OFFICIAL ABSTRACT and CERTIFICATION

Understanding The Role of Microbes in Intestinal Tumor Pathogenesis

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Colorectal cancer (CRC) is the third most deadly cancer worldwide, with 1/2 million deaths yearly. The gut microbiome and its metabolites have been implicated as contributors and regulators of CRC pathogenesis. Treatment with celecoxib, a NSAID and selective COX-2 inhibitor, has been found to reduce the formation of polyps in the gastrointestinal tract of humans and mice. Furthermore, celecoxib has been demonstrated to reduce the rate by which Lgr5-positive stem cells gave rise to differentiated cell types in intestinal crypts through alterations to the gut microbiome. Mice treated with broad-spectrum antibiotics also developed significantly fewer, smaller tumors than untreated mice, hinting that tumor incidence and penetrance were dependent on the gut microbiome.

In this study, methods of immunohistochemistry for Ki-67 and PCR were employed to study the role of celecoxib and antibiotics in modulating microbes and intestinal tumor development in ApcMin/+ mice. A combination of celecoxib and broad-spectrum antibiotics significantly decreased tumor size and number in the mice, more effectively than celecoxib or antibiotics alone. Under antibiotics, the administration of celecoxib resulted in significantly reduced basal proliferation of normal intestinal crypt stem cells. Further experiments exposing HCT116 cells to varying concentrations of murine fecal extract demonstrated that the gut microbiome could directly manipulate inflammation within the gut. This study provides insight into the understanding of the role of gut microbiota in CRC development. As cancer only continues to become a more prevalent issue, targeting the microbiome to prevent inflammation-based CRC development could potentially aid the development of critically-needed chemopreventive strategies.

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