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Novel approaches in combating pancreatitis and development of pancreatic cancer

Acute pancreatitis (AP) is a dangerous; and in up to 5% of cases, it is deadly disease with no specific cure. The leading causes of acute pancreatitis have been identified as gallstone biliary disease and high alcohol intake, while abnormality in calcium signalling in pancreatic acinar cells (PACs) was found to be one of the first events. We have shown previously that bile acids and non-oxidative alcohol metabolites elicit excessive Ca^{2+} release from intracellular stores. Subsequent intracellular Ca^{2+} store depletion activates the opening of Ca^{2+} release activated Ca^{2+} (CRAC) channels in the plasma membrane leading to Ca^{2+} overload, premature activation of pancreatic pro-enzymes, digesting the pancreas and its surroundings. Our recent work using a pancreatic lobule preparation allowed us to investigate potential role for pancreatic stellate cells (PSCs) in AP. AP often results in development of chronic pancreatitis (CP) and increased occurrence of pancreatic cancer (PC). The most common form of PC is pancreatic ductal adenocarcinoma and CP patients are at significant risk of developing PC. Activation of PSCs - during pancreatic injury - induces proliferation as well as secretion of extracellular matrix components, thereby playing an important role in the fibrosis that occurs in CP and PC. PSCs generate substantial Ca2+ signals when challenged with both physiologically and pathologically relevant bradykinin (BK) concentrations by activation via bradykinin receptor type 2 (B2). The major plateau phase of these signals can be markedly reduced by CRAC channel inhibition and, importantly, blockade of PSCs B2 markedly diminishes the extent of acinar necrosis evoked by AP-inducing agents. Our study indicates that combined treatment of pancreatitis with inhibitors of CRAC channels and B2 receptors could be potentially useful against development of PC. This study is beneficial for understanding of new mechanisms that could help combatting AP, transition to CP and development of PC.

Biography

Julia V Gerasimenko has completed her PhD in 1996 from Bogomoletz Institute of Physiology, Kiev, Ukraine. She is a Senior Lecturer in Cardiff School of Biosciences, Cardiff University, UK. She has published 33 papers in reputed journals and has been serving as an Editorial Board Member of repute. She is a Member of Faculty of Gastro-intestinal Physiology, The Physiological Society (UK), British Society for Cell Biology, and European Calcium Society. She was an invited speaker for many scientific conferences in the UK and abroad.

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