EuroSciCon

2nd International Conference on

Cancer Science & Research

March 24-25, 2022
Webinar

Journal of Cancer Science and Research
ISSN: 2576-1447

Friends or foe? The role of different exocrine pancreatic cell types in pancreatic cancer development

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Acute pancreatitis (AP) is a dangerous and deadly disease with no specific cure. 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. The gallstone biliary disease and high alcohol intake are the leading causes of AP. We have shown previously that bile acids and alcohol metabolites induce Ca2+overload in pancreatic acinar cells (PACs), premature intracellular activation of pancreatic pro-enzymes, digesting the pancreas and its surroundings. Recently, we have explored Ca2+ signaling in the different cell types of the pancreatic tissue using a pancreatic lobule preparation. The principal agent evoking Ca2+ signals in pancreatic stellate cells (PSCs) is bradykinin, but in experimental alcohol-related acute pancreatitis, these cells become much less responsive to bradykinin and then acquire sensitivity to trypsin. Our new findings have important implications for understanding of the development of acute pancreatitis and we propose a scheme in which Ca2+ signals in PCSs provide an amplification loop promoting PACs cell death. Initial release of the proteases kallikrein and trypsin from dying acinar cells can, via bradykinin generation and protease-activated receptors, induce Ca2+ signals in PCSs which can then, possibly via nitric oxide generation, cause additional release of proteases, generating a vicious circle. This results in AP and subsequently in CP, increasing chances of PC.

A number of approaches have been successfully tried to alleviate alcohol-induced AP in vitro and in vivo. Inhibitors of store-operated calcium entry and galactose as an oral supplementation have efficiently reduced AP effects. Both approaches are currently the most promising perspectives to develop an effective AP treatment and subsequently reduce probability of CP and PC. Our findings are beneficial for understanding of new mechanisms that could help combatting pancreatic disorders.

Biography

Julia Gerasimenko is a Senior Lecturer at Cardiff School of Biosciences, Cardiff University, UK. She completed her PhD at the Bogomoletz Institute of Physiology, Ukrainian Academy of Science in 1996. Her work has primarily been directed towards elucidating the molecular mechanisms initiating the enigmatic disease Acute Pancreatitis (~20000 admissions to hospital and ~1000 deaths per year in the UK alone). There is currently no treatment for this disease, but Julia's work has opened up new possibilities for rational treatment. She has published 52 research papers (3559 citations, (Scopus), h-index 28) on the disease mechanism in competitive peer-review journals, including Cell, PNAS, Journal of Physiology, Journal of Cell Science and Current Biology. Julia V. Gerasimenko is a Member of Faculty of 1000 (Gastro-intestinal Physiology), The Physiological Society (UK) and the European Calcium Society.

Received: February 28, 2022; **Accepted:** March 02, 2022; **Published:** April 10, 2022