

Resource Summary Report

Generated by [RRID](#) on Sep 8, 2026

LN-229

RRID:CVCL_0393

Type: Cell Line

Proper Citation

RRID:CVCL_0393

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_0393

Proper Citation: RRID:CVCL_0393

Description: Cell line LN-229 is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Female

Disease: Glioblastoma

Defining Citation: [PMID:7693337](#), [PMID:8509230](#), [PMID:9842975](#), [PMID:10416987](#), [PMID:14614447](#), [PMID:14655754](#), [PMID:16697959](#), [PMID:17595512](#), [PMID:19365568](#), [PMID:20504876](#), [PMID:21406405](#), [PMID:22460905](#), [PMID:22570425](#), [PMID:25485619](#), [PMID:25772239](#), [PMID:25877200](#), [PMID:25894527](#), [PMID:25984343](#), [PMID:26496030](#), [PMID:26589293](#), [PMID:27397505](#), [PMID:27412690](#), [PMID:30894373](#), [PMID:30971826](#), [PMID:31068700](#), [PMID:31978347](#), [PMID:35839778](#)

Comments: 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

Organism: Homo sapiens (Human)

Name: LN-229

Synonyms: LN 229, LN229, LNT-229

Cross References: BTO:BTO_0002004, CLO:CLO_0007363, EFO:EFO_0006637, MCCL:MCC:0000288, AddexBio:C0005017/4993, ArrayExpress:E-MTAB-2706, ArrayExpress:E-MTAB-2770, ArrayExpress:E-MTAB-3610, ATCC:CRL-2611, BioGRID_ORCS_Cell_line:929, BioSample:SAMN03471191, BioSample:SAMN03471692, BioSample:SAMN10987619, cancercellines:CVCL_0393,

Cell_Model_Passport:SIDM00684, ChEMBL-Cells:CHEMBL4295402, ChEMBL-Targets:CHEMBL4296459, CLS:305043, Cosmic:849861, Cosmic:1746958, Cosmic:1995485, Cosmic:2302323, Cosmic:2367492, Cosmic:2491102, Cosmic:2516025, Cosmic:2580915, Cosmic-CLP:1240169, DepMap:ACH-000595, EGA:EGAS00001000610, EGA:EGAS00001000978, GDSC:1240169, GEO:GSM101615, GEO:GSM101616, GEO:GSM326200, GEO:GSM397665, GEO:GSM397666, GEO:GSM481416, GEO:GSM887270, GEO:GSM888345, GEO:GSM1374621, GEO:GSM1670049, GEO:GSM2113428, GEO:GSM2113429, GEO:GSM2113430, GEO:GSM2113431, IARC_TP53:30212, LiGeA:CCLE_871, LINCS_LDP:LCL-1354, Lonza:1322, PharmacDB:LN229_841_2019, PRIDE:PXD000589, PRIDE:PXD003790, PRIDE:PXD030304, Progenetix:CVCL_0393, PubChem_Cell_line:CVCL_0393, Ubigene:YC-A021, Wikidata:Q54902771

ID: CVCL_0393

Record Creation Time: 20220427T220730+0000

Record Last Update: 20260905T181034+0000

Ratings and Alerts

No rating or validation information has been found for LN-229.

No alerts have been found for LN-229.

Data and Source Information

Source: [Cellosaurus](#)

Usage and Citation Metrics

We found 854 mentions in open access literature.

Listed below are recent publications. The full list is available at [RRID](#) .

Rivera-Caraballo KA, et al. (2026) Oncolytic HSV-1-Mediated JAG1 Blockade Induces Glioma Senescence-Associated Secretory Phenotype to Increase Macrophage Activation and Cetuximab-Mediated Senolysis. Cancer research, 86(5), 1232.

He M, et al. (2026) Feasibility and optimal imaging time window for intraperitoneal versus intravenous injection of a macrocyclic gadolinium-based contrast agent in mice. The Journal of international medical research, 54(6), 3000605261461956.

Fang C, et al. (2026) CUDC-907 inhibits glioblastoma and enhances glioblastoma sensitivity to temozolomide by inhibiting DNA damage repair. Genes & diseases, 13(5), 101948.

Mazewski C, et al. (2026) Potent Anti-Glioblastoma Effects of Next-Generation MNK Inhibitors. Cancers, 18(13).

- Jia A, et al. (2026) Dual targeting of SLC25A51 and succinate dehydrogenase selectively depletes mitochondrial NAD⁺ to eradicate KRAS-driven AML. *Cell metabolism*, 38(6), 1113.
- Kempynck N, et al. (2026) CREsted: modeling genomic and synthetic cell-type-specific enhancers across tissues and species. *Nature methods*, 23(5), 946.
- Sidhu RS, et al. (2026) Rap1b promotes glioma growth and stemness via regulation of Notch1 and VEGFR2 signaling pathways. *Cellular signalling*, 143, 112457.
- Rurka P, et al. (2026) A novel quinazolinone insulin receptor inhibitor and its synergy with an EGFR inhibitor in glucose-driven glioblastoma. *Molecular oncology*.
- Daly J, et al. (2026) CRISPR activation screens map the genomic landscape of cancer glycome remodeling. *Cell genomics*, 6(4), 101139.
- Su H, et al. (2026) Lipid Droplet-Localized Spindle Apparatus Coiled-Coil Protein 1 Regulates Lipid Droplet Distribution. *Advanced science (Weinheim, Baden-Wurttemberg, Germany)*, e11960.
- Song J, et al. (2026) JOSD1 Deubiquitinates Twist1 and Facilitates Epithelial-Mesenchymal Transition in Glioblastoma. *Cell biochemistry and function*, 44(4), e70208.
- O'Loughlin TA, et al. (2026) mTORC1 activity suppresses ferroptosis through a SCARB1-dependent HDL-tocopherol uptake pathway. *Molecular cell*, 86(8), 1546.
- Song X, et al. (2026) AI-driven network pharmacology and multi-omics validation identify KCNH2 as a prognostic biomarker and candidate therapeutic vulnerability of *Acorus tatarinowii* in glioblastoma. *Frontiers in pharmacology*, 17, 1811032.
- Venkataramani P, et al. (2026) Small-molecule inhibitors of the protein kinase DYRK as potential therapeutic candidates in cancer. *Cell chemical biology*, 33(6), 810.
- Sui M, et al. (2026) ACYP2 Induces Temozolomide Resistance in Glioblastoma by Promoting PARP1-Mediated DNA Damage Repair. *Molecular cancer research : MCR*, 24(1), 7.
- Meena D, et al. (2026) Innate Immune Receptor NLRX1: Potential Modulator of Glioblastoma Pathophysiology. *Journal of cellular physiology*, 241(3), e70163.
- Zhu M, et al. (2026) Targeted degradation of CD24 by a transferrin receptor-engaging bispecific degrader enhances antitumor immunity. *Biochemical pharmacology*, 246, 117721.
- Trautmann RK, et al. (2026) High-Throughput 3D Glioblastoma Model in Glycosaminoglycan Hydrogels for Personalized Therapeutic Screening. *Macromolecular bioscience*, 26(1), e00394.
- Guo D, et al. (2026) Lactate binds and inhibits the innate immune sensor STING to promote tumor immune evasion. *Immunity*, 59(7), 1811.
- Yang M, et al. (2025) Low YTHDC1 Expression Upregulates FSCN1 to Promote Nuclear F-Actin Formation and Facilitate Double-strand DNA Breaks Repair in TMZ-Resistant

Glioblastoma. Advanced science (Weinheim, Baden-Wurttemberg, Germany), e13632.