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Titel des Beitrags:
COX-2 is not required for the development of murine chronic pancreatitis.

Abstract:
Chronic pancreatitis is a severe inflammation of the pancreas associated with destruction of the parenchyma, fibrosis, and persistent abdominal pain. Cyclooxygenase-2 (COX-2) and COX-2-derived prostaglandins, key mediators of the inflammatory response, are elevated in patients with chronic pancreatitis. Previous studies investigated COX-2 as a therapeutic target. These reports showed a reduced pathology in COX-2-deficient mice with a better outcome. Here we compared the role of COX-2 in acute and chronic pancreatic inflammation using the same COX-2(-/-) mouse model of cerulein-induced pancreatitis. In a setting of acute pancreatitis, juvenile COX-2(-/-) mice exhibited a reduced histopathological score compared with wild-type littermates; on the contrary, adult mice did not show any difference in the development of the disease. Similarly, in a setting of chronic pancreatitis induced over a period of 4 wk, adult mice of the two strains showed comparable histological score and collagen deposition. However, the abundance of mRNAs coding for profibrotic genes, such as collagen, ?-smooth muscle actin, and transforming growth factor-? was consistently lower in COX-2(-/-) mice. In addition, comparable histological scores and collagen deposition were observed in wild-type mice treated with a COX-2 inhibitor. We conclude that, in contrast to what was observed in the rat pancreatitis models, COX-2 has a limited and age-dependent
effect on inflammatory processes in the mouse pancreas. These results suggest that COX-2 modulates the inflammatory process during the development of pancreatitis in a species-specific manner. Thus the pathophysiological roles of COX-2 and its therapeutic implications in patients with pancreatitis should be reexamined.