INTEGRATED MORPHOFUNCTIONAL ASSESSMENT OF THE PROSTATE GLAND IN ENVIRONMENTALLY INDUCED UROLITHIASIS AND UNDER NORMAL CONDITIONS: PATHWAYS OF INJURY AND BIOCORRECTION

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Background and Aim: Environmentally induced urolithiasis, driven by chronic exposure to heavy metals, water hardness, and industrial pollutants, is an increasing concern in many regions. While the kidney and upper urinary tract are the primary sites of stone formation, secondary morphofunctional changes in the prostate gland remain insufficiently characterized. The prostate is highly sensitive to systemic oxidative stress, inflammation, and metabolic disturbance. This review and experimental framework aimed to integrate available evidence on structural and functional alterations of the prostate under environmentally induced urolithiasis compared with normal conditions, and to outline the potential of targeted biocorrection.

Materials and Methods: A targeted analysis of experimental animal studies and mechanistic literature was performed using combinations of the terms environmentally induced urolithiasis, prostate morphology, heavy metals (cadmium, lead), oxidative stress, inflammation, androgen receptor, stromal fibrosis, and biocorrection. Evidence from related models of metabolic urolithiasis and heavy-metal toxicity was incorporated when direct prostatic data were limited. A proposed experimental design includes four groups (intact control, environmentally induced urolithiasis, standard therapy, and biocorrection) examined at 28 and 56 days by morphometry, histology, immunohistochemistry, and biochemical assays.

Results: Available data indicate that environmentally induced urolithiasis produces progressive prostatic changes: epithelial hyperplasia with reduced secretory activity, stromal fibrosis, decreased androgen-receptor expression, elevated oxidative-stress markers (increased MDA, reduced SOD and GSH), and upregulation of inflammatory mediators (TNF-α, IL-6). Ultrastructural alterations include mitochondrial damage and reduced secretory granules. Biocorrection protocols combining antioxidant, metabolic, and anti-inflammatory agents (e.g., Tribulus terrestris extract, alpha-lipoic acid, selenium, probiotics) demonstrably attenuate these changes, restoring epithelial architecture, secretory function, and redox balance more effectively than standard litholytic therapy alone. Direct comparative evidence across all four conditions remains limited.

Scientific Novelty: The work proposes an integrated morphofunctional assessment framework that links observable prostatic architecture (epithelial height, stromal volume fraction, secretory activity) to oxidative, inflammatory, hormonal, and regenerative mechanisms. It emphasizes the necessity of a factorial design and an explicit test of interaction between environmental lithogenic factors and biocorrective intervention before claims of synergistic protection can be made.

Conclusion: Environmentally induced urolithiasis induces quantifiable morphofunctional remodeling of the prostate gland through overlapping oxidative, inflammatory, and hormonal pathways. Targeted biocorrection can partially reverse these alterations. Standardized histological scoring, quantitative morphometry, and a focused immunohistochemical panel (androgen receptor, 8-OHdG, NF-κB, cleaved caspase-3, Ki-67, α-SMA) provide a reproducible basis for future experimental and translational studies.

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Keywords: prostate gland; environmentally induced urolithiasis; morphofunctional changes; biocorrection; oxidative stress; heavy metals; immunohistochemistry.

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