

Researchers to publish paper in Molecular Cancer journal

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(Medical Xpress) -- Erxi Wu, assistant professor of pharmaceutical sciences, and Fengfei Wang, research associate in pharmaceutical sciences, co-wrote the article, " α 2-adrenoceptor blockage induces G1/S phase arrest and apoptosis in pancreatic cancer cells via Ras/Akt/NF κ B pathway," which will be published by *Molecular Cancer*.

According to the authors, [pancreatic cancer](#) risk factors, smoking and stress, stimulate nitrosamine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and catecholamines production respectively.

Nitrosamine

4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and catecholamine bind the α -adrenoceptors and induce pancreatic cancer cell proliferation; and they have previously indicated that α -adrenergic antagonists may suppress proliferation and invasion and stimulate apoptosis in pancreatic cancer. To clarify the mechanism of apoptosis induced by α 2-adrenergic antagonist, they hypothesize that blockage of the α 2-adrenoceptor could induce G1/S phase arrest and apoptosis and Ras may be a key player in pancreatic [cancer cells](#).

Their results showed that the α 1 and α 2-adrenoceptor proteins were detected on the cell surface of pancreatic cancer cells from pancreatic carcinoma specimen samples by Immunohistochemistry. The α 2-adrenergic antagonist ICI118,551 significantly induced G1/S phase arrest and apoptosis compared with the α 1-adrenergic antagonist metoprolol, which was determined by the flow cytometry assay. α 2-adrenergic antagonist therapy significantly suppressed the expression of extracellular signal-regulated kinase, Akt, Bcl-2, cyclin D1 and cyclin E and induced the activation of caspase-3, caspase-9 and Bax by Western blotting. Additionally, the α 2-adrenergic antagonist reduced the activation of NF κ B in vitro cultured pancreatic cancer cells.

"The blockage of α 2-adrenoceptor markedly induced pancreatic cancer cells to arrest at G1/S phase and consequently resulted in cell death, which is possibly due to that the blockage of α 2-adrenoceptor inhibited NF κ B, extracellular signal-regulated kinase and Akt pathways. Therefore, their upstream molecule Ras may be a key factor in the α 2-adrenoceptor antagonist induced G1/S phase arrest and apoptosis in pancreatic cancer cells. The new pathway discovered in this study may provide an effective therapeutic strategy for pancreatic cancer," Wu said. Collaborator for this paper is Ma lab at Xi'an Jiaotong University, China. "We have established a productive collaboration with the Ma lab in finding cancer therapeutics and elucidating the mechanisms of the targeted therapy for pancreatic cancer, one of the most lethal malignancies," Wu said.

More information: www.molecular-cancer.com

Provided by North Dakota State University

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