

Resource Summary Report

Generated by [RRID](#) on Aug 27, 2026

HPAC

RRID:CVCL_3517

Type: Cell Line

Proper Citation

RRID:CVCL_3517

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_3517

Proper Citation: RRID:CVCL_3517

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

Sex: Female

Disease: Pancreatic adenocarcinoma

Defining Citation: [PMID:15367885](#), [PMID:20215515](#), [PMID:20418756](#), [PMID:22460905](#), [PMID:22585861](#), [PMID:25167228](#), [PMID:25485619](#), [PMID:25877200](#), [PMID:25939163](#), [PMID:25984343](#), [PMID:26216984](#), [PMID:26589293](#), [PMID:27259358](#), [PMID:27397505](#), [PMID:30894373](#), [PMID:31068700](#), [PMID:35839778](#)

Comments: Characteristics: Established from a nude mouse xenograft (ATCC=CRL-2119)., 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: HPAC

Synonyms: Hpac

Cross References: BTO:BTO_0004121, CLO:CLO_0003814, EFO:EFO_0002197, ArrayExpress:E-MTAB-38, ArrayExpress:E-MTAB-2706, ArrayExpress:E-MTAB-2770, ArrayExpress:E-MTAB-3610, ATCC:CRL-2119, BCRC:60495, BioSample:SAMN03472247, BioSample:SAMN10987698, cancercellines:CVCL_3517, CCRID:1101HUM-PUMC000189, Cell_Model_Passport:SIDM00670, ChEMBL-Cells:ChEMBL3308388, ChEMBL-Targets:ChEMBL612595, CLS:305309,

Cosmic:913306, Cosmic:1571784, Cosmic:1995444, Cosmic:2434094, Cosmic-CLP:1298136, DepMap:ACH-000270, EGA:EGAS00001000610, EGA:EGAS00001000978, GDSC:1298136, GEO:GSM206509, GEO:GSM621897, GEO:GSM784701, GEO:GSM887089, GEO:GSM888160, GEO:GSM1024401, GEO:GSM1374545, GEO:GSM1669892, IARC_TP53:21070, IGRhCellID:HPAC, LiGeA:CCLE_795, LINCS_LDP:LCL-1735, PharmacnoDB:HPAC_564_2019, PRIDE:PXD003198, PRIDE:PXD030304, PRIDE:PXD032263, Progenetix:CVCL_3517, PubChem_Cell_line:CVCL_3517, Wikidata:Q54890226

ID: CVCL_3517

Record Creation Time: 20220427T220440+0000

Record Last Update: 20260815T233941+0000

Ratings and Alerts

No rating or validation information has been found for HPAC.

No alerts have been found for HPAC.

Data and Source Information

Source: [Cellosaurus](#)

Usage and Citation Metrics

We found 56 mentions in open access literature.

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

Kamgar M, et al. (2026) Mutant and Wild-Type RAS Cross-talk and Stoichiometric Deficiencies Are Determinants of Sensitivity to Targeted Therapies in KRASG12R Pancreatic Ductal Adenocarcinoma. Cancer research, 86(8), 2042.

Zhang Z, et al. (2026) A convergent uPAR-positive tumor ecosystem creates broad vulnerability to CAR T cell therapy. Cell, 189(10), 2898.

Miyan J, et al. (2026) The combination of BCL-xL PROTAC and mTOR inhibitor sensitizes pancreatic ductal adenocarcinoma to KRAS G12D inhibitor treatment by enhancing apoptosis induction. bioRxiv : the preprint server for biology.

Ning W, et al. (2026) A bispecific nanobody-drug conjugate targeting TROP2 and c-Met for low-concentration, single-dose treatment of pancreatic cancer. Cell reports. Medicine, 7(4), 102688.

Gong D, et al. (2026) GLIS3 marks a neural-like progenitor cell state that drives metastasis in pancreatic ductal adenocarcinoma. Cell reports, 45(5), 117314.

Bajgain P, et al. (2026) Engineering functionality-optimized fully human B7-H3 CAR T cells for enhanced solid tumor therapy. *Cell reports. Medicine*, 7(5), 102782.

Roach MK, et al. (2026) Vertical inhibition of the autophagy pathway impairs growth and enhances sensitivity to mTORC1 inhibition in pancreatic ductal adenocarcinoma. *bioRxiv : the preprint server for biology*.

Xie L, et al. (2026) Preclinical Characterization and Clinical Activity of RNK08954, a Highly Selective and Orally Bioavailable KRASG12D Inhibitor. *Cancer discovery*, 16(5), 895.

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

Woo SM, et al. (2026) Inhibiting Fatty Acid Oxidation Reverses Autophagy-Mediated Acquired Chemotherapy Resistance in Pancreatic Ductal Adenocarcinoma. *Cancer research*, 86(13), 3194.

Murr J, et al. (2026) The Protein Phosphatase Inhibitor LB100 Targets the Mesenchymal Lineage of Pancreatic Ductal Adenocarcinoma. *MedComm*, 7(6), e70794.

Takano K, et al. (2026) DS-3939a: A TA-MUC1-Directed Antibody-Drug Conjugate with Broad Antitumor Activity. *Molecular cancer therapeutics*, 25(1), 7.

Burge RA, et al. (2026) KRASG12R-Mutant Pancreatic Cancer Features Limited ERK/MAPK Transcriptional Activity and a Distinctive Tumor Microenvironment. *Cancer research*, 86(8), 1868.

Shen S, et al. (2026) ALDH1A3 promotes the lung metastatic organotropism of pancreatic ductal adenocarcinoma through semaphorin signaling. *iScience*, 29(6), 115950.

Zhao B, et al. (2025) PLK1 blockade enhances the anti-tumor effect of MAPK inhibition in pancreatic ductal adenocarcinoma. *Cell reports*, 44(4), 115541.

Lee DKC, et al. (2025) ITGAV and SMAD4 influence the progression and clinical outcome of pancreatic ductal adenocarcinoma. *Molecular oncology*.

Wittenzellner K, et al. (2025) Label-free single-cell phenotyping to determine tumor cell heterogeneity in pancreatic cancer in real time. *JCI insight*, 10(13).

Li H, et al. (2025) Disrupting AGR2/IGF1 paracrine and reciprocal signaling for pancreatic cancer therapy. *Cell reports. Medicine*, 6(2), 101927.

Zhang W, et al. (2025) RACK1 attenuates pancreatic tumorigenesis by suppressing acinar-to-ductal metaplasia through inflammatory signaling modulation. *Cellular oncology (Dordrecht, Netherlands)*.

Maki R, et al. (2025) Efficacy of SOAT1 Inhibitors Against Pancreatic Cancer with TP53 Mutations. *Annals of surgical oncology*, 32(9), 7025.