

**My Account**

Login  
Create Account

**Resources**

View All (813)

Adenoviruses (137)

Antibodies (175)

Bioimages (67)

Genomics Studies (145)

mESC Lines (68)

Mouse Strains (120)

Miscellaneous (46)

Protocols (55)

Research Data (4)

Resource Tags (389)

Visualization (9)

**Research & Cores**

Core Facilities (5)

Research Highlights (5)

Research Networks

Research Objectives

**Information**

About the BCBC

BCBC Events

Branding & Logos

Career Opportunities

Health

NIH hESC Registry

Policies & Guidelines

Member Publications

Research Programs



Research Investigators

Member Directory

Tutorials

## Foxa2 controls vesicle docking and insulin secretion in mature beta-cells - Study GBCO3042

**Genomics Study Specifications**

<b>Study Name</b>	Foxa2 controls vesicle docking and insulin secretion in mature beta-cells
<b>Contact Name</b>	<a href="#">Klaus Kaestner</a> (University of Pennsylvania)
<b>Publication</b>	<a href="http://www.ncbi.nlm.nih.gov/pubmed/17908556">http://www.ncbi.nlm.nih.gov/pubmed/17908556</a>
<b>My Strategies</b>	<a href="#">Return to My Strategies page</a>
<b>Classification</b>	Targets and roles of transcriptional regulators
<b>Links</b>	 <a href="#">Biomaterials Graph</a>  <a href="#">ArrayExpress</a>
<b>BCBC Release Date</b>	September 17, 2007
<b>Public Release Date</b>	September 17, 2007
<b>Citation</b>	Gao N, White P, Doliba N, Golson ML, Matschinsky FM, Kaestner KH. <a href="#">Foxa2 controls vesicle docking and insulin secretion in mature Beta cells</a> . Cell Metab. 2007. 6:267-79

**Synopsis****Study Description**

## Goals

## Approaches

## Results

## Conclusions

## Related Studies

The winged helix transcription factor Foxa2 regulates Pdx1 gene expression and fetal endocrine pancreas development. We show here by inducible gene ablation that Foxa2 inactivation in mature beta-cells induces hyperinsulinemic hypoglycemia in Foxa2loxP/loxP, Pdx1-CreERT2 adult mice. Mutant beta-cells exhibited a markedly increased pool of docked insulin granules, some of which were engaged in sequential or compound exocytosis, consistent with an increased first phase glucose-stimulated insulin secretion. Expression of multiple genes involved in vesicular trafficking, membrane targeting and fuel-secretion pathways is dependent on Foxa2. In addition, impaired cytosolic Ca<sup>2+</sup> oscillations and elevated intracellular cAMP production accompanied this secretory defect, and were likely contributors to the sensitization of the exocytotic machinery. Thus, in the absence of Foxa2, alterations in intracellular second messenger signaling redistribute the insulin granules into the readily releasable pool. We conclude that Foxa2 is required both for the fetal pancreas development and for the function of mature beta-cells.

**Platform types** Expression, Expression microarray

**Platforms** [Show platform Mouse PancChip](#)


**Study Design Type**

- genetic\_modification\_design


**Study Factors** [Show study factors](#)

**Study Assays** [Show study assays](#)

**Access to Study Data****Access Status**

 This resource is publicly viewable.

**Request this Resource**

 Request from a repository

Primary contributor: [Kaestner Lab](#)

Co-contributed by:

- [Stoeckert Lab](#)

**Resource Tags**

forkhead box A2, Foxa2, hyperinsulinemic hypoglycemia, Mouse PancChip 6.1


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 Read more about tags

**Resource History & Actions**

Approved on Sep 17, 2007

Last modified on Aug 02, 2011

 Login to edit or request an edit

**Related resources****BCBC**

No matching resources

**Other Consortia**

No matching resources

Data courtesy of [dkCOIN](#). Only public resources are displayed.

This Study Data is publicly available to all users.

### Gene List(s)

Use the following form(s) to refine the parameters and add the gene list to a strategy:

**Foxa2(loxP/loxP);Pdx1-CreERT2 versus Wild Type - Adult Mouse Islets**

|Fold Change| Greater Than:

Confidence Level: High Confidence  All Results

*For a microarray experiment a result with high confidence has a confidence level of at least 80%.*

*For a ChIP-chip experiment a result with high confidence has a confidence level of at least 90% and all fold changes are positive.*

Reference (Denominator): Wild Type

### Genome Browser


There are no genome browser tracks currently available for this study.

### Lists of Locations

There are no genomic location datasets currently available for this study.

### Repositories


**Kaestner Lab**

 Request this resource

**Stock #:** *Not provided*  
**Availability Notes:** *Not provided*

### Comments

There are no comments for this entry.

 Login to add comments

