

P037 Homeward bound – Islet Transplantation to the pancreas
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The success of islet transplantation as a treatment modality depends upon increasing the number of islets available and increasing transplanted islet survival and function. It is our hypothesis that a portion beta cell failure after transplantation may be ascribed to the transplant site. The following studies were performed in a syngeneic streptozotocin-diabetic rat model. 600, 1000, 2000, and 3200 islets were transplanted into the pancreas, renal, or hepatic subcapsular space. In the pancreas, 600 islets prevented hyperglycemia for over three months of study. In contrast the kidney site required 2000 islets and the liver 3200 islets for long term normoglycemia. Insulin deficiency diabetes is often associated with digestive insufficiency. The transplantation of 800-1000 islets to the pancreas of diabetic rats resulted in the restoration of normal ductal amylase concentrations as well as maintaining normoglycemia. Islet transplantation in the presence of nerve growth factor significantly increased islet reinnervation and nerve-stimulated insulin secretion. These results suggest that islet transplantation to the pancreas offers many advantages over the hepatic site in that fewer islets are required, insulin reaches the liver undiluted via the portal vein, exocrine function is restored, and more normal insulin secretion can be established.