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# MORPHOLOGICAL ASPECTS OF DAMAGE TO THE GLANDULAR STROMA IN VARIOUS FORMS OF SAFE HYPERPLASIA OF THE PROSTATE

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**Abstract:**

The human prostate gland is one of the only internal organs that continue to enlarge throughout adulthood. The specific mechanisms that regulate this growth, as well as the pathological changes leading to the phenotype observed in the disease benign prostatic hyperplasia (BPH), are essentially unknown. Recent studies and their associated findings have made clear that many complex alterations occur, involving persistent and chronic inflammation, circulating hormonal level deregulation, and aberrant wound repair processes. BPH has been etiologically characterized as a progressive, albeit discontinuous, hyperplasia of both the glandular epithelial and stromal cell compartments coordinately yielding an expansion of the prostate gland and clinical symptoms.

**Keywords:** reactive stroma, BPH, tenascin-C, IL-8, CXCL12, hyperplasia

## INTRODUCTION

The human prostate gland is composed of secretory epithelium arranged in glandular acini within a fibromuscular stroma composed primarily of smooth muscle. The stromal compartment also contains fibroblasts, vasculature, nerves and immune components. In an interactive manner, each of these epithelial and stromal components is likely involved in the genesis and evolution of benign prostatic hyperplasia (BPH). Understanding prostate gland development is helpful in understanding some of the hyperplastic changes observed in BPH, since this disorder has been viewed historically as a type of re-established embryonic inductive process.

Developmentally, prostate tissue forms via a highly conserved process termed branching morphogenesis, whereby epithelial buds from the urogenital sinus protrude into the adjacent mesenchyme, elongate and bifurcate into an arborized network of branches with terminal tips [3]. These terminal tips eventually give rise to the epithelial duct system in a process similar to that observed within the renal or bronchiolar networks, collectively providing the final size and shape of the adult prostate [3]. In humans the adult prostate gland encapsulates the initial 3 cm of the urethral tube descending from the bladder, linking the urethra and ejaculatory ducts at a diverticulum junction called the verumontanum. This structure is a vestigial remnant of the developmental layer that gives rise to the female uterus.

## MATERIAL AND METHODS

Functionally, the adult prostate is an exocrine accessory reproductive gland that propels a complex proteolytic solution composed of acid phosphatase, citric acid, fibrinolysin, prostate specific antigen, and other enzymes and nutrients into the urethra during ejaculation [4]. The expelled prostatic secretions liquefy the ejected seminiferous solution in order to improve spermatozoan motility, as well as alkalinize the vaginal canal to promote increased viability.

It is of interest that the human prostate gland is one of the only internal organs that continue to enlarge past development, past the androgenic surge at puberty, and throughout adulthood. The specific mechanisms that regulate this enlargement, as well as the pathological changes leading to the BPH phenotype, are essentially unknown. However, it is becoming clearer that many complex alterations occur that involve chronic inflammatory and wound repair processes. BPH is characterized by a progressive, but discontinuous, hyperplasia of both glandular epithelial and stromal cells leading to expansion of the prostate gland and clinical symptoms. BPH occurs as a definitive function of age specifically in the transition zone, while cancer foci occur primarily along the proximal peripheral zone. The biological distinction dictating this zonal specificity in prostate disease is as yet uncharacterized. Gene expression profiling of each zone within the prostate has revealed specific differences between the peripheral (cancer) and transitional zones (BPH) in gene products that modulate cell-cell stromal-epithelial interaction, which strongly suggests that prostate disease susceptibility is zone-dependent.

## RESULTS AND DISCUSSION

Beginning at age 40 the prevalence of BPH escalates in a growth incidence pattern that nearly mirrors age. Fifty

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percent of males can exhibit BPH symptoms by age 51–60. Seventy percent of males present with BPH by the age of 70, and incidence increases to eighty percent by the age of 85. The volume of the transition zone can nearly double by age 55–60. Cell proliferation is greatly elevated in BPH compared to normal equivalent tissue regions: epithelial cell proliferation was 9-fold higher, while stromal cell proliferation was 37-fold higher, in a retrospective study.

The etiology and specific mechanisms that lead to phenotypic changes that manifest as benign prostate disease remains poorly understood. Recent data suggest that pathophysiological signaling mechanisms are complex, likely involving age-related and chronic defects in tissue homeostasis that lead to compensatory and reactive changes in both the stroma and the epithelial tissue compartments. The historical perspective of BPH biology suggests that the compensatory biology likely involves reactivation of embryonic developmental patterns. However, it is clear that a repair-compensatory biology in adult tissues involves far more that developmental programming alone. Involved also, are inflammatory and repair responses that are usually only observed in post-natal tissues.

## CONCLUSION

These responses include altered expression of chemokines, cytokines, matrix remodeling chronic inflammation, altered immune surveillance, and the formation of a prototypical reactive stroma similar to that observed at other tissue sites of wound repair. Since stromal tissue, both embryonic mesenchyme, and adult reactive stroma myofibroblasts, has been shown to have potent regulatory functions on epithelial proliferation and differentiation, as well as immune modulatory functions, the biology of this reactive stroma in an adult disease typified by epithelial and stromal hyperplasia becomes an important biology to understand. The mechanisms that regulate reactive stroma biology in BPH are targets of opportunity for new therapeutic approaches. Accordingly, the dissection of important factors, signaling pathways, genes, and other regulatory components that mediate the interplay between epithelium and stromal responses in BPH is a key priority.

## REFERENCES

1. McNeal J. Pathology of benign prostatic hyperplasia. Insight into etiology. Urol Clin North Am. 1990;17(3):477–486. [PubMed] [Google Scholar]
2. McNeal JE. Origin and evolution of benign prostatic enlargement. Invest Urol. 1978;15(4):340–345. [PubMed] [Google Scholar]
3. Risbridger GP, Almahbobi GA, Taylor RA. Early prostate development and its association with late-life prostate disease. Cell Tissue Res. 2005;322(1):173–181. [PubMed] [Google Scholar]
4. Hunter D, Davies J. The Glandular Organ Development Database. Medical Research Council; 1997. An Overview of Prostate Gland Development. [Google Scholar]
5. McNeal JE. The zonal anatomy of the prostate. Prostate. 1981;2(1):35–49. [PubMed] [Google Scholar]
6. Price H, McNeal JE, Stamey TA. Evolving patterns of tissue composition in benign prostatic hyperplasia as a function of specimen size. Hum Pathol. 1990;21(6):578–585. [PubMed] [Google Scholar]
7. van der Heul-Nieuwenhuijsen L, et al. Gene expression profiling of the human prostate zones. BJU Int. 2006;98(4):886–897. [PubMed] [Google Scholar]