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## **A role for prostaglandin E<sub>2</sub> and its receptor EP2 in colon carcinogenesis associated with inflammation in mice**

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### **Abstract**

Although cancer is a major cause of death worldwide, the molecular mechanism underlying cancer development as well as pharmaceutical strategies for its disease has not fully been established. Clinical studies have suggested a role for inflammation in the development of cancer. Especially, NSAIDs that inhibit prostaglandin (PG) synthesis reduce the risk of colon cancer, implicating a PG cascade in cancer development. Since the mutation of the Adenomatous Polyposis Coli (APC) gene causes familial adenomatous polyposis resulting in colon cancer formation, mice carrying the APC mutation have been proposed to be a mouse model for carcinogenesis. Our previous report using this model suggested that EP2, one of the PGE receptor subtypes, is critical for polyp formation in APC mutant mice and induces the expression of COX-2, a PG synthase, thus forming a positive feedback loop through COX-2, PGE<sub>2</sub> and EP2. However, this mouse model is not designed to address a role for inflammation in carcinogenesis. Here we have examined a role for PG signaling in another cancer model associated with inflammation, and found that EP2 is critical for colon carcinogenesis in this model. We are currently investigating the mechanism about how EP2 contributes to colon carcinogenesis as well as its associated long-lasting inflammation.