Resource Summary Report

Generated by Kravitz on Sep 10, 2026

HMS LINCS Database

RRID:SCR_006454
Type: Tool

Proper Citation

HMS LINCS Database (RRID:SCR_006454)

Resource Information

URL: http://lincs.hms.harvard.edu/db/

Proper Citation: HMS LINCS Database (RRID:SCR_006454)

Description: Database that contains all publicly available HMS LINCS datasets and information for each dataset about experimental reagents and experimental and data analysis protocols. Experimental reagents include small molecule perturbagens, cells, antibodies, and proteins.

Abbreviations: LINCS, HMS-LINCS, HMS LINCS

Synonyms: NIH LINCS Program, NIH LINCS, Harvard Medical School LINCS Database, LINCS Program, Library of Integrated Network-based Cellular Signatures

Resource Type: data or information resource, data repository, database, service resource, storage service resource

Keywords: tumor, cancer, database, molecular signature, perturbing agent

Related Condition: Cancer, Diseased joint, Autoimmune disease

Funding: NHGRI U54 HG006097;
NIH Common Fund

Availability: Available to the research community

Resource Name: HMS LINCS Database

Resource ID: SCR_006454

Alternate IDs: nlx_156062, r3d100011833

Alternate URLs: http://lincs.hms.harvard.edu/, https://doi.org/10.17616/R3ZK9R

Record Creation Time: 20220129T080236+0000

Record Last Update: 20260905T132554+0000

Ratings and Alerts

No rating or validation information has been found for HMS LINCS Database.

No alerts have been found for HMS LINCS Database.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 15 mentions in open access literature.

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

König LE, et al. (2026) TYK2 mediates neuroinflammation in Alzheimer's disease brains with TDP-43 pathology. Nature communications, 17(1).

Paulino PJIV, et al. (2025) Identification of high-risk signatures and therapeutic targets through molecular characterization and immune profiling of TP53-mutant breast cancer. Journal, genetic engineering & biotechnology, 23(4), 100574.

Tang G, et al. (2025) Exploration of crucial stromal risk genes associated with prognostic significance and chemotherapeutic opportunities in invasive ductal breast carcinoma. Journal, genetic engineering & biotechnology, 23(1), 100448.

Mayr L, et al. (2025) Effective targeting of PDGFRA-altered high-grade glioma with avapritinib. Cancer cell, 43(4), 740.

Saez-Atienzar S, et al. (2024) Mechanism-free repurposing of drugs for C9orf72-related ALS/FTD using large-scale genomic data. Cell genomics, 4(11), 100679.

Kain J, et al. (2022) Mendelian randomization and pathway analysis demonstrate shared genetic associations between lupus and coronary artery disease. Cell reports. Medicine, 3(11), 100805.

Beelontally R, et al. (2017) Identification of compounds with anti-human cytomegalovirus activity that inhibit production of IE2 proteins. Antiviral research, 138, 61.

Lin Y, et al. (2017) Drug target ontology to classify and integrate drug discovery data. Journal of biomedical semantics, 8(1), 50.

Clark NA, et al. (2017) GRcalculator: an online tool for calculating and mining dose-response data. BMC cancer, 17(1), 698.

Williams CA, et al. (2016) Erk5 Is a Key Regulator of Naive-Primed Transition and Embryonic Stem Cell Identity. Cell reports, 16(7), 1820.

Hafner M, et al. (2016) Growth rate inhibition metrics correct for confounders in measuring sensitivity to cancer drugs. Nature methods, 13(6), 521.

Lin JR, et al. (2015) Highly multiplexed imaging of single cells using a high-throughput cyclic immunofluorescence method. Nature communications, 6, 8390.

Vidovi? D, et al. (2014) Large-scale integration of small molecule-induced genome-wide transcriptional responses, Kinome-wide binding affinities and cell-growth inhibition profiles reveal global trends characterizing systems-level drug action. Frontiers in genetics, 5, 342.

Weygant N, et al. (2014) Small molecule kinase inhibitor LRRK2-IN-1 demonstrates potent activity against colorectal and pancreatic cancer through inhibition of doublecortin-like kinase 1. Molecular cancer, 13, 103.

Shao H, et al. (2013) Systematically studying kinase inhibitor induced signaling network signatures by integrating both therapeutic and side effects. PloS one, 8(12), e80832.