ISSN: 2157-2518
Journal of Carcinogenesis & Mutagenesis

OPEN ACCESS Freely available online

Commentary

Immunotherapy in Colitis-Associated Carcinogenesis: Advancements and Challenges from Animal Model Studies

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DESCRIPTION

Inflammatory Bowel Disease (IBD), comprising Crohn's disease and ulcerative colitis, is associated with an increased risk of Colorectal Cancer (CRC). Colitis-Associated Carcinogenesis (CAC) is a process by which chronic inflammation of the colonic mucosa results in the development of colorectal cancer. Animal models of CAC have been developed to better understand the molecular and cellular mechanisms underlying CAC and to develop novel therapies for its prevention and treatment.

Animal models of CAC have been developed to mimic the human disease and to investigate the role of inflammation in the development of CRC. The two most commonly used animal models of CAC are the Azoxymethane (AOM) model and the Dextran Sulfate Sodium (DSS) model.

The AOM model involves the injection of AOM, a potent carcinogen, into mice or rats to induce colon tumors. The injection of AOM is followed by the administration of a pro-inflammatory agent, such as Dextran Sulfate Sodium (DSS), which induces colitis and promotes the development of tumors in the colon.

The DSS model involves the administration of DSS in drinking water to induce colitis in mice or rats. The severity of colitis can be modulated by varying the concentration and duration of DSS administration. Colitis induced by DSS is characterized by epithelial damage, crypt loss, mucosal ulceration, and infiltration of inflammatory cells. The DSS model has the advantage of being relatively inexpensive and easy to perform, and it allows for the study of the effects of chronic inflammation on the development of CRC.

Chronic inflammation is a key factor in the development of CAC. The inflammatory response to chronic colitis leads to the production of Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS), which can damage DNA and other cellular components, leading to mutations and genomic instability. In addition, chronic inflammation leads to the activation of transcription factors such as Nuclear Factor Kappa B (NF-κB) and signal transducer and activator of transcription 3 (STAT3), which promote cell proliferation, survival, and invasion.

The inflammatory response to chronic colitis also leads to the production of cytokines and chemokines, which recruit immune cells to the site of inflammation. Immune cells, such as macrophages, neutrophils, and lymphocytes, produce growth factors and other factors that stimulate the proliferation and survival of epithelial cells. In addition, immune cells produce proteases and other factors that can degrade extracellular matrix, facilitating invasion and metastasis of tumor cells.

Studies using animal models of CAC have identified a number of molecular pathways involved in the development of CRC. These pathways include the Wnt/β-catenin pathway, the tumor protein p53 pathway, the Phosphoinositide 3-Kinase (PI3K)/Akt pathway, and the Mitogen-Activated Protein Kinase (MAPK) pathway. Activation of the Wnt/β-catenin pathway promotes cell proliferation and inhibits apoptosis, leading to the accumulation of mutations and genomic instability. Mutation or loss of the tumor suppressor gene p53 leads to the loss of its tumor suppressor function, allowing cells to escape apoptosis and accumulate additional mutations. Activation of the PI3K/Akt pathway promotes cell survival and growth, while activation of the MAPK pathway promotes cell proliferation and invasion.

Prevention and treatment of CAC

Animal models of CAC have been used to investigate potential therapies for the prevention and treatment of CAC. Dietary interventions, such as the use of high-fiber diets, have been shown to reduce the risk of CAC in animal models. The use of Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) has also been investigated, as they can reduce inflammation and inhibit the production of ROS and RNS. However, long-term use of NSAIDs can lead to gastrointestinal complications, limiting their use as a preventive measure.

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Received: 27-Mar-2023, Manuscript No. JCM-23-21339; **Editor assigned:** 30-Mar-2023, Pre QC No. JCM-23-21339 (PQ); **Reviewed:** 14-Apr-2023, QC No. JCM-23-21339; **Revised:** 20-Apr-2023, Manuscript No. JCM-23-21339 (R); **Published:** 28-Apr-2023, DOI: 10.35248/2157-2518.23.S37.004.

Citation: Ouyang T (2023) Immunotherapy in Colitis-Associated Carcinogenesis: Advancements and Challenges from Animal Model Studies. J Carcinog Mutagen. S37:004.

Another approach to the prevention of CAC is the use of chemopreventive agents. These agents can target specific molecular pathways involved in the development of CRC, such as the Wnt/β-catenin pathway or the PI3K/Akt pathway. For example, inhibitors of the Wnt/β-catenin pathway, such as curcumin and resveratrol, have been shown to reduce tumor formation in animal models of CAC. Similarly, inhibitors of the PI3K/Akt pathway, such as rapamycin, have been shown to reduce tumor formation in animal models of CAC.

Immunotherapy has also emerged as a promising approach for the treatment of CAC. Immunotherapy aims to stimulate the immune system to recognize and attack tumor cells. In animal models of CAC, immunotherapy has been shown to reduce tumor formation and prolong survival. However, more research is needed to optimize the use of immunotherapy in the treatment of CAC.