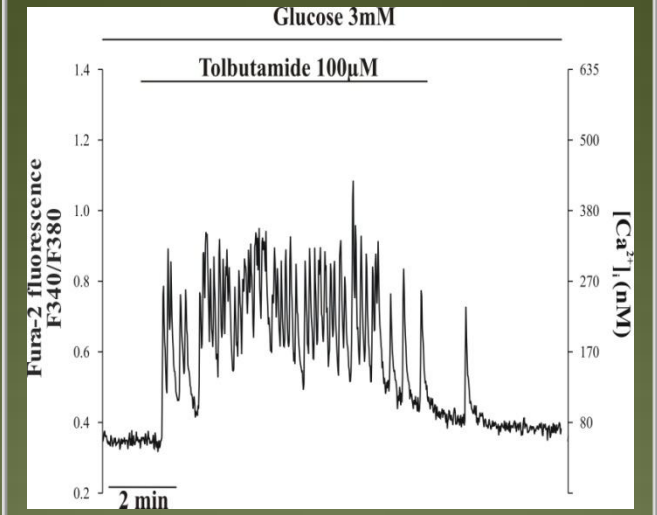
Background

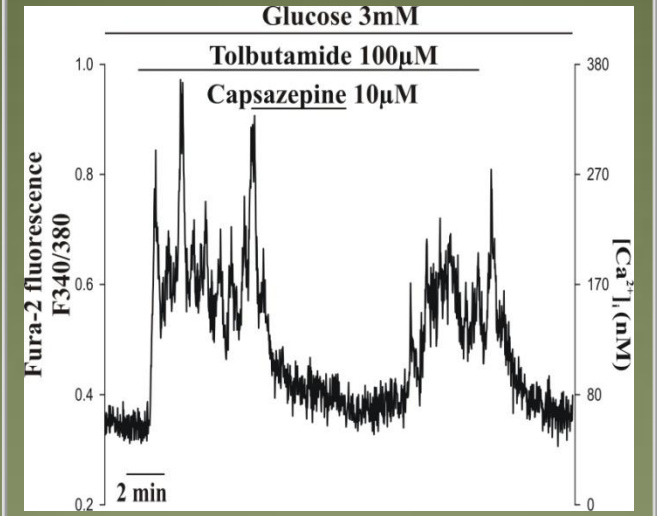
- Increase in the cytosolic Ca^{2+} concentration ($[Ca^{2+}]_i$) in the pancreatic β-cells leads to insulin secretion. Tolbutamide is known to increase the $[Ca^{2+}]_i$ by closing the K_{ATP} channels leading to depolarization of the β-cells and opening of the voltage gated Ca^{2+} channels. It is unclear whether transient receptor potential (TRP) channels are involved in this process.
- The mechanism by which the extracellular adenosine diphosphate ribose (ADPr) increases the $[Ca^{2+}]_i$ is currently unknown.

Aim

- To study whether the TRP channels are involved in tolbutamide-induced $[Ca^{2+}]_i$ increase.
- To identify the surface receptor involved in the ADPr-induced Ca^{2+} increase.

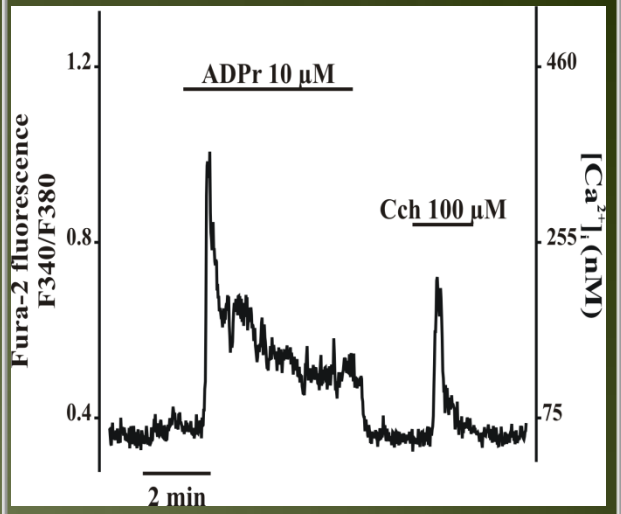
Results 1

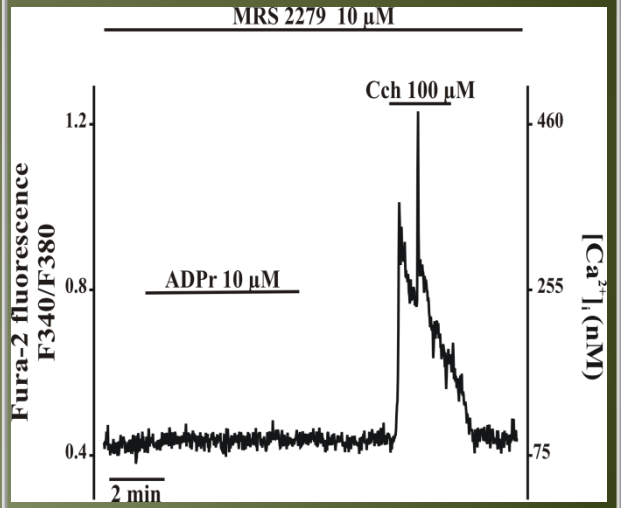




Capsazepine, a selective inhibitor for TRPV1 channel inhibited the tolbutamide-induced $[Ca^{2+}]_i$ increase

Results 2





MRS2279 a selective inhibitor for P2Y1 receptor inhibited the ADPr-induced $[Ca^{2+}]_i$ increase

Role of TRPV1 channel and P2Y1 receptor in Ca^{2+} signalling in β-cells: A study by single cell microfluorometry

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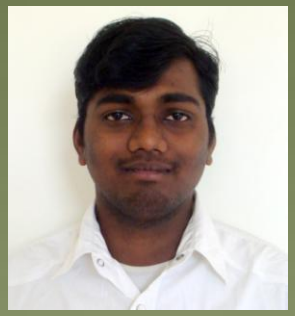
Materials and methods

- A highly differentiated rat insulinoma cell line (S5) that was subcloned from INS-1E cells were used as model for β-cells.
- The $[Ca^{2+}]_i$ changes was measured by Fura-2-based single cell ratiometric microfluorometry using Fura-2.

Acknowledgements

I thank my supervisor Md. Shahidul Islam and Amanda Jabin Fågelskiöld for providing me practical guidance and knowledge in the field of microfluorometry.



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Conclusion

- Depolarization of β-cells by tolbutamide requires Ca^{2+} entry through TRPV1 channels.
- ADPr increases $[Ca^{2+}]_i$ in beta cells by activating the P2Y1 receptors.