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# A Gene Expression Network Model of Type 2 Diabetes Links Cell Cycle - Study GBCO3407

## Genomics Study Specifications

| | |
|---|---|
| **Study Name** | A Gene Expression Network Model of Type 2 Diabetes Links Cell Cycle |
| **Contact Name** | Alan Attie (University of Wisconsin at Madison) |
| **Publication** | http://www.ncbi.nlm.nih.gov/pubmed/18347327 |
| **My Strategies** | Return to My Strategies page |
| **Classification** | Tissue expression, surveys and comparisons |
| **Links** | Biomaterials Graph<br>GEO |
| **BCBC Release Date** | July 07, 2008 |
| **Public Release Date** | July 07, 2008 |
| **Citation** | Keller MP, Choi Y, Wang P, Davis DB, Rabaglia ME, Oler AT, Stapleton DS, Argmann C, Schueler KL, Edwards S, Steinberg HA, Chaibub Neto E, Kleinhanz R, Turner S, Hellerstein MK, Schadt EE, Yandell BS, Kendziorski C, Attie AD. A gene expression network model of type 2 diabetes links cell cycle regulation in islets with diabetes susceptibility. Genome Res. 2008. 18:706-16 |

**Synopsis**

Study Description | Goals | Approaches | Results | Conclusions | Related Studies

Insulin resistance is necessary but not sufficient for the development of type 2 diabetes. Diabetes results when pancreatic beta-cells fail to compensate for insulin resistance by increasing insulin production through an expansion of beta-cell mass or increased insulin secretion. Communication between insulin target tissues and beta-cells may initiate this compensatory response. Correlated changes in gene expression between tissues can provide evidence for such intercellular communication. We profiled gene expression in six tissues of mice from an obesity-induced diabetes-resistant and a diabetes-susceptible strain before and after the onset of diabetes. We studied the correlation structure of mRNA abundance and identified 105 co-expression gene modules. We provide an interactive gene network model showing the correlation structure between the expression modules within and among the six tissues. This resource also provides a searchable database of gene expression profiles for all genes in six tissues in lean and obese diabetes-resistant and diabetes-susceptible mice, at 4 and 10 weeks of age. A cell cycle regulatory module in islets predicts diabetes susceptibility. The module predicts islet replication; we found a strong correlation between ^2 H_2 O incorporation into islet DNA /in vivo/ and the expression pattern of the cell cycle module. This pattern is highly correlated with that of several individual genes in insulin target tissues, including IGF2, which has been shown to promote beta-cell proliferation, suggesting that these genes may provide a link between insulin resistance and beta-cell proliferation. Experiment Overall Design: Type 2 diabetes is a disorder that involves an increased

## Access Status

This resource is publicly viewable.

## Request this Resource

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Primary contributor: Stoeckert Lab

## Resource Tags

Bmp1, bone morphogenetic protein 1, Diabetes, Gdf10, growth differentiation factor 10, Igf2, Igf2bp1, insulin-like growth factor 2, insulin-like growth factor 2 mRNA binding protein 1, nerve growth factor, Ngf, Rosetta/Merck Mouse 44k 1.0 microarray

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## Resource History & Actions

Approved on Jul 07, 2008
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.

demand for insulin brought about by insulin resistance, together with a failure to compensate with sufficient insulin production. Although Insulin resistance occurs in most obese individuals, diabetes is generally forestalled through compensation with increased insulin. This increase in insulin occurs through an expansion of beta-cell mass and/or increased insulin secretion by individual beta-cells. Failure to compensate for insulin resistance leads to type 2 diabetes. One way to understand the pathophysiology of diabetes is to examine the coordinate changes in gene expression that occur in insulin-responsive tissues and pancreatic islets in obese animals that either compensate for insulin resistance or progress to type 2 diabetes. In each case, there are groups of genes that undergo changes in expression in a highly correlated fashion. By identifying groups of correlated transcripts (gene expression modules) during the compensation and development of diabetes, we can gain insight into potential pathways and regulatory networks in obesity-induced diabetes. We study two strains of mice that differ in obesity-induced diabetes susceptibility. In this study, we surveyed gene expression in six tissues of lean and obese C57BL/6 (B6) and BTBR mice aged 4 wks and 10 wks. B6 mice remain essentially non-diabetic at all ages, irrespective of obesity. When obese, BTBR mice become severely diabetic by 10 weeks of age. By analyzing the correlation structure of the genes under three contrast conditions, obesity, strain, and age, we identified gene expression modules associated with the onset of diabetes and provide an interactive co-expression network model of type 2 diabetes. We found a key module that is comprised of cell cycle regulatory genes. In the islet, the expression profile of these transcripts accurately predicts diabetes and is highly correlated with islet cell proliferation.

| | |
|---|---|
| **Platform types** | Expression, Expression microarray |
| **Platforms** | Show platform Rosetta/Merck Mouse 44k 1.0 microarray |
| **Study Design Type** | • clinical_history_design<br>• disease_state_design<br>• individual_genetic_characteristics_design<br>• organism_part_comparison_design<br>• strain_or_line_design |
| **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:

**10wk versus 4wk - Obese BTBR 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): BTBR 4wk Obese Islets processed results

Find Genes

**Obese versus Lean - 4wk B6 Mouse islets**

Obese versus Lean - 10wk B6 Mouse islets

## 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

**Stoeckert Lab**

Request this resource

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

## Comments

*There are no comments for this entry.*

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