Resource Summary Report

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Diabetes Genes

RRID:SCR_008639
Type: Tool

Proper Citation

Diabetes Genes (RRID:SCR_008639)

Resource Information

URL: http://www.diabetesgenes.org

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Description: Our genes and lifestyle factors, such as calorie rich diets and a lack of exercise, contribute to the development of Type 2 diabetes. But what we dont know is the exact nature of the genetic risk and how this interacts with lifestyle factors to cause diabetes. The Diabetes UK Warren 2 Group was formed in 1992 to investigate the genetic basis of Type 2 diabetes. The Group, comprising of researchers from six UK diabetes research centres, began by recruiting families into the study enriched for Type 2 diabetes ie having two or more siblings with the condition. With over 2000 individuals from 843 families in the collection, it is now being used to search for the genes that make people susceptible to Type 2 diabetes. When identifying susceptibility genes it is important to know how the gene affects normal metabolism in relatives without diabetes. In 1999. the Warren 2 Extension study recruited first degree relatives (siblings and children) of the original Warren 2 families, who did not have diabetes, in the knowledge that they would also be enriched with the same susceptibility genes. This study recruited 811 relatives without diabetes (586 offspring and 225 siblings) all of whom have undergone detailed metabolic assessment. A similar study was undertaken in Oxford called the Diabetes In Families (DIF) Study. In 2001, the Warren 2 Trios and Duos Study recruited 500 families from around the country consisting of an individual with Type 2 diabetes and both their parents (trios) or an individual with diabetes, one of their parents and at least 2 siblings (duos). In addition to this, a further 1500 individuals with diabetes were recruited as part of the Warren 2 Cases study. This website is run by the Diabetes Research department and the Centre for Molecular Genetics at the Peninsula Medical School and Royal Devon and Exeter Hospital, Exeter, UK.

Synonyms: Diabetes Genes

Resource Type: data or information resource, database

Keywords: FASEB list

Resource Name: Diabetes Genes

Resource ID: SCR_008639

Alternate IDs: nif-0000-32037

Record Creation Time: 20220129T080248+0000

Record Last Update: 20260811T043331+0000

Ratings and Alerts

No rating or validation information has been found for Diabetes Genes.

No alerts have been found for Diabetes Genes.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 55 mentions in open access literature.

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

Russ-Silsby J, et al. (2026) Loss of function variants in the primate-specific gene ZNF808 cause neonatal, transient and adult-onset diabetes. EBioMedicine, 124, 106113.

Wade AN, et al. (2026) Insights into the genetic aetiology of diabetes in Africa. EBioMedicine, 127, 106265.

Russ-Silsby J, et al. (2025) Global perspectives on monogenic forms of diabetes. Diabetologia, 68(11), 2362.

Donis R, et al. (2025) A homozygous TARS2 variant is a novel cause of syndromic neonatal diabetes. Diabetic medicine : a journal of the British Diabetic Association, 42(3), e15471.

Peghinelli VV, et al. (2024) MODY calculator applied in patients with clinical diagnosis of type 1 diabetes mellitus: Is a higher cutoff needed? Heliyon, 10(16), e36006.

De Sousa SMC, et al. (2024) Identification of monogenic diabetes in an Australian cohort using the Exeter maturity-onset diabetes of the young (MODY) probability calculator and next-generation sequencing gene panel testing. Acta diabetologica, 61(2), 181.

Johnson MB, et al. (2024) Human inherited PD-L1 deficiency is clinically and immunologically less severe than PD-1 deficiency. The Journal of experimental medicine, 221(6).

Alemán-Contreras R, et al. (2024) Utility of Fasting C-Peptide for the Diagnostic Differentiation of Patients with Type 1, Type 2 Diabetes, MODY, and LADA. Life (Basel, Switzerland), 14(5).

Balogun WO, et al. (2024) Implementing genetic testing in diabetes: Knowledge, perceptions of healthcare professionals, and barriers in a developing country. Population medicine, 6.

Welsch S, et al. (2024) A New Tool to Identify Pediatric Patients with Atypical Diabetes Associated with Gene Polymorphisms. Diabetes & metabolism journal, 48(5), 949.

Murphy R, et al. (2023) A Systematic Review of the use of Precision Diagnostics in Monogenic Diabetes. medRxiv : the preprint server for health sciences.

Globa E, et al. (2023) Neonatal and early-onset diabetes in Ukraine: Atypical features and mortality. Diabetic medicine : a journal of the British Diabetic Association, 40(5), e15013.

Harsunen M, et al. (2023) Identification of monogenic variants in more than ten per cent of children without type 1 diabetes-related autoantibodies at diagnosis in the Finnish Pediatric Diabetes Register. Diabetologia, 66(3), 438.

Wyatt RC, et al. (2023) FOXP3 TSDR Measurement Could Assist Variant Classification and Diagnosis of IPEX Syndrome. Journal of clinical immunology, 43(3), 662.

Taylor R, et al. (2023) Aetiology of Type 2 diabetes in people with a 'normal' body mass index: testing the personal fat threshold hypothesis. Clinical science (London, England : 1979), 137(16), 1333.

Murphy R, et al. (2023) The use of precision diagnostics for monogenic diabetes: a systematic review and expert opinion. Communications medicine, 3(1), 136.

Greeley SAW, et al. (2022) ISPAD Clinical Practice Consensus Guidelines 2022: The diagnosis and management of monogenic diabetes in children and adolescents. Pediatric diabetes, 23(8), 1188.

da Silva Santos T, et al. (2022) MODY probability calculator utility in individuals' selection for genetic testing: Its accuracy and performance. Endocrinology, diabetes & metabolism, 5(5), e00332.

Ngoc CTB, et al. (2021) Molecular Genetics, Clinical Characteristics, and Treatment Outcomes of KATP-Channel Neonatal Diabetes Mellitus in Vietnam National Children's Hospital. Frontiers in endocrinology, 12, 727083.

Gaál Z, et al. (2021) A Comprehensive Analysis of Hungarian MODY Patients-Part II: Glucokinase MODY Is the Most Prevalent Subtype Responsible for about 70% of Confirmed Cases. Life (Basel, Switzerland), 11(8).