Resource Summary Report

Generated by RRID on Sep 9, 2026

SU-DIPG-XIII

RRID:CVCL_IT41
Type: Cell Line

Proper Citation

RRID:CVCL_IT41

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_IT41

Proper Citation: RRID:CVCL_IT41

Description: Cell line SU-DIPG-XIII is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Female

Disease: Diffuse intrinsic pontine glioma

Defining Citation: PMID:25939062, PMID:26115193, PMID:28823557, PMID:31439867, PMID:33319914, PMID:34078608, PMID:34215733, PMID:34732238

Category: Cancer cell line

Organism: Homo sapiens (Human)

Name: SU-DIPG-XIII

Synonyms: SU DIPG XIII, SU-DIPGXIII, SU-DIPG-13, DIPG XIII, DIPGXIII, SJHGG054833, Stanford University-Diffuse Intrinsic Pontine Glioma-XIII

Cross References: cancercelllines:CVCL_IT41, GEO:GSM4969590, GEO:GSM4969591, GEO:GSM4969592, GEO:GSM4969593, GEO:GSM4969619, GEO:GSM4969628, GEO:GSM4969629, GEO:GSM4969630, GEO:GSM4969657, GEO:GSM5356026, Wikidata:Q54970736

ID: CVCL_IT41

Record Creation Time: 20220427T221612+0000

Record Last Update: 20260905T182515+0000

Ratings and Alerts

No rating or validation information has been found for SU-DIPG-XIII.

No alerts have been found for SU-DIPG-XIII.

Data and Source Information

Source: Cellosaurus

Usage and Citation Metrics

We found 14 mentions in open access literature.

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

Keane L, et al. (2026) Microglia in diffuse midline glioma contribute to extracellular matrix remodelling and cancer cell invasion. Cell death & disease, 17(1).

Sharma M, et al. (2024) Targeting DNA Repair and Survival Signaling in Diffuse Intrinsic Pontine Gliomas to Prevent Tumor Recurrence. Molecular cancer therapeutics, 23(1), 24.

Kim H, et al. (2024) MTOR modulation induces selective perturbations in histone methylation which influence the anti-proliferative effects of mTOR inhibitors. iScience, 27(3), 109188.

Mota M, et al. (2023) Targeting SWI/SNF ATPases in H3.3K27M diffuse intrinsic pontine gliomas. Proceedings of the National Academy of Sciences of the United States of America, 120(18), e2221175120.

Venneti S, et al. (2023) Clinical Efficacy of ONC201 in H3K27M-Mutant Diffuse Midline Gliomas Is Driven by Disruption of Integrated Metabolic and Epigenetic Pathways. Cancer discovery, 13(11), 2370.

Furth N, et al. (2022) H3-K27M-mutant nucleosomes interact with MLL1 to shape the glioma epigenetic landscape. Cell reports, 39(7), 110836.

Harpaz N, et al. (2022) Single-cell epigenetic analysis reveals principles of chromatin states in H3.3-K27M gliomas. Molecular cell, 82(14), 2696.

Panditharatna E, et al. (2022) BAF Complex Maintains Glioma Stem Cells in Pediatric H3K27M Glioma. Cancer discovery, 12(12), 2880.

Siddaway R, et al. (2022) Splicing is an alternate oncogenic pathway activation mechanism in glioma. Nature communications, 13(1), 588.

Tsai JW, et al. (2022) FOXR2 Is an Epigenetically Regulated Pan-Cancer Oncogene That Activates ETS Transcriptional Circuits. Cancer research, 82(17), 2980.

Keane L, et al. (2021) Inhibition of microglial EZH2 leads to anti-tumoral effects in pediatric diffuse midline gliomas. Neuro-oncology advances, 3(1), vdab096.

Chung C, et al. (2020) Integrated Metabolic and Epigenomic Reprograming by H3K27M Mutations in Diffuse Intrinsic Pontine Gliomas. Cancer cell, 38(3), 334.

Anastas JN, et al. (2019) Re-programing Chromatin with a Bifunctional LSD1/HDAC Inhibitor Induces Therapeutic Differentiation in DIPG. Cancer cell, 36(5), 528.

Xie Q, et al. (2018) N6-methyladenine DNA Modification in Glioblastoma. Cell, 175(5), 1228.