DATA SHEET

PDAC-IO Validated Solution

3D PDAC Tumor–CAF–CD8+ T Cell Tri-culture Workflow with RASTRUM

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

Part of the RASTRUM Validated Solution offering

Context of Use

This validated workflow enables researchers to:

- Evaluate immunotherapy efficacy in the presence of tumor–stroma–immune crosstalk
- Investigate how cancer-associated fibroblasts (CAFs) shape tumor signalling and CD8+ T cell function
- Identify targetable mechanisms of stroma-mediated immune suppression within the tumor microenvironment
- Screen immune checkpoint inhibitors and stroma-targeted combination treatments

Model Overview

This PDAC-IO Validated Solution adds an immune component to the RASTRUM PDAC tumor–CAF Validated Solution, integrating pancreatic cancer cells, CAFs and cytotoxic CD8+ T cells within a defined 3D matrix environment. Because the tumor and CAF compartments are established before T cells are added, early immune-driven changes can be measured against a mature, stroma-conditioned baseline.

Molecular Characterization: RNA sequencing

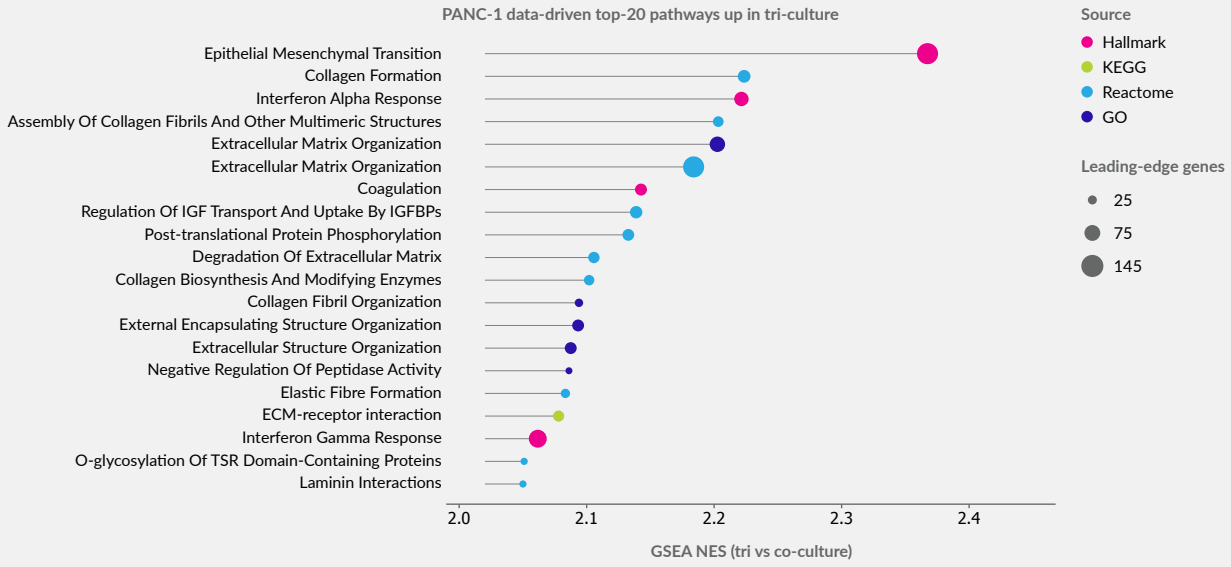


Figure 1: CAFs reprogram tumor–immune signalling in the PDAC-IO model. Top 20 pathways enriched in PANC-1 tri-cultures (PANC-1 + CAFs + CD8+ T cells) versus co-cultures (PANC-1 + CD8+ T cells), ranked by normalised enrichment score (NES). Gene set enrichment analysis (GSEA) was run on the DESeq2 Wald statistic against MSigDB Hallmark, KEGG, Reactome and Gene Ontology Biological Process gene sets; all FDR < 0.001. Dot colour indicates gene set source and dot size indicates the number of leading-edge genes.

Functional Validation: Immunotherapy Response

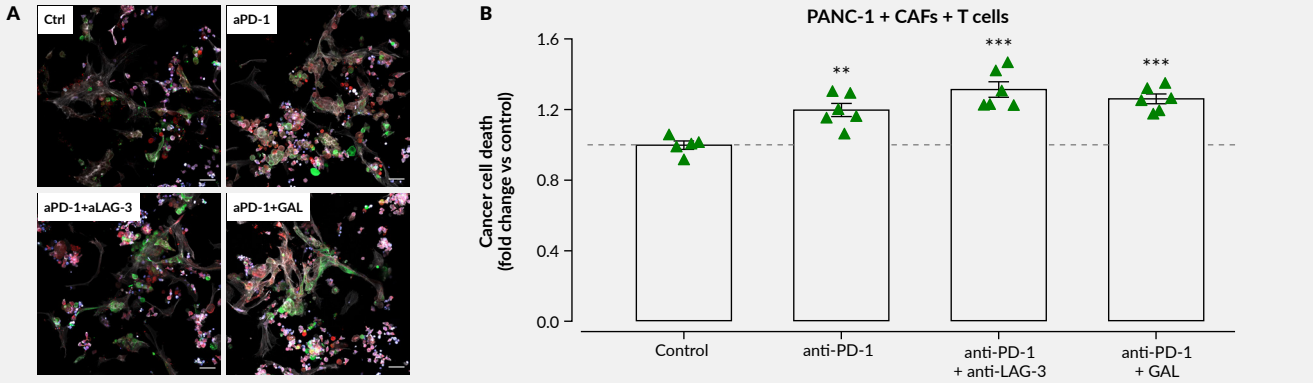


Figure 2: Immunotherapy testing in the PDAC-IO model. To assess immunotherapy response, tri-cultures and co-cultures were treated on day 4 post-print, at the time of CD8+ T cell addition, with anti-PD-1 (10 µg/mL), anti-PD-1 + anti-LAG-3 (10 µg/mL each), anti-PD-1 + the TGF-β receptor I kinase inhibitor galunisertib (0.4 µM) or an IgG4 isotype control. (A) After 24 hr, cultures were fixed and stained for cleaved caspase-3 and pan-cytokeratin to quantify cancer cell death (white: phalloidin, green: pan-CK, red: cleaved caspase-3, purple: T cells). Anti-PD-1 significantly increased cancer cell death versus isotype control, whereas anti-LAG-3 and galunisertib combinations further increased killing. (B) Quantification of cancer cell death in response to IO treatments.

What this model demonstrates

- Stroma reprograms the tumor-immune interface: Adding CAFs switched on EMT, matrix-remodelling and interferon programs, allowing immunotherapy responses to be measured under translationally relevant conditions.
- CAF-associated immunoregulation is captured: compartment-resolved RNA-seq deconvolution suggests CAFs activate TGF-β signalling in CD8+ T cells (data available on request), one of the key barriers to immunotherapy in PDAC.
- Utility for immunotherapy combination screening: Test checkpoint inhibitors alone or with stroma-targeted agents, to develop strategies that overcome CAF-mediated resistance.

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
Immune component	15,000 pre-activated CD8+ T cells/well (HLA-A matched donor), added day 4 post-print
Culture medium	DMEM : FGM-2 : ImmunoCult (1:1:1) with IL-2 and CD3/CD28 activator
Drug treatment	Day 4 post-print with T cell addition, 24 hr exposure

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