

Resource Summary Report

Generated by [dkNET](#) on Sep 2, 2026

Panc 10.05

RRID:CVCL_1639

Type: Cell Line

Proper Citation

RRID:CVCL_1639

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_1639

Proper Citation: RRID:CVCL_1639

Description: Cell line Panc 10.05 is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Male

Disease: Pancreatic ductal adenocarcinoma

Defining Citation: [PMID:9612602](#), [PMID:18000365](#), [PMID:20164919](#), [PMID:22460905](#), [PMID:22585861](#), [PMID:22753594](#), [PMID:25167228](#), [PMID:25984343](#), [PMID:26216984](#), [PMID:27259358](#), [PMID:27397505](#), [PMID:28196595](#), [PMID:29444439](#), [PMID:30894373](#), [PMID:30971826](#), [PMID:31068700](#), [PMID:35839778](#)

Comments: Population: Caucasian., From: Johns Hopkins University School of Medicine; Baltimore; USA., Part of: RAS genetic alteration cell panel (ATCC TCP-1031)., 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: Panc 10.05

Synonyms: Panc-10.05, Panc10.05, PANC-10-05, PANC 1005, PANC1005, Panc1005, Pa16C, PL12, PL-12

Cross References: CLO:CLO_0008380, ArrayExpress:E-MTAB-783, ArrayExpress:E-MTAB-2770, ArrayExpress:E-MTAB-3610, ATCC:CRL-2547, BioGRID_ORCS_Cell_line:489, BioSample:SAMN10988227, cancercellines:CVCL_1639,

Cell_Model_Passport:SIDM01135, ChEMBL-Cells:CHEMBL3307991, ChEMBL-Targets:CHEMBL612583, CLS:300599, Cosmic:925348, Cosmic:1198216, Cosmic:1473147, Cosmic:1509859, Cosmic:2046532, Cosmic:2434097, Cosmic-CLP:925348, DepMap:ACH-000060, EGA:EGAS00001000978, GDSC:925348, GEO:GSM206533, GEO:GSM784707, GEO:GSM887500, GEO:GSM888582, GEO:GSM1024412, GEO:GSM1374809, GEO:GSM1435690, GEO:GSM1435691, GEO:GSM1435692, GEO:GSM1670335, IARC_TP53:27227, LiGeA:CCLE_911, LINCS_LDP:LCL-1743, PharmacDB:Panc10_05_1245_2019, PRIDE:PXD003198, PRIDE:PXD030304, Progenetix:CVCL_1639, PubChem_Cell_line:CVCL_1639, Wikidata:Q54937531

ID: CVCL_1639

Record Creation Time: 20220427T221408+0000

Record Last Update: 20260829T235537+0000

Ratings and Alerts

No rating or validation information has been found for Panc 10.05.

No alerts have been found for Panc 10.05.

Data and Source Information

Source: [CelloSaurus](#)

Usage and Citation Metrics

We found 29 mentions in open access literature.

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

Bandi DSR, et al. (2026) ADT-030, a novel PDE10 inhibitor, demonstrates potent antitumor activity in pancreatic ductal adenocarcinoma. bioRxiv : the preprint server for biology.

Chang WH, et al. (2026) Characterization and therapeutic suppression of KEAP1-NRF2-driven resistance to KRAS inhibitors in pancreatic and lung cancer. bioRxiv : the preprint server for biology.

Assi M, et al. (2026) Extracellular matrix sensing regulates intratumoral heterogeneity of autophagic flux. Cell, 189(6), 1731.

Belanger K, et al. (2026) Cancer-Associated Fibroblasts Promote Epithelial-Mesenchymal Transition and Classical to Basal Subtype Shift in Pancreatic Cancer. Cancer research, 86(14), 3604.

Shrivastava A, et al. (2026) Mutant KRAS-driven selective mRNA translation reveals mechanisms and therapeutic vulnerabilities in cancer. Cell reports, 45(6), 117520.

Johnson JAI, et al. (2025) Human interpretable grammar encodes multicellular systems biology models to democratize virtual cell laboratories. *Cell*, 188(17), 4711.

Raymakers L, et al. (2025) The Efficacy of Targeted Monoclonal IgA Antibodies Against Pancreatic Ductal Adenocarcinoma. *Cells*, 14(9).

Tang J, et al. (2025) CD44 identified as a diagnostic biomarker for highly malignant CA19-9 negative pancreatic cancer. *Cancer letters*, 622, 217713.

Wang S, et al. (2025) IFN- γ -driven UBE2D3 upregulation impairs antigen presentation pathways and anti-tumor immunity in pancreatic cancer. *Nature communications*, 16(1), 10733.

Drizyte-Miller K, et al. (2025) Combination of the MTA-Cooperative PRMT5 Inhibitor BMS-986504 and KRAS Inhibitors Is an Effective Treatment Strategy for MTAP-Deleted KRAS-Mutant Pancreatic Cancer. *Cancer research*, 85(18), 3540.

Long AW, et al. (2024) Heterodimerization of T cell engaging bispecific antibodies to enhance specificity against pancreatic ductal adenocarcinoma. *Journal of hematology & oncology*, 17(1), 20.

Ohara Y, et al. (2024) ELAPOR1 induces the classical/progenitor subtype and contributes to reduced disease aggressiveness through metabolic reprogramming in pancreatic cancer. *International journal of cancer*, 155(3), 569.

Moreno P, et al. (2024) ADRA2A promotes the classical/progenitor subtype and reduces disease aggressiveness of pancreatic cancer. *Carcinogenesis*, 45(11), 845.

Ohara Y, et al. (2024) LMO3 is a suppressor of the basal-like/squamous subtype and reduces disease aggressiveness of pancreatic cancer through glycerol 3-phosphate metabolism. *Carcinogenesis*.

Moreno P, et al. (2024) ADRA2A promotes the classical/progenitor subtype and reduces disease aggressiveness of pancreatic cancer. *bioRxiv : the preprint server for biology*.

Patterson JC, et al. (2023) Plk1 Inhibitors and Abiraterone Synergistically Disrupt Mitosis and Kill Cancer Cells of Disparate Origin Independently of Androgen Receptor Signaling. *Cancer research*, 83(2), 219.

Okano F, et al. (2023) Identification of Membrane-expressed CAPRIN-1 as a Novel and Universal Cancer Target, and Generation of a Therapeutic Anti-CAPRIN-1 Antibody TRK-950. *Cancer research communications*, 3(4), 640.

Ohara Y, et al. (2023) SERPINB3-MYC axis induces the basal-like/squamous subtype and enhances disease progression in pancreatic cancer. *Cell reports*, 42(12), 113434.

Dash S, et al. (2023) MYC/Glutamine Dependency Is a Therapeutic Vulnerability in Pancreatic Cancer with Deoxycytidine Kinase Inactivation-Induced Gemcitabine Resistance. *Molecular cancer research : MCR*, 21(5), 444.

Perurena N, et al. (2023) USP9X mediates an acute adaptive response to MAPK suppression in pancreatic cancer but creates multiple actionable therapeutic vulnerabilities. *Cell reports. Medicine*, 4(4), 101007.