

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

Human skeletal muscle - type 2 diabetes and family history positive individuals - Mexican American - Study GBCO2363

Genomics Study Specifications

Study Name	Human skeletal muscle - type 2 diabetes and family history positive individuals - Mexican American
Contact Name	Mary-Elizabeth Patti (Joslin Diabetes Center and Harvard Medical School)
Publication	http://www.ncbi.nlm.nih.gov/pubmed/12832613
My Strategies	Return to My Strategies page
Classification	Cell stimulation/injury
Links	 Biomaterials Graph  ArrayExpress
BCBC Release Date	April 13, 2009
Public Release Date	April 13, 2009
Citation	Patti ME, Butte AJ, Crunkhorn S, Cusi K, Berria R, Kashyap S, Miyazaki Y, Kohane I, Costello M, Saccone R, Landaker EJ, Goldfine AB, Mun E, DeFronzo R, Finlayson J, Kahn CR, Mandarino LJ. Coordinated reduction of genes of oxidative metabolism in humans with insulin resistance and diabetes: Potential role of PGC1 and NRF1 . Proc Natl Acad Sci U S A. 2003. 100:8466-71

Synopsis**Study Description**

Goals

Approaches

Results



Conclusions

Related Studies

Type 2 diabetes mellitus (DM) is characterized by insulin resistance and pancreatic beta-cell dysfunction. In high-risk subjects, the earliest detectable abnormality is insulin resistance in skeletal muscle. Impaired insulin-mediated signaling, gene expression, and glycogen synthesis, and accumulation of intramyocellular triglycerides have all been linked with insulin resistance, but no specific defect responsible for insulin resistance and DM has been identified in humans. To identify genes potentially important in the pathogenesis of DM, we analyzed gene expression in skeletal muscle from healthy metabolically characterized nondiabetic (family history negative and positive for DM) and diabetic Mexican-American subjects. We demonstrate that insulin resistance and DM associate with reduced expression of multiple nuclear respiratory factor-1 (NRF-1)-dependent genes encoding key enzymes in oxidative metabolism and mitochondrial function. While NRF-1 expression is decreased only in diabetic subjects, expression of both PPARγ coactivator 1-alpha and -beta (PGC1-a/PPARGC1, and PGC1-b/PERC), coactivators of NRF-1 and PPARγ-dependent transcription, is decreased in both diabetic subjects and family history positive nondiabetic subjects. Decreased PGC1 expression may be responsible for decreased expression of NRF-dependent genes, leading to the metabolic disturbances characteristic of insulin resistance and DM.

Platform types

Expression microarray, Expression

Platforms**Access Status**
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Primary contributor: [Stoekert Lab](#)**Resource Tags**
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Resource History & Actions

Approved on Apr 13, 2009
Last modified on Aug 02, 2011

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Show platform Affymetrix
HuGeneFL

Study Design Type

- disease_state_design
- family_history_design
- replicate_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)

There are no gene lists currently available for this study.

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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