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Research Article

Direct Bacteria Translocation, Migration, Seeding Between Gut and Prostate Gland: Hypotesys, Theory and Facts: State of the Art

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ABSTRACT
The hypothesys of the direct translocaton of gut bacteria into the prostate expecially in specific condition if an fascinanting theory : aim of this work is to find literature involved , analyzing and producing an general conclusion useful to better understand the pathogenesys of various kind of prostatitis .
An experimental project is submitted to experimentally verity this process.

KEYWORDS: gut bacteria, prostatitis, direct traslocation indirect traslocation, seedings, migration, leaky gut hypothesys, theory, Literature.

INTRODUCTION

The so called Gut -prostate axis seem not to be connected only in and indirect way but probably it is more complex and it must to be deeply studied:

According T. Prevodoros

"The connection between the gut bacteria GB and prostatitis runs along 3 documented ways:

1. direct seeding: gut microbes migrate to the urinary tract and prostate through shared anatomy, lymphatic channels and circulation.
2. chemistry: dying Gram negative (-) bacteria release LPS, an endotoxin that slips through a weakened barrier and drives body-wide inflammation.
3. immune tone: an imbalanced microbiota pushes the immune system IS toward a pro-inflammatory Th17 profile whose effects reach distant organs, the prostate included."

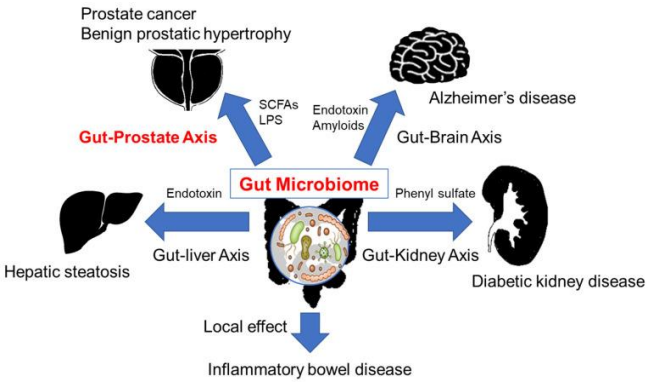

Fig. No. 1: Local effect and gut-distant organ axis via gut microbiome.

The endotoxins and the metabolite from the gut microbiome affect the distant organs, SCFA: short-chain fatty acid, LPS: lipopolysaccharides.

From Kazutoshi Fujita et al 2023.
And related animal model

H. Wolochow et al in past (1966) writed about the passage of bacteria fro mthe intestinal tract trought the lymph and blood in small animals:

TRANSLOCATION OF MICROORGANISMS ACROSS THE INTESTINAL WALL OF THE RAT: EFFECT OF MICROBIAL SIZE AND CONCENTRATION*

H. WOLOCHOW, G. J. HILDEBRAND AND C. LAMANNA†
From the Naval Biological Laboratory, University of California, School of Public Health, Berkeley, California 94625

Previous reports from this laboratory have dealt with the passage of botulinum toxin and of 2 microorganisms from the intestinal tract into the lymph and blood of small animals. Intraduodenally instilled toxin was found in the lymph and exhibited a sedimentation coefficient with the dimensions of protein (Heckly et al, 1960). *Serratia marcescens* and coliphage T1 passed the intestinal barrier into the lymph directly but did not pass into the blood (Hildebrand and Wolo-

instilled into the duodenum separately or in combination: *Schizosaccharomyces pombe*, *Saccharomyces cerevisiae*, *Bacillus cereus*, *Bacillus brevis* spores, *S. marcescens*, and coliphage (strain T1). Whenever feasible, the effect of concentration upon passage of the microorganisms through the intestinal wall was compared in the same animal by hourly instillations of increasing (10-fold) concentrations of the suspended organisms administered in a constant volume (2 to

Kentaro Takezawa et ain 2021 reported also that
"The F/B ratio of the gut microbiota GM was associated with prostate enlargement. Although the detailed mechanisms are unclear, the gut microbiota might affect prostate enlargement."

Cheng Shen et al in 2024
"We found a positive (+) association between prostatitis risk and four gut microorganisms: Faecalibacterium (OR = 1.591, 95% CI: 1.082–2.340, p = 0.018), LachnospiraceaeUCG004 (OR = 1.639, 95% CI: 1.147–2.343, p = 0.007), Sutterella (OR = 1.578, 95% CI: 1.135–2.194, p = 0.007), Gastranaerophilales (OR = 1.476, 95% CI: (1.104–1.972), p = 0.008), this implies that these bacteria may raise the risk of prostatitis."

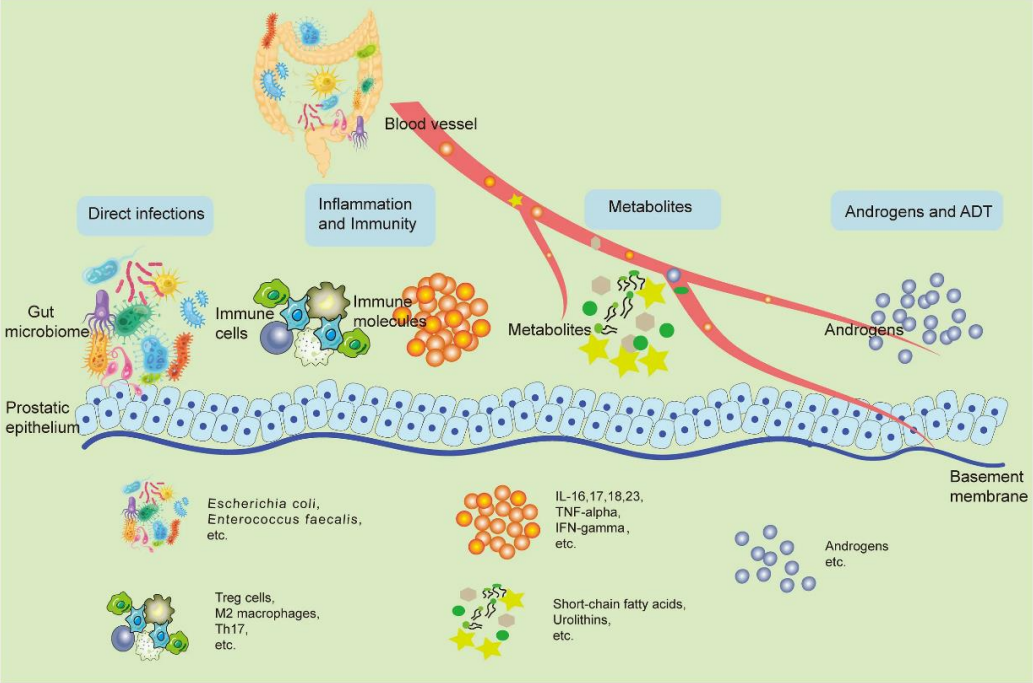

Fig. No. 2: The potential mechanisms involved with the GM and its impact on prostatic disease pathogenesis. The GM can shadow prostate health in both direct and indirect ways. H.Cao et al 2024.

Shang Weon Pak et al 2024 reported that
"The gut and the prostate are connected through various pathways, including the lymphatic system LS , the circulatory system CS , and the shared urinary and GI . This anatomical / functional connection suggests that bacteria from the gut can potentially migrate to the prostate, leading to an inflammation. Dysbiosis, or an imbalance in the gut microbiota GM , might play a role in this process."

By t. kenny 2015 patient.info/health/acute-prostatitis
"Sometimes the prostate is infected by bacteria in the bloodstream BS that have travelled from other infections in the body."

MATERIAL AND METHODS

With an observational point of view various relevant literature are reported for the scope of this work.
All reference comes from Pubmed or other scientific biomedical database.
Figure (1-5) help in the genereal meaning.
An experimental project is submitted
After all this a global conclusioni s finally provided.

RESULTS

From literature

according Jie Chenaet al
"dysregulation of the gut microbiota significantly reduces the SCFA content in feces and impairs the integrity of the gut barrier, leading to the translocation of bacteria and bacterial components such as lipopolysaccharide LPS , mediating the development of prostate inflammation through microbe-associated molecular patterns ."[1]

Jie Chen et al written
"When "gut dysbiosis" occurs, the number of microorganisms producing SCFAs in the gut decreases, and the number of harmful bacteria increases, resulting in damage to the intestinal barrier and "leaky gut" syndrome LGS , which leads to the translocation of bacteria and bacterial component lipopolysaccharide and causes an systemic inflammation. Bacteria and LPS bind to TLRs as MAMPs, upregulate the expression of NF-κB, and activate the NLRP3 inflammasome. Activated NLRP3 activates caspase-1, then activated caspase-1 promotes the maturation of pro-IL-1β and pro-IL-18 to IL-1β and IL-18. LPS can also bind to other PRRs and activate innate immune cells IIC to secrete IL-17, IL-23, TNF-α, and IFN-γ to mediate the occurrence of inflammation. LPS and various inflammatory factors IF travel through the circulation to the prostate and cause an inflammatory response IR in the prostate."[2]

Jhommara Bautista et al
"The concept of the gut-urinary-prostate axis GUPA underscores a hierarchically organized microbial network in which metabolites, immune mediators, and microbial molecules act as shared effectors across compartments. Mechanistically, this axis operates through 3 primary routes: metabolic circulation, immune cross-priming, and direct microbial translocation.

Whole bacteria or bacterial extracellular vesicles BEV can translocate from the gut to the urinary tract UT via hematogenous or lymphatic routes. Identical bacterial genotypes, such as Cutibacterium acnes, Porphyromonas, and Ruminococcus, have been isolated from gut, urine, and prostate samples, suggesting an bidirectional migration and ecological seeding of the prostatic niche. Vesicle-mediated transfer of microbial RNA, metabolites, and quorum-sensing molecules across these compartments may further reinforce the oncogenic signaling networks.

The urinary microbiome UM represents both a conduit and a reflection of prostatic microbial ecology, with high-PSA PCa patients showing reduced prostatic fluid microbial diversity and a differential enrichment of Enterobacter, Lactococcus, Streptococcus compared to controls . These genera, present in both urinary and gut compartments, may signify **translocation events through hematogenous or lymphatic routes LR , or via contiguous mucosal surfaces.**

Current evidence suggests that microbial translocation from both the gut and the genitourinary tract constitutes a plausible source of prostatic colonization PC, expecially in states of impaired epithelial barrier function or immune modulation"[3]

An-Qi Deng et al
"The mechanism through which oral microbiota affects CP/CPPS pathogenesis remains elusive, with **the available evidence principally supporting 2 primary direct pathways, microbial translocation and indirect systemic inflammation.** For the direct pathway, previously mentioned results suggest the hypothesis that hematogenous dissemination HD of oral microorganisms to the prostate triggers histological changes that contribute to the occurrence of CP/CPPS . For the indirect pathway, increasingly circulating levels of periodontal-derived pro-inflammatory factors, including interleukins (IL-1β, IL-2, IL-6, IL-8), TNF-α, and CRP, may act as indirect agents in the onset of CP/CPPS "[4]

Cheng Zha et al
"Bacterial translocation BT :The process of intestinal microorganisms and their metabolites transferring to mesenteric lymph nodes or portal veins through the intestinal mucosal barrier and entering other organs is called "**bacterial translocation BT " . It seems that intestinal microorganisms can directly affect PCa through bacterial translocation or toxins and may also be indirectly affected by intestinal microbiota, including metabolites, immunity, genes, and the effects of anti-tumor drugs.**[5]

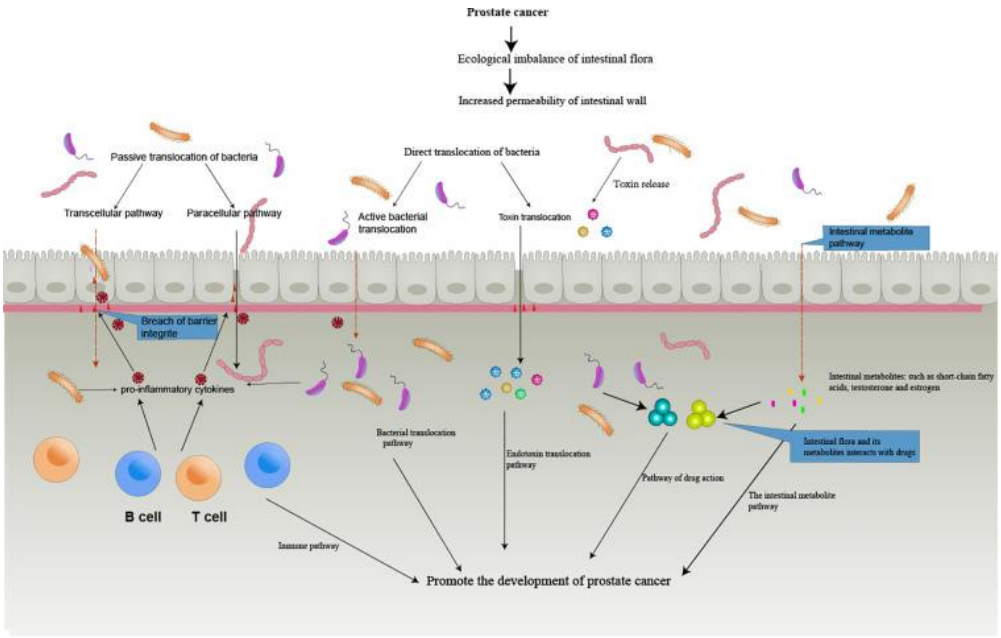

Fig. No. 3: The Intestinal bacterial translocation and toxin translocation.

The imbalance of intestinal flora leads to the destruction of the defense mechanism between the intestinal mucosa and the intestinal immune system, and the release of various inflammatory and cytokines destroys the epithelial cell barrier ECB , leading to the **active and passive translocation of bacteria**, toxins, bacterial metabolites, and interactions with drugs that eventually enter the circulation, thus promoting the formation of tumors. From Front Oncol. 2023 doi: 10.3389/fonc.2023.1196217 Potential role of gut microbiota in prostate cancer: immunity, metabolites, pathways of action? Cheng Zha et al

Xu, Y.. et al.
"Changes in the abundances of specific gut microbiota GM and their metabolites, such as an increased F/B ratio and a decreased abundance of Lactobacillus LB , as well as the levels of inflammatory indicators and markers of intestinal barrier dysfunction IBD , may play a crucial role in the pathogenesis of BPH."[6]

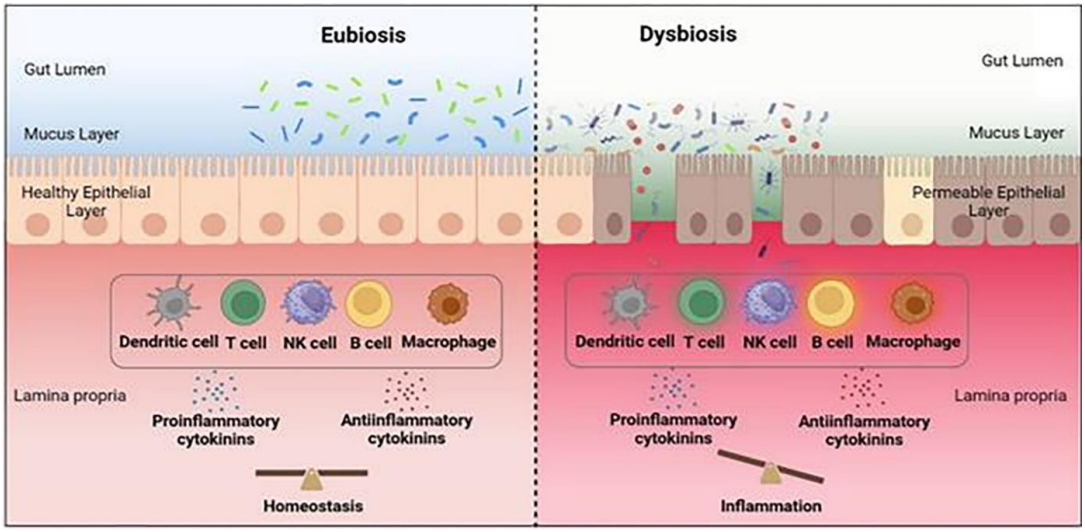

Fig. No. 4: Immune response during eubiosis and dysbiosis gut.

The gut-associated immune system mainly comprises dendritic cells, macrophages, T cells, B cells, natural killer cells, and other subsets. The gut microbiome GM maintains the intact and healthy gut structurally, bearing the lumen, mucus layer, lamina propria, and healthy epithelial cells in the intestinal barrier IB and the associated immune response IR . Eubiosis represents healthy gut immunity and the homeostasis of the immune system through the secretion of balanced pro-inflammatory and anti-inflammatory cytokines. Dysbiosis disrupts membrane integrity and promotes inflammation through the unbalanced secretion of pro-inflammatory and anti-inflammatory cytokines. From Ritis K Shyanti et al 2025

Bangwei Che et al
"some E. coli in the intestinal tract have the ability to adhere to and invade intestinal epithelial cells and survive in macrophages. These E.coli can reach the lamina propria of the intestinal tract through paracellular or cross-cellular pathways . Macrophages may then transfer bacteria from the intestines to lymph nodes, systemic circulation, or even distant organs to produce inflammation . This may also explain why some clinical studies have found that the same kind of E. coli has been detected in the intestines, blood, and urine."[7]

Jhommara Bautista et al
"Microbial translocation and vesicle-mediated trafficking.

Whole bacteria or bacterial extracellular vesicles BEV can translocate from the gut to the urinary tract UT via hematogenous or lymphatic routes LR. Identical bacterial genotypes, such as Cutibacterium acnes, Porphyromonas, and Ruminococcus, have been isolated from gut, urine, and prostate samples**, suggesting bidirectional migration and ecological seeding of the prostatic niche.**

Considering the anatomical and functional continuity between gut, urinary tract UT and prostate, microbial metabolites or whole bacteria may traffic across compartments, integrating local dysbiosis into systemic tumor-promoting networks. Hurst et al. identified anaerobic genera, including novel Porphyromonas, Varibaculum, Peptoniphilus, and Fenollaria species, whose presence in urine sediment, urinary extracellular vesicles UEV , and cancer tissue correlated with higher D'Amico risk groups and metastatic progression, conferring a meta-analysis hazard ratio of 2.60 for disease progression . These organisms may traffic between the urinary tract UT and prostate via ascending infection AI or vesicle-mediated transfer VMT , as suggested by the detection of identical taxa across these niches, with potential prognostic utility."[8]

Romano L. et al
"Along with that, anatomical proximity, cross-organ sensitization,gut dysbiosis, bacterial translocation BT , and immune system overstimulation by bacterial antigens may favor prostate diseases.

An increased intestinal epithelial permeability IEP , which might be accounted for by altered GM and its metabolites, may therefore favor the passage of microorganisms or toxic agents to the LUT, including prostate, thus favoring the development of pathologic conditions. The intestinal epithelial barrier IEB plays a critical role in maintaining the integrity of the GI tract and represents the first line of defense of our body against external noxious agents including toxins and pathogens. IEB is a well-regulated structure, and the integrity of the mucous surface and of the epithelial lining is responsible of its permeability which in turn may influence intestinal homeostasis.

Intestinal epithelial cells IEC are tied to each other by intercellular junctional complexes IJC such as tight junctions, adherence junctions, gap junctions, and desmosomes that are constituted of a set of proteins that exert a crucial role in cell-to-cell adhesion (adherence junctions , desmosomes) and molecular transport systems. Pathological events such as hypoxic injury, inflammation, apoptosis, necrosis may cause dysregulation of paracellular transport and IEB protective regulatory function.

Oxygen free radicals can directly damage mucus and create an pro-inflammatory environment activating cytokines (TNF α, IL-2, IFN-γ), which in turn, damage IEB and result in leakage of diverse antigens into the depth of the intestinal wall IW , thus promoting further secretion of proinflammatory cytokines and their entrance into systemic blood flow via enterohepatic circulation.

This cascade of events is described as the "leaky gut syndrom e"LGS being a vicious circle of pathologic events, where dysbiosis, intercellular junctions' disruption with passage of patho gens and microbial-derived products through the intestinal barrier IB and their systemic translocation, boost of proinflammatory cytokines PC and immune cells overstimulation ICO , are the key components."[9]

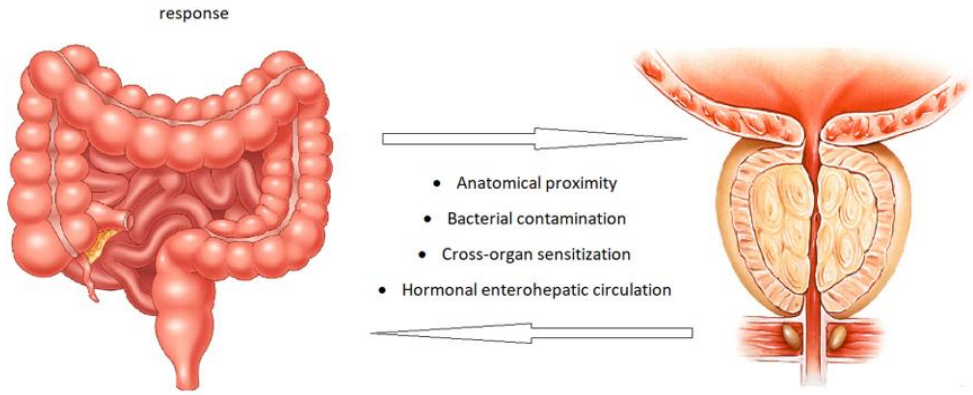

Fig. No. 5: from L.Romano et al 2024 the Gut-prostate axis.

Jinbo Liu et al
"Bacterial translocation BT is a phenomenon in which live bacteria or their products cross the intestinal barrier to other organs or the circulatory system. Gut translocation of bacteria has been reported in both animal models, and clinical trials often accompany acute pancreatitis and are believed to be linked to patient outcome, especially in severe acute pancreatitis."[10]

Cheng Shenet al
"We found a positive (+) association between prostatitis risk and four gut microorganisms: Faecalibacterium (OR = 1.591, 95% CI: 1.082–2.340, p = 0.018), LachnospiraceaeUCG004 (OR = 1.639, 95% CI: 1.147–2.343, p = 0.007), Sutterella (OR = 1.578, 95% CI: 1.135–2.194, p = 0.007), and Gastranaerophilales (OR = 1.476, 95% CI: (1.104–1.972), p = 0.008), this implies that these bacteria may raise the risk of prostatitis. Sensitivity analysis failed to find any proof of pleiotropy at any level. 4 groups of the gut microbiota underwent weighted median analysis, and the directionality of these results agreed with IVW."[11]

Sun, L. et al
"In this study work , we confirmed the existence of BT from the gut to extraintestinal tissues (MLNs, liver, and spleen) in a uremic rat by using GFP-labeled tracer bacteria. Macrophages and GFP-labeled tracer bacteria were colocalized, suggesting that macrophages loaded with bacteria cross the intestinal mucosa IM to promote BT in the uremic state."[12]

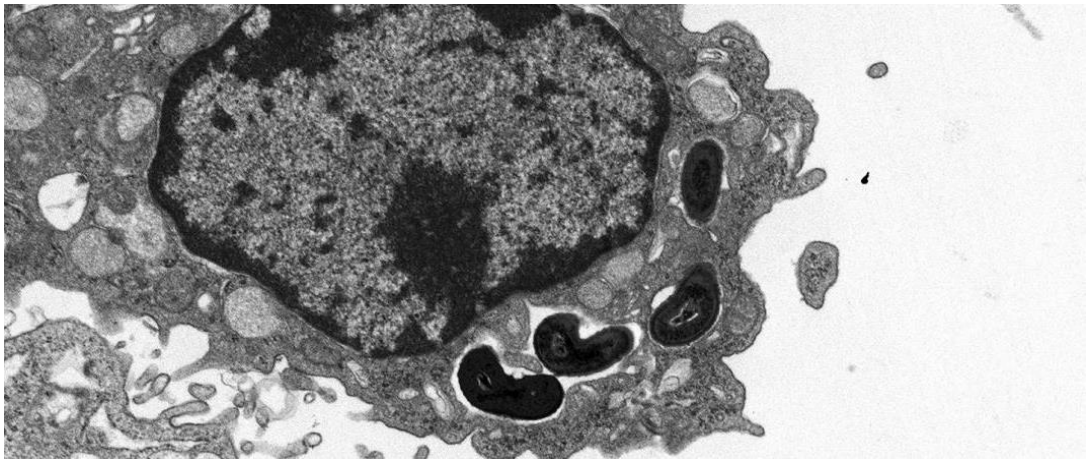

Fig. No. 6: Electron microscopy EM showing a macrophage three hours after infection with the Gram-positive bacteria Listeria innocua. Phagocytosis of live bacteria elicits specific cellular processes and innate immune response not observed with killed bacteria.From J. M. Blander and J. Moretti.

Filipa Mota et al
"Molecular imaging MI for infectious diseases is an emerging field with tremendous potential for both preclinical and clinical applications. Although many targets and screening approaches have been explored, this field would get a major boost from unbiased screens to identify novel biological targets and chemical leads."[13]

Sipho Mdanda et al
"Macrophages serve as effector cells within the innate immune system IIS, performing the critical function of phagocytosing bacteria and secreting both pro-inflammatory and antimicrobial mediators."[14]

From T. PREVEDOROS
"Bacterium or daily habit / What feeds it and why your prostate cares :
Escherichia coli and other Enterobacteriaceae: Refined sugar and low-fiber, ultra-processed eating favor pro-inflammatory gut taxa. This is the same family that forms the intestinal reservoir reseeding the urinary tract UT and prostate.

Klebsiella and Morganella: Potent starch fermenters and histamine producers. Starch-heavy plates are their favorite substrate.

Artificial sweeteners: Saccharin-type sweeteners shift the whole community toward glucose-intolerance-promoting taxa, even when sugar itself is absent.

Prevotella bacteria Gram neg: (protective ally) Thrives on vegetables and fiber. Men with chronic pelvic pain syndrome carry less of it, so a fiber-poor plate starves a defender.

Methanobacteriaceae (protective ally): Methane-producing archaea linked to lower prostatitis risk in genetic data. Slow-transit, fiber-fed guts favor them.

A high intake of sugar tilts the balance toward pro-inflammatory bacteria and away from the producers of short-chain fatty acids, a pattern I broke down in my article on sugar and the gut microbiome.."

Experimental project hypotesys
In order to verify the theory of direct diffusion of gut bacteria in Prostatet tissue (trough macrophages or in other way like blood circulation or other) ti is possible to trace the gut bacteria whit an imaging treacer (PET , SPECT, 18 F Fluorosorbitol or other)

Gruop to be treated: 10 animal male model (mammifer like mouse)

1) Take a gut sample form every patients
2) Trace the gut bacteria (significative in exmaple E.Coli or other)
3) Re -introduce in the gut
4) After a significative time verify with imaging the presence of the treacer in the prostate or in local lymphatic or blood circle

Results: the presence of the treaced bacteria in the prostate means an traslocation of bacteria from the gut

DISCUSSION
Some Escherichia coli in the intestinal tract have the ability to adhere to and invade intestinal epithelial cells and survive in macrophages. Macrophages may then transfer bacteria from the intestines to lymph nodes, systemic circulation, or even distant organs to produce inflammation.[7]

Whole bacteria or bacterial extracellular vesicles can translocate from the gut to the urinary tract via hematogenous or lymphatic routes.[8]

According T.Kenny 2015: “Sometimes the prostate is infected by bacteria in the bloodstream that have travelled from other infections in the body.”

Variuous gut bacteria are involved also as responsible of bacterical prostatitis (even if it can not to be excluded an colonization trought anterior urethra: ascendig retrograde way).

In literature are reported various way related the hypotesys of transclocation of bacteria form the gut to the Prostate galed: trough lynphatic way, ematogen but also direct translocation.

Considering the anatomical and functional continuity between gut, urinary tract and prostate, microbial metabolites or whole bacteria may traffic across compartments, integrating local dysbiosis into systemic tumor-promoting networks.[9]

It was decribed also a vescicle mediate translocation of bacterial components thant can contribute to the inflamation process.

Dysbiosis (imbalance) harms the intestinal barrier. This allows bacteria like Escherichia coli to breach the epithelial layer and enter the lamina propria. Macrophages then ingest these bacteria and travel through the lymphatic system or bloodstream, potentially delivering them to the prostate.

Intestinal epithelial cells are tied to each other by intercellular junctional complexes such as tight junctions, adherence junctions, gap junctions, and desmosomes that are constituted of a set of proteins that exert a crucial role in cell-to-cell adhesion (adherence junctions and desmosomes mainly) and molecular transport systems.

Pathological events such as hypoxic injury, inflammation, apoptosis, necrosis may cause dysregulation of paracellular transport and IEB protective regulatory function.

The anatomical proximity contribute in this process as well as the lympatic and heematogenous circle.

Bacterial translocation is a phenomenon in which live bacteria or their products cross the intestinal barrier to other organs or the circulatory system. Gut translocation of bacteria has been reported in both animal models, and clinical trials often accompany acute pancreatitis and are believed to be linked to patient outcome, especially in severe acute pancreatitis.[10]

We found a positive association between prostatitis risk and four gut microorganisms:[11]

Macrophages can be vehicle transporters, immune signaling. (Macrophages may then transfer bacteria from the intestines to lymph nodes, systemic circulation, or even distant organs to produce inflammation B. CHE et al 2021)

In a uremic rat by using GFP-labeled tracer bacteria. Intriguingly, macrophages and GFP-labeled tracer bacteria were colocalized, suggesting that macrophages loaded with bacteria cross the intestinal mucosa to promote BT in the uremic state.”[12]

Molecular imaging for infectious diseases is an emerging field with tremendous potential for both preclinical and clinical applications.[13]

Macrophages serve as effector cells within the innate immune system, performing the critical function of phagocytosing bacteria and secreting both pro-inflammatory and antimicrobial mediators.”[14]

CONCLUSION
Gut bacteria, prostatitis, direct traslocation concepts, indirect traslocation, seedings, migration, leaky gut hypothesys, theory, Literature are the various faces of the same medal.

The contiguity anatomical way or trough lynphatic or ematogen way are the various mechanims involved with transfer of bacteria or its parts or its bioproducts..

Of this complex exhange way it is to to be considered when study the prostatitis word and also related the reacutization.

CONFLICT OF INTEREST

No

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