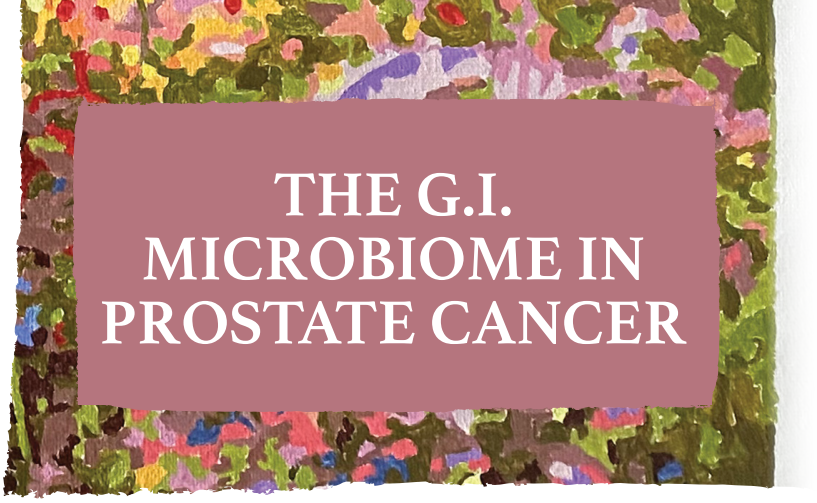
Healing Cancer at the Root
HYPERION
Functional Medicine



THE G.I. MICROBIOME IN PROSTATE CANCER

Prostate cancer generally develops in men in their 50s as a slow growing tumor. Almost all of these are adenocarcinomas which develop from the gland cells inside the prostate. Other types of prostate cancer include small cell carcinoma, other neuroendocrine tumors, sarcomas, and transitional cell carcinoma. Most tend to grow slowly but some can grow and spread quickly.

Diet plays an important role in the development and then the progression of prostate cancer. A high-fat diet (red meat, potatoes, high-fat dairy products) causes G.I. dysbiosis and bacterial metabolites from the G.I. tract to enter the blood circulation, stimulating and promoting prostate cancer growth. Western-style diets are high risk for development prostate cancers when compared to men in Asian countries. Studies have shown that the gut microbiome affects progression and growth of prostate cancer. When Asian men began eating Western style diets high in red meat and fat, the rates of prostate cancer increased. Obesity increases the risk of prostate cancer significantly. Diet is a major factor affecting the gut microbiome, high-fat diets increase gut dysbiosis. The gut wall integrity is impaired, permeability of the gut is increased leading to leaky gut. Metabolites from bacteria in the gut are released into systemic circulation increasing inflammation[1].

Androgen hormone plays a critical role in prostate cancer by binding to the androgen receptor in prostate cancer cells. Androgens are hormones that bind to prostate cancer cells and activate genes that cause the cancer cells to grow. The gut microbiome can affect androgen production, there are specific bacteria in the gut that can produce androgens. Other bacteria have been identified in the G.I. microbiome that can be beneficial[2]. This research supports the critical need for G.I. stool testing in patients with prostate cancer.

The inflammatory state of the G.I. system in the state of dysbiosis increases the risk of a leaky gut allowing microbes, food particles, and toxins to leak out from the intestine itself into the bloodstream. This creates a chronic inflammatory response that affects many systems in the body. The stress on healthy cells from chronic inflammation increases the risk of damage to their DNA and can lead to cancer cell development.

Bacteria in the gut send signals to the mitochondria, this has been observed to alter mitochondrial metabolism, activate immune cells, induce inflammation, and alter the intestinal barrier. Dysbiosis of the microbiome in the gut and the resulting mitochondrial dysfunction are associated with chronic intestinal inflammation and cancer[3].

A comprehensive stool analysis is essential to evaluate the health of the cancer patient's microbiome and the overall function of the G.I. system. Hyperion functional medicine uses stool testing in patients who are in the Phase 1 Functional Oncology Program. The G.I. stool test is used to evaluate the microbiome for dysbiosis, potential for immune dysfunction, inflammatory markers, bacterial metabolites, infectious bacteria, parasites, and overall microbial diversity.

A detailed and stepwise functional treatment plan is made based on the results of the stool test which may include prescribed beneficial probiotics or prebiotic's, G.I. specific nutritional recommendations, pharmaceutical grade supplements to reduce inflammation as indicated, and botanical antibiotic treatment remedies based on the microbiome testing results.

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CANCER ROOTS
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2 - Fujita K, Matsushita M, De Velasco MA, Hatano K, Minami T, Nonomura N, Uemura H. The Gut-Prostate Axis: A New Perspective of Prostate Cancer Biology through the Gut Microbiome. Cancers (Basel). 2023 Feb 21;15(5):1375. doi: 10.3390/cancers15051375. PMID: 36900168; PMCID: PMC10000196.
3 - Jackson DN, Theiss AL. Gut bacteria signaling to mitochondria in intestinal inflammation and cancer. Gut Microbes. 2020 May 3;11(3):285-304. doi: 10.1080/19490976.2019.1592421. Epub 2019 Mar 26. PMID: 30913966; PMCID: PMC7524274.