

# Resource Summary Report

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

## S2-013

RRID:CVCL\_B280

Type: Cell Line

### Proper Citation

RRID:CVCL\_B280

### Cell Line Information

**URL:** [https://web.expasy.org/cellosaurus/CVCL\\_B280](https://web.expasy.org/cellosaurus/CVCL_B280)

**Proper Citation:** RRID:CVCL\_B280

**Description:** Cell line S2-013 is a Cancer cell line with a species of origin Homo sapiens (Human)

**Sex:** Male

**Disease:** Pancreatic ductal adenocarcinoma

**Defining Citation:** [PMID:1319859](#), [PMID:1391733](#), [PMID:11854916](#)

**Comments:** Population: Japanese.

**Category:** Cancer cell line

**Organism:** Homo sapiens (Human)

**Name:** S2-013

**Synonyms:** SUIT2-013, S2013

**Cross References:** cancercellines:CVCL\_B280, TKG:TKG 0709, TOKU-E:4026, Wikidata:Q54951840

**ID:** CVCL\_B280

**Hierarchy:** cvcl\_3172

**Record Creation Time:** 20220427T221513+0000

**Record Last Update:** 20260905T182246+0000

### Ratings and Alerts

No rating or validation information has been found for S2-013.

No alerts have been found for S2-013.

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## Data and Source Information

**Source:** [Cellosaurus](#)

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## Usage and Citation Metrics

We found 5 mentions in open access literature.

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

Lum MA, et al. (2025) Small-molecule modulators of B56-PP2A restore 4E-BP function to suppress eIF4E-dependent translation in cancer cells. The Journal of clinical investigation, 135(4).

Hang H, et al. (2025) SP1-Mediated Glycolytic Reprogramming Promotes Tumorigenesis and Progression in Pancreatic Cancer. Advanced science (Weinheim, Baden-Wurttemberg, Germany), e10071.

Mullen NJ, et al. (2024) DHODH inhibition enhances the efficacy of immune checkpoint blockade by increasing cancer cell antigen presentation. eLife, 12.

He C, et al. (2024) Vitamin B6 Competition in the Tumor Microenvironment Hampers Antitumor Functions of NK Cells. Cancer discovery, 14(1), 176.

Desir S, et al. (2018) Chemotherapy-Induced Tunneling Nanotubes Mediate Intercellular Drug Efflux in Pancreatic Cancer. Scientific reports, 8(1), 9484.