DCAMKL-1: a new horizon for pancreatic progenitor identification

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TYPE 1 DIABETES RESULTS FROM the autoimmune destruction of insulin-producing pancreatic β-cells. Although islet transplantation has shown great potential in the treatment of hyperglycemia in type 1 diabetes patients, availability of donor islets remains a major obstacle (4). Cell replacement strategies aimed at generating functional insulin-producing cells through differentiation of adult pancreatic stem cells/progenitors have been hindered by a lack of suitable markers for their identification.

Pancreatic progenitor cells are important in the regeneration of β-cells and in the development of pancreatic cancer. β-Cell mass undergoes compensatory changes throughout life, particularly during times of increased demand and during injury. This suggests the presence of stem cells/progenitors or some other mechanism of β-cell renewal in the adult pancreas. The lack of demonstrable markers of embryonic β-cell progenitors in the adult pancreas coupled with results from genetic lineage tracing studies showing that β-cells were largely generated by self-replication of preexisting β-cells in adult mice both under physiological conditions and following pancreatic injury (2, 6) cast doubt on the existence of stem cells/progenitors in the pancreas after birth (1). However, several recent studies have demonstrated not only the existence of progenitors in the ductal epithelium for both endocrine and exocrine lineage cells in the adult mouse pancreas (5), but also that some of the new β-cells seen following pancreatic injury in mice arise from progenitors expressing the basic helix-loop-helix transcription factor neurogenin3 (11).

In this issue of American Journal of Physiology, Houchen and colleagues (8) demonstrate not only the existence of progenitors in the ductal epithelium for both endocrine and exocrine lineage cells in the adult mouse pancreas (5), but also that some of the new β-cells seen following pancreatic injury in mice arise from progenitors expressing the basic helix-loop-helix transcription factor neurogenin3 (11). The authors have declared that no conflicts of interest exist.

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