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## Alcohol effects on the pancreas, how to boost our natural defence

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Acute pancreatitis is generally initiated by premature trypsinogen activation in pancreatic acinar cells mediated by excessive intracellular calcium release from internal stores. One of the major causes of acute pancreatitis is excessive alcohol intake; however the molecular mechanism of this severe inflammatory disease is not completely understood. We now show that in two-photon permeabilized pancreatic acinar cells even a low ethanol concentration (10 mM, equivalent of consuming a small amount of alcohol) elicits calcium release from intracellular stores and induces intracellular trypsinogen activation that leads to necrosis. Readmission of the calcium sensor calmodulin (at a normal intracellular concentration) to the permeabilized cells markedly reduced ethanol-induced calcium release and trypsinogen activation. Essentially, calmodulin acts as our natural defence from the medium-high doses of alcohol. It was found that calcium-like peptides (CALPs) can substantially boost the calmodulin-based mechanism and completely protect pancreas from a high dose of alcohol (100 mM) and its metabolites. Very recently it was shown that inhibition of the calcium entry into pancreatic acinar cells can also substantially improve cell survival when they treated with a high dose (100  $\mu$ M) of alcohol metabolite palmitoleic acid ethyl ester (POAEE). It was suggested that CALPs and calcium entry inhibitors or combination of both can be used as a potential therapy against pancreatitis.

### Biography

Oleg V Gerasimenko completed his PhD in 1991 at BogomoletzInst, Kiev, Ukraine before moving to Liverpool, UK in 1993 to join research group lead by Prof. Ole H Petersen. He became Lecturer in 2000 and Reader in 2005 before moving to Cardiff School of Biosciences in 2010. Since then he is a leader of well-equipped MRC funded research group together with Prof Ole H Petersen (Head of School of Biosciences) and Dr. Julia Gerasimenko.

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