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Emergence of Critical Behavior in β-Cell Network MATTHEW WESTACOTT, THOMAS HRAHA, MASON MCCLATCHEY, MARINA POZZOLI, RICHARD BENNINGER, University of Colorado - Denver — The β-cell is a cell type located in the Islet of Langerhans, a micro-organ of the pancreas which maintains glucose homeostasis through secretion of insulin. An electrophysiological process governing insulin release occurs through initial uptake of blood glucose and generation of ATP which inhibits the ATP sensitive potassium channel (K-ATP) causing membrane depolarization (activation). Neighboring β-cells are electrically coupled through gap junctions which allow passage of cationic molecules, creating a network of coupled electrical oscillators. Cells exhibiting hyperpolzarized (inactive) membrane potential affect behavior of neighboring cells by electrically suppressing their depolarization. Here we observe critical behavior between global active-inactive states by increasing the number of inactive elements with the K-ATP inhibitor Diazoxide and a tunable ATP insensitive transgenic mouse model. We show this behavior occurs due to from cell-cell coupling as mice lacking β-cell gap junctions show no critical behavior. Also, a computational β-cell model was expanded to construct a coupled β-cell network and we show this model replicates the critical behavior seen *in-vitro.* While electrical activity of single β-cells is well studied these data highlight a newly defined characteristic of their emergent multicellular behavior within the Islet of Langerhans and may elucidate pathophysiology of Diabetes due to mutations in the K-ATP channel.

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