

Resource Summary Report

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CGEMS

RRID:SCR_008445

Type: Tool

Proper Citation

CGEMS (RRID:SCR_008445)

Resource Information

URL: <http://cgems.cancer.gov>

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Description: The project began as a pilot study to identify inherited genetic susceptibility to prostate and breast cancer. CGEMS has developed into a robust research program involving genome-wide association studies (GWASs) for a number of cancers to identify common genetic variants that affect a person's risk of developing cancer. In collaboration with extramural scientists, NCI's Division of Cancer Epidemiology and Genetics (DCEG) has carried out genome-wide scans for breast, prostate, pancreatic, and lung cancers, while a GWAS of bladder cancer is currently underway. By making the data available to both intramural and extramural research scientists, as well as those in the private sector through rapid posting, NIH can leverage its resources to ensure that the dramatic advances in genomics are incorporated into rigorous population-based studies. Ultimately, findings from these studies may yield new preventive, diagnostic, and therapeutic interventions for cancer. Sponsors: This resource is supported by the U.S. National Institutes Of Health.

Abbreviations: CGEMS

Synonyms: The Cancer Genetic Markers of Susceptibility Project, Cancer Genetic Markers of Susceptibility, The Cancer Genetic Markers of Susceptibility

Resource Type: data or information resource, portal, topical portal

Keywords: cancer, genetic, marker, prostate, breast, research, genome, association, development, scientist, pancreatic, lung, bladder, science, genomics, population, study, therapy, diagnosis

Resource Name: CGEMS

Resource ID: SCR_008445

Alternate IDs: nif-0000-30287

Record Creation Time: 20220129T080247+0000

Record Last Update: 20260912T195704+0000

Ratings and Alerts

No rating or validation information has been found for CGEMS.

No alerts have been found for CGEMS.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 28 mentions in open access literature.

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

Flom LT, et al. (2026) Characterization of Neuronal Ensembles in a Model of Dual-Reward Conditioned Place Preference. eNeuro, 13(6).

Jalili M, et al. (2021) Exploring the Metabolic Heterogeneity of Cancers: A Benchmark Study of Context-Specific Models. Journal of personalized medicine, 11(6).

Ostrom QT, et al. (2018) Sex-specific glioma genome-wide association study identifies new risk locus at 3p21.31 in females, and finds sex-differences in risk at 8q24.21. Scientific reports, 8(1), 7352.

Gao G, et al. (2017) Trans-ethnic predicted expression genome-wide association analysis identifies a gene for estrogen receptor-negative breast cancer. PLoS genetics, 13(9), e1006727.

Wang W, et al. (2017) Pathway-based discovery of genetic interactions in breast cancer. PLoS genetics, 13(9), e1006973.

Quiroz-Z??rate A, et al. (2017) Expression Quantitative Trait loci (QTL) in tumor adjacent normal breast tissue and breast tumor tissue. PloS one, 12(2), e0170181.

Henrion MY, et al. (2015) Common variation at 1q24.1 (ALDH9A1) is a potential risk factor for renal cancer. PloS one, 10(3), e0122589.

Kinnersley B, et al. (2015) Quantifying the heritability of glioma using genome-wide complex trait analysis. Scientific reports, 5, 17267.

Lavender N, et al. (2015) Evaluation of Oxidative Stress Response Related Genetic Variants, Pro-oxidants, Antioxidants and Prostate Cancer. AIMS medical science, 2(4),

Kinnersley B, et al. (2015) Genome-wide association study identifies multiple susceptibility loci for glioma. *Nature communications*, 6, 8559.

Zhang B, et al. (2014) Large-scale genetic study in East Asians identifies six new loci associated with colorectal cancer risk. *Nature genetics*, 46(6), 533.

Savage SA, et al. (2013) Genome-wide association study identifies two susceptibility loci for osteosarcoma. *Nature genetics*, 45(7), 799.

Enciso-Mora V, et al. (2013) Low penetrance susceptibility to glioma is caused by the TP53 variant rs78378222. *British journal of cancer*, 108(10), 2178.

Jiang X, et al. (2012) Mining pure, strict epistatic interactions from high-dimensional datasets: ameliorating the curse of dimensionality. *PloS one*, 7(10), e46771.

Lavender NA, et al. (2012) Interaction among apoptosis-associated sequence variants and joint effects on aggressive prostate cancer. *BMC medical genomics*, 5, 11.

Charoentong P, et al. (2012) Bioinformatics for cancer immunology and immunotherapy. *Cancer immunology, immunotherapy* : CII, 61(11), 1885.

Campa D, et al. (2011) Genetic variability of the mTOR pathway and prostate cancer risk in the European Prospective Investigation on Cancer (EPIC). *PloS one*, 6(2), e16914.

Jiang X, et al. (2011) A bayesian method for evaluating and discovering disease loci associations. *PloS one*, 6(8), e22075.

Batra J, et al. (2011) Association between Prostinogen (KLK15) genetic variants and prostate cancer risk and aggressiveness in Australia and a meta-analysis of GWAS data. *PloS one*, 6(11), e26527.

Lindstrom S, et al. (2011) Characterizing associations and SNP-environment interactions for GWAS-identified prostate cancer risk markers--results from BPC3. *PloS one*, 6(2), e17142.