Resource Summary Report

Generated by dkNET on Apr 27, 2025

COLT-Cancer

RRID:SCR_006485
Type: Tool

Proper Citation

COLT-Cancer (RRID:SCR_006485)

Resource Information

URL: http://colt.ccbr.utoronto.ca/cancer/

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Description: The COLT-Cancer database is a collection of shRNA dropout signatures profiles, covering ~16000 human genes, and derived from more than 70 Pancreatic, Ovarian and Breast human cancer cell-lines using the microarray detection platform developed in the COLT (CCBR-OICR Lentiviral Technology) facility at the Moffat Lab. All shRNA dropout profiles are freely available through download or queries via this website.

Abbreviations: COLT-Cancer

Synonyms: CCBR-OICR Lentiviral Technology Cancer, COLT-Cancer database

Resource Type: service resource, data analysis service, data or information resource, database, production service resource, analysis service resource

Defining Citation: PMID:22102578

Keywords: gene, shrna profile, shrna, functional genetics, cancer, cell line, bio.tools

Related Condition: Pancreatic cancer, Ovarian cancer, Breast cancer

Funding: Ontario Institute for Cancer Research ;
Terry Fox Research Institute ;
Canadian Institutes of Health Research ;
Canada Foundation for Innovation ;
Ontario Research Fund

Availability: Free

Resource Name: COLT-Cancer

Resource ID: SCR_006485

Alternate IDs: biotools:colt-cancer, nlx_149426

Alternate URLs: https://bio.tools/colt-cancer

Record Creation Time: 20220129T080236+0000

Record Last Update: 20250426T055856+0000

Ratings and Alerts

No rating or validation information has been found for COLT-Cancer.

No alerts have been found for COLT-Cancer.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 11 mentions in open access literature.

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

McGee SR, et al. (2017) Network Analysis Reveals A Signaling Regulatory Loop in the PIK3CA-mutated Breast Cancer Predicting Survival Outcome. Genomics, proteomics & bioinformatics, 15(2), 121.

Jaiswal A, et al. (2017) Seed-effect modeling improves the consistency of genome-wide loss-of-function screens and identifies synthetic lethal vulnerabilities in cancer cells. Genome medicine, 9(1), 51.

Kanhaiya K, et al. (2017) Controlling Directed Protein Interaction Networks in Cancer. Scientific reports, 7(1), 10327.

Bridgett S, et al. (2017) CancerGD: A Resource for Identifying and Interpreting Genetic Dependencies in Cancer. Cell systems, 5(1), 82.

Davoli T, et al. (2016) Functional genomics reveals that tumors with activating phosphoinositide 3-kinase mutations are dependent on accelerated protein turnover. Genes

& development, 30(24), 2684.

Pritykin Y, et al. (2015) Genome-Wide Detection and Analysis of Multifunctional Genes. PLoS computational biology, 11(10), e1004467.

Vu V, et al. (2015) Natural Variation in Gene Expression Modulates the Severity of Mutant Phenotypes. Cell, 162(2), 391.

Chen CY, et al. (2014) Dissecting the human protein-protein interaction network via phylogenetic decomposition. Scientific reports, 4, 7153.

Zaman N, et al. (2013) Signaling network assessment of mutations and copy number variations predict breast cancer subtype-specific drug targets. Cell reports, 5(1), 216.

Shao DD, et al. (2013) ATARiS: computational quantification of gene suppression phenotypes from multisample RNAi screens. Genome research, 23(4), 665.

Galperin MY, et al. (2012) The 2012 Nucleic Acids Research Database Issue and the online Molecular Biology Database Collection. Nucleic acids research, 40(Database issue), D1.