Resource Summary Report

Generated by NIF on Sep 10, 2026

T1DBase

RRID:SCR_007959
Type: Tool

Proper Citation

T1DBase (RRID:SCR_007959)

Resource Information

URL: http://t1dbase.org/

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Description: THIS RESOURCE IS NO LONGER IN SERVICE. Documented on August 26,2019. In October 2016, T1DBase has merged with its sister site ImmunoBase (https://immunobase.org). Documented on March 2020, ImmunoBase ownership has been transferred to Open Targets (https://www.opentargets.org). Results for all studies can be explored using Open Targets Genetics (https://genetics.opentargets.org). Database focused on genetics and genomics of type 1 diabetes susceptibility providing a curated and integrated set of datasets and tools, across multiple species, to support and promote research in this area. The current data scope includes annotated genomic sequences for suspected T1D susceptibility regions; genetic data; microarray data; and global datasets, generally from the literature, that are useful for genetics and systems biology studies. The site also includes software tools for analyzing the data.

Synonyms: T1DBase - Type 1 Diabetes Database

Resource Type: data or information resource, data repository, database, resource, service resource, storage service resource

Defining Citation: PMID:20937630

Keywords: genetics, beta cell, gene, variant, region, genomics, gene expression, genome-wide association study, data analysis service, bio.tools

Related Condition: Type 1 diabetes. Diabetes

Funding: Wellcome Trust ;
NIDDK ;
Juvenile Diabetes Research Foundation

Availability: THIS RESOURCE IS NO LONGER IN SERVICE.

Resource Name: T1DBase

Resource ID: SCR_007959

Alternate IDs: nif-0000-03531, biotools:t1dbase

Alternate URLs: https://bio.tools/t1dbase

License: GNU General Public License, Perl Artistic License

Record Creation Time: 20220129T080244+0000

Record Last Update: 20260905T132615+0000

Ratings and Alerts

No rating or validation information has been found for T1DBase .

No alerts have been found for T1DBase .

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 147 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF .

Dule N, et al. (2025) LRRK2 kinase modulates glucose-stimulated insulin secretion via RAB8 phosphorylation and ciliogenesis. Cellular and molecular life sciences : CMLS, 82(1), 276.

Corral-Pujol M, et al. (2023) NOD mouse dorsal root ganglia display morphological and gene expression defects before and during autoimmune diabetes development. Frontiers in endocrinology, 14, 1176566.

Zhang J, et al. (2022) Differences of Circulating CD25hi Bregs and Their Correlations with CD4 Effector and Regulatory T Cells in Autoantibody-Positive T1D Compared with Age-Matched Healthy Individuals. Journal of immunology research, 2022, 2269237.

van Megen KM, et al. (2021) 1,25-dihydroxyvitamin D3 induces stable and reproducible therapeutic tolerogenic dendritic cells with specific epigenetic modifications. Cytotherapy, 23(3), 242.

Kolvenbach CM, et al. (2019) Rare Variants in BNC2 Are Implicated in Autosomal-Dominant Congenital Lower Urinary-Tract Obstruction. American journal of human genetics, 104(5), 994.

Redondo MJ, et al. (2018) A Type 1 Diabetes Genetic Risk Score Predicts Progression of Islet Autoimmunity and Development of Type 1 Diabetes in Individuals at Risk. Diabetes care, 41(9), 1887.

Lindeløv Vestergaard A, et al. (2018) MicroRNAs and histone deacetylase inhibition-mediated protection against inflammatory ?-cell damage. PloS one, 13(9), e0203713.

Oh E, et al. (2018) Syntaxin 4 Expression in Pancreatic ?-Cells Promotes Islet Function and Protects Functional ?-Cell Mass. Diabetes, 67(12), 2626.

Jung Y, et al. (2018) Isl1? Overexpression With Key ? Cell Transcription Factors Enhances Glucose-Responsive Hepatic Insulin Production and Secretion. Endocrinology, 159(2), 869.

Bonifacio E, et al. (2018) Genetic scores to stratify risk of developing multiple islet autoantibodies and type 1 diabetes: A prospective study in children. PLoS medicine, 15(4), e1002548.

Ziegler AB, et al. (2018) JhI-21 plays a role in Drosophila insulin-like peptide release from larval IPCs via leucine transport. Scientific reports, 8(1), 1908.

Woo HJ, et al. (2017) Collective Genetic Interaction Effects and the Role of Antigen-Presenting Cells in Autoimmune Diseases. PloS one, 12(1), e0169918.

Kasela S, et al. (2017) Pathogenic implications for autoimmune mechanisms derived by comparative eQTL analysis of CD4+ versus CD8+ T cells. PLoS genetics, 13(3), e1006643.

Li W, et al. (2017) Network Pharmacology Studies on the Bioactive Compounds and Action Mechanisms of Natural Products for the Treatment of Diabetes Mellitus: A Review. Frontiers in pharmacology, 8, 74.

Banday VS, et al. (2017) Elevated systemic glutamic acid level in the non-obese diabetic mouse is Idd linked and induces beta cell apoptosis. Immunology, 150(2), 162.

Mishra R, et al. (2017) Relative contribution of type 1 and type 2 diabetes loci to the genetic etiology of adult-onset, non-insulin-requiring autoimmune diabetes. BMC medicine, 15(1), 88.

Luan M, et al. (2017) The shared and specific mechanism of four autoimmune diseases. Oncotarget, 8(65), 108355.

Leech CA, et al. (2017) Stromal Interaction Molecule 1 (STIM1) Regulates ATP-sensitive Potassium (KATP) and Store-operated Ca2+ Channels in MIN6 ?-Cells. The Journal of biological chemistry, 292(6), 2266.

Engelmann I, et al. (2017) Persistent coxsackievirus B4 infection induces microRNA dysregulation in human pancreatic cells. Cellular and molecular life sciences : CMLS, 74(20), 3851.

, et al. (2017) Association Between Telomere Length and Risk of Cancer and Non-Neoplastic Diseases: A Mendelian Randomization Study. JAMA oncology, 3(5), 636.