

Resource Summary Report

Generated by [PRECISE-TBI](#) on Sep 7, 2026

GWAS: Catalog of Published Genome-Wide Association Studies

RRID:SCR_012745

Type: Tool

Proper Citation

GWAS: Catalog of Published Genome-Wide Association Studies (RRID:SCR_012745)

Resource Information

URL: <http://www.ebi.ac.uk/gwas/>

Proper Citation: GWAS: Catalog of Published Genome-Wide Association Studies (RRID:SCR_012745)

Description: Catalog of published genome-wide association studies. Genome-wide set of genetic variants in different individuals to see if any variant is associated with trait and disease. Database of genome-wide association study (GWAS) publications including only those attempting to assay single nucleotide polymorphisms (SNPs). Publications are organized from most to least recent date of publication. Studies are identified through weekly PubMed literature searches, daily NIH-distributed compilations of news and media reports, and occasional comparisons with an existing database of GWAS literature (HuGE Navigator). Works with HANCESTRO ancestry representation.

Abbreviations: GWASC

Synonyms: A Catalog of Published Genome-Wide Association Studies, Catalog of Published GWAS, Catalog of published GWAS studies, NHGRI GWAS Catalog, Catalog of Published Genome-Wide Association Studies, GWAS and PGS Catalogs

Resource Type: catalog, data or information resource, database

Defining Citation: [PMID:19474294](#)

Keywords: gene-wide association study, adult, genome, genome-wide association study, single nucleotide polymorphism, publication, literature, phenotype, trait, disease, loci, genetic variant, disorder, snp trait association

Funding: BBSRC ;
NHGRI U24 HG012542;
NHGRI U41 HG007823

Availability: Free, Freely available

Resource Name: GWAS: Catalog of Published Genome-Wide Association Studies

Resource ID: SCR_012745

Alternate IDs: nif-0000-06666, r3d100014209

Old URLs: <http://www.genome.gov/gwastudies>

Record Creation Time: 20220129T080312+0000

Record Last Update: 20260905T132726+0000

Ratings and Alerts

No rating or validation information has been found for GWAS: Catalog of Published Genome-Wide Association Studies.

No alerts have been found for GWAS: Catalog of Published Genome-Wide Association Studies.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 899 mentions in open access literature.

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

He J, et al. (2026) A Comprehensive Assessment of the Causal Relationship between Atopic Conditions and Pancreatic Cancer Risk: A Two-Sample Mendelian Randomization Study. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology, 35(6), 1009.

Yuan C, et al. (2026) Kidney function decline and kidney stone risk: cohort and Mendelian randomization analyses. Renal failure, 48(1), 2641919.

Zhang Y, et al. (2026) A new perspective on population genetics: Deciphering the relationship between genetic variants and disease prevalence in Psoriasis. PloS one, 21(3), e0344204.

McMahon A, et al. (2026) Predicted Effector Gene Aggregation, Standards and Unified Schema (PEGASUS): A Community Framework for Effector Gene Reporting. bioRxiv : the preprint server for biology.

- Chen J, et al. (2026) A HRH1-YAP1 feedback loop drives pancreatic cancer progression and predicts therapeutic response. *Oncology letters*, 32(2), 364.
- Herrera-Rivero M, et al. (2026) Genome-wide association study of atopic and autoimmune comorbidities in alopecia areata. *Frontiers in immunology*, 17, 1810658.
- Mitchell BL, et al. (2026) Genetic exploration of the relationship between liability to psychiatric disorders and acne vulgaris. *European journal of human genetics : EJHG*, 34(4), 565.
- Soremekun O, et al. (2026) Linking the plasma proteome to genetics in individuals from continental Africa provides insights into type 2 diabetes pathogenesis. *Nature genetics*, 58(1), 39.
- Yin Q, et al. (2026) Genetic landscape of morphine response in BXD recombinant inbred mice. *HGG advances*, 7(1), 100535.
- Yang S, et al. (2026) Causal inference and mediating pathways of metabolic syndrome on calcific aortic valve stenosis: linkage disequilibrium score regression and two-sample, two-step mendelian randomization study. *Journal of cardiothoracic surgery*, 21(1).
- Kyomen S, et al. (2026) Cis-regulatory evolution shapes facial diversity in birds and mammals. *Science advances*, 12(19), eaec2511.
- Du M, et al. (2026) Cross-trait genomic modeling reveals the polygenic architecture and systemic impact of MASLD. *Science advances*, 12(7), eaeb5665.
- Deng L, et al. (2026) Impact of gut microbiome, plasma metabolites, peripheral immune cells, and circulating inflammatory protein on chronic spontaneous urticaria: Bidirectional 2-sample Mendelian randomization study and mediation analysis. *The journal of allergy and clinical immunology. Global*, 5(3), 100686.
- Teixeira SK, et al. (2026) Ancestry-informative regulatory variants at KCNB1 modulate adipogenesis and body mass index. *Frontiers in endocrinology*, 17, 1805824.
- Liu S, et al. (2025) DNA methylation associations with cognitive function in early-stage hormone receptor-positive breast cancer patients. *Epigenomics*, 17(13), 879.
- Marino GB, et al. (2025) GeneSetCart: assembling, augmenting, combining, visualizing, and analyzing gene sets. *GigaScience*, 14.
- Xiong Z, et al. (2025) Combined genome-wide association study of facial traits in Europeans increases explained variance and improves prediction. *Nature communications*, 16(1), 6562.
- Zhou R, et al. (2025) Establishing the relationships between obesity and genetically predicted serum micronutrient levels: a multivariable Mendelian randomization analysis. *Eating and weight disorders : EWD*, 30(1), 33.
- Wang FM, et al. (2025) Characterizing aging-related genetic and physiological determinants of spinal curvature. *Communications medicine*, 5(1), 291.

Bradley J, et al. (2025) Novel early-onset Alzheimer-associated genes influence risk through dysregulation of glutamate, immune activation, and intracellular signaling pathways. *Alzheimer's & dementia : the journal of the Alzheimer's Association*, 21(6), e70377.