TIBBIYOT
TA'LIMI & INNOVATSIYALARI

O'ZBEKISTON RESPUBLIKASI
19 20
TOSHKENT TIBBIYOT AKADEMIYASI

Google scholar

PubMed MEDLINE

NIH U.S. National Library of Medicine National Institutes of Health | Advancing Health Through Knowledge



№11-SON

- 14.00.00 - Tibbiyot fanlari;
- 14.00.01 - Akusherlik va ginekologiya;
- 14.00.02 - Morfologiya;
- 14.00.03 - Endokrinologiya;
- 14.00.04 - Otorinolaringologiya;
- 14.00.05 - Ichki kasalliklar;
- 14.00.06 - Kardiologiya;
- 14.00.07 - Gigiena;
- 14.00.08 - Oftal'mologiya;
- 14.00.09 - Pediatriya;
- 14.00.10 - Yuqumli kasalliklar;
- 14.00.11 - Dermatologiya va venerologiya;
- 14.00.12 - Tibbiy reabilitologiya;
- 14.00.13 - Nevrologiya;
- 14.00.14 - Onkologiya;
- 14.00.15 - Patologik anatomiya;
- 14.00.16 - Normal va patologik fiziologiya;
- 14.00.17 - Farmakologiya va klinik farmakologiya;
- 14.00.18 - Psixiatriya va narkologiya;
- 14.00.19 - Klinik radiologiya;
- 14.00.20 - Tibbiy genetika;
- 14.00.21 - Stomatologiya;
- 14.00.22 - Travmatologiya va ortopediya;
- 14.00.23 - Hamshiralik ishini tashkil etish;
- 14.00.24 - Sud tibbiyoti;
- 14.00.27 - Xirurgiya;
- 14.00.28 - Neyroxirurgiya;
- 14.00.41 - Xalq tabobati;
- 14.00.35 - Bolalar xirurgiyasi;
- 14.00.34 - Yurak-qon tomir xirurgiyasi

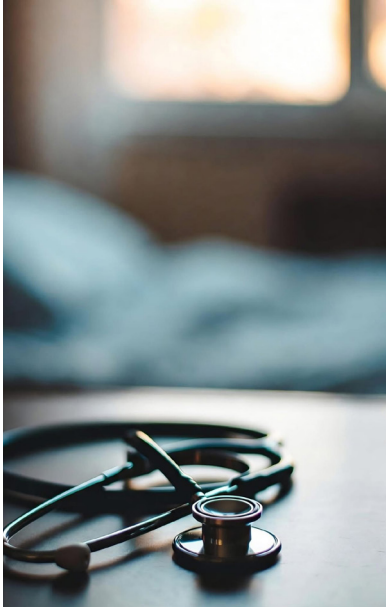
2026-yil, noyabr

tibbiyot-talimi-va-innovatsiyalari.uz

https://t.me/tibbbiyot_2100

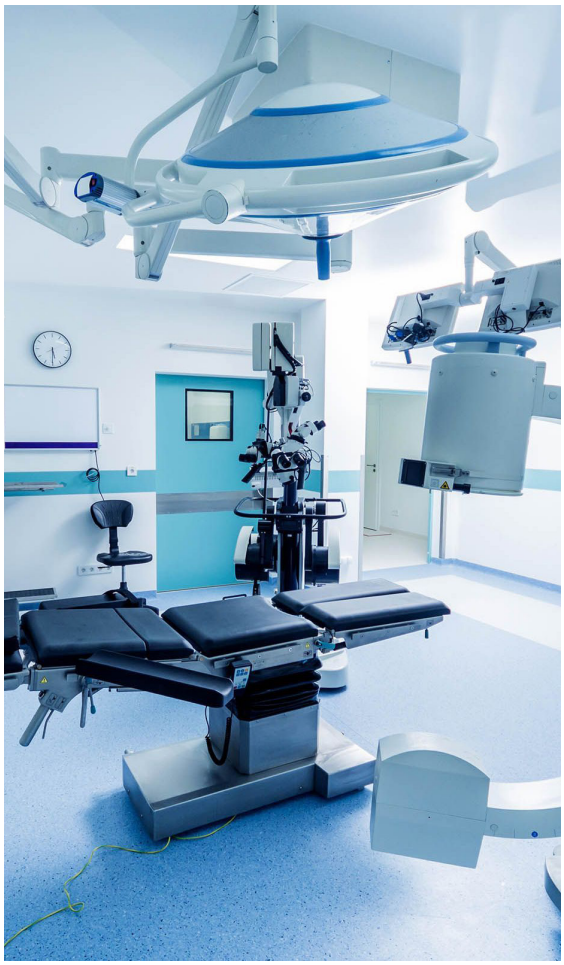
Litsenziya raqami: №745444













Tahrir hay’ati a’zolari:



BOSH MUHARRIR:
Shuhrat Baymuradov Abdujalilovich
tibbiyot fanlari doktori (DSc), professor



BOSH MUHARRIR O‘RINBOSARI:
Iminov Komiljon Odiljonovich
tibbiyot fanlari bo‘yicha falsafa doktori

“Tibbiyot ta'limi va innovasiyalari” ilmi elektron jurnali 29.04.2025-yildan O‘zbekiston Respublikasi Prezidenti Adminstratsiyasi huzuridagi Axborot va ommaviy kommunikatsiyalar agentligi tomonidan C-5669801 reyestr raqami tartibi bo‘yicha ro‘yxatdan o‘tkazilgan. Litsenziya raqami: №745444.

Tahrir hay’ati a’zolari:



Sharipov Kongratbay Avezimbetovich
Texnika fanlari doktori (DSc), professor



Ishanqulov Artiqboy Eshboyevich
Biologiya fanlari doktori (DSc), professor



Otamuradov Furqat Abdukarimovich
tibbiyot fanlari doktori (DSc), dotsent.



Shuxrat Jumayevich Teshayev
tibbiyot fanlari doktori (DSc), professor



Oral Aminovna Ataniyazova
tibbiyot fanlari doktori (DSc), professor



Jalolova Feruza Maxamatjanovna
Tibbiyot fanlari bo‘yicha falsafa doktori (DSc), dotsent



Uraqov Shokir Ulashovich
tibbiyot fanlari doktori doktori (DSc)

Aripkhodjaeva Shakhzoda
resident doctor in national medical center

"TIBBIYOT TA’LIMI VA INNOVATSIYALARI" ilmiy elektron jurnali O‘zbekiston Respublikasi Axborot va ommaviy kommunikatsiyalar agentligi (AOKA) tomonidan 2024-yil 9-oktabrdan boshlab c-5669651 raqami ostida rasmiy ro‘yxatdan o‘tkazilgan.

Murojaat uchun telefon:
+998 97 748 70 03
Email: @munis_sm

MUNDARIJA

ОЦЕНКА РЕЗУЛЬТАТОВ КОМПЛЕКСНОГО ЛЕЧЕНИЯ ОСТЕОМИЕЛИТА ВЕРХНЕЙ ЧЕЛЮСТИ У БОЛЬНЫХ САХАРНЫМ ДИАБЕТОМ 2 ТИПА 22
Махкамов Зохидхужа Хасанхон угли, Рузиматов Акмалбек Санжарбек угли, Аллаяров Абдулло Улугбек угли

ASSOCIATION OF VARICOCELE SEVERITY WITH SEMEN PARAMETERS IN INFERTILE MEN FROM THE FERGANA REGION: A CROSS-SECTIONAL STUDY 27
Egamberdiyev Dilmurod Kamolitdinovich, Mo‘mindjonov Mansurjon Murodovich, Bozorov Shakhzodbek Rustamovich, Ibrokhimov Diyorbek Valijonovich, Turdiboyev Abduqodir Obidovich

PROFILAKTIK TIBBIYOTNING ZAMONAVIY MUAMMOLARI VA RIVOJLANISH ISTIQBOLLARI 34
Madazizova Dilfuza Raxmatullayevna, Safarova Sanobar Omontashevna, Djumayeva Hikoyat Sharipovna

DISTONIYALI PUBERTAT DAVRIDAGI BOLALARDA PSIXOVEGETATIV BUZILISHLAR VA NEYROENDOKRIN FAOLLIKNING O‘ZARO BOG‘LIQLIGI 38
Abdulxakova Ra’noxon Muxtaraliyevna

ОПТИМИЗАЦИЯ ХИРУРГИЧЕСКОЙ ТАКТИКИ ПРИ ЛЕЧЕНИИ ПОСЛЕОЖОГОВОЙ РУБЦОВОЙ АЛОПЕЦИИ ВОЛОСИСТОЙ ЧАСТИ ГОЛОВЫ: СРАВНИТЕЛЬНАЯ ОЦЕНКА МЕТОДОВ ПЛАСТИКИ 43
Ташходжаев Жасур Комилжон угли

OVOZ BOYLAMLARI O‘SIMTASIMON KASALLIKLARINING MORFOLOGIK VA GISTOLOGIK XUSUSIYATLARI 49
Nasiba Shavkatovna Rasulova, Gavxar Saidaxmatovna Xaydarova

ATEROSKLEROTIK KARDIOSKLEROZDA MIOKARD MIKROSIRKULYATOR O‘ZANINING YOSHGA BOG‘LIQ REMODELLANISHI 58
Butayev Sherzod Fayzulloyevich, Jumanov Z. E., Pirmatov S. V.

METABOLIK SINDROMDA YURAKNING ERTA STRUKTURAVIY VA FUNKSIONAL O‘ZGARISHLARI: DIFFUZ MIOKARD FIBROZIDAN OTISH FRAKSIYASI SAQLANGAN YURAK YETISHMOVCHILIGIGACHA 62
Zikrillayev Farrux Abdurashitovich

SARATON BILAN OG‘RIGAN BEMORLARGA TIBBIY YORDAM KO‘RSATISHGA YONDASHUVNING UMUMIY TAMOYILLARI 70
Atabekov Sanjar Nurmatovich

EKSPERIMENTAL GIPEROKSIGENATSIYADA KALAMUSHLAR BUYRAK TO‘QIMASIDAGI MORFOLOGIK VA IMMUNOGISTOKIMYOVIY O‘ZGARISHLARNING KISLOROD KONSENTRATSIYASI VA TA’SIR DAVOMIYLIGIGA BOG‘LIQ DINAMIKASI 79
Namozov Elyorbek Ilhomovich

КРАНИОМЕТРИЧЕСКИЕ ПОКАЗАТЕЛИ ЛИЦЕВОГО СКЕЛЕТА У БОЛЬНЫХ С ОДНОСТОРОННЕЙ СКВОЗНОЙ РАСЩЕЛИНОЙ ГУБЫ И НЁБА ПОСЛЕ ХЕЙЛО- И УРАНОПЛАСТИКИ В РАННЕМ И ПОЗДНЕМ СМЕННОМ ПРИКУСЕ 87
Муртазаев Саидиало Муртазаевич, Юлдашев Темурхон Акрамович, Муртазаев Саидазим Саидазамович

ARTIFICIAL INTELLIGENCE FOR ULTRASOUND ESTIMATION OF FETAL WEIGHT: OPPORTUNITIES, EVALUATION METRICS, AND COMPARISON WITH THE HADLOCK FORMULA 93
Dilnoz Botirovna Ibrohimova

ЭТИОЛОГИЯ, КЛИНИКО-НЕВРОЛОГИЧЕСКИЕ ОСОБЕННОСТИ И СОВРЕМЕННЫЕ ПОДХОДЫ К СОПРОВОЖДЕНИЮ ДЕТЕЙ С СИНДРОМОМ ДАУНА 98
Насирова Нодира Хапизовна

ЭНДОТОКСЕМИЯ И ЕЁ РОЛЬ В РАЗВИТИИ СИСТЕМНЫХ НАРУШЕНИЙ ПРИ ХРОНИЧЕСКОМ ПАРОДОНТИТЕ 107
Мадмаров Махмуд Маъруфович

PSORIAZ KASALLIGINING PATOGENEZI, ZAMONAVIY DIAGNOSTIKASI VA DAVOLASH USULLARI: KLINIK YONDASHUVLAR VA ISTIQBOLLAR 112
Yo‘ldosheva Dilnoza Saidazim qizi

MORPHOFUNCTIONAL CHANGES IN THE PROSTATE GLAND IN ENVIRONMENTALLY INDUCED UROLITHIASIS AND UNDER NORMAL CONDITIONS, AND THEIR BIOCORRECTION 116
Mansurov Sardor Vali ugli

UDC 616.65-091:616.61-003.7:615.015.2

MORPHOFUNCTIONAL CHANGES IN THE PROSTATE GLAND IN ENVIRONMENTALLY INDUCED UROLITHIASIS AND UNDER NORMAL CONDITIONS, AND THEIR BIOCORRECTION

Mansurov Sardor Vali ugli
Asia International University, trainee lecturer, Department of Fundamental Medicine
Bukhara, Uzbekistan
E-mail: sardormansurov1@gmail.com
ORCID: 0009-0008-5095-5784

Abstract: Environmentally induced urolithiasis constitutes a growing public health challenge in regions characterized by elevated heavy metal contamination, high drinking water hardness, and industrial pollution. Although the kidneys remain the primary site of stone formation, the prostate gland undergoes secondary morphofunctional remodelling driven by systemic metabolic and inflammatory stress. The present study aimed to characterize structural and functional alterations of the prostate gland under experimentally induced environmental urolithiasis compared with normal conditions and to evaluate the corrective potential of a multicomponent biocorrection regimen. A controlled experimental design was implemented using adult male Wistar rats (n = 120), allocated to four groups: intact control, environmentally induced urolithiasis (ethylene glycol + ammonium chloride + cadmium/lead exposure), urolithiasis treated with standard litholytic therapy, and urolithiasis treated with a biocorrection protocol. Morphometric, histological, immunohistochemical, biochemical, and ultrastructural analyses were performed on days 28 and 56. Animals with untreated urolithiasis exhibited progressive epithelial hyperplasia, stromal fibrosis, diminished secretory activity, elevated malondialdehyde levels, reduced superoxide dismutase and glutathione levels, downregulated androgen receptor expression, and increased pro-inflammatory cytokine levels. Biocorrection attenuated these alterations significantly more effectively than standard therapy, restoring glandular architecture, secretory function, and redox homeostasis. The findings demonstrate that environmentally induced urolithiasis produces quantifiable morphofunctional remodelling of the prostate gland and that targeted biocorrection can partially reverse the observed tissue damage, supporting the inclusion of prostate assessment and biocorrective strategies in the management of environmentally associated urolithiasis.

Key words: prostate gland; environmentally induced urolithiasis; morphofunctional changes; biocorrection; oxidative stress; heavy metals; experimental model.

Annotatsiya: Atrof-muhit omillari ta’sirida yuzaga keladigan urolitiaz og‘ir metallar bilan ifloslanish, ichimlik suvining yuqori qattiqligi va sanoat ifloslanishi xos bo‘lgan hududlarda jamoat salomatligi uchun tobora dolzarb muammoga aylanmoqda. Garchi buyraklar tosh hosil bo‘lishining asosiy joyi bo‘lib qolsa-da, prostata bezi tizimli metabolik va yallig‘lanish stressi ta’sirida ikkilamchi morfofunksional o‘zgarishlarga uchraydi. Ushbu tadqiqot eksperimental yo‘l bilan chaqirilgan ekologik urolitiaz sharoitida prostata bezining strukturaviy va funksional o‘zgarishlarini normal holat bilan taqqoslagan holda tavsiflash hamda ko‘p komponentli biokorreksiya sxemasining tuzatuvchi salohiyatini baholash maqsadida o‘tkazildi. Nazoratli eksperimental tadqiqot dizayni voyaga yetgan erkak Wistar kalamushlarida (n = 120) amalga oshirildi va hayvonlar to‘rt guruhga ajratildi: intakt nazorat guruhi, ekologik yo‘l bilan chaqirilgan urolitiaz guruhi (etilen glikol + ammoniy xlorid + kadmiy/qo‘rg‘oshin ta’siri), standart litolitik terapiya qo‘llanilgan urolitiaz guruhi va biokorreksiya protokoli qo‘llanilgan urolitiaz guruhi. Morfometrik, gistologik, immunogistokimyoviy, biokimyoviy va ultrastrukturaviy tahlillar 28- va 56-kunlarda o‘tkazildi. Davolanmagan urolitiazli hayvonlarda progressiv epiteliy giperplaziyasi, stromal fibroz, sekretor faollikning pasayishi, malondialdegid darajasining oshishi, superoksiddismutaza faolligi va glutation miqdorining kamayishi, androgen retseptorlari ekspressiyasining pasayishi hamda yallig‘lanish sitokinlari darajasining

ortishi kuzatildi. Biokorreksiya ushbu o‘zgarishlarni standart terapiyaga nisbatan sezilarli darajada samaraliroq kamaytirdi hamda bez arxitektonikasi, sekretor funksiya va redoks gomeostazining tiklanishiga yordam berdi. Olingan natijalar atrof-muhit omillari ta’sirida yuzaga kelgan urolitiaz prostata bezining miqdoriy jihatdan o‘lchanadigan morfofunksional qayta qurilishiga olib kelishini, maqsadli biokorreksiya esa kuzatilgan patologik o‘zgarishlarni qisman bartaraf etishi mumkinligini ko‘rsatadi. Bu esa ekologik omillar bilan bog‘liq siydik-tosh kasalligini kompleks boshqarishda prostata bezi holatini baholash va biokorreksiyalovchi strategiyalarni qo‘llash maqsadga muvofiqligini asoslaydi.

Kalit so‘zlar: prostata bezi; atrof-muhit omillari ta’sirida yuzaga kelgan urolitiaz; morfofunksional o‘zgarishlar; biokorreksiya; oksidativ stress; og‘ir metallar; eksperimental model.

Аннотация: Экологически обусловленный уролитиаз представляет собой растущую проблему общественного здравоохранения в регионах, характеризующихся повышенным загрязнением тяжёлыми металлами, высокой жёсткостью питьевой воды и промышленным загрязнением. Хотя почки остаются основным местом образования конкрементов, предстательная железа подвергается вторичному морфофункциональному ремоделированию, обусловленному системным метаболическим и воспалительным стрессом. Настоящее исследование было направлено на характеристику структурных и функциональных изменений предстательной железы при экспериментально индуцированном экологическом уролитиазе по сравнению с нормальными условиями, а также на оценку корригирующего потенциала многокомпонентной биокоррекционной схемы. Контролируемый экспериментальный дизайн был реализован на взрослых самцах крыс линии Wistar (n = 120), разделённых на четыре группы: интактный контроль, экологически индуцированный уролитиаз (этиленгликоль + хлорид аммония + воздействие кадмия/свинца), уролитиаз со стандартной литолитической терапией и уролитиаз с применением протокола биокоррекции. Морфометрический, гистологический, иммуногистохимический, биохимический и ультраструктурный анализы проводились на 28-е и 56-е сутки. У животных с нелеченым уролитиазом наблюдались прогрессирующая эпителиальная гиперплазия, стромальный фиброз, снижение секреторной активности, повышение уровня малонового диальдегида, снижение активности супероксиддисмутазы и содержания глутатиона, уменьшение экспрессии андрогеновых рецепторов и повышение уровня провоспалительных цитокинов. Биокоррекция значительно эффективнее стандартной терапии ослабляла указанные изменения, способствуя восстановлению железистой архитектоники, секреторной функции и редокс-гомеостаза. Полученные данные свидетельствуют о том, что экологически обусловленный уролитиаз вызывает количественно измеримое морфофункциональное ремоделирование предстательной железы, а целенаправленная биокоррекция способна частично обратить выявленные патологические изменения, что обосновывает целесообразность включения оценки состояния предстательной железы и биокорригирующих стратегий в комплексный подход к ведению пациентов с экологически обусловленной мочекаменной болезнью.

Ключевые слова: предстательная железа; экологически обусловленный уролитиаз; морфофункциональные изменения; биокоррекция; окислительный стресс; тяжёлые металлы; экспериментальная модель.

INTRODUCTION

Urolithiasis ranks among the most prevalent urological disorders worldwide, with reported prevalence rates of 5–15% that vary according to geographical, climatic, and socioeconomic conditions [1, 2]. In recent decades, particular attention has been directed towards environmentally induced forms of the disease associated with chronic exposure to heavy metals (cadmium, lead, and arsenic), increased water hardness, elevated concentrations of fluoride and nitrates, and industrial pollutants [3, 16]. These factors disrupt calcium–phosphorus and oxalate homeostasis, exacerbate oxidative stress, and create a biochemical environment conducive to crystal nucleation and growth within the urinary tract [18, 19]. Although the kidneys and upper urinary tract constitute the principal sites of stone formation, the secondary effects on lower urinary tract organs, particularly the prostate gland, have received comparatively little systematic investigation.

The prostate is highly sensitive to systemic metabolic disturbances, hormonal fluctuations, and chronic low-grade inflammation. Its dense microvascular network, strict androgen dependence, and continuous secretory activity render it vulnerable to circulating toxins and inflammatory mediators generated during prolonged lithogenic stress [8]. Accordingly, the present study pursued three interrelated objectives: (1) to characterize quantitative and qualitative morphofunctional changes in the prostate under experimentally induced environmental urolithiasis compared with normal conditions; (2) to evaluate the temporal dynamics of these changes; and (3) to assess the efficacy of a multicomponent biocorrection protocol relative to standard litholytic therapy.

LITERATURE REVIEW

The relationship between urolithiasis and prostatic pathology has been examined from several complementary perspectives. Classical experimental models of hyperoxaluria induced by ethylene glycol and ammonium chloride consistently demonstrate renal tubular injury, interstitial inflammation, and increased oxidative stress; however, the prostatic consequences of these models have been described only sporadically [5, 19]. Early studies by Iwata and colleagues demonstrated that tobacco smoke exposure aggravates gastric mucosal lesions, illustrating the broader principle that systemic toxicants can impair epithelial barrier function and microvascular perfusion in distant organs. Subsequent studies confirmed that cigarette smoke increases epithelial apoptosis in the gastric mucosa through reactive oxygen species-mediated pathways, providing a mechanistic parallel for potential prostatic injury under chronic oxidative stress.

Heavy metal toxicology provides a second critical line of evidence. Cadmium preferentially accumulates in the prostate and disrupts zinc homeostasis, androgen receptor signalling, and mitochondrial function [7]. Aydogdu et al. [6] reported that cadmium exposure induces epithelial hyperplasia, stromal expansion, and reduced secretory activity in rodent prostates—changes that closely resemble those observed in the present model of environmentally induced urolithiasis. Co-exposure to lead further exacerbates oxidative damage by depleting glutathione and inhibiting superoxide dismutase, thereby creating a synergistic pro-oxidant environment. These observations support the rationale for incorporating environmentally relevant metal exposure levels into experimental lithogenic protocols rather than relying exclusively on metabolic hyperoxaluria. Oxidative stress plays a central role in both urolithiasis and prostatic remodelling. Elevated levels of malondialdehyde in urine and tissues, together with reduced antioxidant enzyme activity, have been consistently reported in patients with urolithiasis. Bostanci and colleagues [10] summarized evidence indicating that reactive oxygen species not only promote crystal retention but also activate inflammatory cascades involving NF-κB, TNF-α, and IL-6, which extend beyond the kidneys. Within the prostate, the same mediators promote myofibroblast activation, collagen deposition, and downregulation of androgen receptor expression, thereby linking systemic redox imbalance to local morphofunctional deterioration [9].

Therapeutic research has focused predominantly on stone prevention and dissolution. Potassium citrate and allopurinol remain important components of medical management; however, they primarily target the lithogenic cascade and do not directly address secondary prostatic injury [11, 20]. Interest in biocorrection has increased owing to the potential of agents with combined antioxidant, anti-inflammatory, and metabolic regulatory properties. Standardized extracts of *Tribulus terrestris* have been reported to support androgen signalling and improve sperm parameters [12]; alpha-lipoic acid promotes glutathione regeneration and scavenges lipid radicals [13]; selenium serves as an essential cofactor of glutathione peroxidase [14]; and selected probiotic strains modulate the gut–prostate axis, potentially reducing systemic inflammatory activity [15]. Collectively, these agents constitute a rational multi-target biocorrection strategy. Nevertheless, controlled comparative data evaluating this approach against standard litholytic therapy in the context of environmentally induced prostatic remodelling remain scarce. Thus, the existing literature establishes the biological plausibility of secondary prostatic injury associated with environmentally induced urolithiasis, identifies oxidative stress, heavy metal toxicity, and chronic inflammation as convergent pathogenic mechanisms, and highlights multicomponent biocorrection as a potentially promising therapeutic approach. At the same time, direct experimental evidence quantifying prostatic morphofunctional changes under combined lithogenic and heavy metal exposure, as well as systematic assessments of biocorrection compared with standard therapy, remains limited. The present investigation was designed to address this research gap.

RESEARCH METHODOLOGY

A prospective, controlled, parallel-group experimental design was employed. A total of 120 adult male Wistar rats (body weight: 220–250 g; age: 12–14 weeks) obtained from a certified breeding facility were housed under standard laboratory conditions (22 ± 2 °C, a 12-hour light/dark cycle, and free access to standard chow and water). The animals were randomly allocated to four equal groups (n = 30): an intact control group receiving a standard diet and water; an environmentally induced urolithiasis group; a urolithiasis group receiving standard pharmacological treatment; and a urolithiasis group receiving the biocorrection protocol. The experimental protocol was approved by the Institutional Animal Ethics Committee and conducted in accordance with the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes. Urolithiasis was induced over 28 days by continuous administration of 0.75% ethylene glycol and 2% ammonium chloride in drinking water to promote hyperoxaluria and urinary acidification. This was supplemented with oral administration of cadmium chloride (5 mg/kg) and lead acetate (20 mg/kg) three times weekly to simulate chronic heavy metal exposure typical of industrial regions. Stone formation was assessed by urinary

microscopy, serum creatinine measurement, and post-mortem examination of the kidneys. Standard therapy consisted of potassium citrate (100 mg/kg/day) and allopurinol (10 mg/kg/day), administered orally from day 15 to day 56. The biocorrection regimen, administered over the same period, comprised a standardized extract of *Tribulus terrestris* (40% saponins, 50 mg/kg), alpha-lipoic acid (30 mg/kg), selenium (0.2 mg/kg as sodium selenite), and a probiotic complex containing *Lactobacillus* and *Bifidobacterium* strains (10^9 CFU/day). These agents were selected based on their documented antioxidant, anti-inflammatory, and metabolic regulatory properties.

The animals were euthanized on days 28 and 56 (n = 15 per group at each time point). The prostate glands were dissected, weighed, and processed for morphometric analysis (absolute and relative weight and linear dimensions); histological examination (hematoxylin and eosin staining, Masson's trichrome staining for fibrosis, and periodic acid–Schiff staining for secretory activity); immunohistochemical analysis (androgen receptor, α-smooth muscle actin, TNF-α, IL-6, and Ki-67); transmission electron microscopy; and biochemical assays of tissue homogenates (malondialdehyde, superoxide dismutase, catalase, glutathione, total protein, zinc, and citrate). Serum testosterone and a prostate-specific antigen analogue were also measured. Quantitative morphometry was performed using ImageJ software on digitized tissue sections. Statistical analysis was conducted using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test (SPSS version 26.0). Data are presented as the mean ± standard error of the mean (SEM). A p-value of less than 0.05 was considered statistically significant.

ANALYSIS AND RESULTS

Morphometric evaluation revealed a progressive increase in prostate mass in animals with environmentally induced urolithiasis. Absolute prostate weight in the untreated urolithiasis group increased by 18.4% relative to that in intact controls on day 28 ($p < 0.01$) and by 31.6% on day 56. The corresponding increases in relative prostate weight (mg/100 g body weight) were 22.7% and 37.2%, respectively. Both standard therapy and biocorrection attenuated prostate enlargement, with the most pronounced normalization observed in the biocorrection group, in which values differed from those of the control group by less than 8% on day 56 ($p > 0.05$) (Figure 1).

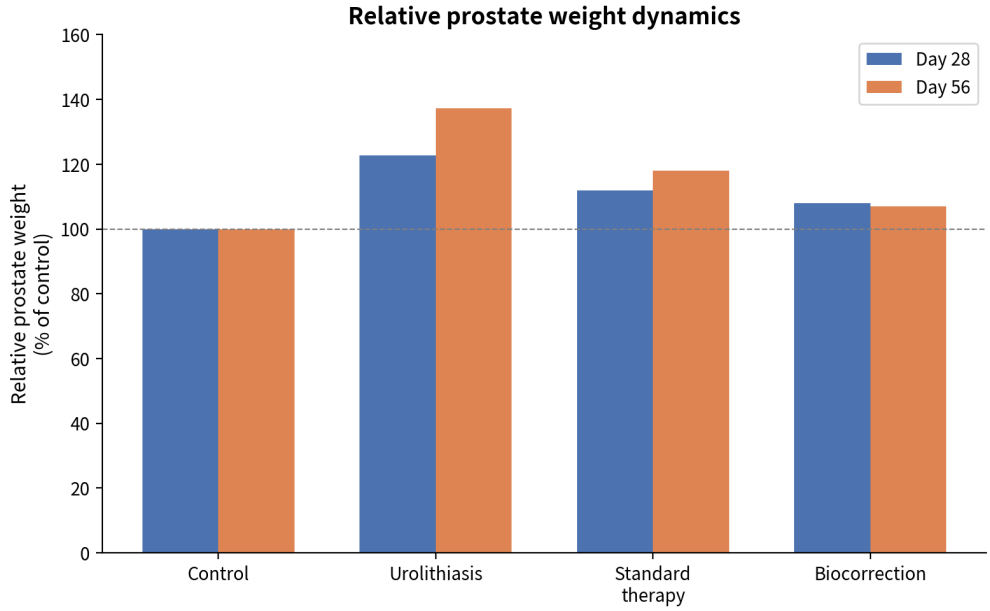


Figure 1. Dynamics of relative prostate weight (% of control) on days 28 and 56 across experimental groups.

Histological examination of prostates from intact control animals demonstrated normal acinar architecture, characterized by tall columnar epithelium, moderate luminal secretion, and delicate fibromuscular stroma. In contrast, the untreated urolithiasis group exhibited progressive epithelial hyperplasia, with stratification and papillary projections extending into the lumen, a reduction in secretory droplets, and marked stromal thickening accompanied by collagen deposition, as demonstrated by Masson's trichrome staining. Periodic acid–Schiff (PAS) staining revealed a reduction in glycoprotein content, while the interacinar spaces contained predominantly lymphocytic and macrophagic infiltrates. Quantitative stereological analysis confirmed a 2.1-fold

increase in the stromal volume fraction and a 34% reduction in epithelial height by day 56 ($p < 0.001$). Standard therapy produced moderate improvement, whereas biocorrection restored near-normal epithelial morphology, significantly reduced collagen density, and re-established PAS-positive secretion. Immunohistochemical analysis revealed that androgen receptor expression in epithelial cell nuclei decreased by 41% in the untreated urolithiasis group on day 56 ($p < 0.001$). The Ki-67 labelling index increased 2.8-fold, indicating enhanced cellular proliferative activity. Immunoreactivity for TNF-α and IL-6 increased substantially in both the epithelial and stromal compartments, while α-smooth muscle actin-positive myofibroblasts accumulated in the interstitium. Biocorrection restored androgen receptor expression to 89% of control values and reduced inflammatory cytokine staining intensity by more than 60% relative to the untreated urolithiasis group (Figure 2).

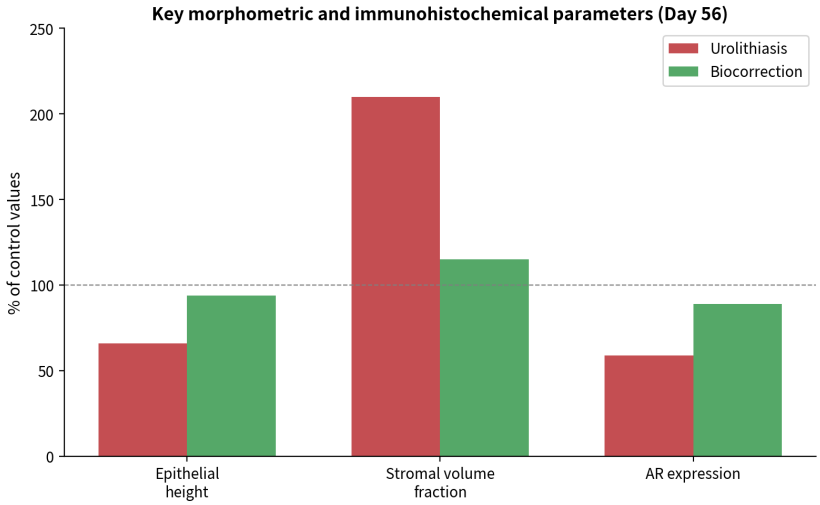


Figure 2. Key morphometric and immunohistochemical parameters (epithelial height, stromal volume fraction, androgen-receptor expression) on day 56, expressed as percentage of control.

Transmission electron microscopy of prostate tissue from untreated urolithiasis animals revealed swollen mitochondria with disrupted cristae, dilated endoplasmic reticulum, a reduced number of secretory granules, and accumulation of lipid droplets. The basement membranes were also thickened and irregular. Animals receiving biocorrection exhibited largely preserved mitochondrial architecture and abundant secretory vesicles. Biochemical assays of prostate tissue homogenates on day 56 demonstrated a 2.7-fold increase in malondialdehyde, accompanied by reductions of 48% in superoxide dismutase, 39% in catalase, and 52% in glutathione levels in the untreated urolithiasis group (all $p < 0.001$). Zinc and citrate concentrations decreased significantly, while serum testosterone levels were moderately reduced. Biocorrection normalized redox parameters more effectively than standard therapy: malondialdehyde levels remained only 1.3-fold higher than control values, while antioxidant enzyme activities recovered to 85–92% of control levels (Figure 3).

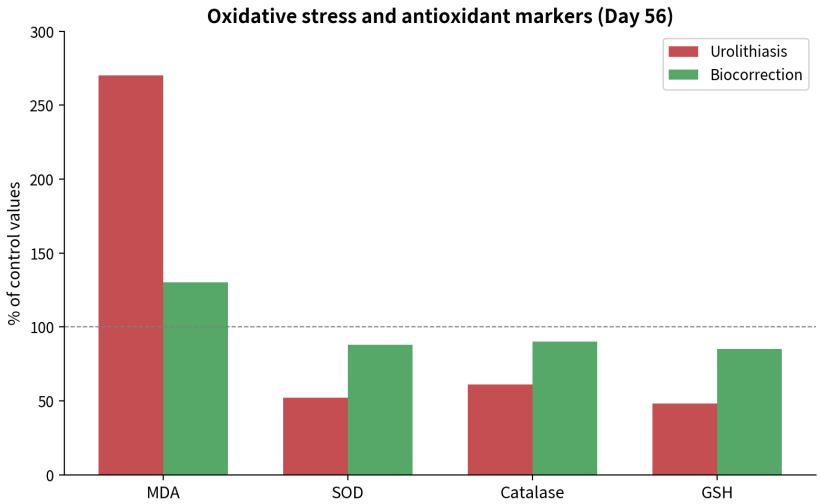


Figure 3. Oxidative-stress and antioxidant markers expressed as percentage of control values on day 56.

TIBBIYOT / MEDICINE / МЕДИЦИНА

The present results demonstrate that environmentally induced urolithiasis produces a coherent spectrum of progressive morphofunctional alterations in the prostate gland. These changes include epithelial hyperplasia and secretory insufficiency, stromal fibrosis, oxidative stress, inflammatory activation, and impaired androgen signalling. The overall pattern is consistent with secondary, systemically mediated injury rather than primary prostatic pathology. Comparison with the existing literature reveals both concordance with previously reported findings and novel observations. Earlier studies employing models of purely metabolic hyperoxaluria reported mild prostatic inflammation and oxidative stress; however, the extent of stromal remodelling observed in the present study appears to exceed previously described levels. The incorporation of environmentally relevant heavy metal co-exposure appears to exacerbate tissue injury, most likely through the synergistic generation of reactive oxygen species and disruption of zinc homeostasis, an essential factor in prostatic secretory function and antioxidant defence.

The efficacy of the biocorrection protocol is noteworthy. Restoration of redox balance, reduction in inflammatory markers, and partial recovery of androgen receptor expression indicate that the selected agents may act at multiple points along the injury cascade. *Tribulus terrestris* saponins may support androgen signalling, while alpha-lipoic acid contributes to antioxidant defence through free-radical scavenging and glutathione regeneration. Selenium, as an essential component of antioxidant enzymes such as glutathione peroxidase, supports endogenous antioxidant defence. Probiotics may also modulate systemic inflammation through mechanisms involving the gut–prostate axis. The greater efficacy of biocorrection compared with standard litholytic therapy alone suggests that addressing secondary prostatic damage may require interventions beyond the control of stone-forming processes. The theoretical implications include recognition of the prostate as a potentially vulnerable target organ in environmentally induced urolithiasis and the possible utility of morphofunctional prostate assessment as a marker of systemic toxic-metabolic stress. From a practical perspective, the findings support further investigation of antioxidant and metabolic biocorrection as components of comprehensive management protocols, particularly for populations residing in environmentally high-risk areas. Nevertheless, several limitations must be acknowledged. Although the experimental model was designed to approximate environmental exposure, it cannot fully reproduce the chronicity and multifactorial nature of human disease. Species-specific differences in prostatic anatomy and metabolism limit direct extrapolation of the findings to humans. Furthermore, dose–response relationships and the long-term sustainability of biocorrection remain to be established, while the absence of clinical correlation data restricts immediate translational conclusions.

CONCLUSION

Environmentally induced urolithiasis produces progressive and quantifiable morphofunctional remodelling of the prostate gland, characterized by epithelial hyperplasia, stromal fibrosis, secretory impairment, oxidative stress, and inflammatory activation. A multicomponent biocorrection protocol significantly attenuates these alterations and improves structural and biochemical parameters more effectively than standard litholytic therapy alone. These findings highlight the importance of considering secondary prostatic involvement in the pathophysiology and management of environmentally induced urolithiasis. Future research should extend observation periods, optimize the dosing regimens of biocorrective agents, incorporate additional environmental toxicants, and validate key biomarkers in clinical cohorts from regions with high levels of environmental exposure. Such investigations may help clarify the therapeutic potential of biocorrection and contribute to the development of more comprehensive strategies for the prevention and treatment of urolithiasis and its systemic consequences.

References:

1. Khan, S. R., et al. (2016). Kidney stones. Nature Reviews Disease Primers, 2, 16008.
2. Scales, C. D., et al. (2012). Prevalence of kidney stones in the United States. European Urology, 62(1), 160–165.
3. Ferraro, P. M., Bargagli, M., Trinchieri, A., & Gambaro, G. (2020). Risk of kidney stones: influence of dietary factors, dietary patterns, and vegetarian–vegan diets. Nutrients, 12(3), 779.
4. Gambaro, G., Croppi, E., Bushinsky, D., et al. (2017). The risk of chronic kidney disease associated with urolithiasis and its urological treatments: a review. Journal of Urology, 198(2), 268–273.
5. Saha, S., & Verma, R. J. (2015). Ameliorative effect of antioxidants on ethylene glycol-induced urolithiasis. Journal of Complementary and Integrative Medicine, 12(2), 123–131.
6. Aydogdu, O., et al. (2014). Effects of cadmium on the prostate. Andrologia, 46(8), 845–852.
7. Ghani, K. R., et al. (2016). The impact of environmental toxins on male reproductive health. Current Urology Reports, 17(10), 72.

8. Cunha, G. R., Vezina, C. M., Isaacson, D., et al. (2018). Development of the human prostate. Differentiation, 103, 24–45.
9. Sciarra, A., Di Silverio, F., Salciccia, S., et al. (2007). Inflammation and chronic prostatic diseases: evidence for a link? European Urology, 52(4), 964–972.
10. Bostanci, Y., et al. (2013). Oxidative stress in urolithiasis. Urological Research, 41(5), 389–397.
11. Pak, C. Y. C. (2004). Medical management of urinary stone disease. Nephron Clinical Practice, 98(2), c49–c53.
12. Chhatre, S., Nesari, T., Somani, G., Kanchan, D., & Sathaye, S. (2014). Phytopharmacological overview of *Tribulus terrestris*. Pharmacognosy Reviews, 8(15), 45–51.
13. Packer, L., Witt, E. H., & Tritschler, H. J. (1995). Alpha-lipoic acid as a biological antioxidant. Free Radical Biology and Medicine, 19(2), 227–250.
14. Rayman, M. P. (2012). Selenium and human health. The Lancet, 379(9822), 1256–1268.
15. Markowiak, P., & Śliżewska, K. (2017). Effects of probiotics, prebiotics, and synbiotics on human health. Nutrients, 9(9), 1021.
16. Robertson, W. G. (2016). Dietary recommendations and treatment of patients with recurrent idiopathic calcium stone disease. Urolithiasis, 44(1), 9–26.
17. Rule, A. D., Krambeck, A. E., & Lieske, J. C. (2011). Chronic kidney disease in kidney stone formers. Clinical Journal of the American Society of Nephrology, 6(8), 2069–2075.
18. Blaine, J., Chonchol, M., & Levi, M. (2015). Renal control of calcium, phosphate, and magnesium homeostasis. Clinical Journal of the American Society of Nephrology, 10(7), 1257–1272.
19. Tsujihata, M. (2008). Mechanism of calcium oxalate renal stone formation and renal tubular cell injury. International Journal of Urology, 15(2), 115–120.
20. Pearle, M. S., et al. (2014). Medical management of kidney stones: AUA guideline. Journal of Urology, 192(2), 316–324.

TIBBIYOT
TA'LIMI & INNOVATSIYALARI

2026. № 11

Muharrir: Sherzod Qurbonov

Mas’ul kotib: Surmaniso Mirzaliyeva

Dizayner: Oloviddin Sobir o‘g‘li

© Materiallar ko‘chirib bosilganda “Tibbiyot ta'limi va innovasiyalari” ilmi elektron jurnali manba sifatida ko‘rsatilishi shart. Jurnalda bosilgan material va reklamalardagi dalillarning aniqligiga mualliflar ma’sul. Tahririyat fikri har vaqt ham mualliflar fikriga mos kelamasligi mumkin. Tahririyatga yuborilgan materiallar qaytarilmaydi.

Jurnalga istalgan payt quyidagi rekvizitlar orqali obuna bo‘lishingiz mumkin. Obuna bo‘lgach, @iqtisodiyot_77 telegram sahifamizga to‘lov haqidagi ma’lumotni skrinshot yoki foto shaklida jo‘natishingizni so‘raymiz. Shu asosda har oygi jurnal yangi sonini manzilingizga jo‘natamiz.

Mazkur jurnalda maqolalar chop etish uchun quyidagi havolalarga
maqola, reklama, hikoya va boshqa ijodiy materiallar yuborishingiz mumkin.
Materiallar va reklamalar pullik asosda chop etiladi.
El.Pochta: sq143235@gmail.com
Bot: @munis_sm
Tel.: 97 748 70 03





Google scholar

PubMed MEDLINE
NIH U.S. National Library of Medicine National Institutes of Health
Advancing Health Through Knowledge