DPSG ABSTRACT FORM

Deadline: April 15th, 2012 for Lille, France

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Basic Science	x	**Clinical Research**		**Epidemiology & Databases**		**Health Care Delivery**	

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ABSTRACT TITLE and Co-Authors:

PANCREATIC BETA CELL PROGENITORS RESIDENT WITHIN ISLETS CONTRIBUTE TO REGENERATION FOLLOWING BETA CELL LOSS, BUT FAIL TO DIFFERENTIATE IN ANIMALS BORN TO MOTHERS FED A LOW PROTEIN DIET DURING PREGNANCY

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Regeneration of beta cell mass following diabetes-associated loss can occur despite a low intrinsic proliferation rate. We hypothesized that beta cell regeneration is achieved partly by the mobilization of an immature beta cell progenitor population resident within islets, and that the mobilization of these cells would be compromised in animals born to mothers exposed to a low protein diet in early life. Balb/c mice were fed a control (C) (20% protein) or an isocaloric LP (8% protein) diet during gestation until birth and C diet thereafter. At birth pups were injected i.p. with 35 mg/kg STZ (or a vehicle sham) from days 1 to 5 for each dietary treatment. Pups were sacrificed 7, 14 and 30 days of age and the pancreata removed for histological analysis. In C-fed animals treated with STZ the beta cell mass had recovered to control values by 30 days, but regeneration did not occur in LP-fed animals that had received STZ. We identified islet cells immunoreactive for the transcription factor Pdx-1, but not insulin, as putative beta cell progenitors, and these accounted for 7±1% of Pdx-1-positive cells in islets from C-fed mice. The frequency of Pdx-1^{+}/Ins^{-} cells was not altered by LP diet, or STZ. However, in LP-fed mice that had received STZ there was a significant increase in the number of Pdx-1^{+}/Ins^{-} cells (18±2%, $p<0.001$) despite a failure of beta cell regeneration. Results suggest that a differentiation of resident beta cell progenitors can contribute to the rapid beta cell regeneration seen following STZ. However, while progenitors are present in LP-fed mice they accumulate and fail to differentiate, resulting in a reduced beta cell mass. A deficiency in progenitor differentiation into functional beta cells is likely to contribute to the impaired beta cell phenotype seen in offspring born to LP-fed mothers, and the risk of future diabetes.

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- Submitting an abstract does not constitute or guarantee registration; abstract acceptance will be notified on or before June15th.