Protective role of complement C3 against cytokine-mediated beta cell apoptosis

Background and aims: Type 1 diabetes is a chronic autoimmune disease characterized by pancreatic islet inflammation and β-cell destruction by pro-inflammatory cytokines and other mediators. The complement system, a major component of the immune system, has been recently shown to also act in metabolic organs, such as liver, adipose tissue, and pancreas. In the present study we identified complement C3 as an important hub of a cytokine-modified complement network in human islets and characterized the role of C3 in β-cell survival.

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Contributors: Dos Santos, R. S., Marroqui, L., Grieco, F. A., Marselli, L., Henz, S. R., Marchetti, P., Wernersson, R., Eizirik, D. L.
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