

Editorial

An Advance Therapy of Colitis Colorectal Cancer

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Colorectal Cancer (CRC) is the third most common cancer in men and the second in women worldwide. Chronic inflammation is the most important risk factor for its development as it favors neoplastic transformation by enhancing epithelial cell turnover in the colonic mucosa [1]. Conventional therapies including surgery, current chemotherapy (5-Fluorouracil, Oxaliplatin, Doxorubicin (Dox), among others) and radiotherapy are frequently inadequate due to their side effects (cytotoxicity, chemo and radio-resistance, failure in treatment efficacy and potency) [2]. Dox (anthracycline antitumor antibiotic) intercalates between base pairs in the DNA helix and preventing DNA replication and inhibiting topoisomerase II which leads to cell death by apoptosis [3]. Drug Metformin (Met) inhibit cancer development and tumor growth by activation of AMP-Activated Protein Kinase (AMPK) which negatively regulates mTOR (Mammalian Target of Rapamycin) activation [4]. It also blocks a metabolic stress response that stimulates the inflammatory pathway associated with a wide variety of cancers [5]. Dox & met both are inducers of apoptosis and autophagy. Na-oxamate (Ox) enhances the antitumor effects of met and dox by affecting the ability of the tumor cells to proliferate under hypoxia with a final induction of apoptosis, autophagy [6]. Ox is LDHA inhibitors which induce G2/M cell cycle arrest and promote apoptosis through enhancement of mitochondrial ROS generation, lead to inhibition of aerobic glycolysis [7].

Treatment of colitis-related CRC with the triple therapy (Met-Ox-Dox) resulted in significant inhibition of tumor growth by means of inhibition of the p-mTOR, autophagy induction, glycolysis inhibition and apoptotic cell death. These results highlight the importance of targeting aberrant energetic metabolism of cancer cells and searching for novel antineoplastic drugs as promising strategies to have a clinical application in a near future and the response to 'genome instability and mutation' especially linked to DNA-intercalating characteristics of Dox, remains to be briefly described [8].

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