tubular epithelium, manifesting as diminished membranous staining, a heterogeneous and focal distribution, and focal fragmentation. These morphological alterations reflected impaired intercellular adhesion alongside dystrophic and degenerative changes (total cells: 216; positive cells: 53; negative cells: 166; positive expression: 24.5%; total area: 853,200 px²) (Figure 5).



Total number of cells detected	216
Positive cells	53
Negative cells	166
Positive expression	24.5%
Total area	853200 px^2

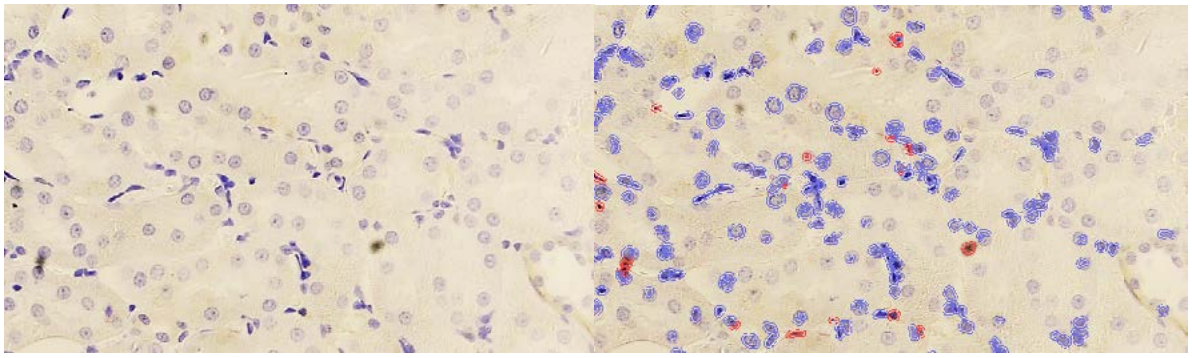
Figure 5. Immunohistochemical evaluation of E-cadherin expression in the renal tissue following zinc deficiency. DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

Combined (Mg+Fe+Se+Zn) deficiency. The combined deficiency group demonstrated a profound reduction in E-cadherin expression within the renal tubular epithelium, characterized by a marked diminution of membranous staining, a diffuse and non-linear distribution, and near-complete loss of expression in certain areas. Such alterations were accompanied by severe disruption of intercellular adhesion, epithelial disorganisation, and exacerbation of dystrophic and degenerative changes (total cells: 240; positive cells: 35; negative cells: 209; positive expression: 14.6%; total area: 839,592 px²) (Figure 6).

Relative to individual micronutrient deficiencies, combined deficiency exerted a more pronounced detrimental effect on the expression of adhesion molecules in the renal tissue (Figure 7).

These findings confirm that micronutrient imbalance compromises the morphofunctional stability of the nephron epithelium, holding significant scientific and clinical relevance for the early diagnosis and prevention of renal pathologies.

These results corroborate previous findings identifying E-cadherin as an essential mediator of renal epithelial cell adhesion. Yang et al. demonstrated that E-cadherin is abundantly expressed in the distal tubules, collecting ducts, and renal medulla of rats, highlighting its role in maintaining epithelial architecture and intercellular cohesion [26].



Total number of cells detected	240
Positive cells	35
Negative cells	209
Positive expression	14.6%
Total area	839592 px^2

Figure 6. Immunohistochemical evaluation of E-cadherin expression in the renal tissue following combined (Mg+Fe+Se+Zn) micronutrient deficiency. DAB chromogenic staining. ×200. Quantification performed using QuPath 0.5.1. E-cadherin–positive cells are highlighted in red.

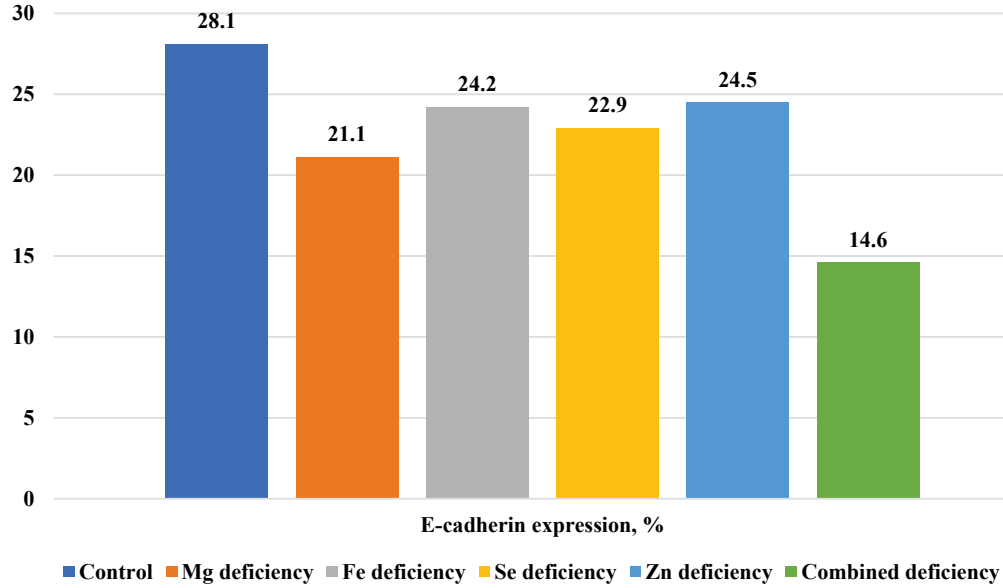


Figure 7. E-cadherin expression in control and experimental groups (%)

The reduction in E-cadherin levels observed across all micronutrient-deficient groups aligns with the established paradigm that E-cadherin loss or dysfunction is a critical driver of tubular epithelial-mesenchymal transition (EMT) and subsequent renal fibrosis. Zhang et al. demonstrated that E-cadherin disruption directly triggers EMT in renal tubular epithelial cells via Slug-mediated transcriptional repression [27], while Rabino et al. showed that E-cadherin signalling suppression promotes EMT and renal tubular fibrosis during hypoxia [28].

Our findings regarding zinc deficiency closely parallel those of Ume et al., who reported that zinc supplementation prevents high glucose-induced E-cadherin loss in renal tubular NRK-52E cells, whereas zinc deficiency exacerbates EMT-associated alterations [30]. Furthermore, recent experimental evidence confirms that zinc deficiency induces glomerulosclerosis and renal interstitial fibrosis via E-cadherin downregulation and Wnt3a/β-catenin pathway activation in rats [22].

Investigating iron deficiency in the HK-2 cell line, Zhang et al. showed that the disruption of intracellular iron homeostasis is associated with decreased E-cadherin levels and EMT activation via HIF-1α-dependent mechanisms, a finding consistent with the present observation of reduced E-cadherin expression in iron-deficient animals [29].

A recent review concluded that magnesium counteracts renal fibrosis by attenuating oxidative stress, inflammation, and pro-fibrotic signalling, suggesting that magnesium deficiency may compromise epithelial stability and diminish cell adhesion markers such as E-cadherin [19]. Similarly, selenium exerts nephroprotective and anti-apoptotic effects in experimental models of renal injury, wherein selenium-dependent antioxidant systems play a crucial role in maintaining tubular cell viability and conferring protection against EMT [24, 25].

The significantly more pronounced reduction observed in the combined deficiency group suggests that the simultaneous depletion of multiple micronutrients exerts

a cumulative or synergistic detrimental effect on epithelial adhesion, representing the earliest immunomorphological manifestation of nephron epithelial instability.

Conclusion

1. Dietary deficiencies of magnesium, iron, selenium, and zinc significantly reduce E-cadherin expression in the renal tubular epithelium of rats, resulting in disrupted intercellular adhesion and epithelial disorganisation.

2. The reduction in E-cadherin expression was proportional to the severity of micronutrient deficiency, decreasing from 28.1% in the control group to 21.1% (Mg), 24.2% (Fe), 22.9% (Se), 24.5% (Zn), and 14.6% in the combined deficiency group.

3. The combined deficiency of multiple trace elements exerts a cumulative detrimental effect on adhesion molecule expression in renal tissue, indicating synergistic impairment of nephron epithelial integrity.

4. These alterations represent early immunomorphological markers of nephron epithelial instability associated with nutritional imbalance, holding potential significance for the diagnosis and prevention of nutrition-related renal diseases.

Prospects for further research. Future investigations should include the assessment of additional EMT markers (N-cadherin, α-SMA, and vimentin) alongside E-cadherin to characterise the full spectrum of morphological alterations in alimentary microelementosis. Dose-response studies employing graded levels of individual and combined deficiencies, together with the evaluation of reversibility following micronutrient supplementation, would provide further insight into the clinical relevance of these findings. Extending the experimental model to female animals and various age groups would improve the generalisability of the results.

Author contributions. R. Baymuradov: study concept and design, experimental work, immunohistochemical analysis, image quantification using QuPath, statistical processing, manuscript preparation and final editing; Sh. Teshayev: scientific supervision, critical review of the methodology, interpretation of the results, and final approval of the manuscript. All authors have read and approved the final version of the manuscript.

Conflict of interest. The authors declare no conflict of interest regarding the preparation and publication of this article.

Use of artificial intelligence. Artificial intelligence was not used in the preparation of this manuscript.

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ЕКСПРЕСІЯ Е-КАДГЕРИНУ В ТКАНИНІ НИРОК ПРИ РІЗНИХ ВИДАХ АЛІМЕНТАРНОГО МІКРОЕЛЕМЕНТОЗУ

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Резюме.

Аліментарний мікроелементоз є поширеним станом, пов'язаним із порушенням клітинної адгезії та структурної цілісності ниркової тканини. Е-кадгерин – ключовий адгезивний глікопротеїн нефронного тубулярного епітелію – є чутливим маркером його ранніх ушкоджень.

Мета. Вивчити зміни експресії Е-кадгерину в нирковій тканині щурів за різних видів аліментарного мікроелементозу (дефіцит магнію, заліза, цинку, селену та їх поєднання) методом імуногістохімії.

Матеріали та методи. 90 самців білих щурів поділено на 6 груп: контроль і 5 дослідних (дефіцит Mg, Fe, Zn, Se, комбінований) – 12 тижнів на сертифікованих дієтах ALTROMIN. Імуногістохімічне дослідження (Ventana Benchmark XT; антитіла до

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Е-кадгерину), кількісна оцінка за H-score та % позитивних клітин (QuPath 0.5.1). Усі дослідження були виконані відповідно до Міжнародних настанов щодо біомедичних досліджень за участю тварин (EEC, Страсбург, 1985), Європейської конвенції про захист хребетних тварин (ETS № 123, Страсбург, 1986), Настанов з догляду та використання лабораторних тварин у біомедичних дослідженнях (ILAR, DELS) та Директиви 2010/63/EU. Статистичний аналіз: Microsoft Excel, IBM SPSS Statistics v.23; параметричні та непараметричні методи. Для міжгрупових порівнянь використовували критерій Манна–Вітні та t-тест Стьюдента. Статистично значущим вважали рівень $p < 0,05$. Дослідження виконані в рамках наукового плану Бухарського державного медичного інституту під назвою «Розробка нових підходів до ранньої діагностики, лікування та профілактики патологічних станів, що впливають на здоров'я населення Бухарського регіону після COVID-19 (2022-2026)».

Результати. Усі форми мікроелементного дефіциту призводили до зниження експресії Е-кадгерину: від 28,1% (контроль) до 21,1% (Mg), 24,2% (Fe), 22,9% (Se), 24,5% (Zn) і 14,6% при комбінованому дефіциті. Поєднаний дефіцит мав кумулятивний негативний ефект.

Висновки. Аліментарний мікроелементоз спричиняє кількісно вимірюване порушення експресії Е-кадгерину в нирковій тканині щурів з кумулятивним ефектом при комбінованому дефіциті. Ці зміни є ранніми імуноморфологічними маркерами нестабільності епітелію нефрону.

Ключові слова: Е-кадгерин; нирки; дефіцит мікроелементів; магній; залізо; селен; цинк; імуногістохімія; аліментарний мікроелементоз.

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