ICOVAMENIB IN DIABETES

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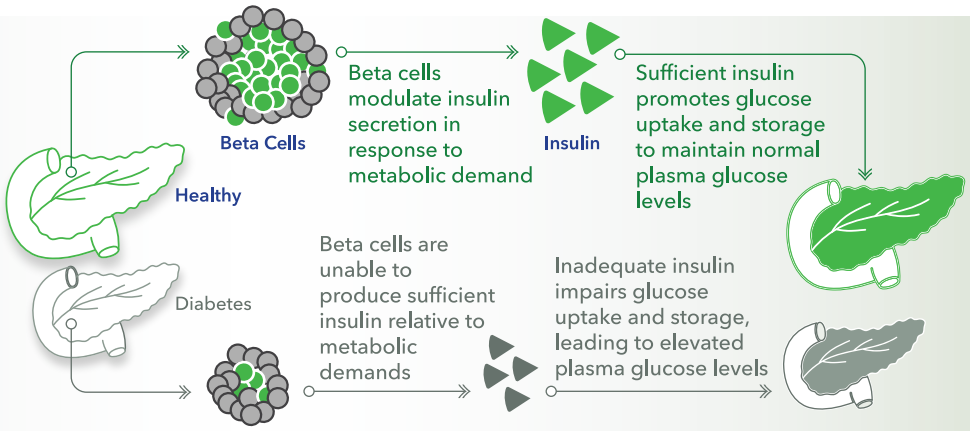
What is the biological cause of diabetes?

A progressive decline in pancreatic beta-cell mass and function.

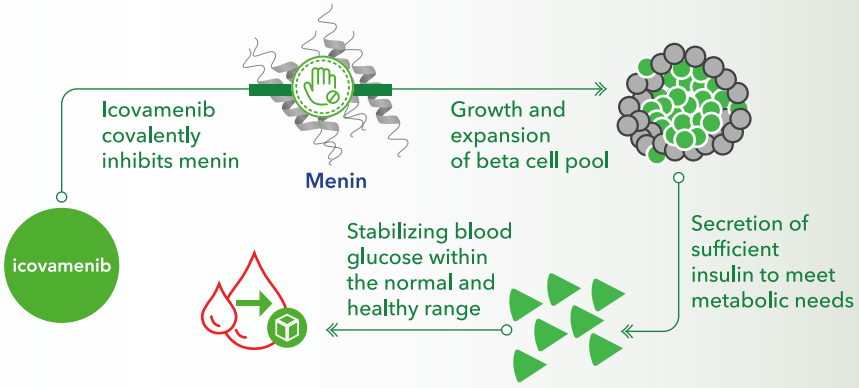
Diminished beta-cell capacity leads to inadequate insulin secretion relative to metabolic demands, resulting in chronic hyperglycemia. This persistent elevation in blood glucose can induce widespread microvascular damage through mechanisms such as oxidative stress and advanced glycation end-product formation.



How is icovamenib intended to impact beta cells?

Icovamenib inhibits menin, an important protein that controls beta-cell mass.

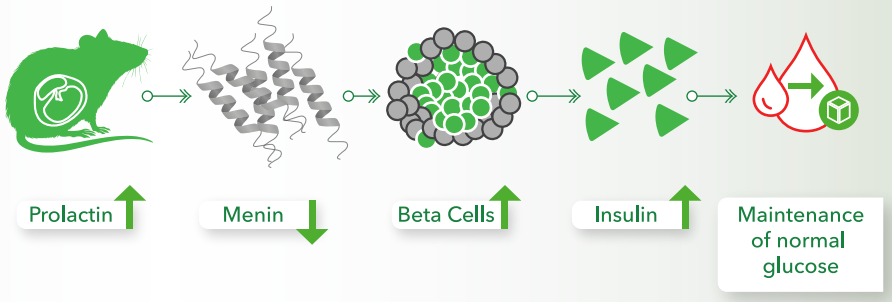
Icovamenib is a first-in-class investigational oral molecule in clinical development directly targeting menin. Icovamenib explores the potential to cure diabetes by naturally regenerating insulin-producing beta cells through the potent and durable inhibition of menin.



Is there a naturally occuring proof of concept?

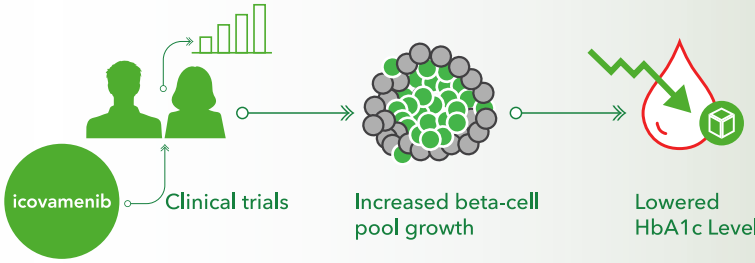
Stanford University researchers have demonstrated preclinically that during pregnancy, the hormone prolactin and placental lactogen, acting through the prolactin receptor, downregulate menin, resulting in the proliferation of maternal pancreatic beta cells, increased insulin production, and the maintenance of normal glucose levels to prevent gestational diabetes.

*Menin Controls Growth of Pancreatic ß-Cells in Pregnant Mice and Promotes Gestational Diabetes. Science, (2007), 801-806, 318



How do you measure success in diabetes?

Based on preclinical studies, our goal with icovamenib is to increase the pool of functional beta cells after several weeks of dosing, which could potentially lead to sufficient natural insulin production. Cinical trials are underway to investigate how this effect may, over time, translate into normalized glucose levels and lowered HbA1c for people with diabetes. Results of ongoing and future clinical trials will be necessary to fully assess the potential impact of the investigational agent, icovamenib.



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