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Galactose can reduce side effects of Asparaginase-based drugs for childhood ALL

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Abstract:

Asparaginase-based drugs are very successful against childhood acute lymphoblastic leukaemia (ALL), however, they can induce Acute pancreatitis (AP) as a side effect and force clinicians to discontinue the treatments. AP is a frequent human disease with substantial mortality with no specific therapy. Previous investigations into the mechanisms of AP established that intracellular ATP loss is a crucial factor leading to calcium overload and necrosis. We have recently reported that glucose metabolism is severely inhibited under AP conditions due to the inhibition of hexokinases. ATP loss and calcium exacerbate each other and lead to necrosis. We have found that replacing or supplementing glucose with galactose has markedly reduced the loss of ATP, calcium overload, and subsequent necrosis in vitro. Galactose as an oral enhancement has viably secured against AP in two distinctive mouse models of AP. In both cases, galactose has markedly reduced pancreatic histology scores, acinar necrosis, and inflammation. We suggest that galactose oral supplements may be used to protect against AP and therefore improve the efficacy of the childhood ALL treatments.



Biography:

Oleg Gerasimenko has completed his Ph.D. in 1991 from the Bogomoletz Institute of Physiology and postdoctoral studies from The University of Liverpool, UK. He is a Reader at Cardiff School of Biosciences, Cardiff University, UK, and a Fellow of The Physiological Society UK. He has published 90 papers in reputed journals (h-index 41) and has been serving as an Editorial board member of Scientific Reports and Pflugers Archiv.

Speaker Publications:

1. "Galactose protects against cell damage in mouse models of acute pancreatitis". Journal of Clinical Investigation, 128(9), pp. 3769-3778

2."Ca2+ signalling underlying pancreatitis". Cell Calcium, 70, pp. 95-101.

3. "Calcium and adenosine triphosphate

control of the cellular pathology: asparaginase-induced in pancreatitis elicited via protease-activated receptor 2". Philos Trans R Soc of Lond B Biol Sci. 5;371(1700). pii: 20150423.

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