Propositions to the Thesis

The Role of Prox1 during mouse pancreas organogenesis

1) Compensatory mechanisms involving the activity of the one-cut transcription factor OC-2 may be partially responsible for the recovery of the endocrine phenotype in the *Prox1*-deficient pancreas.
This Thesis; Vanhorenbeeck et al. *Dev Biol* **305**, 685-94 (2007); Margagliottiet al. *Dev Biol* **311**, 579-89 (2007).

2) Our proposal that the ensuing pancreatitis of *Prox1*$^{loxP/loxP}$*;Pdx1-Cre* mice could result of congenital ductal alterations, bears on sufficient corroborating scientific evidence.
This Thesis; Vonlaufen et al., *J Gastroenterol Hepatol* **23**, 1339-48 (2008); Hue Su et al *HPB (Oxford)* **8**, 264-86 (2006); Schulzke et al. *Ann N Y Acad Sci* **1165**, 294-300 (2009); Weber et al., *Lab Invest* **88**, 1110-20 (2008); Zhang et al. *Mech. Dev.* 126: 958-973 (2009).

3) The decreased size of most islets in the pancreas of *Prox1*$^{loxP/loxP}$*;Pdx1-Cre* suggests a role for Prox1 in islet cell migration and/or adhesion.
This Thesis; Sosa-Pineda et al., *Nat Genet* **25**(3): 254-255; Papoutsi et al., *Cell Tissue Res* **330**(2): 209-220 (2007); Kamiya etal., *Hepatology* **48**(1): 252-264 (2008); Wigle et al., *Cell* **98**(6): 769-778 (1999); Miralles et al., *J Cell Biol* **143**(3): 827-836 (1998); Cirulli et al., *J Cell Biol* **150**(6): 1445-1460 (2000).

4) Although Prox1 is not expressed in differentiated pancreatic exocrine cells, some of the developmental defects of *Prox1*-deficient pancreata suggest a role of this transcription factor in pancreatic exocrine progenitors.
This Thesis.

5) Opposite effects of the loss of Prox1 function on the cell cycle in different developing organs suggest tissue specific function of this transcription factor.
Wigle et al., *Nat Genet* 21(3): 318-322 (1999); Dyer et al., *Nat Genet* 34(1): 53-58 (2003); Misra et al., *Dev Dyn* 237(2): 393-402 (2008); Sosa-Pineda et al., *Nat Genet* 25(3): 254-255 (2000); Kamiya et al., *Hepatology* 48(1): 252-264 (2008); Wang et al., *Dev Biol* 286(1): 182-194 (2005).

6) In contrast to what the authors claim, poor efficiency associated with direct protein delivery using penetrins precludes widespread use of this method to generate pluripotent stem (PS) cells.
Kim et al., Cell Stem Cells. 4:472-476 (2009); Zhou et al., Cell Stem Cell. 4:381-384 (2009).

7) Before showing that hematopoietic stem cells derived from human iPS cells can be used for transplantation in vivo, the work of Lengerke et al. seems promising but is as yet preliminary.
Lengerke et al., *Hematopoietic Stem Cells VII: Ann.N.Y.Acad.Sci.* 1176:219-227 (2009).

8) The conclusion of Barth et al that Jarid2 overexpression contributes to the heart outflow tract phenotype of *Nkx2.5* null mice is at best only partially supported by the experimental evidence.
Barth et al., *Dev. Dyn.* 239:2024-2033 (2010).

9) Le May et al.'s study showing that oxidative stress predisposes both sexes to diabetes in ERαKO mice challenges the idea of female sex-specific protection from diabetes by estradiol.
Le May et al., *Proc Natl Acad Sci U S A* **103**(24): 9232-9237 (2006); Wild et al., *Diabetes Care* **27**(5): 1047-1053 (2004); Louet et al., *Curr Atheroscler Rep* **6**(3): 180-185 (2004).

10) Johnson et al.'s proposal that the bone marrow "could be a potential source of germ cells that could sustain oocyte production in adulthood" is novel and interesting. However, confirmation that oocytes generated from bone marrow or peripheral blood can be fertilized and undergo proper embryonic development is necessary to validate their potential therapeutic value.
Johnson et al., *Cell* **122**(2): 303-315 (2005).

11) You know you are from Chicago when you think 35 ^{o}F (2 ^{o}C) is great weather to wash your car.