DATA SHEET

PDAC Validated Solution

3D PDAC Tumor–CAF Co-culture Workflow with RASTRUM

Accelerate the development of effective therapies for pancreatic ductal adenocarcinoma (PDAC) using a reproducible 3D tumor–stroma model designed for translational research workflows

Part of the RASTRUM Validated Solution offering

Context of Use

This validated workflow enables researchers to:

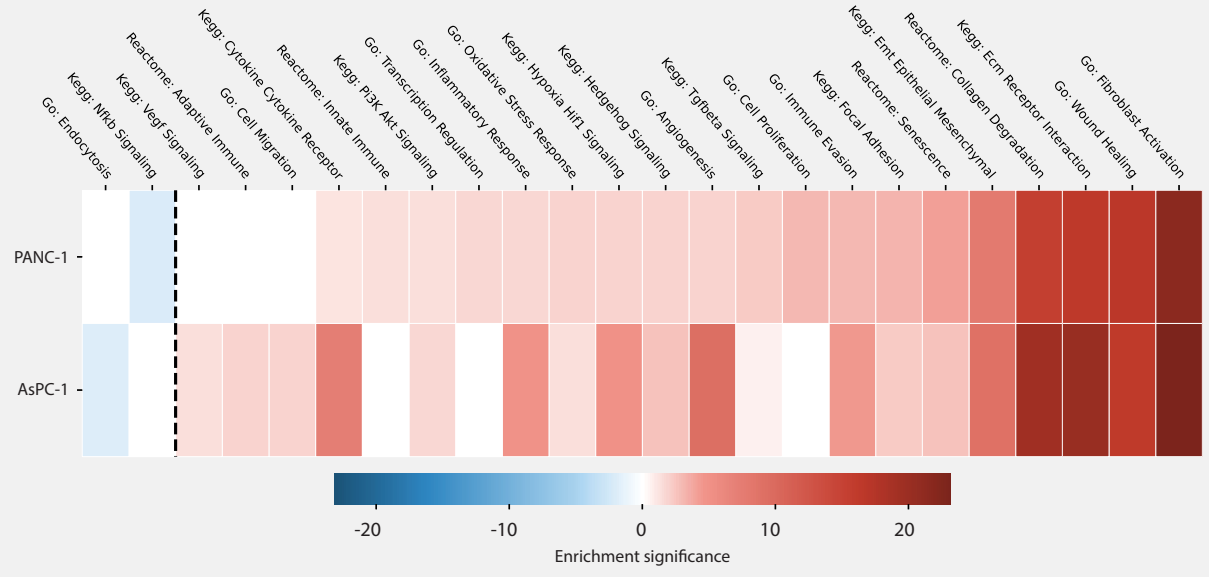
- Evaluate therapeutic efficacy in the presence of tumor–stroma crosstalk
- Investigate stromal contributions to tumor progression and drug resistance
- Identify targetable resistance mechanisms within the tumor microenvironment
- Screen pathway-targeted therapies and combination treatments

Model Overview

This PDAC Validated Solution recreates tumor–stroma interactions by integrating pancreatic cancer cells with cancer-associated fibroblasts (CAFs) within a defined 3D matrix environment.

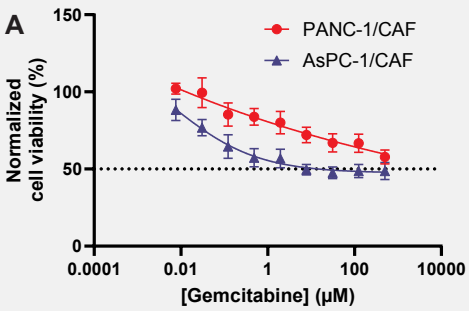
To incorporate broad coverage of key PDAC signalling pathways and provide canonical TGF-β-driven (PANC-1) or TGF-β-null (AsPC-1) contexts, we combined either PANC-1 or AsPC-1 cancer cells with primary PDAC CAFs (G018). This approach also allows drug efficacy comparisons between primary and metastatic contexts.

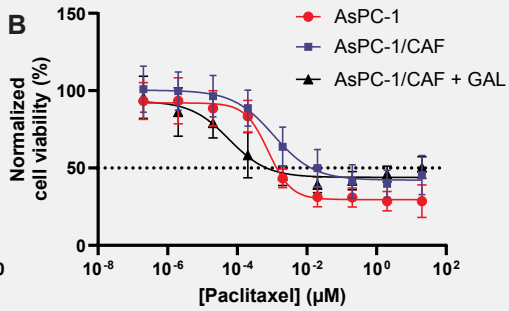
Molecular Characterization: RNA sequencing



RNA-seq of RASTRUM-generated PDAC models reveals pathway activation associated with tumor–stroma signaling in co-cultures compared to monocultures.
Differential gene expression analysis highlights pathways related to fibroblast activation, extracellular matrix remodeling, and therapeutic resistance.

Functional Validation: Drug Response





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IC50 values (μM):		
AsPC-1	AsPC-1/CAF	AsPC-1/CAF +GAL
0.00191	0.146	0.001245

Tumor–stroma interactions alter therapeutic response.
RASTRUM co-culture models demonstrate increased resistance to paclitaxel relative to monoculture conditions, consistent with stromal-mediated chemoprotective effects observed in PDAC. Addition of the TGFBR1 inhibitor galunisertib (GAL) reverses the chemoprotective effects in co-cultures.

What this model demonstrates

- **Stromal signaling is context-dependent:** Screen your compounds in biologically rich contexts. RNA-seq analysis reveals distinct pathway enrichment profiles between PANC-1 and AsPC-1 cell lines, highlighting how tumor genotype shapes tumor–stroma crosstalk and downstream resistance mechanisms.
- **CAF co-culture drives chemoprotection:** AsPC-1 cells co-cultured with CAFs demonstrated that stromal interactions significantly reduce chemotherapy sensitivity, making your findings translationally relevant.
- **Utility for combination therapy screening:** Develop treatment strategies that counteract CAF-mediated drug resistance or that remain efficacious in the presence of tumor–stroma signaling.

Access the full workflow documentation

- Complete protocol pack
- Reagents and materials list
- Experimental workflow guidance

Model format	Imaging Model
Matrix	Px03.84 (3 kPa, RGD, YIGSR, GFOGER, HA, full-length fibronectin)
Cell density	Cancer: 2M/mL; CAF: 10M/mL
Co-culture ratio	1:5 tumor : CAF
Drug treatment	Day 4 post-print, 72 hr exposure

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