

Chemical Pancreatectomy using Ethanol Infusion ID: 4181

Featured Innovator: George Gittes, MD

Chronic pancreatitis affects nearly 200,000 people in the US. These patients suffer severe and intractable pain — often managed with narcotics — and are at a greatly increased risk of pancreatic cancer. They all eventually develop diabetes. The most reliable way to treat pancreatitis is surgical removal, but this approach is highly invasive, highly morbid, and actually increases the risk of diabetes. We have devised a minimally-invasive chemical method that removes the problematic pancreatic tissue without compromising the crucial insulin-producing aspects of the gland.

Technology Description

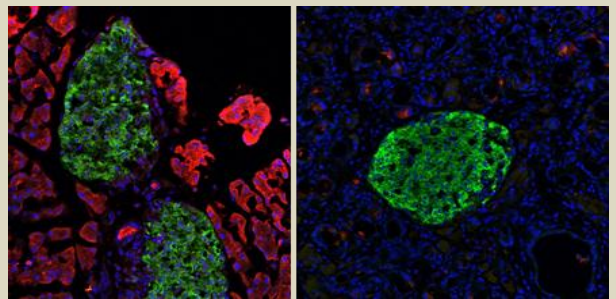
By one theory of chronic pancreatitis, the exocrine pancreas — which produces digestive enzymes — creates a toxic environment that then kills off the otherwise healthy insulin-producing islets of the endocrine pancreas. We discovered that infusing pure ethanol into the pancreatic duct of a mouse leads to total destruction of the problematic exocrine pancreas while leaving the endocrine pancreas intact. In a model of chronic pancreatitis, ethanol infusion halted pancreatic islet destruction and improved insulin production. As opposed to traditional pancreatectomy, our method can be performed endoscopically for minimal invasiveness. Also, because ethanol infusion spares the hormonal functions of the pancreas, our method could treat the painful and carcinogenic aspects of pancreatitis while also reducing patients' risk of developing diabetes.

Advantage

- Minimally invasive
- Eliminates cancer risk
- Alleviates pain
- Reduces risk of diabetes

Applications

Chronic pancreatitis



Histology of control pancreas on the left (green for insulin, red for amylase) and one week after ethanol infusion on the right. Note that the exocrine tissue is absent, but the islets are intact after ethanol infusion.

Stage of Development

- *in vivo* mouse data
- *in situ* primate surgical protocol

IP Status

Provisional patent application filed

Innovator



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Dr. Gittes completed his surgical internship and residency at the University of California at San Francisco. Then he completed a fellowship at the Children's Mercy Hospital in Kansas City, MO. He continued on at the Children's Mercy Hospital, the University of Missouri Kansas City School Hospital, and the University of Missouri Kansas City School of Medicine, where he held the Thomas M. Holder/Keith W. Ashcraft Chair in Pediatric Surgical Research. Then Dr. Gittes joined the faculty at the University of Pittsburgh in 2005. His research interests include Smad regulation of pancreatic islet formation.

Education

MD, Harvard Medical School
AB, Harvard College

Publications

- Xiao X, Guo P, Shiota C, Zhang T, Coudriet GM, Fischbach S, Prasad K, Fusco J, Ramachandran S, Witkowski P, Piganelli JD, Gittes GK (2018). Endogenous Reprogramming of Alpha Cells into Beta Cells, Induced by Viral Gene Therapy, Reverses Autoimmune Diabetes. *Cell Stem Cell*, 22(1), 78-90.
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- Xiao X, Chen Z, Shiota C, Prasad K, Guo P, El-Gohary Y, Paredes J, Welsh C, Wiersch J, Gittes GK (2013). No evidence for β cell neogenesis in murine adult pancreas. *The Journal of Clinical Investigation*, 123(5), 2207-2217.
- Gittes GK (2009). Developmental biology of the pancreas: a comprehensive review. *Developmental Biology*, 326(1), 4-35.

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