Resource Summary Report

Generated by NIF on Sep 10, 2026

NCI-H1792

RRID:CVCL_1495
Type: Cell Line

Proper Citation

RRID:CVCL_1495

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_1495

Proper Citation: RRID:CVCL_1495

Description: Cell line NCI-H1792 is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Male

Disease: Lung adenocarcinoma

Defining Citation: PMID:1311061, PMID:8806092, PMID:11030152, PMID:12068308, PMID:18083107, PMID:20164919, PMID:20215515, PMID:22460905, PMID:22961666, PMID:24135919, PMID:25485619, PMID:25877200, PMID:25984343, PMID:26589293, PMID:27397505, PMID:28196595, PMID:29444439, PMID:29681454, PMID:30894373, PMID:31068700, PMID:31467290, PMID:31803961, PMID:31978347, PMID:35839778

Comments: Population: Caucasian., Part of: NCI RAS program mutant KRAS cell line panel., Part of: MD Anderson Cell Lines Project., Part of: COSMIC cell lines project., Part of: Cancer Dependency Map project (DepMap) (includes Cancer Cell Line Encyclopedia - CCLE).

Category: Cancer cell line

Organism: Homo sapiens (Human)

Name: NCI-H1792

Synonyms: H1792, H-1792, NCIH1792

Cross References: BTO:BTO_0005836, CLO:CLO_0008019, EFO:EFO_0002266, ArrayExpress:E-MTAB-38, ArrayExpress:E-MTAB-783, ArrayExpress:E-MTAB-2706, ArrayExpress:E-MTAB-2770, ArrayExpress:E-MTAB-3610, ATCC:CRL-5895, BioGRID_ORCS_Cell_line:537, BioSample:SAMN03470834, BioSample:SAMN10987908,

cancercelllines:CVCL_1495, Cell_Model_Passport:SIDM00771, ChEMBL-Cells:CHEMBL3308816, ChEMBL-Targets:CHEMBL1075525, CLS:305835, Cosmic:722044, Cosmic:724868, Cosmic:877246, Cosmic:877433, Cosmic:903603, Cosmic:980986, Cosmic:1032458, Cosmic:1146889, Cosmic:1152513, Cosmic:1891896, Cosmic:1995541, Cosmic-CLP:724868, DepMap:ACH-000496, EGA:EGAS00001000610, EGA:EGAS00001000978, EGA:EGAS00001002554, GDSC:724868, GEO:GSM171848, GEO:GSM171849, GEO:GSM253305, GEO:GSM434329, GEO:GSM784206, GEO:GSM784257, GEO:GSM794334, GEO:GSM817092, GEO:GSM827476, GEO:GSM887382, GEO:GSM888460, GEO:GSM1670192, IARC_TP53:506, IGRhCellID:NCIH1792, LiGeA:CCLE_358, LINCS_LDP:LCL-1644, PharmacoDB:NCIH1792_1039_2019, PRIDE:PXD030304, Progenetix:CVCL_1495, PubChem_Cell_line:CVCL_1495, Wikidata:Q54907864

ID: CVCL_1495

Record Creation Time: 20220427T220838+0000

Record Last Update: 20260905T181242+0000

Ratings and Alerts

No rating or validation information has been found for NCI-H1792.

No alerts have been found for NCI-H1792.

Data and Source Information

Source: Cellosaurus

Usage and Citation Metrics

We found 41 mentions in open access literature.

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

Baars B, et al. (2026) RAS Mutation-Specific Responses to Paralog- and State-Selective RAS Inhibitors. Molecular cancer research : MCR, 24(1), 60.

Treekitkarnmongkol W, et al. (2026) RASH3D19 mediates RAS activation through a positive feedback loop in KRAS-mutant cancer. Nature cell biology, 28(1), 197.

Wang P, et al. (2025) Glecirasib, a Potent and Selective Covalent KRAS G12C Inhibitor Exhibiting Synergism with Cetuximab or SHP2 Inhibitor JAB-3312. Cancer research communications, 5(5), 792.

Palmer CL, et al. (2025) CDK4 selective inhibition improves preclinical anti-tumor efficacy and safety. Cancer cell, 43(3), 464.

Jameson NM, et al. (2025) The Selective WEE1 Inhibitor Azenosertib Shows Synergistic Antitumor Activity with KRASG12C Inhibitors in Preclinical Models. Cancer research

communications, 5(2), 240.

Park J, et al. (2025) MYC plus class IIa HDAC inhibition drives mitochondrial dysfunction in non-small cell lung cancer. Cell reports, 44(6), 115722.

Feng J, et al. (2025) A pan-KRAS inhibitor and its derived degrader elicit multifaceted anti-tumor efficacy in KRAS-driven cancers. Cancer cell.

Wang Y, et al. (2025) Mutation of SMARCA4 Induces Cancer Cell-Intrinsic Defects in the Enhancer Landscape and Resistance to Immunotherapy. Cancer research, 85(11), 1997.

Lang PA, et al. (2025) Optimized arenaviruses with tumor-tropic mutations promote safe anti-tumor efficacy via sustainable immune modulatory properties. Cell reports. Medicine, 6(10), 102411.

Gebru MT, et al. (2025) Loss of UXS1 Selectively Depletes Pyrimidines and Induces Replication Stress in KEAP1-Mutant Lung Cancer. Cancer research, 85(23), 4806.

Daley BR, et al. (2025) SOS1 Inhibition Enhances the Efficacy of KRASG12C Inhibitors and Delays Resistance in Lung Adenocarcinoma. Cancer research, 85(1), 118.

Fukuda K, et al. (2024) Targeting WEE1 enhances the antitumor effect of KRAS-mutated non-small cell lung cancer harboring TP53 mutations. Cell reports. Medicine, 5(6), 101578.

Geroyska S, et al. (2024) N-Myristoytransferase Inhibition Causes Mitochondrial Iron Overload and Parthanatos in TIM17A-Dependent Aggressive Lung Carcinoma. Cancer research communications, 4(7), 1815.

Greenwood HE, et al. (2024) Imaging NRF2 activation in non-small cell lung cancer with positron emission tomography. Nature communications, 15(1), 10484.

Morel M, et al. (2024) FBXL16 promotes cell growth and drug resistance in lung adenocarcinomas with KRAS mutation by stabilizing IRS1 and upregulating IRS1/AKT signaling. Molecular oncology, 18(3), 762.

Han X, et al. (2024) Activation of polyamine catabolism promotes glutamine metabolism and creates a targetable vulnerability in lung cancer. Proceedings of the National Academy of Sciences of the United States of America, 121(13), e2319429121.

Kotagiri S, et al. (2024) Enhancer reprogramming underlies therapeutic utility of a SMARCA2 degrader in SMARCA4 mutant cancer. Cell chemical biology, 31(12), 2069.

Graham K, et al. (2024) Discovery of YAP1/TAZ pathway inhibitors through phenotypic screening with potent anti-tumor activity via blockade of Rho-GTPase signaling. Cell chemical biology, 31(7), 1247.

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.

Liu C, et al. (2023) Cell-to-cell variability in Myc dynamics drives transcriptional heterogeneity in cancer cells. Cell reports, 42(4), 112401.