Resource Summary Report

Generated by FDI Lab - SciCrunch.org on May 6, 2024

NCI-H3122

RRID:CVCL_5160
Type: Cell Line

Proper Citation

(RRID:CVCL_5160)

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_5160

Proper Citation: (RRID:CVCL_5160)

Description: Cell line NCI-H3122 is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Male

Defining Citation: PMID:11030152, PMID:12759538, PMID:18594010, PMID:20679594, PMID:22961666, PMID:23344087, PMID:24675041, PMID:26361996, PMID:27397505, PMID:29444439, PMID:29681454, PMID:30894373, PMID:30971826, PMID:31068700, PMID:31803961, PMID:35839778

Comments: Omics: Transcriptome analysis by microarray., Omics: SNP array analysis., Omics: Protein expression by reverse-phase protein arrays., Omics: DNA methylation analysis., Omics: Deep quantitative proteome analysis., Omics: Deep proteome analysis., Omics: Deep exome analysis., Omics: CRISPR phenotypic screen., Population: Caucasian., Part of: COSMIC cell lines project., Part of: Cancer Dependency Map project (DepMap) (includes Cancer Cell Line Encyclopedia - CCLE).

Category: Cancer cell line

Name: NCI-H3122

Synonyms: H3122, H-3122, NCIH3122

Cross References: CLO:CLO_0037033, ArrayExpress:E-MTAB-3610, BioGRID_ORCS_Cell_line:962, BioSample:SAMN10989614, cancercelllines:CVCL_5160, Cell_Model_Passport:SIDM00137, ChEMBL-Cells:CHEMBL4295436, ChEMBL-

Targets:CHEMBL4296475, CLS:300484, Cosmic:755472, Cosmic:914951, Cosmic:1146912, Cosmic:2245571, Cosmic-CLP:1240190, DepMap:ACH-000337, EGA:EGAS00001000082, EGA:EGAS00001000978, GDSC:1240190, GEO:GSM171874, GEO:GSM171875, GEO:GSM253349, GEO:GSM434307, GEO:GSM794370, GEO:GSM1374739, GEO:GSM1374740, GEO:GSM1670238, IGRhCellID:H3122GEO, LINCS_LDP:LCL-1675, NCI-DTP:NCI-H3122, PharmacoDB:NCIH3122_1104_2019, PRIDE:PXD002556, PRIDE:PXD030304, Progenetix:CVCL_5160, PubChem_Cell_line:CVCL_5160, Wikidata:Q54908028

ID: CVCL_5160

Ratings and Alerts

No rating or validation information has been found for NCI-H3122.

No alerts have been found for NCI-H3122.

Data and Source Information

Source: Cellosaurus

Usage and Citation Metrics

We found 12 mentions in open access literature.

Listed below are recent publications. The full list is available at FDI Lab - SciCrunch.org.

Pelos G, et al. (2024) Fast proliferating and slowly migrating non-small cell lung cancer cells are vulnerable to decitabine and retinoic acid combinatorial treatment. International journal of cancer, 154(6), 1029.

Rodina A, et al. (2023) Systems-level analyses of protein-protein interaction network dysfunctions via epichaperomics identify cancer-specific mechanisms of stress adaptation. Nature communications, 14(1), 3742.

Hondo N, et al. (2023) MEK inhibitor and anti-EGFR antibody overcome sotorasib resistance signals and enhance its antitumor effect in colorectal cancer cells. Cancer letters, 567, 216264.

Saifullah , et al. (2022) Integrated analysis of ALK higher expression in human cancer and downregulation in LUAD using RNA molecular scissors. Clinical & translational oncology : official publication of the Federation of Spanish Oncology Societies and of the National Cancer Institute of Mexico, 24(9), 1785.

Kim M, et al. (2021) A MET-PTPRK kinase-phosphatase rheostat controls ZNRF3 and Wnt signaling. eLife, 10.

Bosch-Barrera J, et al. (2021) Silibinin Suppresses Tumor Cell-Intrinsic Resistance to Nintedanib and Enhances Its Clinical Activity in Lung Cancer. Cancers, 13(16).

Yan P, et al. (2020) Molecular Stressors Engender Protein Connectivity Dysfunction through Aberrant N-Glycosylation of a Chaperone. Cell reports, 31(13), 107840.

Yenerall P, et al. (2020) RUVBL1/RUVBL2 ATPase Activity Drives PAQosome Maturation, DNA Replication and Radioresistance in Lung Cancer. Cell chemical biology, 27(1), 105.

Gurtner K, et al. (2020) Radioresistance of KRAS/TP53-mutated lung cancer can be overcome by radiation dose escalation or EGFR tyrosine kinase inhibition in vivo. International journal of cancer, 147(2), 472.

Kurppa KJ, et al. (2020) Treatment-Induced Tumor Dormancy through YAP-Mediated Transcriptional Reprogramming of the Apoptotic Pathway. Cancer cell, 37(1), 104.

Mollaoglu G, et al. (2018) The Lineage-Defining Transcription Factors SOX2 and NKX2-1 Determine Lung Cancer Cell Fate and Shape the Tumor Immune Microenvironment. Immunity, 49(4), 764.

Watanabe H, et al. (2013) Integrated cistromic and expression analysis of amplified NKX2-1 in lung adenocarcinoma identifies LMO3 as a functional transcriptional target. Genes & development, 27(2), 197.