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Student Session

DGK δ deficiency in mouse pancreatic β -cells alleviates STZ-induced diabetes.

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Diacylglycerol kinase (DGK) phosphorylates diacylglycerol (DAG) to phosphatidic acid. We have previously demonstrated that DGK δ acts as a negative regulator of β -cell proliferation. However, it is still unknown whether DGK δ deficiency in pancreatic β -cells alleviates diabetes. In the present study, we investigated the effect of β -cell specific DGK δ deficiency on hyperglycemia in streptozotocin (STZ)-induced diabetic mice. We administered STZ to 5-wk-old mice and performed measurement of body weight and blood glucose level for 60 days. At 11 days after STZ administration, there was no significant difference in serum insulin concentrations, pancreatic insulin content, or β -cell area between β -cell specific DGK δ knockout (β DGK δ KO) and control mice. At 60 days, in contrast, blood glucose level was significantly lower and serum insulin level and pancreatic insulin content were significantly higher in β DGK δ KO mice than in control mice. In morphological analysis, a significant increase in β -cell area was observed in β DGK δ heteroKO mice compared with control mice. These results suggest that DGK δ deficiency in pancreatic β -cells prevents STZ-induced hyperglycemia, which is likely due to maintained β -cell mass resulting from promoted β -cell proliferation.