ATCC's patient-derived 2-D & 3-D cancer models make translational oncology a reality for the scientific community

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Poster 3405

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Human Cancer Model Initiative (HCMI)

The Human Cancer Models Initiative (HCMI) is a global collaboration led by the National Cancer Institute (NCI), with partners including Cancer Research UK, the Wellcome Sanger Institute, Hubrecht Organoid Technology, and NCI-funded institutions such as the Broad Institute and Cold Spring Harbor Laboratory. The initiative develops next-generation, patient-derived cancer models that better capture the genomic and phenotypic complexity of human tumors compared to traditional cell lines. ATCC® is the sole distributor of the HCMI portfolio, providing more than 330 fully characterized 2-D and 3-D cancer models derived from over 28 tissue types, including colorectal, pancreatic, brain, and esophageal cancers as well as rare cancers like Wilms tumor and Ewing's sarcoma. These models reflect diverse clinical backgrounds and retain high genomic fidelity, preserving over 80% of oncogenic drivers and maintaining transcriptional and epigenetic landscapes comparable to patient tumors. By offering clinically relevant models with available sequencing data and patient metadata through the HCMI portal, this work supports improved preclinical testing, biomarker discovery, precision oncology research, and studies of tumor heterogeneity and health disparities.

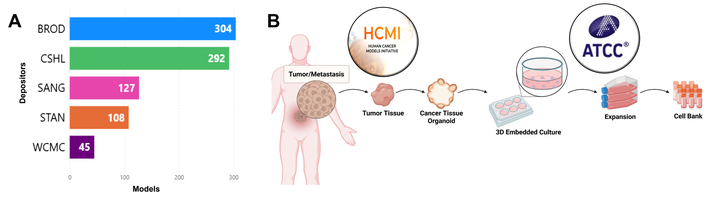

Figure 1: The Human Cancer Models Initiative (HCMI). (A) Institutes involved with depositing HCMI organoid/spheroid models at ATCC®. (B) ATCC® initiates the production pipeline and generates cell banks from this material. Figure created with BioRender.com.

Production Workflow

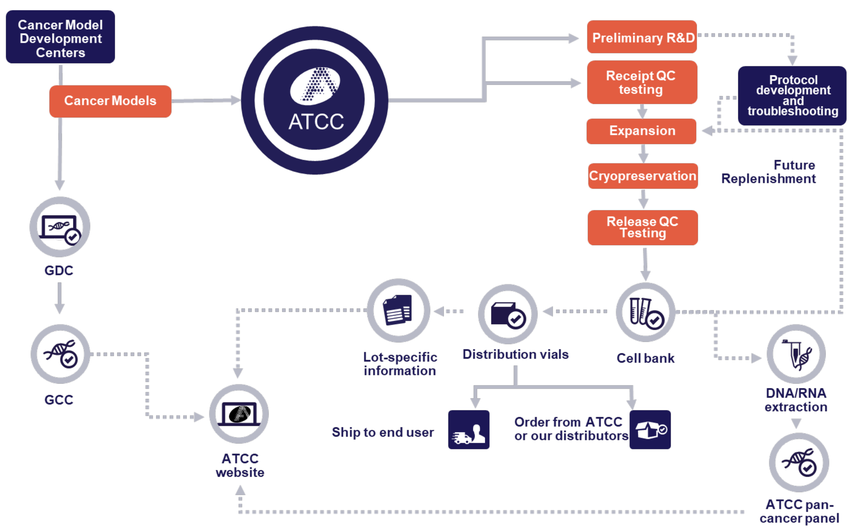

Figure 2: Our production workflow outlines the end-to-end process for manufacturing and cell banking process for HCMI models. This workflow visually walks through key stages in preliminary R&D and model generation protocol development at their respective academic/institutional sites, transitioning then to accessioning, expansion, quality testing, cryopreservation of cell banks, and eventual distribution and shipment to end users by ATCC®. This workflow also presents ATCC's role in quality control, DNA analysis, comparative analysis with GDC data generation, and future replenishments, emphasizing the rigorous steps involved in releasing high-quality cancer models to the research community.

HCMI Portfolio Breakdown

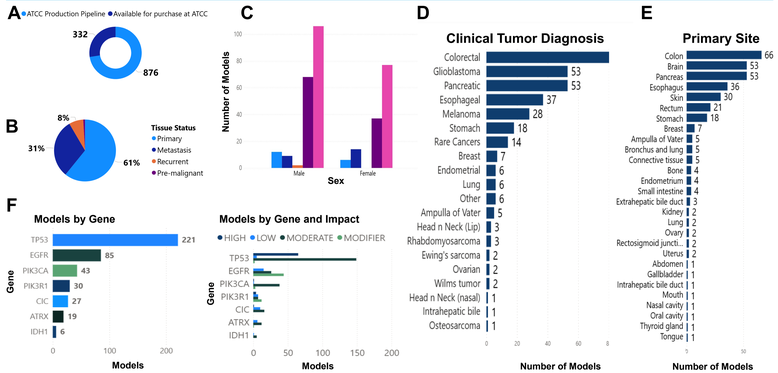

Figure 3: Overview of the diversity and clinical relevance of the Human Cancer Models Initiative (HCMI) portfolio. (A) The portfolio includes an extensive and diverse set of existing next-generation models alongside forthcoming releases of models in development. (B) The tissue status distribution illustrates the proportion of models derived from primary, metastatic, or recurrent tumor samples, emphasizing the portfolio's ability to reflect disease heterogeneity. (C) Demographic summaries offer insight into patient characteristics, including age, sex, and other available clinical metadata, underscoring the portfolio's representation of diverse patient populations. (D) The charts summarize the clinical tumor diagnoses represented in the collection, (E) capturing major cancer types from various primary sites. (F) Variant calling results were cross-referenced with the CLINVAR clinical variant archive to classify each mutation by gene, gene class (e.g., oncogene, tumor suppressor), and variant type (e.g., missense, frameshift, splice-site). The integration of CLINVAR enables rigorous interpretation of genomic events by linking mutations to established clinical significance categories and known pathogenic molecular mechanisms.

HCMI – Colorectal Cancer Organoids

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ATCC® No.	Cancer Type	KRAS Mutant	Histological Subtype	Type	Acquisition Site	Gender	Age	Clinical Stage
PDM-43™	Colorectal	G12D	Adenocarcinoma	Primary	Rectum	Female	61	Stage IIIB
PDM-415™	Colorectal	G13D	Adenocarcinoma	Metastatic	Peritoneum	Female	48	Stage IIIB
PDM-417™	Colorectal	-	Adenocarcinoma	Metastatic	Pelvis	Male	39	-
PDM-418™	Colorectal	-	Adenocarcinoma	Metastatic	Liver	Female	54	Stage IVC
PDM-419™	Colorectal	G12D	Adenocarcinoma	Metastatic	Liver	Male	62	Stage I
PDM-364™	Colorectal		Adenocarcinoma	Primary	Sigmoid colon	Female	70	
PDM-1™	Colorectal	G12A	Adenocarcinoma	Primary	Cecum	Male	75	Stage I
PDM-42™	Colorectal	G12C	Adenocarcinoma	Metastatic	Liver	Female	51	Stage IV
PDM-7™	Colorectal	G12V	Adenocarcinoma	Primary	Sigmoid colon	Male	75	Stage IIIA
PDM-155™	Colorectal	G12D	Adenocarcinoma	Primary	Cecum	Male	71	Stage IIA
PDM-61™	Colorectal	-	Adenocarcinoma	Primary	Ascending colon	Male	71	Stage IIIB
PDM-418™	Colorectal	-	Adenocarcinoma	Metastatic	Liver	Female	54	Stage IVC

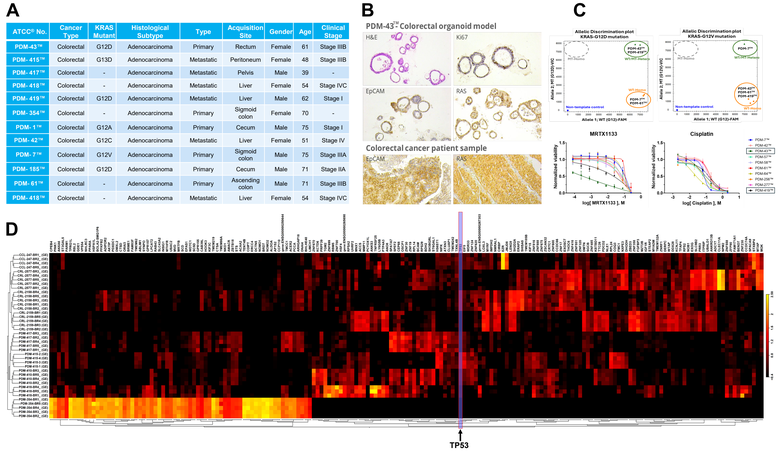

Figure 4: Colorectal cancer organoid model characterization. (A) List of colorectal cancer models that show unique KRAS mutations. (B) ATCC® PDM-43™ histopathology as compared to colorectal cancer patient samples showing H&E, Ki67, EPCAM, and RAS markers (C) The KRAS G12D mutant organoid ATCC® PDM-43™, as seen also from the allelic discrimination plot, exhibited marked sensitivity to the KRAS G12D inhibitor MRTX1133, demonstrating a pronounced cytotoxic response relative to KRAS wild-type or alternate-KRAS-mutation organoids. (D) Heatmap shows gene expression pattern across the cell models. Red-induced, Black-reduced. ATCC® provides Organoid Growth Kit 1A (ATCC® ACS-7100™)/Organoid Growth Kit 1D (ATCC® ACS-7103™)* for colorectal organoid culture. **For relevant growth media components and instructions, visit model specific information on ATCC's website.*

HCMI – Pancreatic Cancer Organoids

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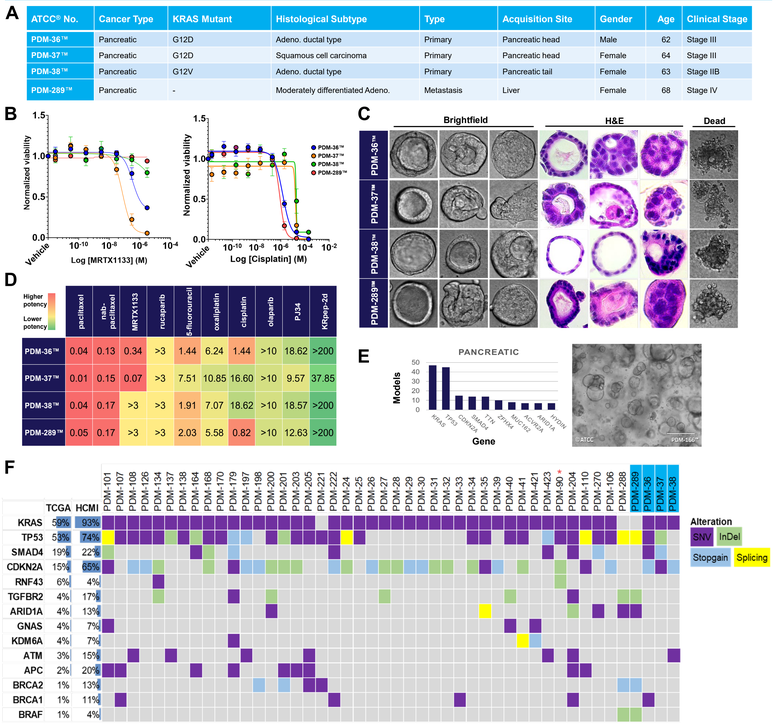
ATCC® No.	Cancer Type	KRAS Mutant	Histological Subtype	Type	Acquisition Site	Gender	Age	Clinical Stage
PDM-36™	Pancreatic	G12D	Adeno. ductal type	Primary	Pancreatic head	Male	62	Stage III
PDM-37™	Pancreatic	G12D	Squamous cell carcinoma	Primary	Pancreatic head	Female	64	Stage III
PDM-38™	Pancreatic	G12V	Adeno. ductal type	Primary	Pancreatic tail	Female	63	Stage IIB
PDM-289™	Pancreatic	-	Moderately differentiated Adeno.	Metastasis	Liver	Female	68	Stage IV



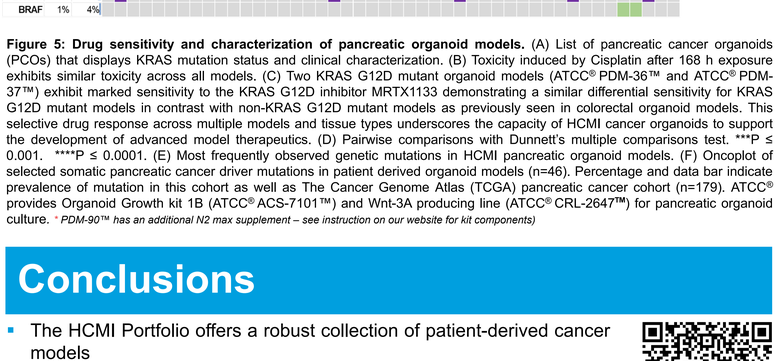


Figure 5: Drug sensitivity and characterization of pancreatic organoid models. (A) List of pancreatic cancer organoids (PCOs) that displays KRAS mutation status and clinical characterization. (B) Toxicity induced by Cisplatin after 168 h exposure exhibits similar toxicity across all models. (C) Two KRAS G12D mutant organoid models (ATCC® PDM-36™ and ATCC® PDM-37™) exhibit marked sensitivity to the KRAS G12D inhibitor MRTX1133 demonstrating a similar differential sensitivity for KRAS G12D mutant models in contrast with non-KRAS G12D mutant models as previously seen in colorectal organoid models. This selective drug response across multiple models and tissue types underscores the capacity of HCMI cancer organoids to support the development of advanced model therapeutics. (D) Pairwise comparisons with Dunnett's multiple comparisons test. ***P ≤ 0.001. ****P ≤ 0.0001. (E) Most frequently observed genetic mutations in HCMI pancreatic organoid models. (F) Oncoplot of selected somatic pancreatic cancer driver mutations in patient derived organoid models (n=46). Percentage and data bar indicate prevalence of mutation in this cohort as well as The Cancer Genome Atlas (TCGA) pancreatic cancer cohort (n=179). ATCC® provides Organoid Growth kit 1B (ATCC® ACS-7101™) and Wnt-3A producing line (ATCC® CRL-2647™) for pancreatic organoid culture. ** PDM-90™ has an additional N2 max supplement – see instruction on our website for kit components)*

Conclusions

- The HCMI Portfolio offers a robust collection of patient-derived cancer models
- Advanced cell models enable more predictive and mechanistic drug-response profiling
- HCMI ensures consistency and scientific quality
- Overall portfolio advances precision oncology and supports the development of next-generation cancer therapeutics

Explore HCMI Models

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