Acta Clin Croat (Suppl. 2) 2024; 63:56-62
doi: 10.20471/acc.2024.63.s2.9

Professional Paper

MICROBIOME AND PROSTATE CANCER

Ivana Mareković[1,2]

[1]Clinical Department of Clinical Microbiology, Prevention and Infection Control,University Hospital Centre Zagreb, Zagreb, Croatia
[2]School of Medicine University of Zagreb, Zagreb, Croatia

SUMMARY – According to scientific research, the microbiome and the likelihood of developing cancer are causally related. Current evidence indicates the probable absence of bacteria in a disease-free prostate. There is still a lack of conclusive evidence linking the diversity of gut microbiota and prostate cancer. The development and spread of prostate cancer seem to be significantly influenced by inflammation. With next generation sequencing methods, different bacterial species were found in the gut microbiome of patients with prostate cancer. The microbiome can affect the effectiveness of various chemotherapeutic drugs by altering the immune system and metabolic processes. Patients with and without radiation enteropathy have different microbiomes. The possibility of risk assessment, prevention, or treatment of radiation-induced enteropathy can be revealed by microbiome testing before and during pelvic radiation. It is essential to understand the complex relationship between the microbiome and prostate cancer. The manipulation of gut microbiome may enhance the effectiveness of cancer treatments. The opportunity to use the microbiome as a target for the prevention of this cancerous condition as well as an additional intriguing option in the management of prostate cancer and a more comprehensive therapeutic approach should not be missed.

Key words: *prostate cancer; microbiome; treatment; prevention; inflammation*

Introduction

According to Whipps et al., the term microbiome refers to a distinctive microbial community that inhabits a plausible, well-defined environment that has distinct physio-chemical features but also contains its "theatre of activity". The latter includes the full range of molecules made by the microorganisms, including their structural components (nucleic acids, proteins, lipids, polysaccharides), metabolites (signaling molecules, toxins, organic and inorganic molecules), and molecules produced by coexisting hosts and structured by the surrounding environmental conditions[1]. The term microbiome should be distinguished from the term metagenome, the latter being a grouping of genes and genomes from the microbiota. The microbiota consists of the living organisms (microorganisms themselves) of an ecosystem or a particular area[2].

The gastrointestinal system contains 99% of the human microbiome, which has both local and long-distance effects. The host is protected from pathogenic bacteria by the microbiome, which also controls metabolic processes. The microbiome is crucial for the growth of innate and adaptive immune systems as well as the preservation of homeostasis[3]. The impact of the human gut microbiota on health and disease has been studied throughout the past ten years. The invention of next-generation sequencing, which can recognize taxa at the species level as well as bacterial metabolic path-

Correspondence to:
Prof. Dr. Sc. Ivana Mareković
E-mail: imarekov@kbc-zagreb.hr

ways, made this possible. Intestines contain 10^{13}-10^{14} bacteria, and each person's gut microbiome is distinct, depending on a variety of factors such as food, exercise, genetics, gender, race, and geography. The "gut-liver axis" or the "gut-brain axis" relates to the connection between the gut microbiome and distant organs (liver, brain) via immunological and metabolic systems. Numerous disorders, including rheumatoid arthritis, Alzheimer's disease, and ulcerative colitis, have been linked to the gut microbiota[4].

Previous research has indicated that microbial infections are responsible for 15% to 20% of all cancer cases[5]. Scientific research has demonstrated a causal relationship between the microbiome and the likelihood of developing cancer. This relationship is bidirectional, as both the microbiome and cancer have the potential to alter it. Genomic integration, ongoing inflammation, genotoxicity, and immunological regulation are crucial mechanisms by which the microbiome influences the evolution of cancer[4]. Recent research has shown that chemicals produced by the gut microbiota influence the development and spread of prostate cancer, providing evidence of a "gut-prostate axis"[6]. Bacteria have long been thought to be a source of chronic, low-grade inflammation which may induce prostate cancer. It was in 1863 that Rudolf Virchow noted leucocytes in neoplastic tissues and made a connection between inflammation and cancer[7].

Urogenital and gut microbiomes as contributing factors in prostate cancer pathogenesis

The inability to obtain tissue samples free of disease made it challenging to determine whether microbiota are present in the prostatic tissue of healthy men. It seems that healthy prostatic tissue does not contain any bacteria, as was demonstrated by a study conducted on tissue samples in 18 organ donors. Specifically, samples used in this study underwent 16S rDNA PCR testing for bacteria but came back negative[8]. Components with antimicrobial activity present in the prostatic fluid including zinc, defensins, immunoglobulins, complement proteins, and lactoferrin, could be responsible for the absence of bacteria in these tissue samples. In comparison, when tissue samples from patients with benign prostate hyperplasia (BPH) and prostate cancer were tested, PCR detected bacterial rDNA[9].

According to studies, the microbiome can have a direct and indirect impact on the occurrence and progress of prostate cancer. Directly, the microbiome localized to the urogenital system influences prostate tissue and cancer pathogenesis. This mechanism is related to long-term inflammatory urinary tract diseases such as chronic prostatitis and BPH. The oral and gut microbiomes, as well as any changes to them, may indirectly affect how prostate cancer develops and spreads[10,11]. Metabolic activities carried out by the gut microbiota may be the cause of systemic inflammation leading to tumor development[3]. Therefore, inflammation appears to play an important role in the development and progression of prostate cancer. The prostate tissue frequently has a lesion called proliferative inflammatory atrophy. It is believed that the presence of inflammation in the tissue causes the formation of tumors through high levels of reactive oxygen species and genetic changes and instability[12,13].

Several studies examining prostatic cancer tissue and adjacent benign tissue found a connection btween prostate cancer and different bacterial genera including gram-positive (*Propionibacterium* species, *Staphylococcus* species) and gram-negative bacteria (phyla Proteobacteria, Firmicutes, Actinobacteria, Bacteroidetes; genera *Pseudomonas, Escherichia, Acinetobacter*)[14,15,16,17]. Different cellular genes connected to oncogenesis and their expression could be affected, as described by Banerjee et al., when *cagA* sequences specific for *Helicobacter* integrate into specific chromosomes of prostate tumor cells[16]. It has been suggested that *Propionibacterium acnes*, which is now known as *Cutibacterium acnes*, contributes to the growth of prostate cancer[18,19]. When analyzing surgical and biopsy samples, Miyake et al. found an independent link between prostate cancer and its advanced stages and *Mycoplasma genitalium*[20]. In a recent study, Salachan et al. discovered a significant excess of *Shewanella* in tissue samples from malignant prostates and hypothesized that this pathogen is responsible for downregulating toll-like receptors (TLRs) and reduced response of the host's immunological system caused by this pathogen[21].

When analyzing urine samples, Shrestha et al. found that *Streptococcus, Anaerococcus, Actinobaculum, Varibaculum*, and *Propionibacterium* were more common in urine samples from patients with prostate cancer[22]. However, possible urine sample contamination should be considered when interpreting the results. Before molecular sequencing methods were discovered and started to be used for microbiome research, there was a long-standing belief that urine from a healthy human urinary tract is sterile. This viewpoint was cer-

tainly impacted by the limitations of bacterial culture as a method that was used as a standard in clinical practice[9].

Several studies have explained the link between specific species of gut microbiomes and prostate cancer risk. Patients with and without cancer showed a significant difference when components of their gut microbiome were analyzed and compared in rectal swab samples, as described by Liss et al. *Bacteroides* and *Streptococcus* spp. were found in large numbers in patients with prostate cancer, but in comparison with controls, microbial diversity was not significantly different[23]. Golombos et al. demonstrated a difference in the gut microbiomes when patients with prostate cancer and BPH were compared: a relatively high number of *Bacteroides massiliensis* was found in patients with prostate cancer, whereas *Faecalibacterium prausnitzii* and *Eubacterium rectale* were found in patients with BPH[24]. Patients with prostate cancer have a greater abundance of *Bacteroides* spp. but lower alpha diversity in comparison with non-cancer patients[25,26]. Alpha diversity is the term used when evaluating sequencing data from a specific sample of the gut microbiome and can be described as within-sample diversity. In contrast, beta diversity measures the similarity or difference between populations (between samples)[27]. Another study conducted in the United States did not show a difference in alpha diversity between patients[23].

Viral causes have also been suggested for prostate cancer. Infections with polyomaviruses, human papilloma virus (HPV), and human cytomegalovirus (HCMV) are associated with infection of human prostate tissue and with a higher incidence of prostate cancer[28,29]. In a study by Banerjee et al. viruses were detected in formalin-fixed tissues of patients with prostate cancer. Of the viruses detected, 41% were known tumorigenic viruses, including high-risk HPV type 16 and 18 (present in >80% of samples) as well as HCMV. The associations between HPV 18, Kaposi's sarcoma-associated herpesvirus, and Polyomaviridae with lower Gleason score have been demonstrated[16].

The carcinogenic potential of microbial products has been investigated[10]. Metabolites of microbiota are also significant. Matsushita et al. demonstrated that the insulin growth factor-1 signaling pathway is used by intestinal bacteria and their metabolites, short-chain fatty acids (SCFAs), to promote the growth of prostate cancer in a mouse model. Additionally, the relative abundances of the SCFA-producing bacterial genera *Rikenellaceae*, *Alistipes*, and *Lachnospira* were markedly higher in the high-risk group. They suggested that the gut microbiota profile might be a new, helpful indica-

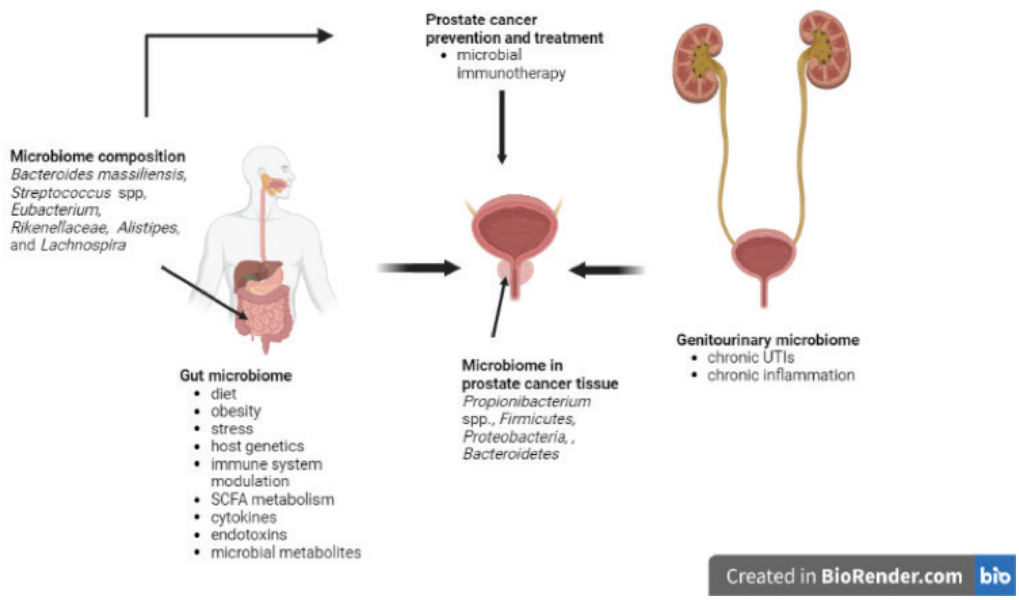

Figure 1. The complex interaction between gut microbiota and development, progression, and treatment of prostate cancer

SCFA=short chained fatty acids; UTI=urinary tract infection

tor for finding high-risk prostate cancer and may have an impact in the carcinogenesis of this malignant condition[30]. Manipulation of SCFA levels in the intestinal tract by altering the microbiota may be considered a possible strategy for the treatment and prevention of cancer[3]. The complex interaction between gut microbiota and development, progression, and treatment of prostate cancer is shown in **Figure 1**.

Microbiome and prostate cancer treatment and prevention

Management of prostate cancer is based on pharmacotherapy, particularly androgen deprivation therapy (ADT), surgery, chemotherapy, and radiotherapy. However, new options are needed, particularly in patients with higher stages of prostate cancer resistant to primary chemotherapeutic regimens.

A growing body of research has shown that the microbiome can impact the effectiveness of various medications, including chemotherapeutic agents. Cyclophosphamide is one of a number of clinically significant cancer medications whose capacity to elicit anti-tumor immune responses contributes to their therapeutic efficacy. According to a study by Viaud et al., cyclophosphamide shortens gut villi in mouse models, which then permits the translocation of specific gram-positive bacterial species (*Lactobacillus johnsonii, L. murinus*, and *Enterococcus hirae*) into secondary lymphoid organs (lymph nodes, tonsils, and the spleen) where they can stimulate the production of type T17 helper[31]. Through the control of immune cytokines and the release of innate myeloid cells, Lida et al. demonstrated that immunomodulation of chemotherapy drugs is helped by the intestinal microbiome and is mediated via immune cytokines balancing and innate myeloid cell release. Consequently, healthy commensal microbiota, which mediates its effects by modifying the functions of tumor-infiltrating myeloid-derived cells in the tumor microenvironment, is necessary for optimal responses to cancer therapy[32]. Additionally, bacteria in the gut control metabolic processes (reduction, hydrolysis, dihydroxylation, and dealkylation) that influence the effectiveness of various chemotherapeutic drugs. It has been shown that *Mycoplasma hyorhinis* can metabolize the prostate cancer drug gemcitabine into an inactive metabolite, reducing the drug's effectiveness[33].

However, there is a two-way relationship between the microbiome and chemotherapy. Chemotherapy has an effect on the makeup of the microbiome as well, as was shown in a study by Sfanos et al. The authors demonstrated variable gut microbiota compositions of men with prostate cancer receiving oral androgen deprivation therapy (ADT), such as bicalutamide, enzalutamide, and abiraterone acetate. One of the differences was a higher abundance of species such as *Akkermansia muciniphila* and *Ruminicoccaceae* spp.[26]. According to a study by Samykutty et al., oral abiraterone acetate administration causes a higher population of *A. muciniphila* to predominate in the fecal microbiome. In addition, they found that the biosynthesis pathway for vitamin K2, a potential anti-cancer agent, is consistently activated in gastrointestinal samples exposed to abiraterone acetate, which may contribute to the drug's effectiveness[34]. The findings of these studies demonstrate that the microbiome can alter the immune system and metabolic processes, which can affect the efficacy of various chemotherapeutic drugs. This makes the microbiome a target that may be modified to improve treatment response. In the future, clinicians may be able to identify which patients are more likely to respond favorably or adversely to particular cancer therapeutics by sampling and categorizing the fecal microbiome profiles of their patients[9]. Therefore, the manipulation of the gut microbiome may enhance the effectiveness of cancer treatments.

When radiation therapy is used, side effects such as gastrointestinal toxicity are the main cause for concern. According to a study by Reis Ferreira et al, patients with and without radiation enteropathy have different microbiomes. Additionally, in patients with progressive radiation enteropathy, microbiota diversity dynamically decreased over time. Late radiation enteropathy and low diversity are clearly related, and *Clostridium*, *Roseburia*, and *Phascolarctobacterium* abundances are higher in radiation enteropathy. The possibility of risk assessment, prevention, or treatment of radiation-induced enteropathy could be revealed by microbiome testing before and during pelvic radiation[35].

Research on bacterial immunotherapy is ongoing. Specific microorganisms can interact with vital processes such as proliferation and/or apoptosis and deliver exogenous genes to prostate cancer cells or their metabolites. Nonpathogenic bacteria or viruses currently have the potential to be used as targeted therapies. For instance, *Escherichia coli* can produce TNF-α, which can cause tumor cell apoptosis in mouse tumors. However, because of the severe systemic side effects,

this type of treatment is not in practical use yet. After potential biosafety issues and side effects are resolved, microbial immunotherapy may be able to overcome the limitations of conventional therapy[36].

The possible clinical application of testing prostate microorganisms would be an indication or recommendation for re-biopsy or close follow-up after negative results of the initial prostate biopsy[22]. Another practical and potential intervention would be targeting infectious agents with antibiotics to lower the risk of prostate cancer, given that the information obtained from non-invasive sampling (urine samples) accurately reflects the microbial environment in the prostate[22].

Numerous studies have examined the role of diet and associated obesity in raising the risk of prostate cancer. With gut dysbiosis and the release of gut microbial metabolites such as SCFAs and phospholipids that affect internal organs, a high-fat diet can change the composition of the gut's microbial population. The gut microbiome can be improved through dietary interventions, which can stop or delay the onset of prostate cancer. Promoting the consumption of prebiotics and/or probiotics, for instance, may encourage healthy gut flora that may lower the risk of developing prostate cancer[36].

Conclusion

According to recent research that suggests atrophy and inflammation have a role in prostate carcinogenesis, the microbiome may contribute to the development of an inflammatory environment in prostate tissue that may support the growth and progression of prostate cancer. There is still a lack of conclusive evidence linking the diversity of gut microbiota and prostate cancer. Understanding the intricate relationship between the microbiome and prostate cancer is crucial. It is important to elucidate the complex interaction between the microbiome and prostate cancer. We should not pass up the chance to use the microbiome as a target for the prevention of this cancerous condition as well as an additional intriguing option in the management of prostate cancer and a more comprehensive therapeutic approach.

References

1. Whipps J, Lewis K, Cooke R. Mycoparasitism and plant disease control. In: Burge M, editor. Fungi Biol Control Syst. Manchester University Press; 1988. p.161-87.
2. Berg G, Rybakova D, Fischer D, Cernava T, Vergès MC, Charles T, et al. Microbiome definition re-visited: old concepts and new challenges. Microbiome. 2020 Jun 30;8(1):103. doi: 10.1186/s40168-020-00875-0.
3. Yang HJ, Kim JH. Role of microbiome and its metabolite, short chain fatty acid in prostate cancer. Investig Clin Urol. 2023 Jan;64(1):3-12. doi: 10.4111/icu.20220370.
4. Schwabe RF, Jobin C. The microbiome and cancer. Nat Rev Cancer. 2013 Nov;13(11):800-12. doi: 10.1038/nrc3610.
5. Gopalakrishnan V, Helmink BA, Spencer CN, Reuben A, Wargo JA. The Influence of the Gut Microbiome on Cancer, Immunity, and Cancer Immunotherapy. Cancer Cell. 2018 Apr 9;33(4):570-80. doi: 10.1016/j.ccell.2018.03.015.
6. Fujita K, Matsushita M, Banno E, De Velasco MA, Hatano K, Nonomura N, et al. Gut microbiome and prostate cancer. Int J Urol. 2022 Aug;29(8):793-8. doi: 10.1111/iju.14894.
7. Balkwill F, Mantovani A. Inflammation and cancer: back to Virchow? Lancet. 2001 Feb 17;357(9255):539-45. doi: 10.1016/S0140-6736(00)04046-0.
8. Hochreiter WW, Duncan JL, Schaeffer AJ. Evaluation of the bacterial flora of the prostate using a 16S rRNA gene based polymerase chain reaction. J Urol. 2000 Jan;163(1):127-30.
9. Porter CM, Shrestha E, Peiffer LB, Sfanos KS. The microbiome in prostate inflammation and prostate cancer. Prostate Cancer Prostatic Dis. 2018 Sep;21(3):345-54. doi: 10.1038/s41391-018-0041-1.
10. Wheeler KM, Liss MA. The Microbiome and Prostate Cancer Risk. Curr Urol Rep. 2019 Sep 7;20(10):66. doi: 10.1007/s11934-019-0922-4.
11. Katongole P, Sande OJ, Joloba M, Reynolds SJ, Niyonzima N. The human microbiome and its link in prostate cancer risk and pathogenesis. Infect Agent Cancer. 2020 Aug 31;15:53. doi: 10.1186/s13027-020-00319-2.
12. Miyake M, Tatsumi Y, Ohnishi K, Fujii T, Nakai Y, Tanaka N, et al. Prostate diseases and microbiome in the prostate, gut, and urine. Prostate Int. 2022 Jun;10(2):96-107. doi: 10.1016/j.prnil.2022.03.004.
13. Sfanos KS, Yegnasubramanian S, Nelson WG, De Marzo AM. The inflammatory microenvironment and microbiome in prostate cancer development. Nat Rev Urol. 2018 Jan;15(1):11-24. doi: 10.1038/nrurol.2017.167.
14. Cavarretta I, Ferrarese R, Cazzaniga W, Saita D, Lucianò R, Ceresola ER, et al. The Microbiome of the Prostate Tumor Microenvironment. Eur Urol. 2017 Oct;72(4):625-631. doi: 10.1016/j.eururo.2017.03.029.
15. Feng Y, Ramnarine VR, Bell R, Volik S, Davicioni E, Hayes VM, et al. Metagenomic and metatranscriptomic analysis of human prostate microbiota from patients with prostate cancer. BMC Genomics. 2019 Feb 18;20(1):146. doi: 10.1186/s12864-019-5457-z.
16. Banerjee S, Alwine JC, Wei Z, Tian T, Shih N, Sperling C, et al. Microbiome signatures in prostate cancer. Carcinogenesis. 2019 Jul 6;40(6):749-64. doi: 10.1093/carcin/bgz008.
17. Australian Prostate Cancer BioResource; Yow MA, Tabrizi SN, Severi G, Bolton DM, Pedersen J, Giles GG, et al. Characterisation of microbial communities within aggressive prostate cancer tissues. Infect Agent Cancer. 2017 Jan 13;12:4. doi: 10.1186/s13027-016-0112-7.

18. Fassi Fehri L, Mak TN, Laube B, Brinkmann V, Ogilvie LA, Mollenkopf H, et al. Prevalence of Propionibacterium acnes in diseased prostates and its inflammatory and transforming activity on prostate epithelial cells. Int J Med Microbiol. 2011 Jan;301(1):69-78. doi: 10.1016/j.ijmm.2010.08.014.
19. Chen Y, Wei J. Identification of Pathogen Signatures in Prostate Cancer Using RNA-seq. PLoS One. 2015 Jun 8;10(6):e0128955. doi: 10.1371/journal.pone.0128955.
20. Miyake M, Ohnishi K, Hori S, Nakano A, Nakano R, Yano H, et al. *Mycoplasma genitalium* Infection and Chronic Inflammation in Human Prostate Cancer: Detection Using Prostatectomy and Needle Biopsy Specimens. Cells. 2019 Mar 2;8(3):212. doi: 10.3390/cells8030212.
21. Salachan PV, Rasmussen M, Fredsøe J, Ulhøi B, Borre M, Sørensen KD. Microbiota of the prostate tumor environment investigated by whole-transcriptome profiling. Genome Med. 2022 Jan 25;14(1):9. doi: 10.1186/s13073-022-01011-3
22. Shrestha E, White JR, Yu SH, Kulac I, Ertunc O, De Marzo AM, et al. Profiling the Urinary Microbiome in Men with Positive versus Negative Biopsies for Prostate Cancer. J Urol. 2018 Jan;199(1):161-71. doi: 10.1016/j.juro.2017.08.001.
23. Liss MA, White JR, Goros M, Gelfond J, Leach R, Johnson-Pais T, et al. Metabolic Biosynthesis Pathways Identified from Fecal Microbiome Associated with Prostate Cancer. Eur Urol. 2018 Nov;74(5):575-82. doi: 10.1016/j.eururo.2018.06.033.
24. Golombos DM, Ayangbesan A, O'Malley P, Lewicki P, Barlow L, Barbieri CE, et al. The Role of Gut Microbiome in the Pathogenesis of Prostate Cancer: A Prospective, Pilot Study. Urology. 2018 Jan;111:122-8. doi: 10.1016/j.urology.2017.08.039.
25. Alanee S, El-Zawahry A, Dynda D, Dabaja A, McVary K, Karr M, et al. A prospective study to examine the association of the urinary and fecal microbiota with prostate cancer diagnosis after transrectal biopsy of the prostate using 16sRNA gene analysis. Prostate. 2019 Jan;79(1):81-7. doi: 10.1002/pros.23713.
26. Sfanos KS, Markowski MC, Peiffer LB, Ernst SE, White JR, Pienta KJ, et al. Compositional differences in gastrointestinal microbiota in prostate cancer patients treated with androgen axis-targeted therapies. Prostate Cancer Prostatic Dis. 2018 Nov;21(4):539-48. doi: 10.1038/s41391-018-0061-x.
27. Hills RD Jr, Pontefract BA, Mishcon HR, Black CA, Sutton SC, Theberge CR. Gut Microbiome: Profound Implications for Diet and Disease. Nutrients. 2019 Jul 16;11(7):1613. doi: 10.3390/nu11071613.
28. Samanta M, Harkins L, Klemm K, Britt WJ, Cobbs CS. High prevalence of human cytomegalovirus in prostatic intraepithelial neoplasia and prostatic carcinoma. J Urol. 2003 Sep;170(3):998-1002. doi: 10.1097/01.ju.0000080263.46164.97.
29. Martinez-Fierro ML, Leach RJ, Gomez-Guerra LS, Garza-Guajardo R, Johnson-Pais T, Beuten J, et al. Identification of viral infections in the prostate and evaluation of their association with cancer. BMC Cancer. 2010 Jun 24;10:326. doi: 10.1186/1471-2407-10-326.
30. Matsushita M, Fujita K, Motooka D, Hatano K, Fukae S, Kawamura N, et al. The gut microbiota associated with high-Gleason prostate cancer. Cancer Sci. 2021 Aug;112(8):3125-35. doi: 10.1111/cas.14998.
31. Viaud S, Saccheri F, Mignot G, Yamazaki T, Daillère R, Hannani D, et al. The intestinal microbiota modulates the anticancer immune effects of cyclophosphamide. Science. 2013 Nov 22;342(6161):971-6. doi: 10.1126/science.1240537.
32. Iida N, Dzutsev A, Stewart CA, Smith L, Bouladoux N, Weingarten RA, et al. Commensal bacteria control cancer response to therapy by modulating the tumor microenvironment. Science. 2013 Nov 22;342(6161):967-70. doi: 10.1126/science.1240527.
33. Vande Voorde J, Liekens S, Balzarini J. Mycoplasma hyorhinis-encoded purine nucleoside phosphorylase: kinetic properties and its effect on the cytostatic potential of purine-based anticancer drugs. Mol Pharmacol. 2013 Dec;84(6):865-75. doi: 10.1124/mol.113.088625.
34. Samykutty A, Shetty AV, Dakshinamoorthy G, Kalyanasundaram R, Zheng G, Chen A, et al. Vitamin k2, a naturally occurring menaquinone, exerts therapeutic effects on both hormone-dependent and hormone-independent prostate cancer cells. Evid Based Complement Alternat Med. 2013;2013:287358. doi: 10.1155/2013/287358.
35. Reis Ferreira M, Andreyev HJN, Mohammed K, Truelove L, Gowan SM, Li J, et al. Microbiota- and Radiotherapy-Induced Gastrointestinal Side-Effects (MARS) Study: A Large Pilot Study of the Microbiome in Acute and Late-Radiation Enteropathy. Clin Cancer Res. 2019 Nov 1;25(21):6487-500. doi: 10.1158/1078-0432.CCR-19-0960.
36. Kustrimovic N, Bombelli R, Baci D, Mortara L. Microbiome and Prostate Cancer: A Novel Target for Prevention and Treatment. Int J Mol Sci. 2023 Jan 12;24(2):1511. doi: 10.3390/ijms24021511.

Sažetak

MIKROBIOM I KARCINOM PROSTATE

I. Mareković

SAŽETAK: Znanstvena istraživanja pokazala su uzročnu povezanost između mikrobioma i nastanka različitih vrsta karcinoma. Trenutni rezultati ukazuju na vjerojatnu odsutnost bakterija u zdravom tkivu prostate. Još uvijek nema pouzdanih dokaza koji bi povezali sastav, karakteristike i raznovrsnost crijevnog mikrobioma nastankom karcinoma prostate. Čini se da upala ima ključnu ulogu u razvoju i progresiji ove maligne bolesti. Primjenom sekvenciranja sljedeće generacije različite bakterijske vrste detektirane su u crijevnom mikrobiomu pacijenata s karcinomom prostate. Mikrobiom može utjecati na učinkovitost različitih kemoterapeutika moduliranjem imunološkog sustava i metaboličkih procesa. U procjeni rizika za razvoj enteropatije povezane sa zračenjem, te za procjenu mogućnosti prevencije i liječenja, u budućnosti bi se moglo koristiti testiranje mikrobioma prije i za vrijeme terapije zračenjem. Budućim istraživanjima važno je u potpunosti razjasniti složenu interakciju mikrobioma i karcinoma prostate. Djelovanje na crijevni mikrobiom moglo bi se koristiti u svrhu pospješenja učinkovitosti liječenja karcinoma i njegove prevencije.

Ključne riječi: *karcinom prostate, mikrobiom, liječenje, prevencija, upala*