

Resource Summary Report

Generated by [ASWG](#) on Aug 2, 2026

deCODE genetics

RRID:SCR_003334

Type: Tool

Proper Citation

deCODE genetics (RRID:SCR_003334)

Resource Information

URL: <http://www.decode.com/>

Proper Citation: deCODE genetics (RRID:SCR_003334)

Description: A biopharmaceutical company applying its discoveries in human genetics to develop drugs and diagnostics for common diseases. They specialize in gene discovery - their population approach and resources have enabled them to isolate key genes contributing to major public health challenges from cardiovascular disease to cancer. The company's genotyping capacity is now one of the highest in the world. They have a large population-based biobank containing whole blood and DNA samples with extensive relevant phenotypic information from around 120.000 Icelanders. In the company's work in more than 50 disease projects, their statistical and informatics departments have established themselves in data processing and analysis. deCODE genetics is widely recognized as a center of excellence in genetic research.

Abbreviations: deCODE

Synonyms: Islensk Erfdagreining EHF, Islensk Erfdagreining

Resource Type: commercial organization

Keywords: biopharmaceutical, genetics, drug, diagnostic, genotyping, phenotype, data processing, analysis, genetic variant, risk factor, genome, blood, dna, biobank, single nucleotide polymorphism

Related Condition: Schizophrenia, Cardiovascular disease, Cancer, Type 2 diabetes, Atrial fibrillation, Heart attack

Availability: Free, Freely available

Resource Name: deCODE genetics

Resource ID: SCR_003334

Alternate IDs: nif-0000-31959, ISNI: 0000 0004 0618 6889, grid.421812.c, Wikidata: Q493712

Alternate URLs: <https://ror.org/04dzdm737>

Record Creation Time: 20220129T080218+0000

Record Last Update: 20260801T190220+0000

Ratings and Alerts

No rating or validation information has been found for deCODE genetics.

No alerts have been found for deCODE genetics.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 57 mentions in open access literature.

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

Li H, et al. (2025) Multi-ancestry sequencing-based genome-wide association study of C-reactive protein in 513,273 genomes. Nature communications, 16(1), 3892.

Hu M, et al. (2025) Identifying Key Biomarkers Related to Immune Response in the Progression of Diabetic Kidney Disease: Mendelian Randomization Combined With Comprehensive Transcriptomics and Single-Cell Sequencing Analysis. Journal of inflammation research, 18, 949.

Lv R, et al. (2025) Investigating causal relationships between plasma proteins and lung adenocarcinoma: result from proteomics and Mendelian randomization study. Discover oncology, 16(1), 42.

Li R, et al. (2025) Mendelian randomization study on the causal relationships among fasting blood glucose, plasma proteins, and squamous cell lung cancer. Discover oncology, 16(1), 588.

Chen W, et al. (2025) Causal Effect of Obstructive Sleep Apnea on Sick Sinus Syndrome: A Bidirectional Mendelian Randomization Study. Nature and science of sleep, 17, 689.

Chen Q, et al. (2025) Exploring the causal relationship between iron status and subarachnoid hemorrhage based on two sample mendelian randomization. Scientific reports, 15(1), 15549.

Fu J, et al. (2025) CCNE1 Exerts a Protective Effect on Parkinson's Disease by Regulating Ferroptosis-Related Proteins. Brain and behavior, 15(12), e71110.

Malomane DK, et al. (2025) Patterns of population structure and genetic variation within the Saudi Arabian population. *bioRxiv : the preprint server for biology*.

Sun H, et al. (2025) A multi-omics target study for glioblastoma multiforme (GBM) based on Mendelian randomization analysis. *IBRO neuroscience reports*, 18, 400.

Xu G, et al. (2025) Causality of Childhood and Adult Body Mass Index on Sick Sinus Syndrome: A Mendelian Randomization Study. *Cureus*, 17(3), e80913.

Fan W, et al. (2025) Mendelian randomization analysis of plasma proteins reveals potential novel tumor markers for gastric cancer. *Scientific reports*, 15(1), 3537.

Fu J, et al. (2025) Assessing the causal relationship between the plasma proteome and epilepsy: A Mendelian randomization study. *Epilepsia open*, 10(6), 1966.

Li J, et al. (2024) Proteome-wide Mendelian randomization identifies potential therapeutic targets for nonalcoholic fatty liver diseases. *Scientific reports*, 14(1), 11814.

Xu H, et al. (2024) Exploring the causal effect of complement and IgA nephropathy-a Mendelian randomization study. *Renal failure*, 46(2), 2436632.

Olafsdottir TA, et al. (2024) Sequence variants influencing the regulation of serum IgG subclass levels. *Nature communications*, 15(1), 8054.

Liu X, et al. (2024) Integrative genomic analysis of RNA-modification-single nucleotide polymorphisms associated with kidney function. *Heliyon*, 10(20), e38815.

Saevarsdottir S, et al. (2024) Start codon variant in LAG3 is associated with decreased LAG-3 expression and increased risk of autoimmune thyroid disease. *Nature communications*, 15(1), 5748.

Yu K, et al. (2024) Integrated analyses of single-cell transcriptome and Mendelian randomization reveal the protective role of FCRL3 in multiple sclerosis. *Frontiers in immunology*, 15, 1428962.

Pahlevan Kakhki M, et al. (2024) A genetic-epigenetic interplay at 1q21.1 locus underlies CHD1L-mediated vulnerability to primary progressive multiple sclerosis. *Nature communications*, 15(1), 6419.

Song J, et al. (2024) Identifying new biomarkers and potential therapeutic targets for breast cancer through the integration of human plasma proteomics: a Mendelian randomization study and colocalization analysis. *Frontiers in endocrinology*, 15, 1449668.