New Therapy to Treat Pancreatitis

Published date: June 1, 2016

Technology description

Pancreatitis is an inflammation of the pancreas, including acute pancreatitis (AP) and chronic pancreatitis (CP). Other than pain management, no standard therapeutic strategy is currently available. The research team lead by Dr. Bashoo Naziruddin has discovered that Withaferin A (WA) administration can effectively protect mice from Cerulein-induced pancreatitis by blocking NF-B activation and translocation and thus reducing expression of pro-inflammatory cytokines. Therefore WA could be a novel therapeutic agent for pancreatitis. Market Pancreatitis is an inflammation of the pancreas that includes two main types, acute pancreatitis (AP) and chronic pancreatitis (CP). Globally, in 2013 about 17 million cases of pancreatitis, resulted in 123,000 deaths. AP occurs in about 30 per 100,000 people a year. New cases of CP develop in about 8 per 100,000 people a year and currently affect about 50 per 100,000 people in the United States. People with AP are normally treated with IV fluids and pain medications in the hospital. In up to 25% of AP patients, the pancreatitis can be severe and patients may need to be admitted to an intensive care unit (ICU). CP can result in reversible loss of pancreatic acinar cells, and subsequently islet cells, and can be difficult to treat. Aside from pain management, there is no standard therapeutic strategy for reducing inflammation in pancreatitis. There is a huge unmet medical need for new therapies for treating pancreatitis. Technology Withaferin A (WA) is a plant-derived compound with strong anti-inflammatory and anti-oxidant properties. WA was applied in cerulean-induced pancreatitis mice model: WA prevented damage to pancreatic parenchyma caused by repeated injection of cerulein and reduced fibrosis of pancreas. Cerulein induced CP revealed activation and translocation of NF-B into the nucleus of pancreatic acinar cells. NF-B translocation was inhibited by WA administration. Proliferation marker Ki67 were increased by cerulein administration and clearly inhibited by WA treatment. Infiltration of leukocytes as a result of inflammatory cytokine/chemokine production due to cerulein treatment was abolished by WA. Induction of pro-inflammatory and pro-apoptotic genes by cerulein was inhibited by WA.

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