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Original Article

Renal mechanisms of calcium and phosphates homeostasis in rats at the early stages of alloxan-induced experimental diabetes mellitus under conditions of pharmacological blockade of the renin–angiotensin–aldosterone system

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Abstract

The objectives of the research were to study the peculiarities of the intrarenal mechanisms of calcium and phosphates homeostasis regulation against the background of pharmacological blockade of the intrarenal renin-angiotensin system at the early stages of alloxan-induced experimental diabetes mellitus. The experiments were carried out on 26 white non-linear mature male rats – 16 animals with 11-day long alloxan-induced experimental diabetes mellitus and 10 intact animals of the control. Pharmacological blockade of renin–angiotensin–aldosterone system was induced by captopril administration to 8 diabetic rats. The results of the present experimental study evidence that in addition to its well-known effects on intrarenal hemodynamics, the renin–angiotensin–aldosterone system plays an important role in the modulation of sodium-dependent tubular transport processes of calcium and phosphates. The increase of sodium-dependent excretion of calcium and phosphates in rats with 11-day alloxan-induced experimental diabetes mellitus is predominantly of a hyperdynamic–hyperperfusion nature. Limitation of sodium ions reabsorption is accompanied by a decrease in the intensity of sodium-dependent calcium and phosphates reabsorption processes. Despite the inability of pharmacological RAAS blockade to eliminate the hemodynamic-hyperperfusion consequences of hyperfiltration, compensatory-functional renal mechanisms of calcium and phosphates metabolism remain preserved at this stage of experimental alloxan-induced diabetes mellitus, ensuring effective maintenance of their homeostasis.

Keywords: alloxan, experimental diabetes mellitus, calcium, phosphates, renin–angiotensin–aldosterone system

Introduction

Since the kidneys are known to play a key role in the regulation of mineral metabolism, alterations in their functional state inevitably affect both the intensity and the pattern of calcium and phosphorus metabolism [1]. The assessment of renal mechanisms governing calcium and phosphates homeostasis becomes particularly relevant in diabetes mellitus (DM), when their imbalance results not only from dysregulation of calciotropic factors, but also from renal dysfunction in response to metabolic, hemodynamic, and dyscirculatory hyperglycemia-induced processes. Transtubular transport of calcium and phosphates in DM is characterized by

changes in the intensity of tubule-specific reabsorption of cations, including sodium-dependent one, and depends on the duration of the pathological process, as we have demonstrated previously under conditions of alloxan-induced hyperglycemia [2]. Consequently, the regulatory mechanisms involved in their initiation and progression require a thorough investigation, particularly at the early stages of diabetic renopathy development. One of the mentioned mechanisms is activation of the renin–angiotensin–aldosterone system (RAAS). The multifactorial impact of RAAS on the progression of renal pathology has been extensively studied by both national and international researchers, including those in the context of DM. Nevertheless, regardless numerous scientific evidences concerning the pathogenetic role of RAAS in the progression of diabetic kidney disease (DKD) [3, 4], the extent and nature of RAAS involvement in the regulation of calcium–phosphate metabolism at the early stages of DM development still require certain specification.

The objectives of the research were to study the peculiarities of the intrarenal mechanisms of calcium and phosphates homeostasis regulation against the background of pharmacological blockade of the intrarenal renin-angiotensin system at the early stages of alloxan-induced experimental diabetes mellitus (EDM).

Material and methods

The experiments were carried out on 26 white non-linear mature male rats, weighted 0.18–0.20 kg, kept under identical standard vivarium conditions. Experimental diabetes mellitus (EDM) was induced in 16 animals by a single intraperitoneal administration of diabetogenic dose of alloxan (Alloxan monohydrate, "Acros Organics", Belgium) – 160 mg/kg of the body weight – preceeded by 12-hour deprivation of food with preserved access to water *ad libitum*. 10 days after alloxan administration, the rats were withdrawn from the experiment (group 1). To determine the role of RAAS in the pathogenesis of renal dysfunction under diabetic conditions, an aqueous solution of the angiotensin-converting enzyme inhibitor (ACEi) captopril (Kaptopril, KRKA, Slovenia) was administered intraperitoneally to 8 alloxan-diabetic rats (group 2) at a dose of 10 mg/kg of the body weight on 11th day of the experiment [5]. Two hours after ACEi administration, all rats with alloxan-induced diabetes, as well as 10 intact control animals, were loaded with water at a volume of 5% of the body weight, urine was collected during 2 hours, after which animals were euthanized by decapitation under light ether anesthesia.

The level of glucose in the blood samples was determined by One Touch Ultra glucometer (LifeScan, USA) and further only the data of rats with persistent hyperglycemia exceeding 7.0 mmol/L were considered.

After assessment of water-induced 2-hour diuresis (in ml/100 g of the body weight for 2 hours), urine creatinine concentration (in a reaction with picric acid according to Folin's method) and plasma creatinine concentration (according to A.K.Merzon's method) were determined [6], GFR was calculated based on endogenous creatinine clearance [6]. The concentration of sodium in urine samples as well as in the renal cortex dissected after decapitation [7], was detected by the flame photometry method, the calcium urine content – by the intensity of coloration in the presence of o-cresolphthalein complexone, the level of phosphates in urine – by photometry of the phosphoromolybdate complex. The indices of electrolyte excretion were calculated, correlated with the unit of functioning nephron (its absolute values were calculated per 100 µl of glomerular filtrate [GF]) [6, 8], as well as the calculation of calcium-phosphorus, calcium- and phosphorus-creatinine ratios was made to assess the degree of calciuria and phosphaturia, sodium-calcium and sodium-phosphorus urinary ratios in urine in order to evaluate sodium-dependent mechanisms of tubular transport of cations.

The data obtained were statistically processed with determination of the mean value and standard errors. The non-parametric Mann-Whitney rank test, provided by the software "Statistica for Windows", "Version 8.0", was used to assess the probability of difference between the studied groups [9].

The research was carried out in compliance with the provisions of the EU Directive No. 609 (1986) and the Order of the Ministry of Health of Ukraine No. 690 of 09/23/2009 "On Measures to Further Improve Organizational Standards for Work with Experimental Animals".

Results and discussion

Analysis of intrarenal calcium and phosphates transport at the early stages of alloxan-induced EDM under conditions of captopril administration detected a decreasing tendency in the urinary calcium–phosphate ratio (by 32.8% compared to alloxan-diabetic rats of *group* 1, and by 23.6% in comparison with the control

animals), resulted both from a slight reduction in urinary calcium concentration and from an increase in urinary phosphorus concentration (Table 1). The tendency toward attenuation of the calciuric response of the kidneys of rats with 11-day alloxan-induced diabetes [2] persisted after pharmacological blockade of RAAS as well – calcium concentration in urine of alloxan-diabetic animals of *group* 2 was 16.5% lower than a corresponding index in rats of *group* 1 and 23.8% lower than control values. Despite a significant raise of diuresis and glomerular filtration rate (GFR) after pharmacological blockade of intrarenal RAAS (by 42.9% and 46.5%, respectively, compared to alloxan-diabetic rats of *group* 1) (Figure 1), and the consequently expected augmentation of the calcium filtration charge, there were no excessive urinary calcium excretion observed in rats at this stage of the experiment. Thus, the insignificant increase in calcium excretion after captopril

Table 1: Characteristics of renal mechanisms of calcium and phosphates homeostasis in rats with 11-day alloxane-induced experimental diabetes with underlying pharmacological blockade of RAAS (X±Sx).

Indices	Groups, number of animals		
	Control, n=10	11-day EDM (group 1), n=8	11-day EDM+captopril (group 2), n=8
Calcium concentration in urine, mmol/L	1.26±0.03	1.15±0.11 p>0.3	0.96±0.04 p<0.001 p_1>0.1
Excretion of calcium, μmol per 2 hours	3.72±0.24	3.99±0.49 p>0.6	4.69±0.42 p=0.05 p_1>0.2
Standardized excretion of calcium, μmol/100 ml of GF	1.05±0.04	0.84±0.14 p>0.1	0.65±0.07 p<0.001 p_1>0.2
Phosphates concentration in urine, mmol/L	4.64±0.48	4.34±0.48 p>0.6	4.65±0.63 p>0.9 p_1>0.6
Excretion of phosphates, μmol per 2 hours	13.48±1.44	14.92±1.76 p>0.5	21.95±2.57 p<0.01 p_1<0.05
Standardized excretion of phosphates, μmol/100 ml of GF	3.86±1.44	3.09±0.50 p>0.2	3.12±0.50 p>0.2 p_1>0.9
Calcium-phosphorus ratio in urine, un.	0.304±0.038	0.283±0.037 p>0.8	0.229±0.026 p>0.1 p_1>0.3
Calcium-creatinine ratio in urine, un.	1.34±0.05	0.77±0.12 p<0.01	0.70±0.09 p<0.001 p_1>0.7
Phosphorus-creatinine ratio in urine, un.	4.93±0.50	2.82±0.34 p<0.05	3.33±0.59 p>0.09 p_1>0.5
Sodium-calcium ratio in urine, un.	0.57±0.04	3.62±0.32 p<0.001	4.17±0.39 p<0.001 p_1>0.4
Sodium-phosphorus ratio in urine, un.	0.171±0.024	0.991±0.111 p<0.001	0.933±0.114 p<0.001 p_1>0.6

Note: Intergroup differences were assessed using the non-parametric Mann-Whitney test. p – probability of discrepancy of indices relative to control group; p_1 – probability of discrepancy of indices relative to group 1; n – number of animals.

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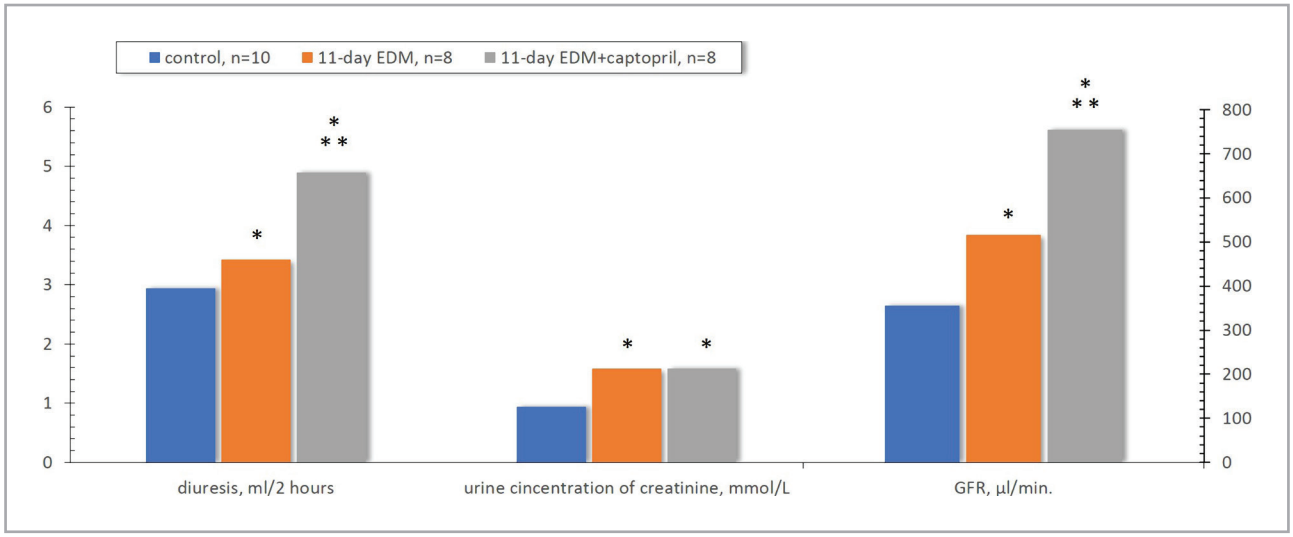

Figure 1: Characteristics of renal excretory function in rats with 11-day alloxan-induced experimental diabetes mellitus under conditions of pharmacological blockade of the RAAS. * – probability of discrepancy of indices relative to control group; ** – probability of discrepancy of indices relative to group 1.

administration to diabetic rats (by 17.5%) was not realized when the excretion rate was correlated with the unit of functioning nephron: calcium excretion standardized by the volume of GF decreased reliably under captopril effect (by 22.6% compared to the corresponding parameter in *group* 1 and by 38.1% in comparison with the control value). Moreover, a 10.0% reduction of the urinary calcium–creatinine ratio in rats with 11-day EDM after captopril administration accompanied by practically unchanged creatininuria intencity (Figure 1), indicates the involvement of tubular mechanisms in maintaining of calcium homeostasis in alloxan-diabetic rats at this stage of the experiment.

The proximal convoluted and straight tubules (reabsorbing 60–70% of filtered calcium), the thick ascending part of the Henle's loop (additional absorption of 20% of filtered calcium) and distal tubules (5–10% of calcium reabsorption) are known to be the main areas of calcium reabsorption. However, the tubular calcium transport and the nature of its reabsorption differs substantially in certain segments of the nephron [2, 10–15]. Calcium reabsorption in the proximal convoluted and straight tubules occurs predominantly via energy-independent transport, which is isosmotic – calcium, sodium and water are reabsorbed simultaneously [2, 16, 17]. The reabsorption of filtered calcium in the thick part of the Henle's loop occurs both as its passive transport through the intercellular spaces (in the medullar part) and via active transport mechanisms (in the cortical part). At the same time, calcium reabsorption in the distal convoluted tubules takes place entirely by active transcellular transport [18].

The latter begins with calcium entry to the tubular cell through calcium channels in the apical cellular membrane by potent concentration gradient and completes with the removal of calcium from the tubular cell across the basolateral membrane due to the cooperative function of Ca2+-ATPase and Na+/Ca2+ exchanger, which transports three sodium ions into the cells in exchange for one calcium ion from cells to the interstitium [2, 16, 19–21]. Considering the above, sodium-dependent mechanisms, in our opinion, play an important role in the processes of renal calcium handling.

Investigation of the role of sodium-dependent mechanisms in the processes of renal calcium homeostasis in rats with 11-day EDM demonstrated a 13.0% elevation of the urinary sodium–calcium ratio following captopril administration, attributable specifically to a reduction of calcium concentration in urine, since urinary sodium levels in rats with this duration of EDM remained practically unchanged under conditions of pharmacological RAAS blockade (Figure 2). Considering a decrease of proximal tubular sodium reabsorption by single nephrons (by 9.7%) in response to captopril administration and probable, parallel to sodium, limitation of proximal calcium reabsorption [2, 11, 13–16] it can be suggested that it is distal calcium transport mechanisms, including sodium-dependent ones, that provides calcium retention in the body.

Analysis of transtubular sodium and calcium transport under conditions of pharmacological RAAS blockade in rats with 11-day EDM demonstrated that increase in final urine volume and GFR, accompanied

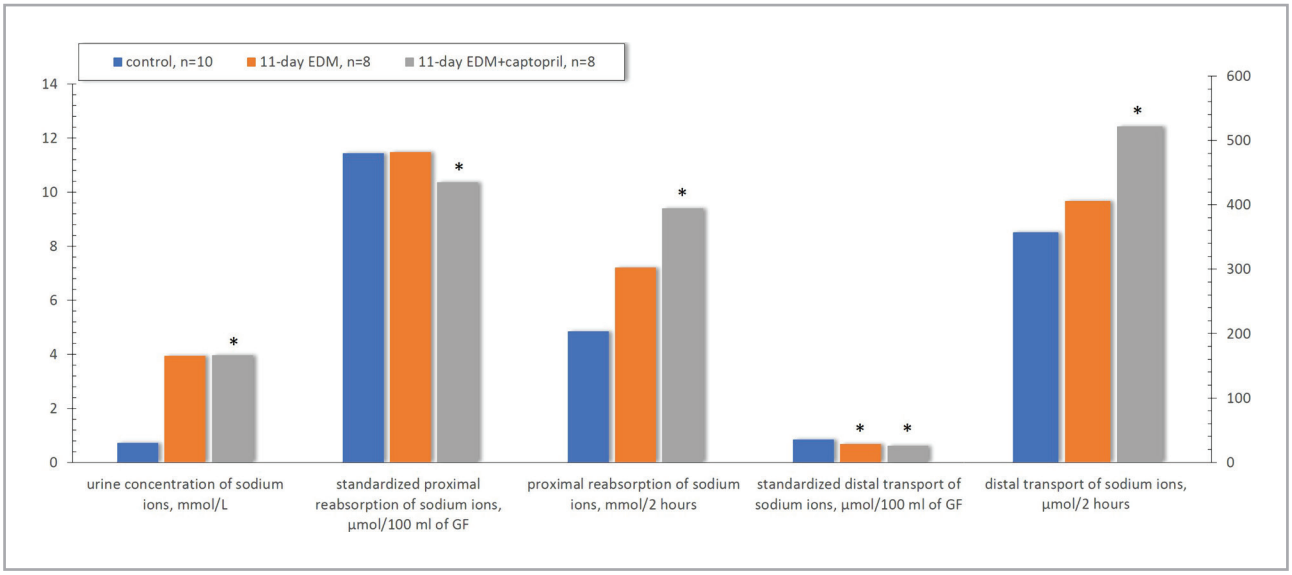

Figure 2: Characteristics of tubular sodium transport in rats with 11-day alloxan-induced experimental diabetes mellitus under conditions of pharmacological blockade of the RAAS. * – probability of discrepancy of indices relative to control group; ** – probability of discrepancy of indices relative to group 1.

by an elevated filtered sodium load of nephron, results in maximal tension of tubulo-tubular feedback and a relative inability of the distal tubules to ensure retention of sodium ions that have escaped reabsorption in the proximal tubules. Limitation of aldosterone-dependent distal sodium reabsorption under the action of captopril (Figure 2) leads to a reduction of intracellular sodium content and creates a favorable gradient for Na+/Ca2+ countertransport, thereby ensuring active calcium release from tubular epithelial cells and, consequently, preventing excessive urinary calcium loss (Table 1) [2, 17, 19, 21, 22].

This assumption is supported by a decrease in sodium ion content in the renal cortex of rats with 11-day alloxan-induced EDM under conditions of pharmacological RAAS blockade (it remained 2.1-fold lower than control values) (Figure 3). The absence of significant changes after captopril administration excludes the involvement of RAAS activation in the development of tubulointerstitial disturbances at the early stages of alloxan-induced EDM.

The study of renal mechanisms of phosphate homeostasis at the early stages of alloxan-induced EDM showed a significant increase in phosphorus excretion under conditions of pharmacological RAAS blockade (group 2), exceeding the value observed in alloxan-diabetic animals of group 1 (by 47.1%) and that of intact animals (by 62.8%) (Table 1). When correlated with the unit of functioning nephron, phosphate excretion in group 2 remained 19.2% lower than the corresponding control value and was practically unchanged after pharmacological RAAS blockade, indicating that the increased filtration charge of phosphate was causative for the enhanced phosphates excretion against the background of intensified diuresis following captopril administration.

This assumption is supported by the slight elevation of the urinary phosphorus–creatinine ratio in rats

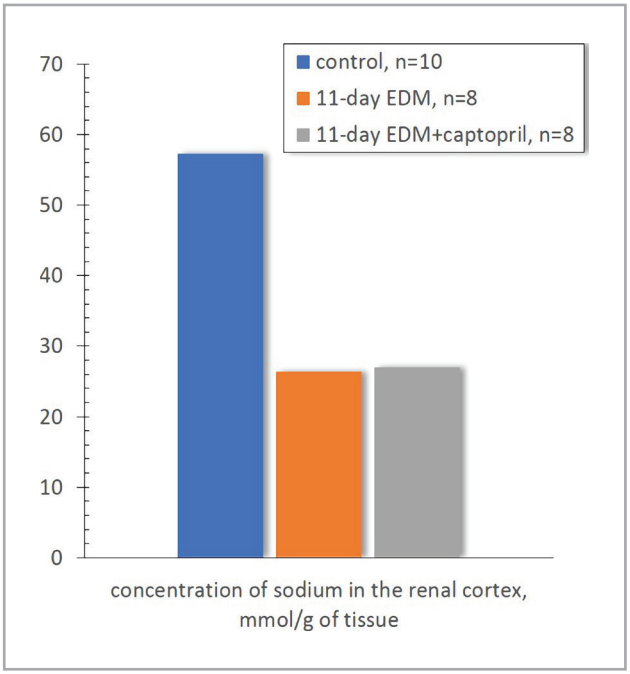

Figure 3: Sodium ions content in renal tissue of rats with 11-day alloxan-induced experimental diabetes mellitus under conditions of pharmacological blockade of the RAAS. * – probability of discrepancy of indices relative to control group; ** – probability of discrepancy of indices relative to group 1.

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with 11-day alloxan-induced EDM after captopril administration (by 15.3%), mainly due to a minor increase in urinary phosphates concentration accompanied by nearly unchanged creatininuria (Figure 1). Nevertheless, the phosphorus–creatinine ratio remained 48.1% lower than that of control rats.

Apparently, as the phosphates filtration load increases, the proximal tubular transport maximum is rapidly reached, phosphates reabsorption becomes saturated, and, as filtration exceeds the reabsorptive capacity of sodium–phosphate transporters, relative phosphate reabsorption decreases and urinary phosphate excretion increases. Thus, urinary phosphate concentration after captopril administration increased by 7.1% and nearly reached the level observed in intact rats.

On the one hand, this finding indicates sufficiently effective renal mechanisms of phosphates homeostasis at this stage of the experiment. On the other hand, it emphasizes their dependence on tubular sodium transport processes, since the urinary sodium–phosphorus ratio tended to decrease in rats with 11-day EDM (by 5.9% *vs*. pre-captopril values), proportionally to the reduction in proximal sodium reabsorption standardized by GF, while urinary sodium ion concentration remained unchanged under the influence of captopril.

Since only about 10% of filtered phosphates is reabsorbed in the distal tubules [1, 2, 15, 23–30], captopril administration and blockade of aldosterone-dependent sodium reabsorption processes had no substantial effect on renal phosphate homeostasis.

Conclusion

This study that, beyond its well-known effects on intrarenal hemodynamics, the renin–angiotensin–aldosterone system plays an important role in the modulation of sodium-dependent tubular transport processes of calcium and phosphates. The increase of sodium-dependent excretion of calcium and phosphates in rats with 11-day alloxan-induced experimental diabetes mellitus is predominantly of a hyperdynamic–hyperperfusion nature. Despite captopril-induced reduction in efferent arteriolar tone and a consequent decrease of intraglomerular pressure, diabetes-associated dilation of the afferent arteriole, accompanied by enhanced renal blood flow and renal hyperperfusion combined with augmented ultrafiltrate volume due to water-induced diuresis, leads to intensification of renal excretory function. Glomerular hyperfiltration, resulting in proximal tubular overload with ultrafiltrate containing high sodium ions concentrations, probably, causes exceeding of the reabsorptive capacity of the tubular segment of the nephron, reflected primarily on the proximal tubules and, due to functional tension of tubulo-tubular feedback, – on the distal tubules of the nephron as well. Limitation of sodium ions reabsorption is accompanied by a decrease in the intensity of sodium-dependent calcium and phosphates reabsorption processes. However, despite the inability of pharmacological RAAS blockade to eliminate the hemodynamic-hyperperfusion consequences of hyperfiltration, compensatory-functional renal mechanisms of calcium and phosphates metabolism remain preserved at this stage of experimental alloxan-induced diabetes mellitus, ensuring effective maintenance of their homeostasis. Further research is needed to clarify the role of RAAS activation in the progression of tubulointerstitial injury during hyperglycemia.

Conflicts of interests

The authors declare no conflict of interest.

References

1. Blaine J, Chonchol M, Levi M. Renal Control of Calcium, Phosphate, and Magnesium Homeostasis. Clin J Am Soc Nephrol 10: 1257-1272, 2015. doi: 10.2215/CJN.09750913.
2. Olenovych O, Gozhenko A, Tkach Ye. Peculiarities of transtubular transport of calcium and phosphates in the dynamics of the development of alloxan-induced experimental diabetes mellitus. Romanian Journal of Diabetes Nutrition and Metabolic Diseases 31(4):411-19, 2024. https://doi.org/10.46389/rjd-2024-1736.
3. Siragy HM, Carey RM. Role of the intrarenal renin-angiotensin-aldosterone system in chronic kidney disease. Am J Nephrol 31(6):541-50, 2010. doi:10.1159/000313363.
4. Triebel H, Castrop H. The renin angiotensin aldosterone system. Pflugers Arch - Eur J Physiol 476:705–13, 2024. https://doi.org/10.1007/s00424-024-02908-1.
5. Boychuk TM, Olenovych OA, Gozhenko AI. Method for modeling experimental blockade of the intrarenal renin–angiotensin system. Patent of Ukraine №146192, published January 28, 2021 (in Ukrainian).
6. Mahalias VM, Mikhieiev AO, Rohovyi YuYe et al. Modern methods of experimental and clinical studies of the central research laboratory of Bukovinian State Medical Academy. Medical Academy, Chernivtsi, 42p, 2001 (in Ukrainian).
7. Boychuk TM, Olenovych OA, Gozhenko AI. Method for diagnosis of tubulointerstitial syndrome. Patent of Ukraine №145836, published January 7, 2021 (in Ukrainian).
8. Schuck O. Examination of kidney function. Avicenum, Prague, 344p, 1981.

9. Korda MM, Kashuba OM. Basics of medical statistics and conducting computer statistical analysis of data with statistical programs. TNMU «Ukrmedknyha», Ternopil, 120p, 2021 (in Ukranian).
10. Friedman PA. Mechanisms of renal calcium transport. Exp Nephrol 8:343-50, 2000.
11. Jeon US. Kidney and calcium homeostasis. Electrolyte Blood Press 6(2):68–76, 2008. doi:10.5049/EBP.2008.6.2.68.
12. Lambers TT, Bindels RJ, Hoenderop JG. Coordinated control of renal Ca2+ handling. Kidney Int 69: 650-654, 2006.
13. Mensenkamp AR, Hoenderop JG, Bindels RJ. Recent advances in renal tubular calcium reabsorption. Curr Opin Nephrol Hypertens 15: 524-529, 2006.
14. Moor MB, Bonny O. Ways of calcium reabsorption in the kidney. Am J Physiol Renal Physiol 310:F1337-F1350, 2016. doi:10.1152/ajprenal.00273.2015.
15. Mount DB, Yu ASL. Transport of inorganic solutes: sodium, chloride, potassium, magnesium, calcium, and phosphate. In: Brenner & Rector's The Kidney, 8th ed; Brenner BM (ed). Philadelphia, Saunders, pp 185–192, 2008.
16. Blaustein MP, Lederer WJ. Sodium/calcium exchange: its physiological implications. Physiol Rev 79(3):763–854, 1999.
17. Bonny O, Edwards A. Calcium reabsorption in the distal tubule: regulation by sodium, pH, and flow. Am J Physiol Renal Physiol 304: F585-F600, 2013.
18. Loffing J, Loffing-Cueni D, Valderrabano V, et al. Distribution of transcellular calcium and sodium transport pathways along mouse distal nephron. Am J Physiol Renal Physiol 281: F1021-F1027, 2001.
19. Brini M, Calì T, Ottolini D, Carafoli E. The plasma membrane Ca^{2+} ATPase and the plasma membrane sodium/calcium exchanger cooperate in the regulation of cell calcium. Am J Physiol Cell Physiol 278:C661–C6, 2011.
20. Iwamoto T, Kita S. Hypertension, Na+/Ca2+ exchanger, and Na+, K+-ATPase. Kidney Int 69(12):2148-54, 2006. doi:10.1038/sj.ki.5000421. Epub 2006 Apr 26.
21. Matchkov VV, Gustafsson H, Rahman A, Briggs Boedtkjer DM, Gorintin S, Hansen AK et al. Interaction between Na+/K+-pump and Na+/Ca2+-exchanger modulates intercellular communication. Circ Res 100(7):1026-35, 2007. doi:10.1161/01.RES.0000262659.09293.56.
22. Weidong W, Chunling L et al. Reduced expression of renal Na+ transporters in rats with PTH–induced hypercalcemia. Am J Physiol Renal Physiol 286:534-45, 2004.
23. Biber J, Hernando N, Forster I. Phosphate transporters and their function. Annu Rev Physiol. 75:535-50, 2013. doi:10.1146/annurev-physiol-030212-183748.
24. Blaine J, Weinman EJ, Cunningham R. The regulation of renal phosphate transport. Adv Chronic Kidney Dis 18: 77-84, 2011.
25. Fenollar-Ferrer C, Forrest LR. Structural models of the NaPi-II sodium-phosphate cotransporters. Pflugers Arch - Eur J Physiol 471:43–52, 2019. https://doi.org/10.1007/s00424-018-2197-x
26. Hernando N, Forster IC, Biber J, Murer H. Molecular characteristics of phosphate transporters and their regulation. Exp Nephrol 8(6):366-75, 2000. doi:10.1159/000020691.
27. Levi M, Gratton E, Forster IC, Hernando N, Wagner CA, Biber J, et al. Mechanisms of phosphate transport. Nat Rev Nephrol 15(8):482–500, 2019. doi:10.1038/s41581-019-0159-y.
28. Marks J, DebnamES, Unwin RJ. Phosphate homeostasis and the renal-gastrointestinal axis. Am J Physiol Renal Physiol 299:F285-F296, 2010.
29. Murer H, Biber J. A molecular view of proximal tubular inorganic phosphate (Pi) reabsorption and of its regulation. Pflugers Arch 433(4):379-89, 1997. doi:10.1007/s004240050292.
30. Segawa H, Shiozaki Y, Kaneko I, Ken-ichi M. The Role of Sodium-Dependent Phosphate Transporter in Phosphate Homeostasis. J Nutr Sci Vitaminol 61 Suppl:S119-21, 2015. doi:10.3177/jnsv.61.S119. PMID: 26598821.

https://doi.org/10.46389/rjd-2026-2151