Beta Cell Interest Group (BIG)

Current and ongoing beta cell research is presented in this weekly seminar by faculty, postdoctoral fellows and students. If you are interested in attending the Beta Cell Interest Group (BIG) seminars and joining the BIG community, please contact David Jacobson.

Keywords: big Beta cell

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Meeting Details

Start Date / Time	April 11, 2018 at 9:00 AM
End Date / Time	April 11, 2018 at 9:55 PM
Duration	12 hour(s) 55 minutes
Location	512 Light Hall
Presenter Name	Matt Dickerson (Jacobson's lab)
Presentation Title	Exploring the roles of ligand-gated ion channels in alpha-cell calcium handling and glucagon secretion
Status	This meeting has already occurred

Meeting Agenda/Notes

During the pathogenesis of type 2 diabetes (T2D) a rise in pancreatic alpha-cell glucagon (GCG) secretion increases hepatic glucose production and exacerbates hyperglycemia. It is well established that calcium influx through voltage-dependent calcium channels (VDCCs) is required for alpha-cell GCG secretion and that islet calcium signaling becomes dysfunctional under stressful conditions such as T2D. Thus, it is critical to fully characterize the mechanisms that govern alpha-cell calcium handling to develop new treatments to normalize GCG secretion and alleviate hyperglycemia during T2D. Interestingly, alpha-cells express calcium-activated potassium (KCa) channels, which are activated by an elevation in intracellular calcium leading to plasma membrane potential (Vm) hyperpolarization and VDCC closure. Here for the first time we investigate the role of KCa channels in alpha-cell calcium handing and GCG secretion. Inhibition of KCa channels transiently depolarized alpha-cell Vm and increased calcium influx; however, prolonged inhibition ultimately resulted in decreased calcium influx and reduced GCG secretion. This was presumably due in part to voltage-dependent inactivation of P/Q-type VDCCs that are closely linked to GCG granule exocytosis. These results demonstrate that periodic alpha-cell Vm hyperpolarization due to KCa channel activation serves to prevent voltage-dependent inactivation of P/Q-type VDCCs and facilitate GCG secretion.