

Resource Summary Report

Generated by [dkNET](#) on Sep 3, 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,

cancer cell lines: 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, PharmacDB: 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: 20260829T234730+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 [dkNET](#).

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

Trekitkarnmongkol 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.

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

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.

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.

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

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.

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-Myristoyltransferase 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.

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

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.

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.