BioMed Research Journal
BMRJ, 8(1): 15
www.scitcentral.com

ISSN: 2578-8892

Abstract: Open Access

Exploring the Potential of Prostaglandin Pathways in Cancer Prevention and Therapy

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Published July 23, 2024

ABSTRACT

Prostaglandins (PGs), prostaglandin-endoperoxide synthases (PTGS, cyclooxygenases), and PG receptors play crucial roles in cancer development and progression by mediating profound effects in carcinogenesis. We focus on the status of PG signaling pathways in modulating cancer progression and aim to shed light on the mechanisms of PG generation and the roles of individual mediators and their receptors in modulating inflammation response and tumor progression. Understanding the molecular mechanisms of PG action holds significant clinical relevance for cancer chemoprevention and therapy. As an illustrative example, our study delves into the mechanistic role of the COX1/2-Driven thromboxane A2 pathway in suppressing Barrett's esophagus (BE) and esophageal cancer development. COX2 and thromboxane A2 synthase (TBXAS), along with Thromboxane A2 Receptor (TBXA2R), are found to be highly expressed in BE and esophageal adenocarcinoma (EAC) patients, accompanied by a significant elevation of circulating TXA2 levels. Acetylsalicylic acid (ASA) demonstrates efficacy in suppressing BE and EAC growth by targeting the TXA2 pathway. Moreover, biopsy analyses from 49 patients with similar baseline characteristics reveal that ASA substantially reduces serum TXA2 levels, leading to decreased inflammation. Furthermore, deletion or targeting of the TXA2 pathway efficiently suppresses BE and EAC growth in surgical mouse models of esophagoduodenostomy and in patient-derived xenograft (PDX) mouse models. Mechanistic studies demonstrate that the TXA2 signaling pathway can mediate BE and EAC progression through multiple signaling pathways, such as the MAPK and NF-kB pathways. This study underscores the importance of the COX1/2-driven TXA2 pathway in BE and EAC pathophysiology and sets the stage for the introduction of a TXA2-targeting strategy for EAC prevention and early detection. In conclusion, our findings suggest that targeting PG pathways offer promising avenues for cancer prevention and therapy.

Keywords: Prostaglandins (PGs), Carcinogenesis, PG signaling pathways, Inflammation response, Tumor progression

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Citation: Zhang T. (2024) Exploring the Potential of Prostaglandin Pathways in Cancer Prevention and Therapy. BioMed Res J 8(S1): 15.