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Transcription profiling of mouse pancreatic islets on day 14.5 of pregnancy - Study GBCO3735

Genomics Study Specifications

Study Name	Transcription profiling of mouse pancreatic islets on day 14.5 of pregnancy
Contact Name	Klaus Kaestner (University of Pennsylvania)
Publication	http://www.ncbi.nlm.nih.gov/pubmed/19574445
My Strategies	Return to My Strategies page
Classification	Cell stimulation/injury; Pancreas development and growth; Islet/beta-cell stimulation/injury
Links	Biomaterials Graph ArrayExpress
BCBC Release Date	January 19, 2010
Public Release Date	January 19, 2010
Citation	Rieck S, White P, Schug J, Fox AJ, Smirnova O, Gao N, Gupta RK, Wang ZV, Scherer PE, Keller MP, Attie AD, Kaestner KH. The transcriptional response of the islet to pregnancy in mice. Mol Endocrinol. 2009. 23:1702-12
Synopsis	Study Description | Goals | Approaches | Results | Conclusions | Related Studies The inability of the beta-cell to meet the demand for insulin brought about by insulin resistance leads to type 2 diabetes. In adults, beta-cell replication is one of the mechanisms thought to cause the expansion of beta-cell mass. Efforts to treat diabetes require knowledge of the pathways that drive facultative beta-cell proliferation in vivo. A robust physiological stimulus of beta-cell expansion is pregnancy, and identifying the mechanisms underlying this stimulus may provide therapeutic leads for the treatment of type 2 diabetes. The peak in beta-cell proliferation during pregnancy occurs on day 14.5 of gestation in mice. Using advanced genomic approaches, we globally characterize the gene expression signature of pancreatic islets on day 14.5 of gestation during pregnancy. We identify a total of 1,907 genes as differentially expressed in the islet during pregnancy. We demonstrate that the islets ability to compensate for relative insulin deficiency during metabolic stress is associated with the induction of both proliferative and survival pathways. A comparison of the genes induced in three different models of islet expansion suggests that diverse mechanisms can be recruited to expand islet mass. The identification of many novel genes involved in islet expansion during pregnancy provides an important resource for diabetes researchers to further investigate how these factors contribute to the maintenance of not only islet mass, but ultimately beta-cell mass.
Platform types	Expression, Expression microarray
Platforms	Show platform Agilent Whole Mouse Genome Microarray 4x44K [G4122F]
Study Design Type	• stimulus_or_stress_design

Access Status

This resource is publicly viewable.

Request this Resource

Request from a repository

Primary contributor: Kaestner Lab

Resource Tags

Agilent Whole Human Genome Microarray 4x44K [G4112F], Birc5, Bmp1, Cdk4, Cish, Fbxl17, Fbxl21, Fbxo27, Fbxw15, Gdf3, Hopx, Il1rn, Itk, MIP-GFP transgenic mouse, Myc, Ngfr, Nupr1, Pap, Pax8, Prl2c5, Prlr, Reg3a, Reg3b, Socs2, Tnfrsf11b, Tph1, Tph2

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Resource History & Actions

Approved on Jan 19, 2010
Last modified on Aug 02, 2011

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Related resources

BCBC

No matching resources

Other Consortia

No matching resources

Data courtesy of dkCOIN. Only public resources are displayed.

Study Factors	Show study factors
Study Assays	Show study assays

Access to Study Data

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:

E14.5 pregnant versus non-pregnant mouse islets

|Fold Change| Greater Than: 1.5

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): Non-pregnant

Find Genes

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.

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