AI-Powered Discovery of Natural Compounds for Benign Prostatic Hyperplasia Treatment

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Article

AI-Powered Discovery of Natural Compounds for Benign Prostatic Hyperplasia Treatment

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Abstract

Background. Benign prostatic hyperplasia (BPH) is a common condition in aging men that causes urinary symptoms. Current treatments have limitations, necessitating new approaches. Single-cell RNA sequencing (scRNA-seq) provides detailed insights into cellular activity, while Artificial Intelligence (AI) techniques such as large language models (LLMs) can analyze complex queries to identify natural substances that may downregulate overexpressed genes, providing a novel approach for the treatment of BPH. ***Method.*** Single-cell RNA sequencing (scRNA-seq) data (*GSE226237*) were obtained from the National Institutes of Health (NIH) Gene Expression Omnibus (GEO) repository. Gene expression profiles from three large prostates were compared with those from three small prostates to identify differentially expressed genes associated with larger prostate tissue. These genes were evaluated as potential therapeutic targets. Candidate natural compounds that may modulate the activity of overexpressed genes were generated using a structured prompt submitted to the large language model ChatGPT. ***Results.*** Single-cell RNA sequencing analysis comparing three large prostates with three small prostates identified distinct gene expression differences associated with prostate enlargement. Several genes involved in immune regulation and cellular structure were downregulated in large BPH, while genes related to epithelial integrity, protein synthesis, and proliferation such as SLC14A1, KRT5, KRT14, KRT15, PRAC1, CSTA, RPL36A, GABARAP, RPS17, and FXYD3 were upregulated. To explore potential therapeutic interventions, these overexpressed genes were analyzed using ChatGPT 5.4 to identify candidate natural compounds capable of modulating their activity. The resulting gene/compound associations include natural products such as resveratrol, curcumin, epigallocatechin gallate (EGCG), genistein, quercetin, sulforaphane, berberine, ashwagandha, and lycopene, highlighting promising directions for natural product based modulation of BPH-associated gene expression. ***Conclusions***. These findings highlight molecular changes in BPH progression and show how single-cell transcriptomics reveals gene expression linked to epithelial proliferation, immune modulation, and metabolism. Integrating AI with omics data offers a framework for identifying potential natural therapeutics, though these AI-generated recommendations remain hypothesis-generating rather than definitive.

Keywords: benign prostatic hyperplasia; large language models; natural products; single-cell RNA sequencing

Introduction

Benign prostatic hyperplasia (BPH) is a common condition in aging males, marked by the non-cancerous enlargement of the prostate gland. This often results in urinary symptoms that can significantly diminish quality of life. Existing treatments, ranging from medications to invasive procedures, are frequently limited by side effects and inconsistent effectiveness, highlighting the pressing need for innovative therapeutic strategies. Recent breakthroughs in single-cell RNA sequencing (scRNA-seq) have transformed our understanding of cellular heterogeneity and the

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molecular mechanisms driving diseases like BPH. By offering a high-resolution view of gene expression at the single-cell level, scRNA-seq enables the identification of critical cellular populations and molecular pathways involved in disease progression. At the same time, advances in large language models (LLMs) in Artificial Intelligence (AI) provide robust tools for analyzing complex datasets and generating insights for drug repurposing. This study investigates the integration of scRNA-seq data and LLMs to enhance drug repurposing for BPH treatment. By combining the precision of scRNA-seq with the analytical power of LLMs, we aim to uncover novel therapeutic targets and reposition existing drugs to address unmet clinical needs. Building on this approach, we also explore the potential of integrating omics data with LLM to identify natural compounds capable of downregulating overexpressed genes, demonstrating a promising pathway for the development of safe and effective treatments for conditions such as BPH.

Data

We obtained scRNA-seq data (*GSE226237*) from the NIH/NCBI GEO repository to compare gene expression in the single-cell sequencing data from three small prostates (<80mL volume) and three large prostates (>80mL volume). Human prostate transition zone tissue resected during Thulium laser enucleation of the prostate (ThuLEP) were mechanically and enzymatically digested, scRNA-seq was performed for all 6 samples. We applied a t-test to log-normalized scRNA-seq expression data to compare cells from small and large prostate samples.

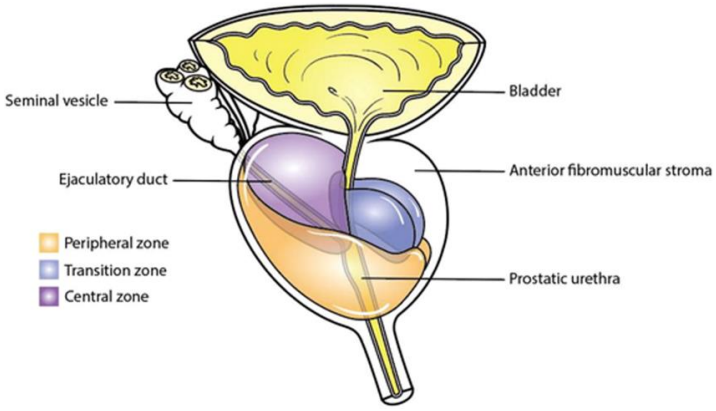

Figure 1. – Prostate gland zones [1].

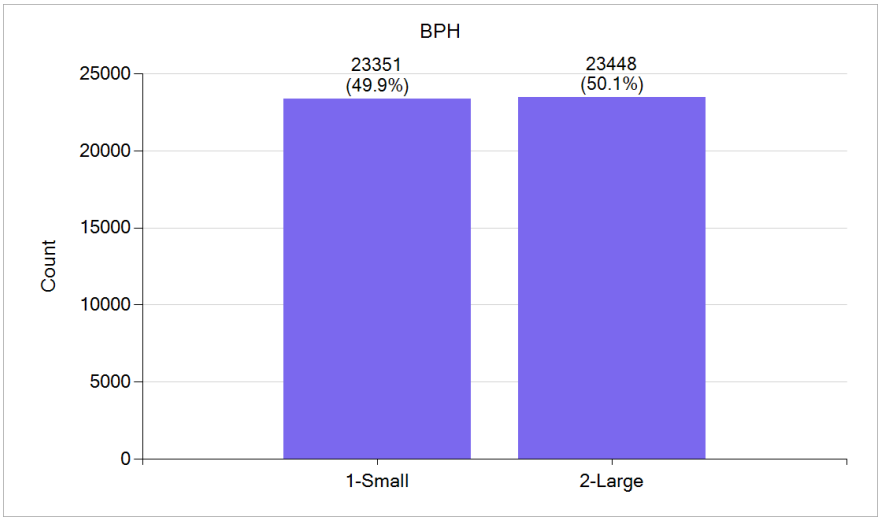

Figure 2. *GSE226237* contains 23351 small BPH and 23448 large BPH single cells.

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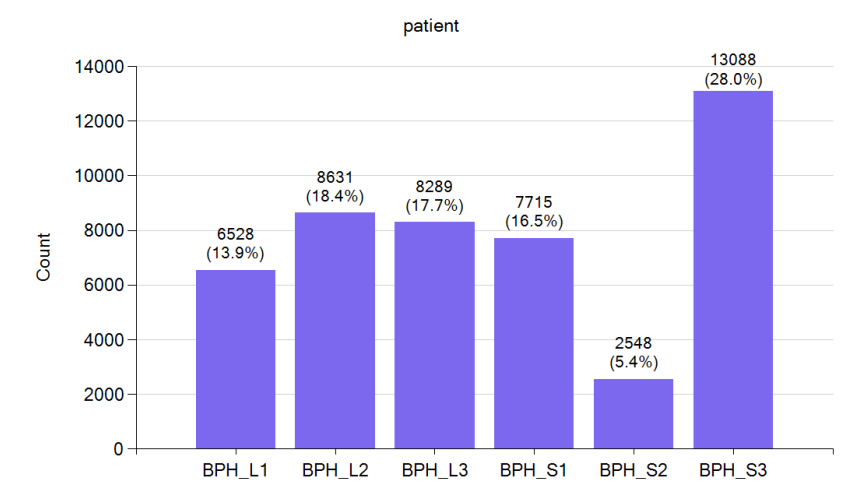

Figure 3. the *GSE226237* single-cell data were obtained from three patients with small BPH and three patients with large BPH.

Differential Gene Expression – Small BPH compared to Large BPH single cells

Table 1 presents the top 10 genes that are either downregulated or upregulated when comparing small BPH and large BPH single cells. Downregulation and upregulation refer to the decrease or increase in gene expression, respectively.

Table 1. Top 10 downregulated and upregulated genes through single-cell gene analysis in large BPH compared to small BPH single cells.

Small BPH compared to Large BPH single cells			
Downregulated in Large BPH		**Upregulated in Large BPH**	
Gene Symbol	**Gene Name**	**Gene Symbol**	**Gene Name**
HLA-B	Major Histocompatibility Complex, Class I, B	*SLC14A1*	Solute Carrier Family 14 Member 1 (Kidd Blood Group)
HLA-A	Major Histocompatibility Complex, Class I, A	*KRT5*	Keratin 5
HLA-E	Major Histocompatibility Complex, Class I, E	*KRT14*	Keratin 14
TSC22D3	TSC22 Domain Family Member 3	*KRT15*	Keratin 15
AHNAK	AHNAK Nucleoprotein	*PRAC1*	PRAC1 Small Nuclear Protein
MYH9	Myosin Heavy Chain 9	*CSTA*	Cystatin A
VIM	Vimentin	*RPL36A*	Ribosomal Protein L36a
ITGA1	Integrin Subunit Alpha 1	*GABARAP*	GABA Type A Receptor-Associated Protein
HLA-C	Major Histocompatibility Complex, Class I, C	*RPS17*	Ribosomal Protein S17
TXNIP	Thioredoxin Interacting Protein	*FXYD3*	FXYD Domain Containing Ion Transport Regulator 3

In large BPH, several genes are downregulated, indicating a potential reduction in their activity as the condition progresses. Among them are the HLA-A, HLA-B, HLA-C and HLA-E genes, which are key components of the major histocompatibility complex (MHC) class I system, responsible for presenting intracellular antigens to the immune system. Studies have demonstrated a marked upregulation of HLA-DR expression, notably in prostate epithelial cells and infiltrating CD45+ immune cells, in BPH, in contrast to its minimal expression in normal prostate tissue [2]. The

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glucocorticoid-induced leucine zipper (TSC22D3-2) is a widely expressed dexamethasone-induced transcript that has been proposed to be important in immunity, adipogenesis, and renal sodium handling based on in vitro studies [3]. AHNAK a nucleoprotein plays a critical role in the regulation of calcium channels, blood-brain barrier formation, embryonic development, lipid metabolism, membrane repair, inflammatory responses and other processes [4]. Conversely, it has been reported that the pathogenesis of BPH is associated with a unique type of myosin heavy chain proliferation [5] and the mesenchymal marker vimentin (VIM) is expressed at higher levels in hyperplastic human prostatic epithelium compared to normal tissue [6]. Modulating adhesion molecule expression such as integrin (ITGA1) can regulate cell proliferation, apoptosis, EMT, and fibrotic processes, engaged in the development of prostatic hyperplasia [7]. The role of TXNIP is not clear in BPH but acts as a tumor suppressor in human prostate cancer [8].

Genes upregulated in large BPH reflect enhanced activity of structural and regulatory proteins. SLC14A1, involved in urea transport, becomes more active, perhaps in response to increased metabolic demands in the larger prostate tissue. However, it has been shown that the expression of SLC14A1 is significantly reduced in prostate cancer cells and tissue comparing to normal prostate epithelial cell and para-cancerous tissue [9]. The keratin genes KRT5, KRT14, and KRT15, all associated with epithelial cell structure and integrity, show increased expression, possibly indicating greater structural reinforcement as the tissue expands [10]. Selective expression of KRT13 and PRAC1 was also documented in prostate stem cells relative to progenitors and knockdown experiments confirmed their critical role in maintaining stemness properties [11]. Cystatins (CSTA) are a family of protease inhibitors, found both inside and outside cells, that specifically inhibit cysteine cathepsins. These lysosomal cysteine proteases play a role in various biological processes, such as protein degradation and post-translational modifications [12]. Additionally, prostate cells require a constant supply of proteins for growth and division. Ribosomal proteins (RPL36A and RPS17) facilitate this process by ensuring adequate protein synthesis [13].

Overexpressed Genes in BPH and AI-Driven Natural Therapeutics

Table 2 highlights the top 10 overexpressed genes in BPH and the corresponding natural compounds proposed to regulate their activity. The compounds were identified using *ChatGPT* with the following prompt:

> "*Act as an expert in naturopathic medicine, nutrigenomics, and molecular biology. A list of genes is overexpressed in benign prostatic hyperplasia (BPH). For each gene, recommend one natural compound (such as a phytochemical, plant extract, vitamin, mineral, or natural metabolite) that may downregulate the gene's expression or inhibit its biological pathway. Base your recommendations on known molecular mechanisms, signaling pathways, or published experimental evidence when possible. Prioritize compounds known to influence inflammation, androgen signaling, cell proliferation, oxidative stress, or prostate biology, as these are relevant to BPH. If direct evidence for a specific gene is unavailable, suggest a compound that modulates the closest relevant pathway or transcriptional regulator. Output format (table): Gene | Natural Substance | Proposed Mechanism of Downregulation | Supporting Evidence (if known) Rules: Provide exactly one natural substance per gene. Prefer well-known bioactive compounds over vague herbal mixtures. Keep explanations concise (1–2 sentences per mechanism).*"

Table 2. Top 10 overexpressed genes in the large BPH and the recommended natural products by ChatGPT.

Overexpressed Genes in BPH and Natural Compounds Recommended by ChatGPT		
Gene	Natural Compound	Rationale
SLC14A1	Resveratrol from red grapes	Experimental studies show resveratrol reduces inflammatory signaling and alters gene expression profiles in prostate and epithelial cells. [14]

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KRT5	Curcumin from turmeric	Multiple in-vitro studies show curcumin suppresses keratinocyte proliferation and downregulates keratin-associated pathways. [15]
KRT14	Epigallocatechin gallate (EGCG) from green tea	EGCG shown to regulate keratinocyte gene expression and reduce epithelial hyperplasia in experimental models. [16]
KRT15	Genistein from soy	Studies in prostate and epithelial cells show genistein alters differentiation markers and suppresses proliferation. [17]
PRAC1	Quercetin from onions or apples	Quercetin reported to inhibit prostate cell proliferation and modulate androgen-responsive gene pathways. [18]
CSTA	Sulforaphane from broccoli sprouts	Sulforaphane from cruciferous vegetables widely shown to regulate cysteine-protease inhibitor pathways and antioxidant genes. [19]
RPL36A	Berberine from Berberis species	Evidence shows berberine downregulates ribosome biogenesis and protein synthesis pathways in proliferating cells. [20]
GABARAP	Ashwagandha	Resveratrol is widely reported to regulate autophagy signaling and associated gene expression. [21]
RPS17	Epigallocatechin gallate (EGCG) from green tea	Experimental studies demonstrate EGCG inhibits ribosome biogenesis and cell growth signaling. [16]
FXYD3	Lycopene from tomatoes	Lycopene has been reported to influence prostate epithelial gene expression and reduce proliferation in prostate models. [22]

Discussion

Benign prostatic hyperplasia (BPH) is a multifactorial condition characterized by progressive enlargement of the prostate gland driven by complex interactions between epithelial cells, stromal cells, immune signaling, and metabolic pathways. The present single-cell transcriptomic analysis comparing small and large BPH tissues reveals distinct gene expression patterns that provide insight into molecular mechanisms associated with disease progression. Specifically, the observed differential expression suggests that large BPH is characterized by changes in immune regulation, cytoskeletal remodeling, epithelial proliferation, and increased protein synthesis, all of which may contribute to the expansion of prostate tissue.

A notable finding is the downregulation of several major histocompatibility complex (MHC) class I genes, including *HLA-A, HLA-B, HLA-C, and HLA-E*, in large BPH. These genes play an essential role in antigen presentation and immune surveillance. Their reduced expression may reflect alterations in immune signaling within hyperplastic prostate tissue. Previous studies have reported increased expression of other immune markers, such as *HLA-DR*, in prostate epithelial cells and infiltrating immune cells in BPH, indicating that immune regulation in the prostate may shift during disease progression. Such changes could contribute to a chronic inflammatory microenvironment that supports tissue remodeling and cellular proliferation. Other downregulated genes identified in large BPH include *TSC22D3*, *AHNAK*, *MYH9*, *VIM*, *ITGA1*, and *TXNIP*, each of which plays roles in cellular signaling, structural organization, or metabolic regulation. *TSC22D3* is a glucocorticoid-responsive gene implicated in immune modulation and cellular stress responses. *AHNAK* has been associated with calcium channel regulation and membrane repair, processes important for cellular homeostasis. The reduction of *MYH9* and *VIM* expression is notable because both proteins are involved in cytoskeletal organization and cellular motility, which may reflect altered structural dynamics in enlarged prostate tissue. Additionally, *ITGA1*, an integrin involved in cell adhesion and extracellular matrix interactions, has been implicated in regulating cell proliferation, apoptosis, and

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epithelial-mesenchymal transition (EMT), all processes relevant to tissue remodeling in BPH. Although the role of *TXNIP* in BPH remains unclear, its established tumor-suppressive function in prostate cancer suggests that its downregulation could influence redox regulation and metabolic pathways within hyperplastic prostate cells.

In contrast, the genes upregulated in large BPH largely reflect enhanced epithelial structure, increased cellular proliferation, and elevated biosynthetic activity. For example, *SLC14A1*, a urea transporter, may be upregulated in response to increased metabolic activity within enlarged prostate tissue. Interestingly, previous studies have shown that *SLC14A1* expression is reduced in prostate cancer compared with normal prostate tissue, suggesting that its expression may differ between benign and malignant prostate conditions. Several keratin genes, including *KRT5*, *KRT14*, and *KRT15*, were also significantly upregulated in large BPH. These genes encode intermediate filament proteins that contribute to epithelial cell structure and integrity. Their increased expression may reflect expansion of basal epithelial cell populations or enhanced structural reinforcement as prostate tissue grows. Keratin expression is often associated with stem and progenitor cell populations, and previous studies have demonstrated that keratin-associated genes and prostate-associated transcripts such as *PRAC1* are selectively expressed in prostate stem cells and contribute to the maintenance of stemness properties. The increased expression of these genes in large BPH may therefore indicate expansion of progenitor-like epithelial populations within hyperplastic tissue. Additional upregulated genes include *CSTA*, *RPL36A*, *GABARAP*, *RPS17*, and *FXYD3*, which are associated with protease regulation, protein synthesis, autophagy, and ion transport. *CSTA* encodes a cysteine protease inhibitor that regulates cathepsin activity and may influence extracellular matrix remodeling and inflammatory processes. The upregulation of ribosomal proteins such as *RPL36A* and *RPS17* suggests increased translational activity to support cell growth and proliferation within enlarged prostate tissue. *GABARAP* is involved in intracellular trafficking and autophagy, processes that help maintain cellular homeostasis under stress conditions. Meanwhile, *FXYD3* regulates ion transport through Na^+/K^+-ATPase modulation and has been implicated in epithelial cell physiology and proliferation.

In addition to characterizing gene expression changes associated with BPH progression, this study also explored the potential use of artificial intelligence to generate hypotheses for natural therapeutics targeting overexpressed genes. Using a structured prompt, a large language model was queried to identify natural compounds that may downregulate or modulate the activity of genes upregulated in BPH. The proposed compounds, including *resveratrol*, *curcumin*, *epigallocatechin gallate* (EGCG), *genistein*, *quercetin*, *sulforaphane*, *berberine*, *ashwagandha*, and *lycopene*, have previously been reported to influence biological pathways relevant to prostate health, such as inflammation, oxidative stress, androgen signaling, and cellular proliferation.

Several of these compounds have well-documented biological activities that may be relevant to BPH pathophysiology. For example, *resveratrol* exhibits anti-inflammatory and antioxidant properties and has been shown to modulate epithelial cell proliferation. *Curcumin* and *EGCG* are known to regulate inflammatory pathways such as NF-κB signaling and have demonstrated anti-proliferative effects in various epithelial tissues. *Genistein*, a soy isoflavone, can influence estrogen receptor signaling and has been investigated for its potential protective effects in prostate disorders. *Sulforaphane*, derived from cruciferous vegetables, activates the Nrf2 antioxidant pathway and may modulate protease activity and inflammatory signaling. Similarly, *berberine* has been shown to regulate metabolic and protein synthesis pathways, while *lycopene*, a carotenoid abundant in tomatoes, has long been associated with prostate health and reduced oxidative stress.

It is important to emphasize that the AI-generated compound recommendations presented in this study should be considered hypothesis-generating rather than definitive therapeutic suggestions. While many of the identified compounds have documented biological activities and potential benefits for prostate health, direct experimental validation is necessary to confirm whether these substances can specifically regulate the expression of the identified genes in BPH tissue.

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Nonetheless, this approach illustrates the potential value of integrating transcriptomic data with artificial intelligence tools to accelerate the identification of candidate therapeutic compounds.

Summary

Benign prostatic hyperplasia (BPH) is a multifactorial condition involving complex interactions between epithelial, stromal, immune, and metabolic processes that lead to progressive prostate enlargement. In this study, single-cell transcriptomic analysis comparing small and large BPH tissues revealed distinct gene expression patterns associated with disease progression. Large BPH was characterized by downregulation of immune-related and structural genes, including several MHC class I genes and regulators of cytoskeletal organization and cellular signaling, alongside upregulation of genes related to epithelial structure, basal cell expansion, protein synthesis, and metabolic activity. These findings suggest that immune modulation, epithelial proliferation, and increased biosynthetic activity contribute to prostate tissue growth in advanced BPH. In addition, an artificial intelligence–based hypothesis generation approach was used to identify natural compounds that may modulate overexpressed genes, highlighting several bioactive substances with known anti-inflammatory, antioxidant, and anti-proliferative properties. Although these compounds require experimental validation, the study demonstrates how integrating single-cell transcriptomics with AI tools can provide new insights into BPH biology and generate potential therapeutic hypotheses.

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