Abstract Example

Cyclic AMP Enhances Beta Cell Network Activity in mouse pancreas

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Insulin secretion from beta cells is triggered upon increase in $[Ca^{2}]_{c}$ and can be further amplified by cAMP, which has been previously described to act through PKA- or Epac2-dependent pathways. Since the precise mechanism of action is not fully understood we assessed the $[Ca^{2}]_{c}$ dynamics in beta cell populations with electro- and opto-physiological approaches combined with the acute tissue slice technique, supported by network-based analyses. In the absence of forskolin, substimulatory glucose concentration failed to increase $[Ca^{2}]_{c}$, while stimulatory glucose concentration evoked a transient calcium increase followed by synchronized high frequency Ca^{2} oscillations. Addition of forskolin to the substimulatory glucose concentration triggered a delayed high frequency Ca^{2}oscillations. In high glucose concentration the addition of forskolin further increased the frequency of Ca^{2} oscillations. Despite a modest decrease in durations of individual oscillations, the relative active time increased by more than 50 %. Furthermore, the beta cell functional networks become denser in the forskolin regime, suggesting a higher degree of synchronicity. To determine which of the two aforementioned pathways was responsible for augmented Ca^{2} oscillations, the same sets of experiments were performed on pancreatic slices from mice lacking the Epac2 protein. In this case, a qualitatively very similar behaviour was observed compared with WT littermates. These results corroborate previously published data describing that phosphorylation of several targets by PKA is responsible for the cAMP-augmented Ca^{2} oscillations in pancreatic beta cells.

KEY WORDS: Insulin, cyclic AMP, pancreatic beta cells