



Role of Bacteria in Stimulation and Inhibition of Carcinoma

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DESCRIPTION

Cancer, which develops from the growth of malignant cells into masses known as tumours, continues to be one of the leading causes of morbidity and mortality in the world. Tumors cause DNA mutations that result in the acquisition of epigenetic changes that promote oncogenesis and carcinogenesis with a variety of diseases. Through a process known as metastasis, some of these tumours are migrating from their tissue to different areas of the body. Tumors can be grouped based on the tissue and/or organ from which they originate. Carcinomas, for instance, are cancers that spread through tissues that cover all the body organs. Not all cancers are malignant, some are referred as nonsolid cancers including leukaemia, lymphoma, and myeloma.

Alcohol, smoking, and sunlight are a few of the extrinsic factors that increase the risk of developing cancer. Men are more likely to develop lung, stomach, prostate, and colorectal cancers. Women are more likely to develop lung, breast, cervix, and colorectal cancers. According to this theory, cell transformation is a process by which healthy cells change into cancer cells. Despite substantial research in this area, the exact mechanism behind cancer is still unknown. With this knowledge, it was impossible to comprehend how the tumour developed and how the bacteria might have colonised the tumour or caused it.

Bacteria affect the cell cycle and their capacity to attack the immune system and cause immune suppression through a variety of mechanisms, such as inflammation, lymphoproliferation, and the induction of hormones that boost epithelial cell proliferation. Bacterial chronic infections are significant cancer-related risk factors. Proinflammatory cytokines like tumour necrosis factor and chemokines are secreted during inflammation, which attracts additional cells to the site of infection and intensifies the immune response. As a result, renewed cell division is stimulated, which can result in mutations, deletions, or translocations as damaged DNA promotes the growth of cancer cells.

The DNA damage resembles cancer caused by changed genes that control normal cell regulation and ultimately apoptosis. Gastric

and colorectal cancers have been linked to the bacteria *Helicobacter pylori* and later *Fusobacterium nucleatum*. According to several studies, individuals with colorectal cancer have higher than average amounts of the bacteria that cause inflammatory bowel illnesses and can also create toxins and carcinogenic chemicals.

Although the mechanism by which bacteria kill cancer cells is poorly known, Bacteriophage Cancer Therapy (BCT) entirely opens doors for the treatment of cancer. When researchers Busch and Fehleisen discovered that individuals with erysipelas infections caused by *Streptococcus* develop carcinomas, they made the first discovery of the link between cancer and bacteria. The human body's commensal bacteria, or microbiota, are essential for functional regulation and healthy survival. The microbiota, on the other hand, regulates cancer during its predisposing conditions, beginning with immune response susceptibility, genetic instability, progression, and interaction with therapy stages. DNA vaccines and antitumor metabolites are two more therapeutic strategies used by microorganisms to manipulate cancer.

Some bacterial strains have been found to cause cancer, including *Helicobacter pylori*, which has been linked to gastric cancer; *Salmonella typhi*, which has been linked to hepatobiliary carcinoma; *Campylobacter jejuni*, which has been linked to small intestinal lymphomas; *Chlamydia psittaci*, which has been linked to ocular lymphomas; *Mycobacterium tuberculosis*, and *Citrobacter rodentium* with human colorectal cancer. Microbes may cause approximately 16% of all cancers worldwide; liver and gastrointestinal tract cancers, in particular, have been linked to bacteria. Among these, colonic cancer was linked to the presence of *Streptococcus bovis* endocarditis. However, it is challenging to pinpoint the precise roles that the microbiome and the host play in the emergence of cancer. This is due to the wide variety of interactions that exist between them.

It has been demonstrated that pathogenic bacteria alter and take advantage of the location of the human host cell in various ways over the course of the infection cycle. It was acknowledged that cancer does not behave as an infectious or contagious disease when it was learned that bacteria are the primary cause of many

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infectious diseases. The idea that bacterial participation causes cancer was therefore rejected. In addition to having a complicated structure that can trigger the immune system, pathogenic bacteria have alterations on their outer surfaces that allow them to evade the immune system and increase their chances of significant survival.

CONCLUSION

Some bacteria have also shown the synthesis of tumour proteins through products of metabolism that have direct myogenic or mutagenesis effects, in addition to promoting cancer by impairing the host's natural defence mechanisms such as inflammation and antigen recognition. Due to the tumour

selectivity and broad gene packaging capabilities of bacteria, targeting tumours is the best strategy to administer therapeutic doses. Due to its high specificity, capacity to manage after ingestion and conditions in several live experiments, bacterial therapy has demonstrated noteworthy effects despite the fact that traditional cancer treatments are still the majority of treatments. By concentrating on bacterial therapy and employing genetically modified bacteria alone or in combination with traditional cancer treatments, researchers can implement a medicine with no side effects and potentially permanently humanity from cancer. Bacteria are anticipated to offer a distinct immunotherapy treatment technique that can be improved by cutting-edge genetic engineering of bacterial strains, despite the fact that the core process varies.