Beta Cell Interest Group (BIG) Seminar

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: beta cell BIG

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Meeting Details

Start Date / Time	November 13, 2013 at 9:00 AM
End Date / Time	November 13, 2013 at 10:00 AM
Duration	1 hour(s)
Location	512 Light Hall
Presenter Name	Troy Hutchens (Piston lab)
Presentation Title	“The role of EphA4 receptor signaling in the glucose-inhibition of glucagon secretion”
Status	This meeting has already occurred

Meeting Agenda/Notes

Juxtacrine signaling through the EphA-ephrin-A signaling pathways has been shown to play an important role in both the basal and glucose dependent hormone secretion from islet β-cells. Since the α-cell is closely related to the β-cell, we speculate that a similar mechanism plays a role in basal glucagon secretion and GIGS. Interestingly, both human and mouse α-cells only express a single Eph receptor, EphA4. In preliminary studies pharmacologically inhibiting EphA4 receptor signaling, glucagon secretion was elevated at basal glucose and secretion was inappropriately stimulated in response to glucose. These two findings mirror the glucagon secretion patterns observed in both sorted α-cells and diabetic patients, indicating the importance of EphA4 receptor signaling in mediating glucagon secretion at basal and elevated glucose. *We hypothesize that EphA4 receptor signaling in α-cells is required for the appropriate suppression of glucagon secretion at basal glucose and for GIGS. Additionally, we hypothesize EphA4 receptor signaling is permissive for additional paracrine inhibitory signals (insulin and/or somatostatin).*