

Figure S1. Coregulators are depleted from samples following reverse coimmunoprecipitations (In reference to Figure 2). These controls illustrate that almost all the coregulator was removed in the co-IP supernatants with the Brg1, Mi2b, Tif1b, or Baf170 antibodies.





Figure S2. Brg1 and Brm are broadly expressed in the developing pancreas and the adult mouse and human pancreas (In reference to Figure 3). Immunofluorescence analysis of A) Brg1, B) Brm, insulin, and Pdx1 in embryonic day (E) 12.5, 15.5, 18.5, and 2 month old mouse pancreas sections demonstrating Brg1 and Brm co-expression with Pdx1 in the embryonic and adult mouse pancreas. C,D) Representative immunofluorescence staining analysis of insulin, C) BRG1, and D) BRM in human normal and T2DM pancreas paraffin sections. There was no obvious difference in BRG1 or BRM levels in normal and T2DM islets. Nuclei are counterstained with DAPI (blue). Scale bars indicate 20µM.



Figure S3. Pdx1 interacts with the Brg1-Swi/Snf and Brm-Swi/Snf complexes in developing and adult mouse pancreas (In reference to Figure 4). The PLA was performed with antibodies to Pdx1 and A) Brg1 or B) Brm in E12.5, E18.5, and 2 month old mouse pancreata. The wide cellular distribution of the Pdx1:Brg1 and Pdx1:Brm signals reproduces their general distribution in E12.5 pancreatic epithelium, while enrichment at E18.5 and 2 months reflects Pdx1High levels in insulin+ cells. Representative images are included for each time point; arrows indicate PLA signals and the lower magnification insets are provided to orient each image. Scale bar corresponds to 10µM. Right panel in A, B): Graphical representations showing the distribution of the PLA signals per cell nucleus. E12.5 quantification includes all pancreatic epithelium, while E18.5 and 2 month quantification is of insulin+ nuclei. N>3, *P<0.05.



Figure S4. Pdx1 does not interact with the Ldb1 coregulator (In reference to Figure 4). No PLA signal was detected under low or high glucose conditions between Pdx1 and the IsI1 transcription factor coregulator, Ldb1.



Figure S5. PDX1, BRG1 and BRM antibodies do not enrich for target gene sequences in HeLa cells (In reference to Figure 5). A) BRM antibodies failed to immunoprecipitate bona fide target sequences of the CD44 promoter from HeLa cell chromatin (Banine et al., 2005). B) Neither PDX1 nor BRG1 antibodies immunoprecipitate target sequences of the INSULIN or MAFA (data not shown) promoters in BRG1+, PDX1- HeLa cells. N=2 for each ChIP.



Figure S6. Schematic illustrating how physiological and pathophysiological conditions influence PDX1:SWI/SNF recruitment and INSULIN gene expression (In reference to Figures 4 & 7). PDX1 (blue circle) differentially recruits BRG1- (green) or BRM-SWI/SNF (red) complexes to activate or repress target gene genes, respectively. A,B) In response to changes physiological glucose concentrations, PDX1 preferentially recruits BRM-SWI/SNF (inset) at low, fasting levels of glucose, which uses energy from ATP-hydrolysis to mobilize histones and reduce binding/activity of other trans-activators (and/or increase trans-repressor(s)), consequently reducing gene expression. B) In fed, high glucose stimulating conditions, selective binding of PDX1 to BRG1-SWI/SNF (inset) improves access of trans-activators and RNA Polymerase II to promote gene activation. C) The retention of PDX1:BRM binding over PDX-1:BRG1 in T2DM-cells (Figure 6) strongly suggests that PDX1 target genes are negatively regulated by BRM-SWI/SNF under such pathophysiological conditions.





Figure S7. Nuclear Pdx1, Brg1 and Brm protein levels are unaffected by blood glucose concentration in vivo (In reference to Figure 4). Immunofluorescence analysis of mouse islet β -cell nuclei reveals that Pdx1, Brg1 (top) and Brm (bottom) protein content is not altered in either low glucose (16hr fast) or high glucose (16hr fast + glucose injection) conditions. Scale bar corresponds to 20µM.