

# Resource Summary Report

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## DGAP

RRID:SCR\_003036

Type: Tool

### Proper Citation

DGAP (RRID:SCR\_003036)

### Resource Information

**URL:** <http://www.diabetesgenome.org>

**Proper Citation:** DGAP (RRID:SCR\_003036)

**Description:** Produce resources to unravel the interface between insulin action, insulin resistance and the genetics of type 2 diabetes including an annotated public database, standardized protocols for gene expression and proteomic analysis, and ultimately diabetes-specific and insulin action-specific DNA chips for investigators in the field. The project aims to identify the sets of the genes involved in insulin action and the predisposition to type 2 diabetes, as well as the secondary changes in gene expression that occur in response to the metabolic abnormalities present in diabetes. There are five major and one pilot project involving human and rodent tissues that are designed to: \* Create a database of the genes expressed in insulin-responsive tissues, as well as accessible tissues, that are regulated by insulin, insulin resistance and diabetes. \* Assess levels and patterns of gene expression in each tissue before and after insulin stimulation in normal and genetically-modified rodents; normal, insulin resistant and diabetic humans, and in cultured and freshly isolated cell models. \* Correlate the level and patterns of expression at the mRNA and/or protein level with the genetic and metabolic phenotype of the animal or cell. \* Generate genomic sequence from a panel of humans with type 2 diabetes focusing on the genes most highly regulated by insulin and diabetes to determine the range of sequence and expression variation in these genes and the proteins they encode, which might affect the risk of diabetes or insulin resistance. The DGAP project will define: \* the normal anatomy of gene expression, i.e. basal levels of expression and response to insulin. \* the morbid anatomy of gene expression, i.e., the impact of diabetes on expression patterns and the insulin response. \* the extent to which genetic variability might contribute to the alterations in expression or to diabetes itself.

**Abbreviations:** DGAP

**Synonyms:** The Diabetes Genome Anatomy Project, Diabetes Genome Anatomy Project

**Resource Type:** data or information resource, narrative resource, database, experimental protocol, resource

**Defining Citation:** [PMID:19786482](#)

**Keywords:** gene, insulin action, predisposition, gene expression, metabolic abnormality, diabetes, insulin resistance, genetics, insulin, genetic variation, proteomics, genomics, affymetrix oligonucleotide array, microarray, protein, genomic sequence, data set

**Related Condition:** Type 2 diabetes, Normal, Insulin resistance

**Funding:** NIDDK

**Availability:** THIS RESOURCE IS NO LONGER IN SERVICE

**Resource Name:** DGAP

**Resource ID:** SCR\_003036

**Alternate IDs:** nif-0000-30414

**Record Creation Time:** 20220129T080216+0000

**Record Last Update:** 20260801T190216+0000

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## Ratings and Alerts

No rating or validation information has been found for DGAP .

No alerts have been found for DGAP .

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## Data and Source Information

**Source:** [SciCrunch Registry](#)

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## Usage and Citation Metrics

We found 9 mentions in open access literature.

**Listed below are recent publications.** The full list is available at [ASWG](#) .

Kumar A, et al. (2017) SVMRFE based approach for prediction of most discriminatory gene target for type II diabetes. Genomics data, 12, 28.

Gao S, et al. (2013) NCOA5 haploinsufficiency results in glucose intolerance and subsequent hepatocellular carcinoma. Cancer cell, 24(6), 725.

Li X, et al. (2010) Bio-informatics analysis of a gene co-expression module in adipose tissue containing the diet-responsive gene Nnat. BMC systems biology, 4, 175.

Baudot A, et al. (2009) Translational disease interpretation with molecular networks. *Genome biology*, 10(6), 221.

Doria A, et al. (2008) The emerging genetic architecture of type 2 diabetes. *Cell metabolism*, 8(3), 186.

Liu M, et al. (2007) Network-based analysis of affected biological processes in type 2 diabetes models. *PLoS genetics*, 3(6), e96.

Drake TA, et al. (2006) Integrating genetic and gene expression data: application to cardiovascular and metabolic traits in mice. *Mammalian genome : official journal of the International Mammalian Genome Society*, 17(6), 466.

Gunton JE, et al. (2005) Loss of ARNT/HIF1beta mediates altered gene expression and pancreatic-islet dysfunction in human type 2 diabetes. *Cell*, 122(3), 337.

Sethi JK, et al. (2004) A burst of energy in metabolic disease research. *Genome biology*, 5(6), 327.