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Commentary

Embryonic Development And Adult Homeostasis

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INTRODUCTION

ontroversy over whether pancreatic islet cells arise from adult stem or progenitor-like cells actually predates the discovery of insulin, and the recent use of islet transplantation to treat diabetes has only intensified interest in this question. Recent breakthroughs, particularly those based on Cre-loxP lineage-tracing in the mouse, have resolved some aspects of this controversy, but not all. We now know that insulin-producing β-cells and other islet cells derive from multipotent progenitors in the embryo, but that their maintenance and expansion in postnatal life is driven primarily by proliferation of existing differentiated cells. This appears to be true even during regeneration, and seems to apply to the exocrine acinar cells as well as islets. Following pancreatic duct ligation, however, islet precursors re-appear in the injured pancreas, arising from ducts and differentiating into new islet cells. Thus, while the pancreas does not normally rely on classical stem cells, a stem cell-like mechanism for new islet differentiation may be inducible under specific circumstances. Understanding the signals that promote β-cell formation in the embryo and adult should facilitate efforts to derive clinically-useful β-cells in vitro, either from adult ducts or embryonic stem cells. Type I diabetes is caused by the autoimmune destruction of pancreatic β-cells, and has emerged as a case study for stem cell-based "regenerative medicine." Its selling point is the idea that the location of β-cells within the pancreas is irrelevant to their ability to regulate blood sugar through insulin release. Moved elsewhere, so long as they have access to the circulation, β-cells should function to maintain glucose homeostasis – a hypothesis amply supported by animal studies and now the basis for clinical islet transplantation in humans .Islet transplantation confronts formidable hurdles as a treatment for type I diabetes, such as blocking autoimmunity and preserving graft function. When these difficulties are overcome, however, the approach will still be hampered by the scarcity of cadaver-derived islets. Three potential solutions have been proposed: first, to enhance replication of islet cells in vitro, "stretching" the limited supply; second, to isolate adult stem cells from the pancreas that can expand and produce new β-cells; third, to manipulate Embryonic Stem (ES) cells so that they adopt a β-cell identity.The first of these approaches, recently discussed elsewhere is beyond the scope of this review, although we will discuss the contribution of β-cell proliferation to islet regeneration following injury. With respect to the second approach, we will consider the existence of stem cells in the adult pancreas, and discuss how they might be exploited clinically. Finally, we will discuss recent advances toward β-cell derivation from ES cells, and consider how this approach might benefit both basic and clinical researchers. As any of these strategies will depend on understanding normal pancreas development and homeostasis, it is here that we will begin our review. he mammalian pancreas arises from two evaginations in the posterior foregut, one dorsal and one ventral, which expand into the tail and head, respectively, of the mature organ. The first evidence of pancreas development, at embryonic day 8.5 (E8.5) in the mouse, is expression of the homeodomain transcription factor Pdx1 within the cells that will evaginate to form the pancreatic buds Shortly thereafter, these undifferentiated progenitor cells begin to express the bHLH transcription factor as well as the digestive Genetic lineage-tracing, using the Cre-loxP system, indicates that essentially all mature pancreatic cells, including acini, ducts and islets, arise from these Pdx1+/Ptf1a+ progenitor cells Moreover, in both mice and humans, pancreas growth and differentiation are nearly abolished when either of these genes is mutated Although there have been few thorough studies of human embryonic and fetal pancreas development, the weight of evidence indicates overall similarity to that of rodents Space does not permit a full discussion of the complexities of pancreas development, which have been reviewed elsewhere We will focus instead on the issue of pancreatic lineages, as it relates directly to the question of pancreatic stem cells

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