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Gene expression in PBMCs from children with diabetes - Study GBCO3203

Genomics Study Specifications

Study Name	Gene expression in PBMCs from children with diabetes
Contact Name	Ellen C. Kaizer (University of Texas Southwestern Medical Center)
Publication	http://www.ncbi.nlm.nih.gov/pubmed/17595242
My Strategies	Return to My Strategies page
Classification	Tissue expression, surveys and comparisons
Links	Biomaterials Graph ArrayExpress
BCBC Release Date	April 13, 2009
Public Release Date	April 13, 2009
Citation	Kaizer EC, Glaser CL, Chaussabel D, Banchereau J, Pascual V, White PC. Gene expression in peripheral blood mononuclear cells from children with diabetes. J Clin Endocrinol Metab. 2007. 92:3705-11
Synopsis	Study Description | Goals | Approaches | Results | Conclusions | Related Studies

Objective: We hypothesized that type 1 diabetes (T1D) is accompanied by changes in gene expression in peripheral blood mononuclear cells (PBMCs) due to dysregulation of adaptive and innate immunity, counterregulatory responses to immune dysregulation, insulin deficiency and hyperglycemia. Research Design and Methods: Microarray analysis was performed on PBMCs from 43 patients with newly diagnosed T1D, 12 patients with newly diagnosed type 2 diabetes (T2D) and 24 healthy controls. One and four month follow-up samples were obtained from 20 of the T1D patients. Results: Microarray analysis identified 282 genes differing in expression between newly diagnosed T1D patients and controls at a false discovery rate of 0.05. Changes in expression of interleukin-1 (IL1B), early growth response gene 3 (EGR3), and prostaglandin-endoperoxide; synthase 2 (PTGS2) resolved within four months of insulin therapy and were also observed in T2D suggesting that they resulted from hyperglycemia. With use of a knowledge base, 81/282 genes could be placed within a network of interrelated genes with predicted functions including apoptosis and cell proliferation. IL1B and the MYC oncogene were the most highly-connected genes in the network. IL1B was highly overexpressed in both T1D and T2D, whereas MYC was dysregulated only in T1D. Conclusion: T1D and T2D likely share a final common pathway for beta cell dysfunction that includes secretion of interleukin-1 and prostaglandins by immune effector cells, exacerbating existing beta cell dysfunction, and causing further hyperglycemia. The results identify several targets for disease-modifying therapy of diabetes and potential biomarkers for monitoring treatment efficacy. Experiment Overall Design: We obtained blood samples from 24 healthy volunteers,

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Approved on Apr 13, 2009
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43 newly diagnosed T1D patients and 12 newly diagnosed T2D patients. All study participants were between the ages of 2 and 18 years. We collected samples one and four months after diagnosis from the last 20 of the T1D patients. For each time point one sample did not pass quality control and was dropped from the analysis. Patients with T2D were distinguished from T1D on the basis of age, body habitus, Experiment Overall Design: presence (11/12 patients) of acanthosis nigricans, family history of type 2 diabetes (11/12 patients), and absence of autoantibodies to insulin, IA-2, and GAD65. We allowed low titers of insulin antibodies in T2D patients (< 4 U/mL), which have been previously reported. All but two Experiment Overall Design: of the T1D patients with positive anti-insulin antibodies were also positive for at least one additional autoantibody.

Platform types	Expression microarray, Expression
Platforms	Show platform Affymetrix HG-U133A
Study Design Type	• disease_state_design • time_series_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

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

Comments

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