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 Manpreet Kaur Saini, Sankar Nath Sanyal
<https://doi.org/10.1139/bcb-2014-0016>

ABSTRACT

An angiogenic pathway was studied that involved stromal tissue degradation with matrix metalloproteinases (MMPs), vesicular endothelial growth factor-A (VEGF-A), and hypoxia inducible factor-1 α (HIF-1 α) mediated growth regulation in a complex interaction with chemokines, such as monocyte chemoattractant protein-1 (MCP-1) and macrophage inflammatory protein-1 β (MIP-1 β). Gene and protein expression was studied with real-time PCR, Western immunoblot, and immunofluorescence. Morphological and histopathological analysis of tumor was done, as also the activity of MMPs and HIF-1 α by gelatin zymography and ELISA. Binding interactions of proteins were studied by molecular docking. Piroxicam, a traditional NSAID and C-phycoerythrin, a biliprotein from *Spirulina platensis*, were utilized in the chemoprevention of DMH-induced rat colon cancer. A significant number of tumors was evident in DMH treated animals, while with piroxicam and C-phycoerythrin, the number and size of tumors/lesions were reduced. Colonic tissues showed severe dysplasia, tubular adenoma, and adenocarcinoma from DMH, with invasive features along with signet ring cell carcinoma. No occurrence of carcinoma was detected in either of the drug treatments or in a combination regimen. An elevated VEGF-A, MMP-2, and MMP-9 level was observed, which is required for metastasis and invasion into surrounding tissues. Drugs induced chemoprevention by down-regulating these proteins. Piroxicam docked in VEGF-A binding site of VEGF-A receptors i.e., VEGFR1 and VEGFR2, while phycoerythrin (a chromophore of C-phycoerythrin) docked with VEGFR1 alone. HIF-1 α is up-regulated which is associated with increased oxygen demand and angiogenesis. MCP-1 and MIP-1 β expression was also found altered in DMH and regulated by the drugs. Anti-angiogenic role of piroxicam and C-phycoerythrin is well demonstrated.

Keywords: [MMPs](#), [VEGF-A](#), [HIF-1 \$\alpha\$](#) , [MCP-1](#), [MIP-1 \$\beta\$](#)

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