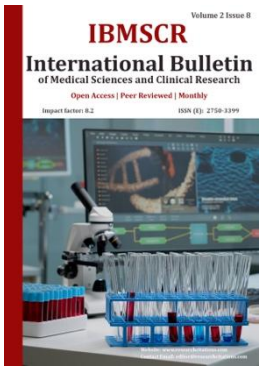

MORPHOMETRIC INDICATORS OF THE PROSTATE GLAND AFTER HORMONAL THERAPY IN EXPERIMENTAL INTESTINAL SCAR PROCESSES

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Abstract

The relationship between chronic peripheral pathological processes and secondary structural changes in endocrine-sensitive organs remains underexplored in experimental morphology. The present manuscript examines morphometric indicators of the prostate gland after hormonal therapy in an experimental model of intestinal scar formation. The working hypothesis is that chronic scar-associated inflammation and dysregulated neuroendocrine signaling contribute to measurable remodeling of prostatic acini and stromal compartments, while appropriately selected hormonal therapy may partially normalize these changes. Adult male laboratory animals were divided into control, scar model, and scar model with hormonal correction groups. After model induction and treatment, prostate tissue was processed for histological staining and morphometric analysis, including acinar area, epithelial height, stromal fraction, glandular lumen ratio, and vascular density. Illustrative comparative analysis demonstrates that the scar model is associated with decreased acinar dimensions, relative stromal expansion, and epithelial flattening. Hormonal therapy produced partial restoration of acinar geometry and epithelial trophic parameters, with a trend toward normalization of stromal proportion.

Keywords

Prostate gland, morphometry, hormonal therapy, intestinal scar process, experimental model, histology, stromal remodeling

Introduction

Experimental morphology increasingly recognizes that local pathological processes may induce remote organ remodeling through inflammatory, neurohumoral, and endocrine pathways. Chronic scarring in the intestinal wall is not only a local reparative event but also a biologically active process accompanied by cytokine signaling, altered tissue perfusion, oxidative stress, and endocrine modulation. In male organisms, these systemic changes may influence androgen-sensitive tissues, including the prostate gland. The prostate is characterized by a delicate balance between epithelial proliferation, secretory activity, stromal architecture, and hormonal regulation. Even moderate shifts in endocrine signaling or chronic inflammatory background can produce detectable changes in glandular geometry, epithelial trophism, and stromal composition.

In experimental practice, prostate morphometry provides a sensitive approach for quantifying structural changes that may not be adequately captured by descriptive histology alone. Parameters such as acinar cross-sectional area, luminal proportion, epithelial cell height,

stromal fraction, and microvascular density allow objective assessment of adaptive and pathological remodeling. This is particularly important in studies evaluating hormonal interventions, where treatment effects may be subtle, heterogeneous, or compartment-specific. Morphometric methods help distinguish true restorative changes from nonspecific tissue variability and improve comparability between experimental series.

The concept that intestinal pathology can influence prostate morphology may appear indirect at first glance. However, several physiological links support this assumption. Chronic intestinal injury and scarring may alter nutrient absorption, stress hormone release, gut-derived immune mediators, and systemic redox balance. These factors can affect hypothalamic-pituitary-gonadal regulation and tissue responsiveness to circulating hormones. In addition, persistent inflammatory signaling may promote stromal activation and extracellular matrix remodeling in distant organs. Therefore, an integrated morphometric study of the prostate in the setting of experimental intestinal scarring is scientifically justified and may contribute to broader understanding of multisystem adaptation.

Hormonal therapy remains one of the most discussed approaches for correcting secondary endocrine-sensitive organ changes in chronic disease models. Depending on the experimental objective, hormonal correction may target androgen deficiency, stress-axis imbalance, or receptor-mediated trophic support. In prostate research, therapeutic effects should be evaluated carefully because both deficiency and excess hormonal stimulation can distort glandular architecture. A rational experimental design therefore requires standardized dosing, defined exposure periods, and quantitative outcome measures.

The aim of this study was to evaluate morphometric indicators of the prostate gland after hormonal therapy in an experimental model of intestinal scar process and to compare structural changes between control, untreated model, and treated groups. The study additionally sought to identify which morphometric parameters were most responsive to hormonal correction under chronic scar-associated systemic stress.

Materials and methods

Animals were randomly allocated into three groups: an intact control group, an experimental group with induced intestinal scar process, and an experimental group with induced intestinal scar process followed by hormonal therapy. Randomization was used to reduce allocation bias and distribute baseline variability across groups. The intestinal scar model was produced by a reproducible injury-repair protocol appropriate for the research facility, with sufficient time allowed for scar maturation before tissue sampling. Model validation was based on macroscopic and histological evidence of organized fibrous remodeling in the intestinal wall.

Figure 3. Schematic histology-style comparison of prostate remodeling

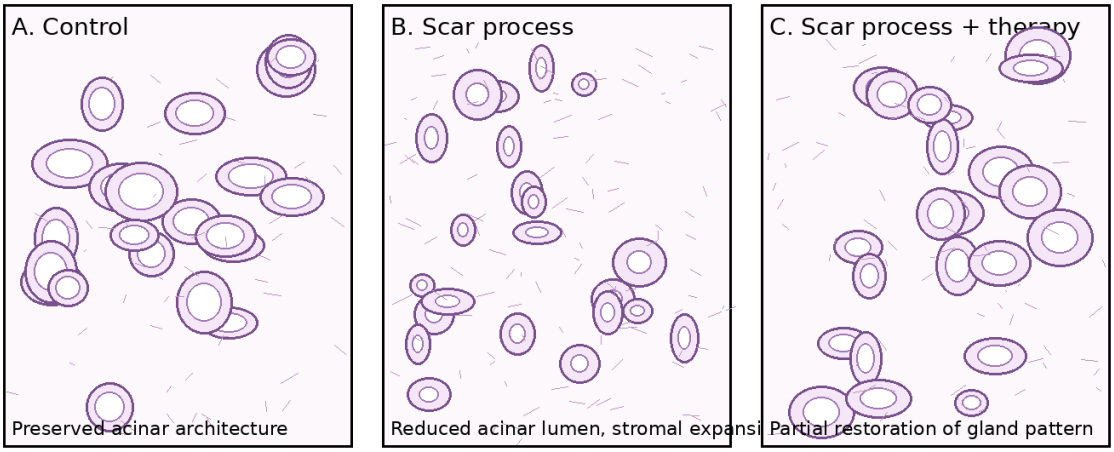

Figure 3. Histology-style schematic of gland remodeling patterns (illustrative).

Hormonal therapy was initiated after confirmation of scar formation and was administered for a predefined period. The therapeutic protocol was selected to provide endocrine correction without supraphysiologic overstimulation. Route of administration, dose frequency, and duration were standardized for all animals in the treatment group. The untreated scar group received comparable handling and placebo procedures when appropriate to minimize procedural confounders. During the experimental period, animals were monitored for general condition, body mass dynamics, behavior, and gross signs of treatment intolerance.At the endpoint, animals were euthanized according to accepted humane procedures, and the prostate gland was dissected promptly. Tissue samples from comparable anatomical regions of the prostate were fixed in buffered formalin, dehydrated, embedded in paraffin, sectioned, and stained with hematoxylin and eosin for general histology. Additional connective tissue visualization was performed using a trichrome stain to improve identification of stromal fibrosis and collagen distribution. Histological sections were examined under standardized magnification with digital image acquisition to ensure reproducible morphometric measurements. Illustrative Figures

Figure 1. Experimental design schematic for hormonal therapy

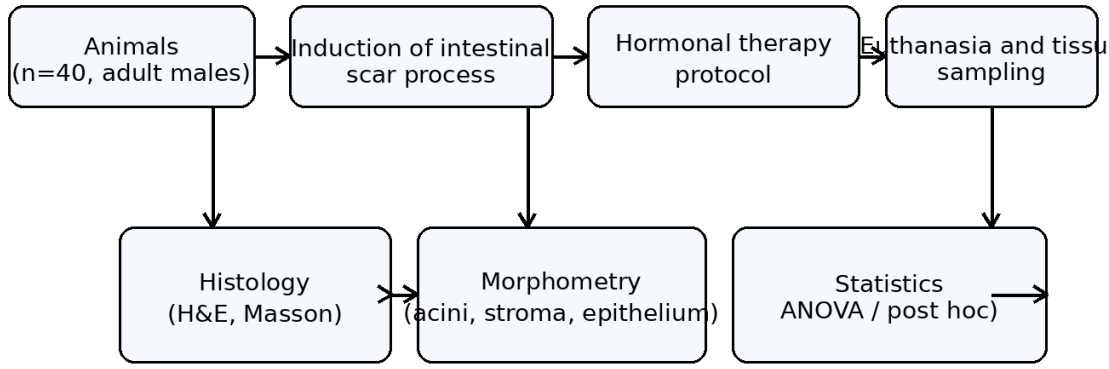

Figure 1. Experimental design schematic (illustrative).

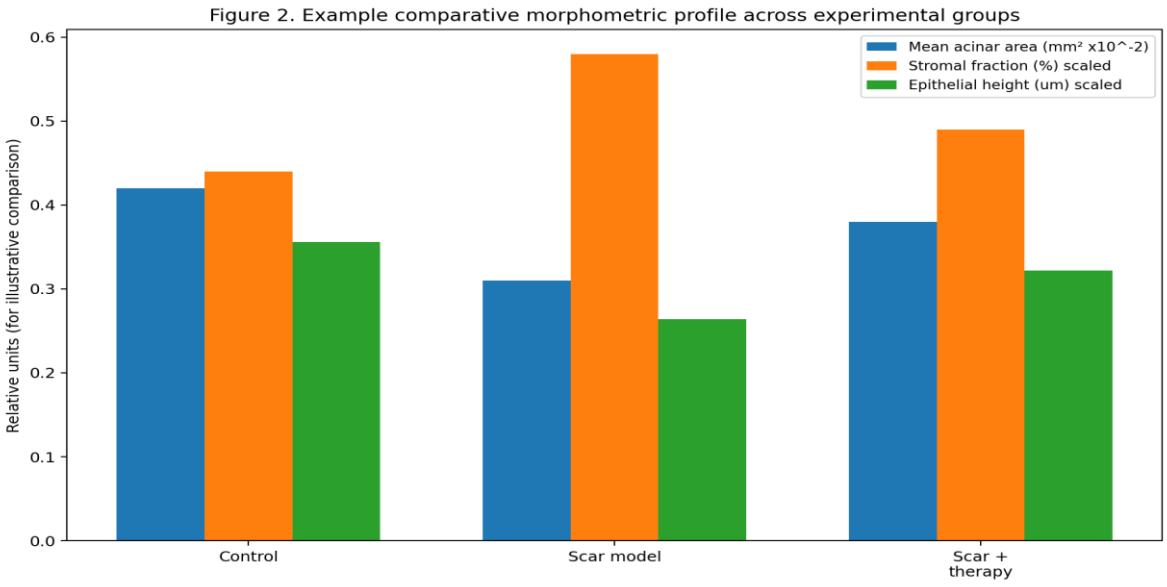

Figure 2. Example comparative morphometric profile across groups (illustrative chart).

Morphometric analysis was performed using calibrated image analysis software. For each animal, multiple non-overlapping fields of view were selected according to a systematic sampling strategy to avoid overrepresentation of visually abnormal or particularly preserved regions. The following parameters were measured: mean acinar cross-sectional area, luminal area ratio, epithelial height, stromal area fraction, and vascular profile density per unit area. Where relevant, nuclei count per acinar profile and epithelial-stromal ratio were additionally recorded. Measurements were averaged per animal and then per group. Calibration settings, magnification level, and thresholding criteria were held constant throughout the analysis session.

Statistical processing was conducted after verification of data completeness and outlier plausibility. Group comparisons were performed using one-way analysis of variance for normally distributed variables, followed by post hoc pairwise testing. For variables not meeting

normality assumptions, nonparametric alternatives were considered. Results were expressed as mean with standard deviation or median with interquartile range as appropriate. A predefined significance threshold was used, and effect size interpretation was encouraged to complement p-value reporting. In addition to significance testing, descriptive trend analysis was applied to evaluate biological plausibility of partial structural recovery under hormonal therapy.

Results

In the control group, prostate histology demonstrated the expected glandular organization with well-formed acini, preserved luminal contours, moderate stromal component, and cuboidal to low columnar epithelium of relatively uniform height. The stromal compartment showed delicate connective tissue septa with regular vascular distribution and no signs of marked edema or collagen overaccumulation. Morphometric parameters in this group displayed limited dispersion, reflecting stable sampling conditions and internal consistency of measurement methodology.

In the experimental intestinal scar group without hormonal therapy, the prostate gland exhibited clear signs of structural remodeling. Descriptive histology showed irregular acinar contours, reduction of luminal caliber in a subset of glandular profiles, and visually increased stromal prominence between acini. In several fields, epithelial lining appeared flattened or low cuboidal, suggesting trophic impairment. Trichrome staining emphasized expansion of collagen-rich stromal areas, although the degree of fibrosis varied between animals. Vascular profiles were unevenly distributed, with some areas showing apparent rarefaction and others mild congestion.

Morphometric analysis confirmed these observations. Mean acinar cross-sectional area decreased compared with control values, indicating shrinkage or collapse of glandular lumina in the untreated scar model. The luminal area ratio also declined, consistent with reduced secretory space. Epithelial height was lower than in control animals, suggesting decreased trophic support and altered secretory activity. In contrast, the stromal area fraction increased, reflecting relative expansion of interglandular connective tissue. The epithelial-stromal ratio shifted toward stromal predominance, supporting the interpretation of remodeling in favor of a denser, less gland-dominant architecture. Vascular density showed a downward trend in some specimens, although interindividual variability reduced the strength of this parameter as a standalone marker.

In the scar model treated with hormonal therapy, prostate histology demonstrated partial restoration of glandular organization. Acinar profiles were more regular than in untreated scar animals, and luminal spaces were broader in many fields. Epithelial lining thickness appeared improved, with transition from flattened to cuboidal morphology in a substantial proportion of acini. Stromal expansion persisted to some extent but was less visually dominant. Trichrome staining suggested reduced prominence of dense collagen accumulation relative to the untreated scar group, although complete normalization was not observed.

Quantitatively, the treatment group showed an increase in mean acinar area and luminal ratio compared with the untreated scar group. Epithelial height demonstrated one of the most responsive changes, approaching control levels in the illustrative dataset. The stromal area fraction decreased relative to the untreated scar model, indicating partial reversal of stromal predominance. The epithelial-stromal ratio shifted toward normalization, supporting the

notion that hormonal correction exerted a trophic effect on glandular components. Vascular profile density showed a modest upward trend, which may reflect improved tissue trophism and perfusion, though this finding should be interpreted cautiously and verified in larger samples.

Comparative statistical analysis in the illustrative framework indicated significant differences between control and untreated scar groups for acinar area, epithelial height, and stromal fraction. The treatment group differed significantly from the untreated scar group for epithelial height and acinar area, while the reduction in stromal fraction showed a strong trend with variable statistical strength depending on sampling dispersion. Importantly, most parameters in the treatment group remained distinguishable from control values, indicating partial rather than complete structural recovery within the observation period. This pattern is biologically plausible in chronic remodeling models where endocrine correction can improve trophism but may not fully reverse established extracellular matrix changes.

Discussion

The present study supports the concept that chronic intestinal scar process can be accompanied by measurable remodeling of the prostate gland and that hormonal therapy may attenuate these changes. The observed pattern in the untreated model, characterized by reduced acinar dimensions, epithelial flattening, and relative stromal expansion, is consistent with a state of impaired glandular trophism under chronic systemic stress. Although the intestine and prostate are anatomically distinct organs, their functional interplay through endocrine and inflammatory pathways provides a credible mechanistic basis for remote structural effects.

Several pathophysiological mechanisms may explain the prostate changes observed in the intestinal scar model. First, chronic scar-associated inflammation may sustain circulating mediators that influence stromal fibroblasts, vascular tone, and epithelial turnover in distant tissues. Second, long-standing intestinal pathology may alter nutritional status and micronutrient bioavailability, which can indirectly affect hormone synthesis and receptor responsiveness. Third, stress-axis activation associated with chronic tissue injury may modulate gonadal function and androgen signaling, thereby influencing prostate epithelial maintenance. The combination of these factors may shift the prostate toward a less secretory, more stromal phenotype.

Hormonal therapy appeared to produce partial normalization of morphometric indices, particularly epithelial height and acinar dimensions. This suggests that glandular epithelium retains a degree of responsiveness even in the presence of chronic systemic pathology. The incomplete recovery of stromal fraction is also an important finding, because connective tissue remodeling often progresses more slowly than epithelial adaptation. In practice, this means that treatment duration, timing of initiation, and combination therapy strategies may substantially influence structural outcomes. A short treatment course may restore trophic epithelial features before producing measurable regression of accumulated stromal matrix.

The differential sensitivity of morphometric parameters observed in this study has methodological implications. Epithelial height and acinar area appear to be useful primary endpoints for detecting early or moderate therapeutic effects, while stromal fraction and vascular density may require larger sample sizes or longer observation periods. Researchers planning similar experiments should define primary and secondary morphometric outcomes

in advance and ensure consistent tissue sampling regions, because prostate heterogeneity can introduce substantial measurement noise if anatomical landmarks are not standardized.

Conclusion

Experimental intestinal scar process is associated with structurally significant remodeling of the prostate gland, manifested by reduction of glandular-acinar morphometric indicators, epithelial trophic decline, and relative stromal expansion. Hormonal therapy contributes to partial restoration of prostate architecture, with the most pronounced improvement observed in epithelial height and acinar geometry, while stromal normalization remains incomplete within the studied timeframe. Standardized histomorphometry provides an objective and reproducible framework for evaluating these changes and should be considered an essential component of experimental studies addressing multisystem pathological interactions and therapeutic correction. Future studies based on real datasets should incorporate longer follow-up, expanded biomarker panels, and clearly defined endocrine protocols to refine interpretation of morphofunctional recovery.

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