Building Better Cancer Models to Advance Precision Medicine

AACR Spotlight 2026

Agenda

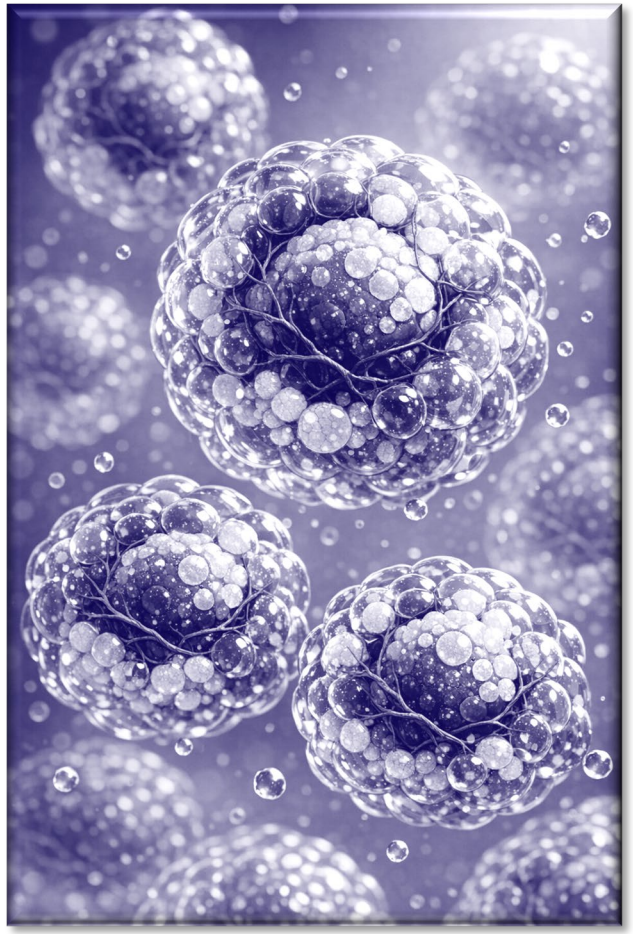


Image generated by Microsoft Copilot

- Carolina Lucchesi

  Building Better Cancer Models to Advance Precision Medicine

- Claudia K. Petritsch, PhD:

  Cell Plasticity-Driven Immune Evasion in Patient-Derived Glioma Models

- Benjamin D. Hopkins, PhD:

  Patient Derived Tumor Organoid for Therapeutic Modeling in Cancer

Presenters

Carolina Lucchesi, PhD
Principal Scientist, Head of Microphysiological Systems, ATCC

Claudia K. Petritsch, PhD
Associate Professor in Research, Director Pediatric Cancer Model Development Center, Sr. Scientist in Neuroscience, Stanford University

Benjamin David Hopkins, PhD
Assistant Professor of Research in Systems and Computational Biomedicine, Weill Cornell Medical College

About Us

ATCC is a global leader in providing authenticated, high-quality biological resources and standards for industry, academia, and government.

- Founded in 1925, ATCC is a private, nonprofit, global biological resource center and standards organization that provides scientists with the biomaterials and resources they need to conduct critical life science research.
- World's trusted, premier biological materials resource and standards development organization:
  - 4,000+ cell lines
  - 80,000+ microorganisms
  - Genomic and synthetic nucleic acids
  - Media, sera, and reagents
  - Advanced cell models
  - Standards

Building Better Cancer Models to Advance Precision Medicine

AACR Spotlight 2026

Modernization of the ATCC in vitro cell model portfolio

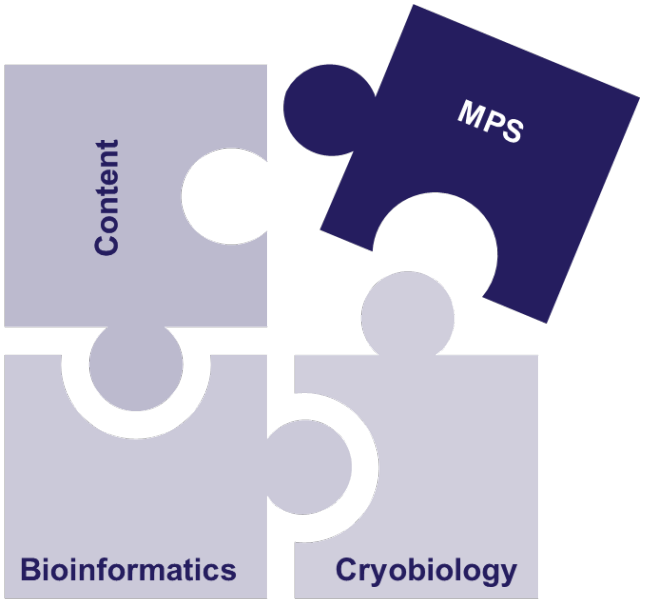
Image generated by Microsoft Copilot

Ensuring the Future ATCC is investing in key technologies to ensure its products and services remain the definitive standards in biological research

Present ATCC R&D teams are actively developing new products to meet the needs of the scientific community

Past ATCC cell and micro collections were historically deposited by academic and other research scientists

ATCC LEADING BIOLOGICAL STANDARDS®



Research & Development

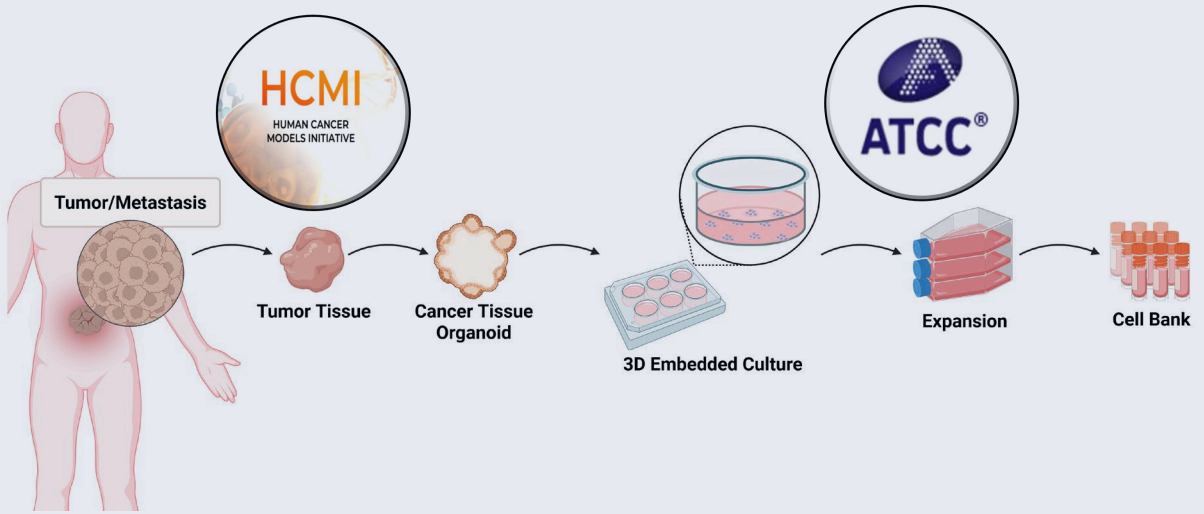
ATCC's Human Cancer Models Initiative

- The Human Cancer Models Initiative (HCMI) is an international consortium dedicated to generating patient-derived cancer models to facilitate cancer research.
- **ATCC is the sole distributor of HCMI models**

Founders and Members

- Broad Institute
- Cancer Research UK
- Cold Spring Harbor Laboratories
- Cornell University
- Hubrecht Organoid Technology Foundation
- National Cancer Institute
- Stanford University
- Wellcome Sanger Institute and others

ATCC's HCMI Workflow



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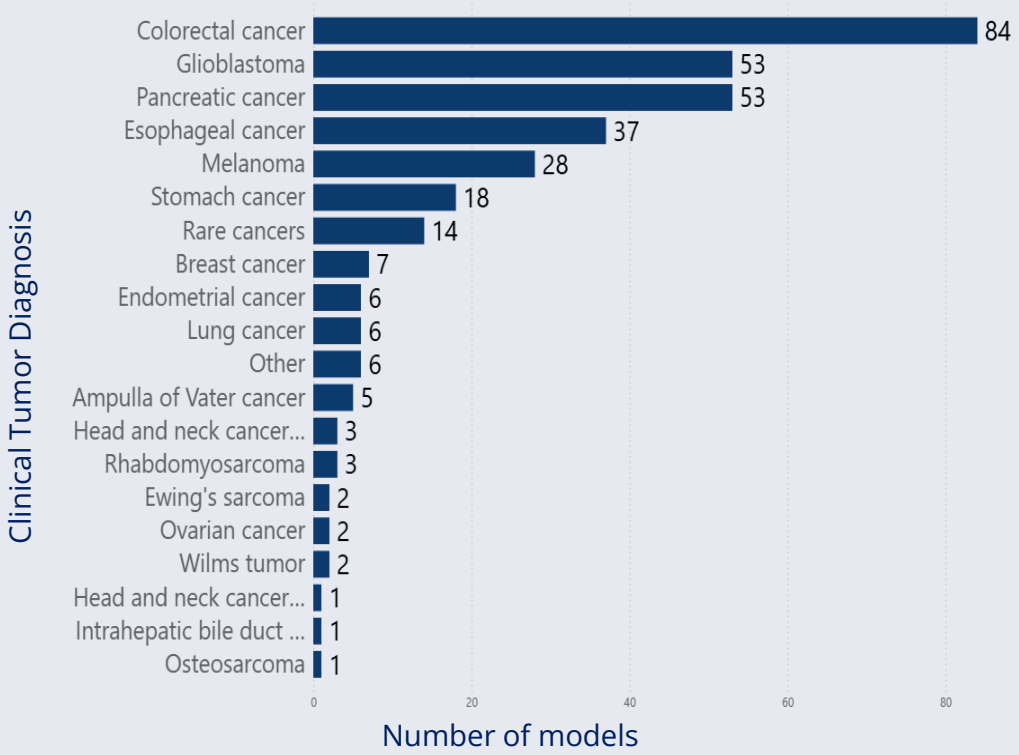
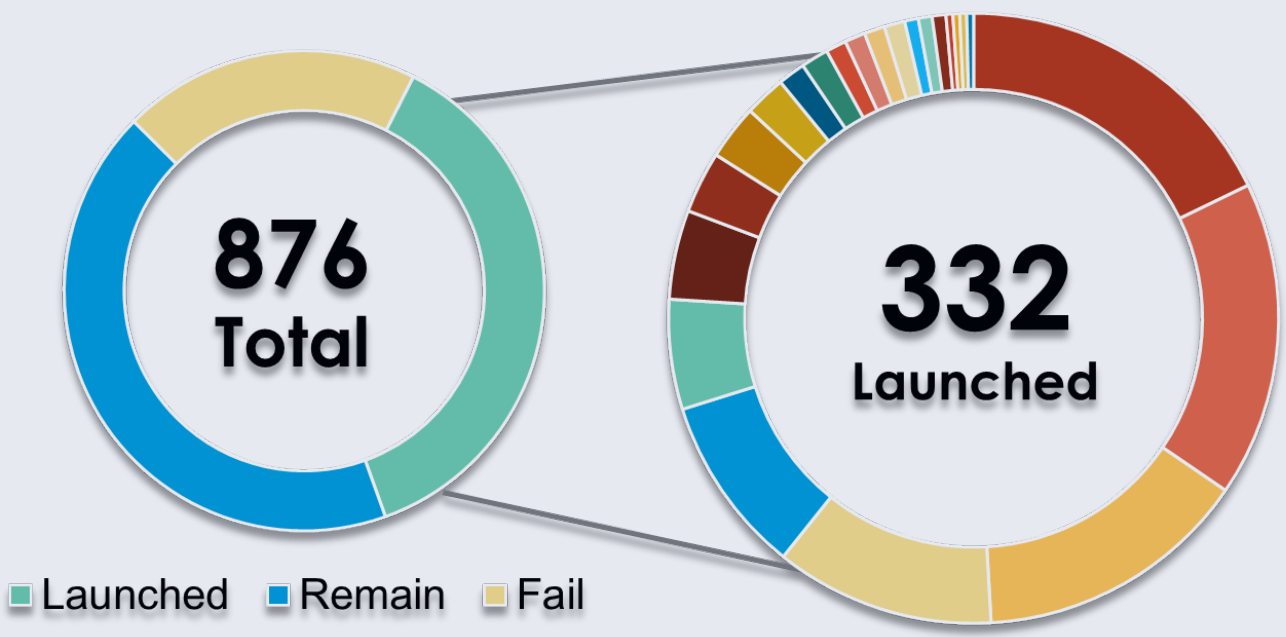
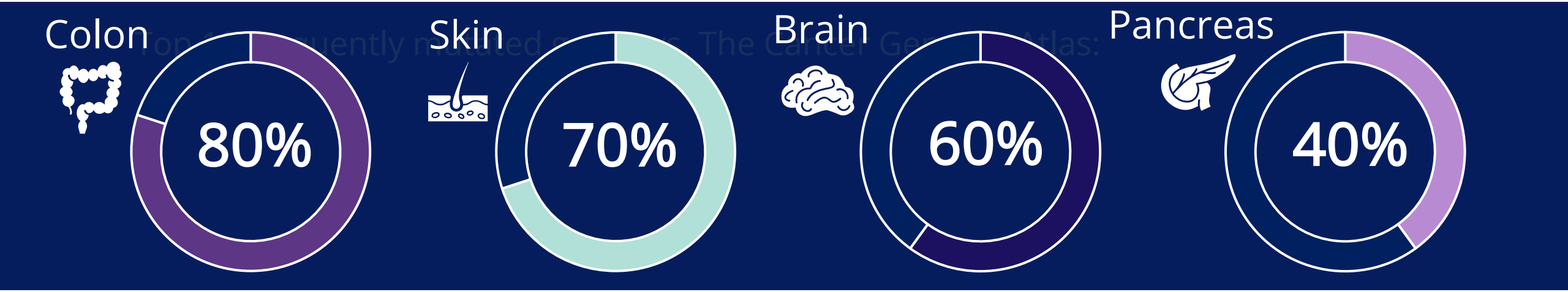
HCMI Portfolio Model Diversity





Collection includes models derived from rare adult and pediatric cancer such as rhabdomyosarcoma, leiomyosarcoma, Ewing sarcoma and Will tumor

Model Relevance



Top 10 genes captured in the HCMI models:

APC	LRP1B
TP53	ZFHX4
KRAS	CSMD3
MUC16	ROBO2
FAT4	ALK

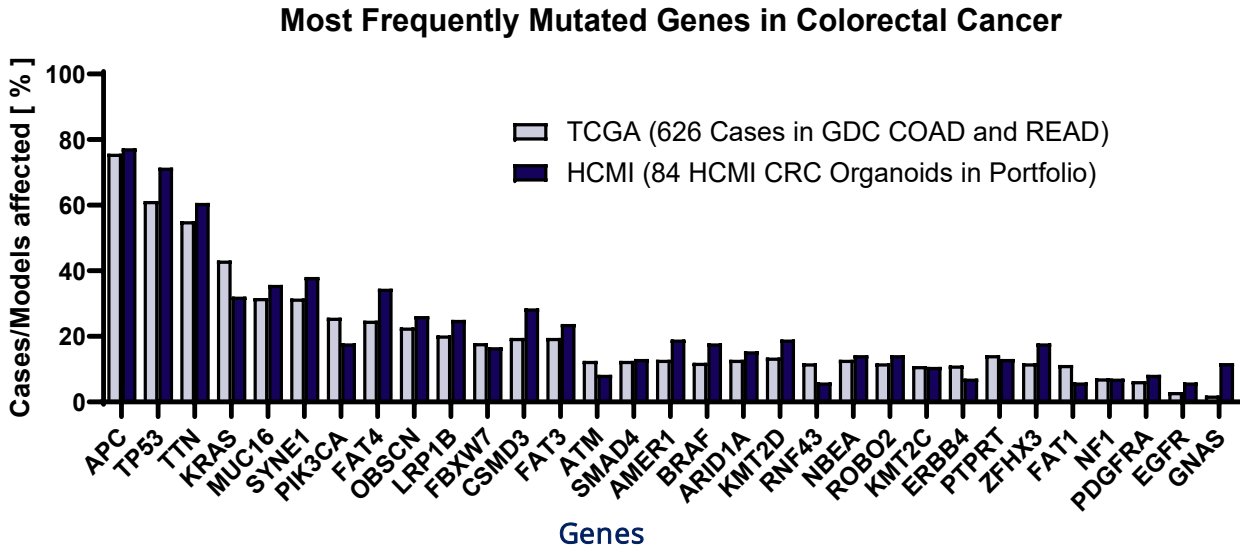
TP53, BRAF, NRAS, NF1	
MUC16	GRIN2A
LRP1B	MECOM
CSMD3	FAT4
FAT3	DCC
FAM135B	COL1A1

PTEN	PIK3CA
TP53	CSMD3
EGFR	BCOR
CNTNAP2	NF1
PIK3CR1	FAT1

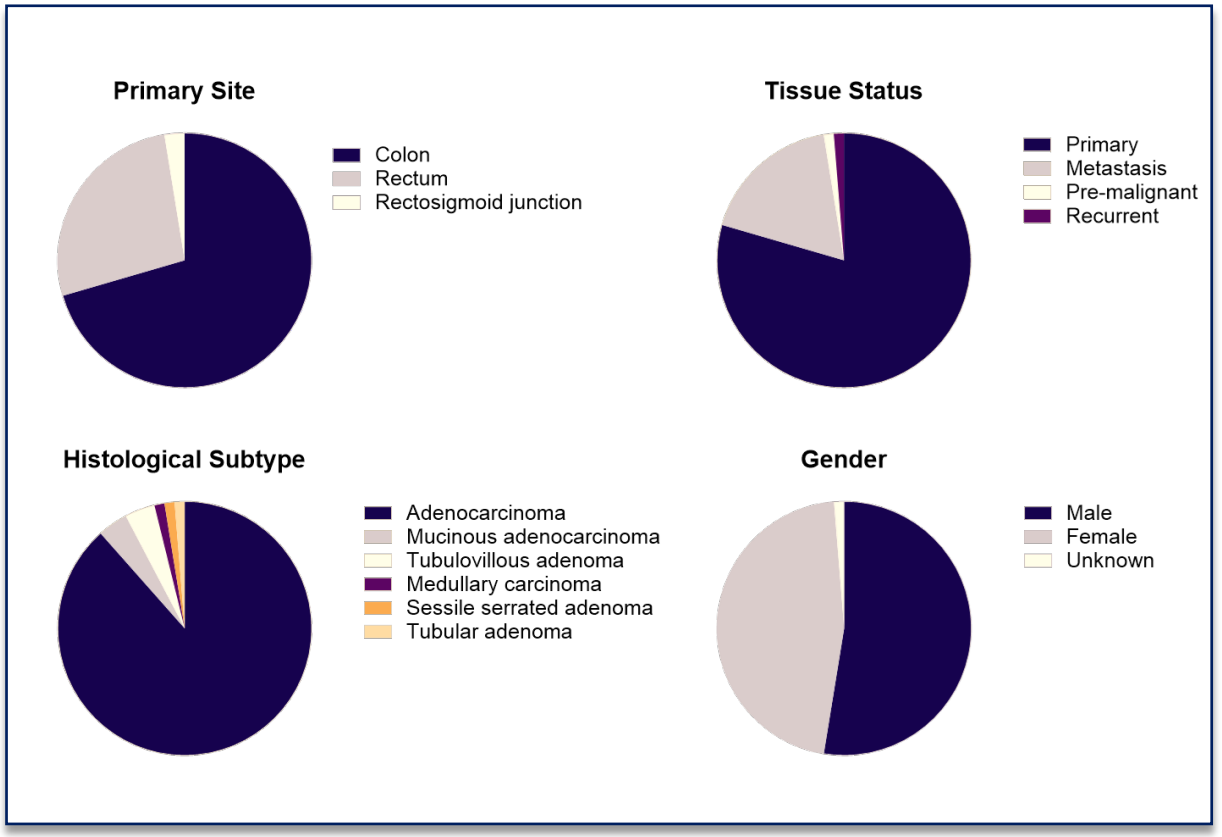
KRAS	MUC4
TP53	MUC16
SMAD4	KMT2D
CDKN2A	FAT3
ACVR2A	RHOH

Canonical mutation	Matching mutation

HCMI colorectal cancer model cohort

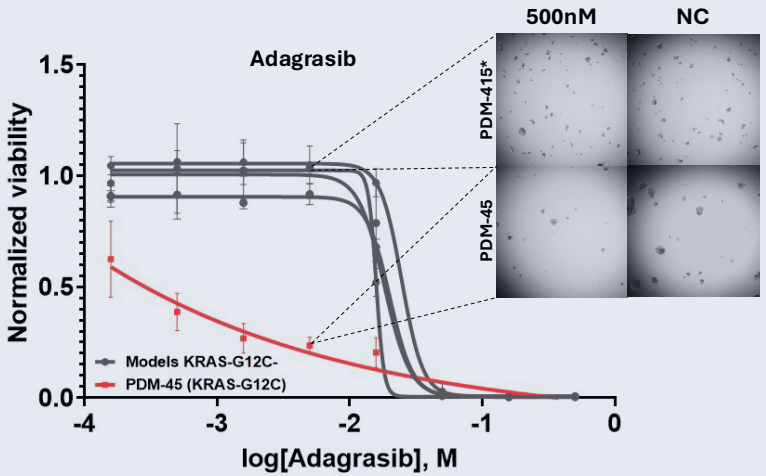


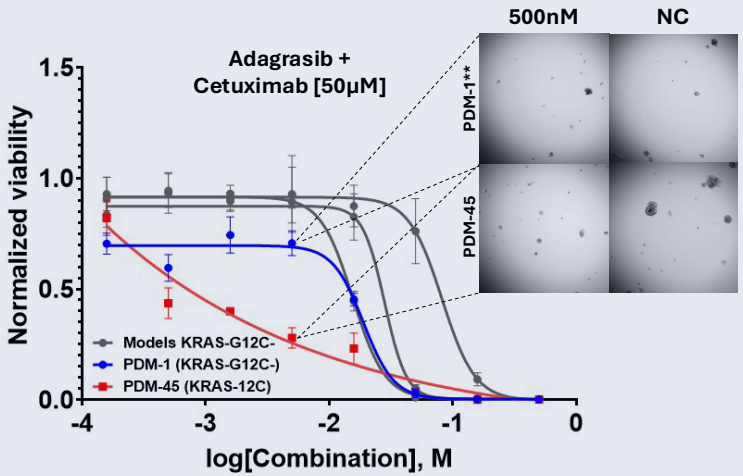
Diverse population dynamics and gene mutations in our colorectal cancer model



Response to Adagrasib or Adagrasib+Cetumimab

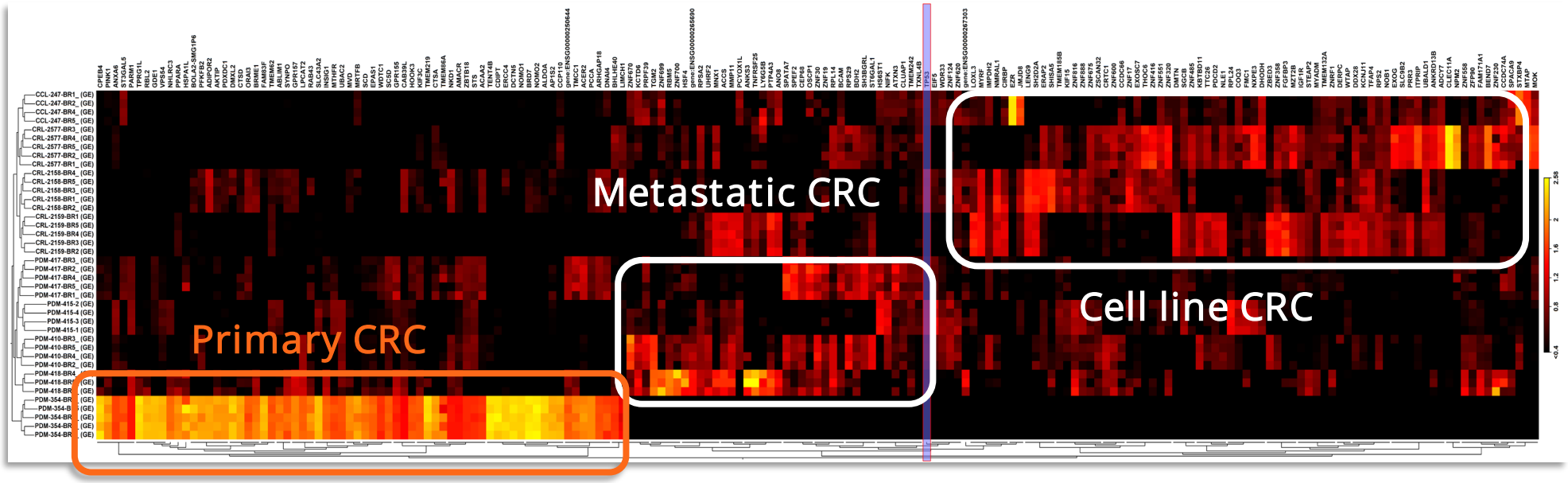
ATCC® Model	Cancer Type	Histological Subtype	Type	Acquisition Site	Gender	Race	Age	Clinical Stage	KRAS Status
PDM-1™	Colorectal	Adenocarcinoma	Primary	Cecum	Male	White	75	Stage I	KRAS-G12A
PDM-45™	Colorectal	Adenocarcinoma	Primary	Transverse colon	Male	--	80	Stage IIIB	KRAS-G12C
PDM-354™	Colorectal	Adenocarcinoma	Primary	Sigmoid colon	Female	--	70	--	No KRAS Mutation
PDM-410™	Colorectal	Adenocarcinoma	Metastatic	Liver	Female	Black	56	Stage IVA	No KRAS Mutation
PDM-415™	Colorectal	Adenocarcinoma	Metastatic	Peritoneum	Female	Black	48	Stage IIIB	KRAS-G13D

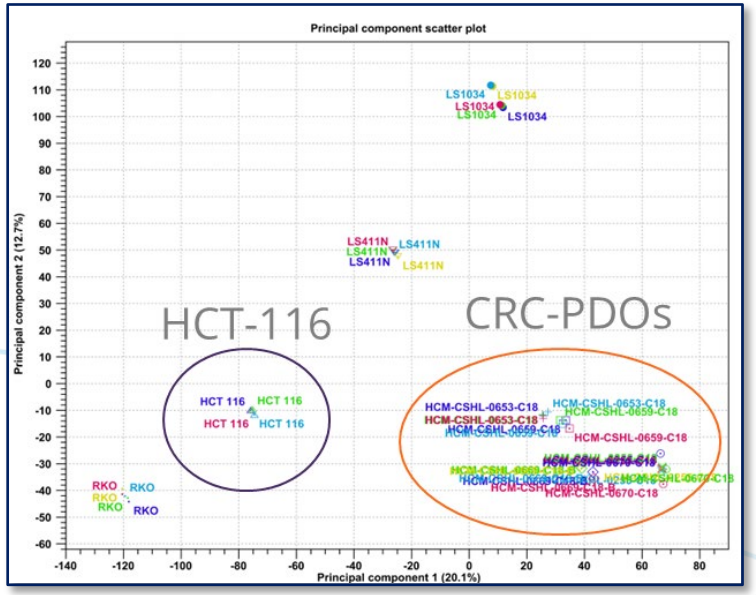




The KRAS-G12C model shows clear sensitivity to Adagrasib as a single agent, consistent with mutation-specific targeting.

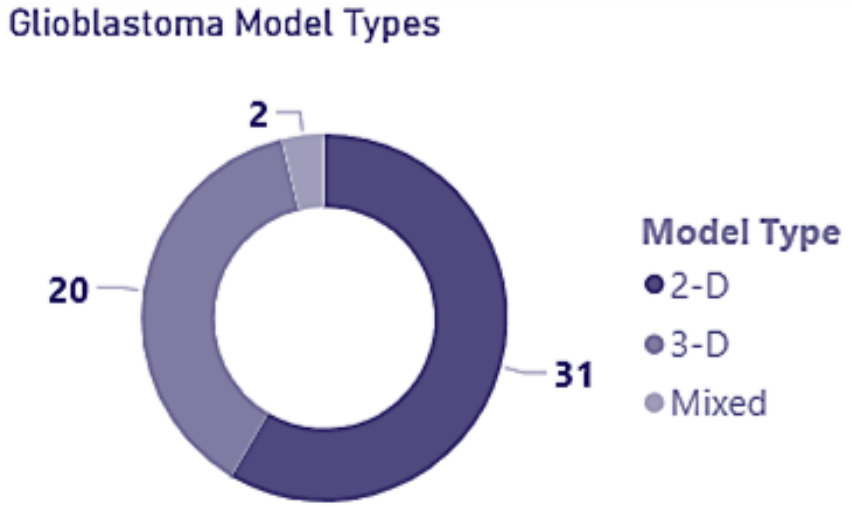
Transcriptomic Clustering Distinguishes Organoids from Conventional Cell Lines

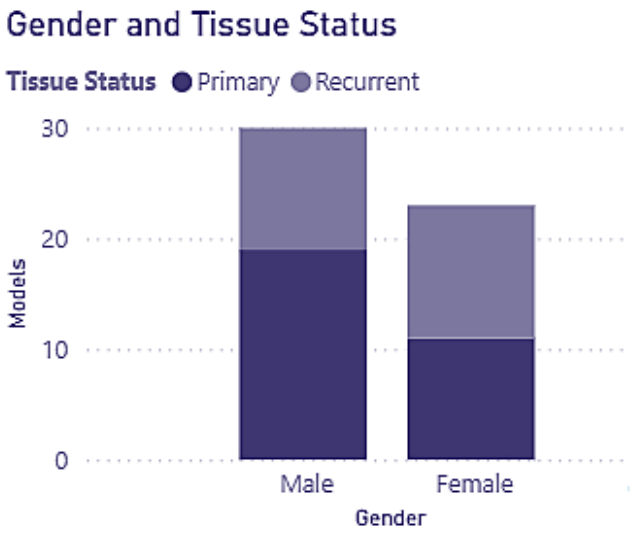


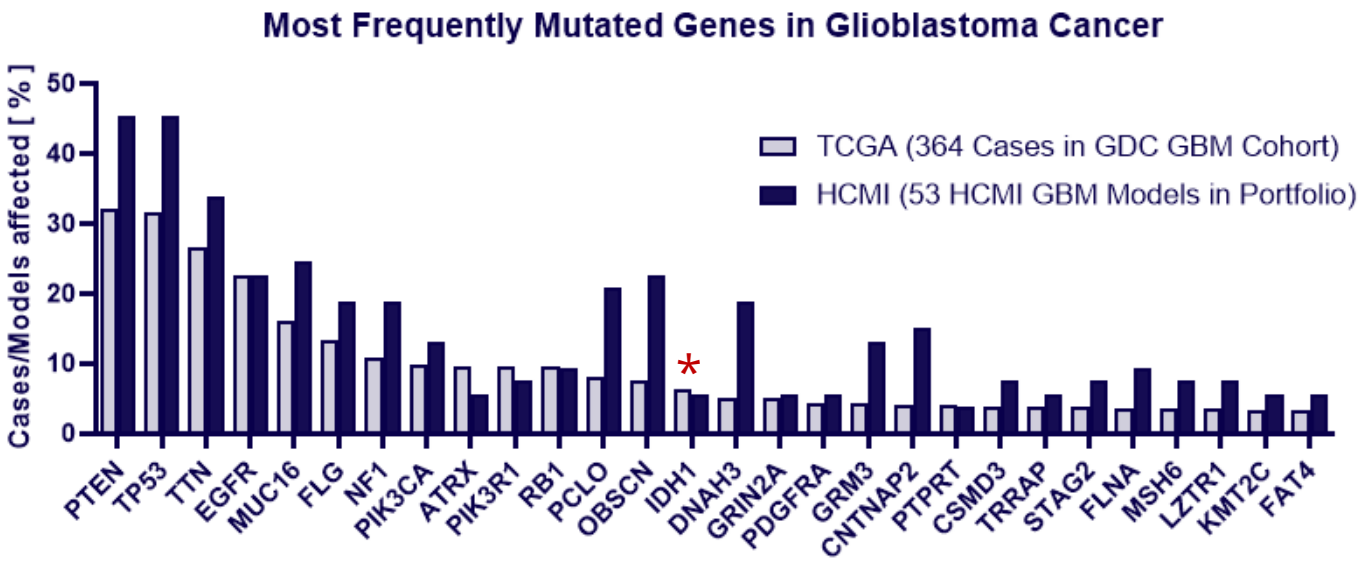


Patient-derived colorectal cancer organoids cluster by disease state (primary vs. metastatic) and exhibit gene expression profiles that are markedly distinct from standard cell lines (e.g., HCT116), demonstrating superior preservation of patient-specific tumor biology

Diversity of Glioblastoma Models





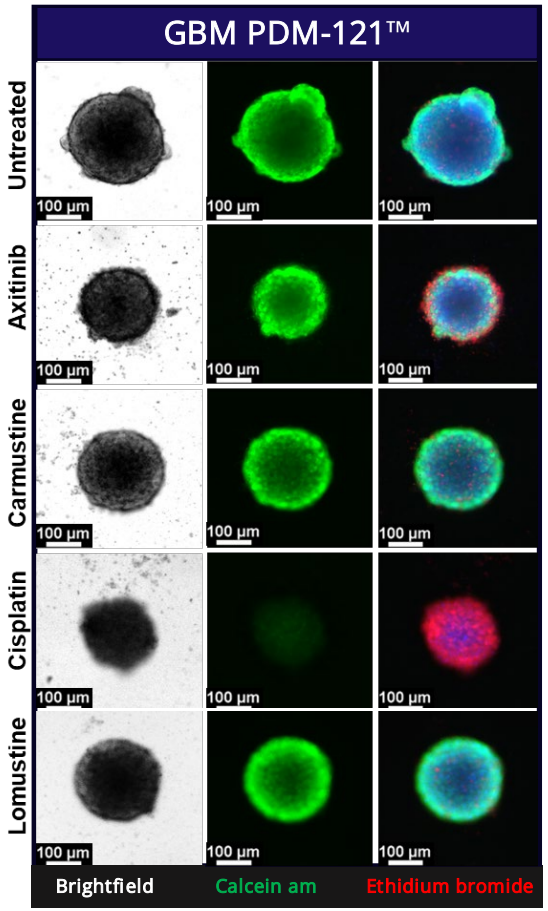


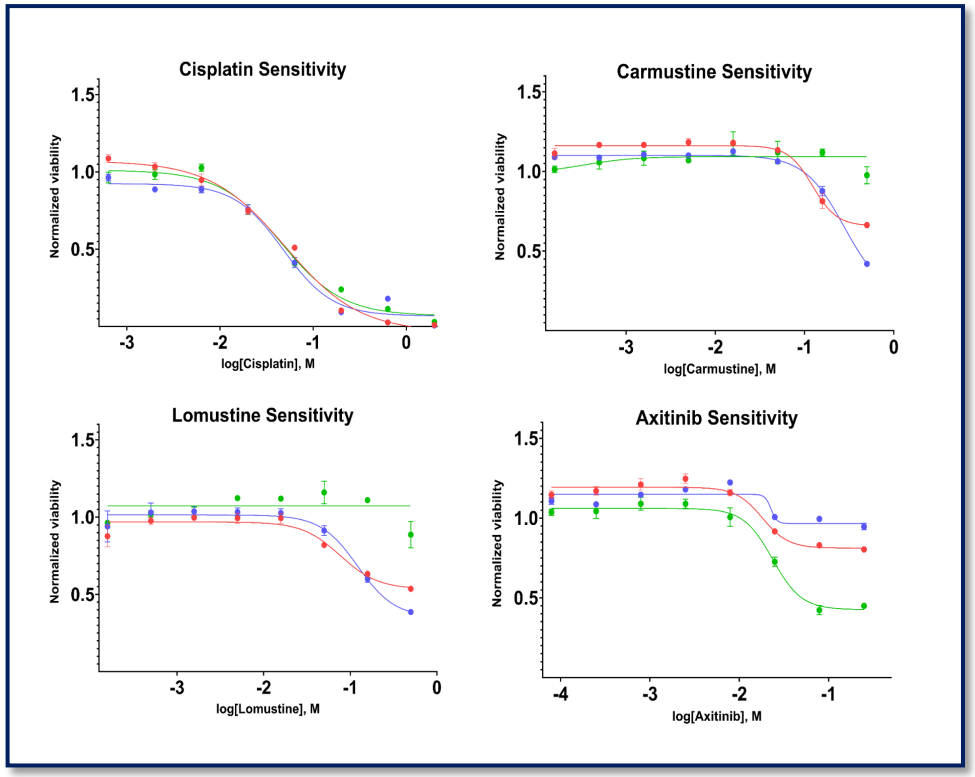
Diverse population dynamics and gene mutations in our glioblastoma model

**AACR Poster focused on IDH1*

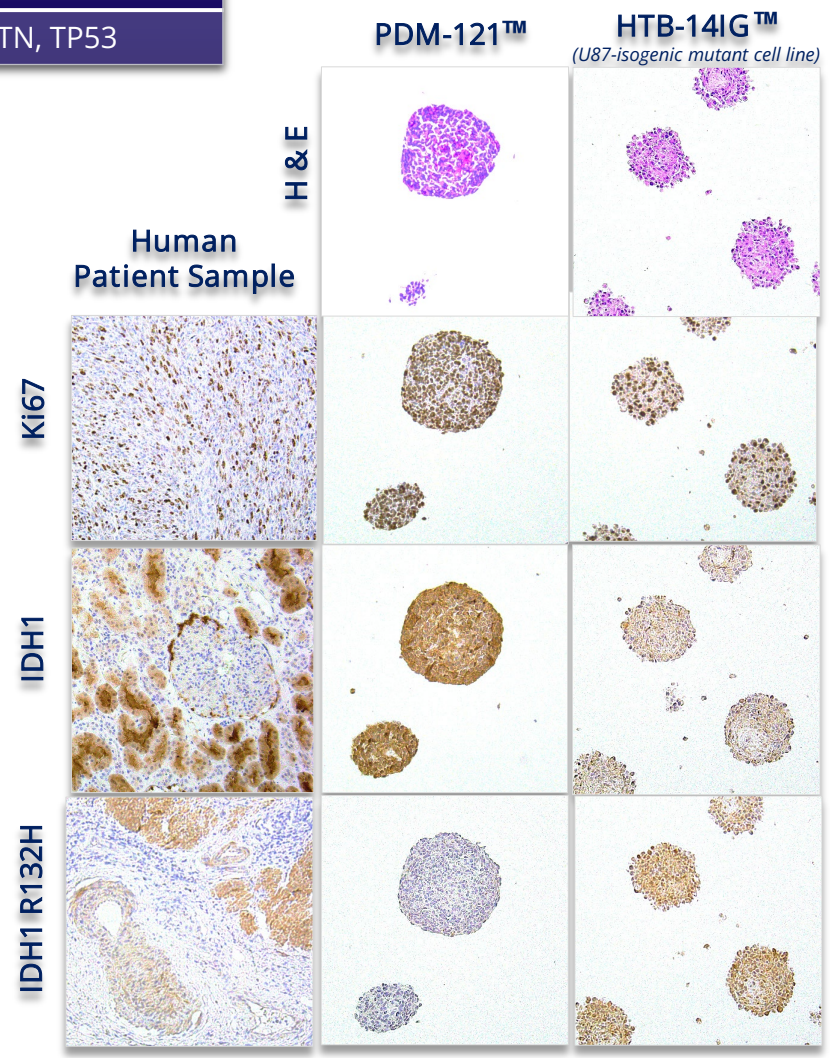
Pediatric Glioblastoma

ATCC Part #	Cancer Type	Disease Status	Type	Acquisition Site	Gender	Race	Age	Tissue Status	Key Mutations
PDM-121	Glioblastoma	Progressive	3D Spheroid	Brain	Female	White	11	Primary	IDH1, TTN, TP53





IDH1-driven drug resistance tested in pediatric primary neurospheres



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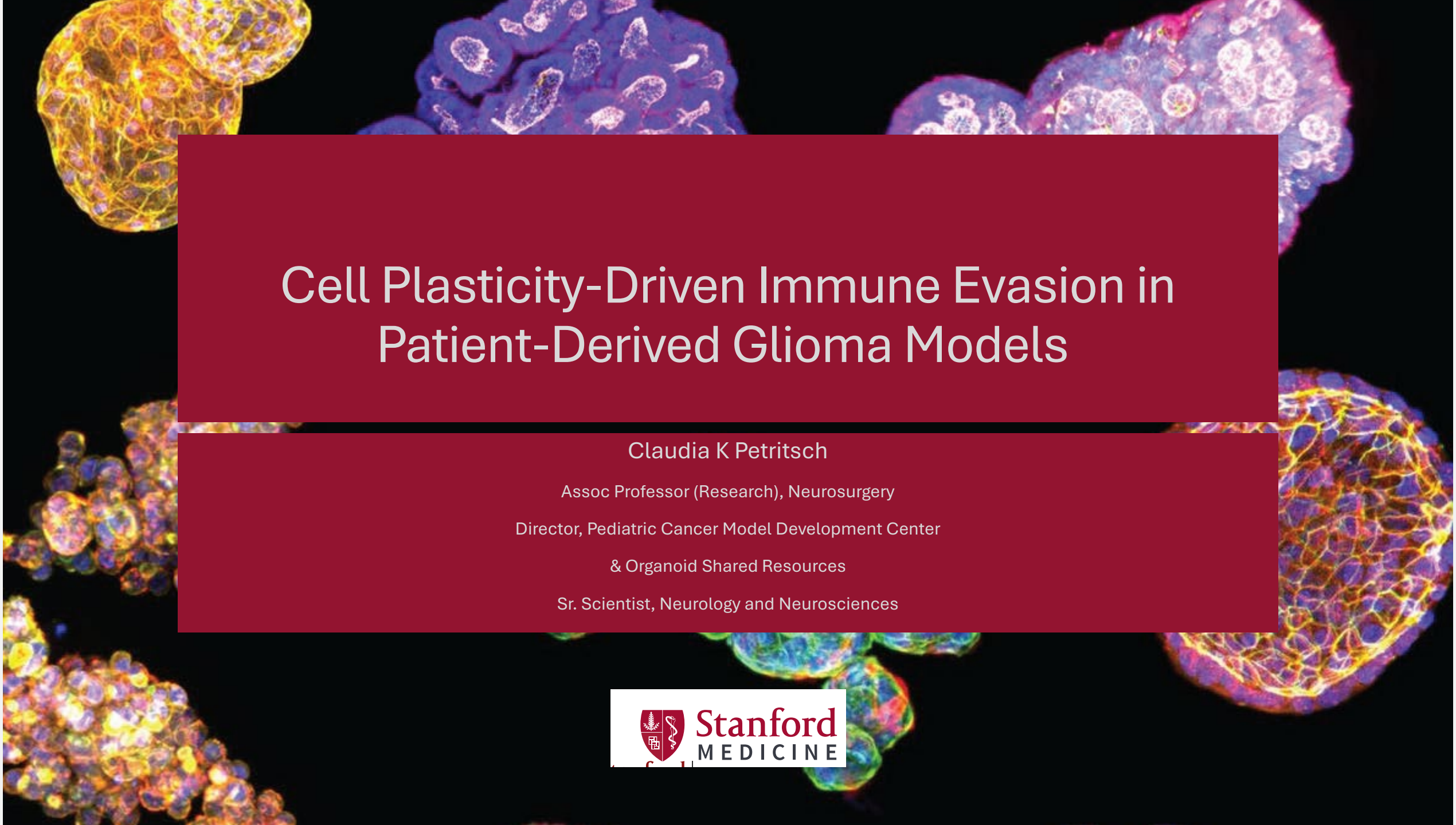

Cell Plasticity-Driven Immune Evasion in Patient-Derived Glioma Models

Claudia K Petritsch

Assoc Professor (Research), Neurosurgery

Director, Pediatric Cancer Model Development Center

& Organoid Shared Resources

Sr. Scientist, Neurology and Neurosciences

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Disclosures

- No Disclosures

Topics covered

- Why do oncology drugs fail in the clinic?
- Why do we need better cell culture models, especially for childhood brain tumors?
- What are better (next-generation) cell culture models?
- BRAF V600E-mutant high-grade patient-derived models: a paradigm for precision medicine – insights achieved by next-generation cell culture models
- The Stanford Pediatric Cancer Model Development Center

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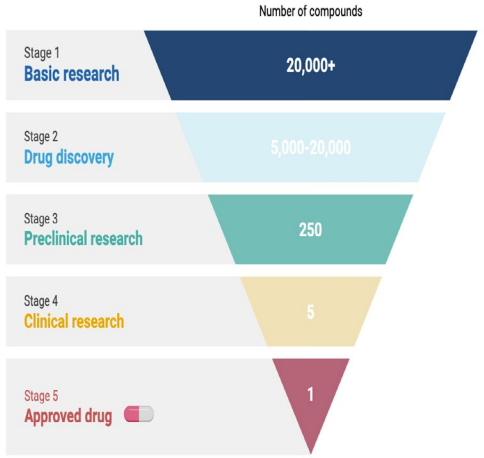
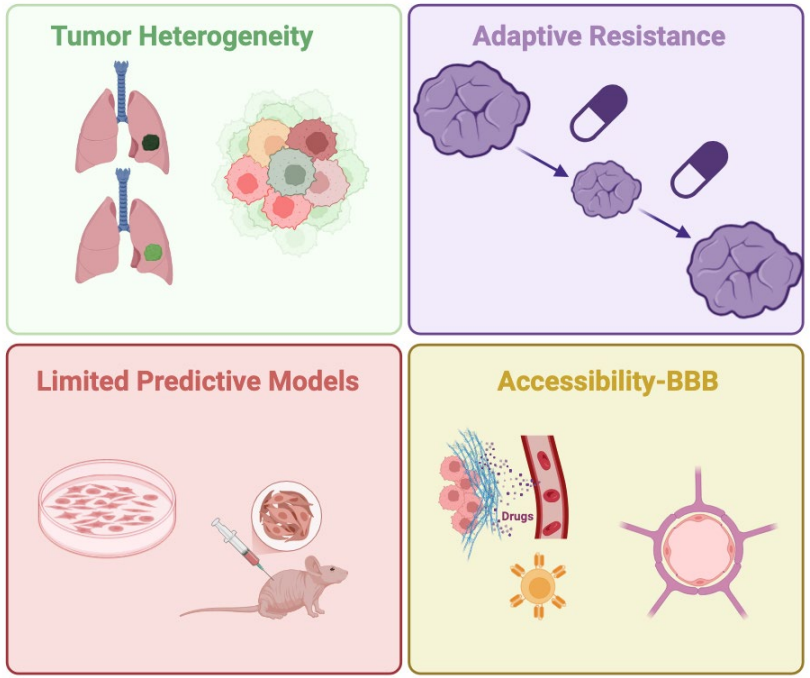
Low Prediction Models: Barrier for Oncology Drug Development

1 Oncology drug development attrition rates



2 Lack of efficacy causes for oncology drugs



~50-60% of drugs fail in the clinic due to lack of efficacy
FDA Approval can take up to 13 years and cost up to $2.5b

Advantages of Human Next-Generation Cell Culture Models

1 Conventional Cell Culture Models

Tumor/Healthy tissue

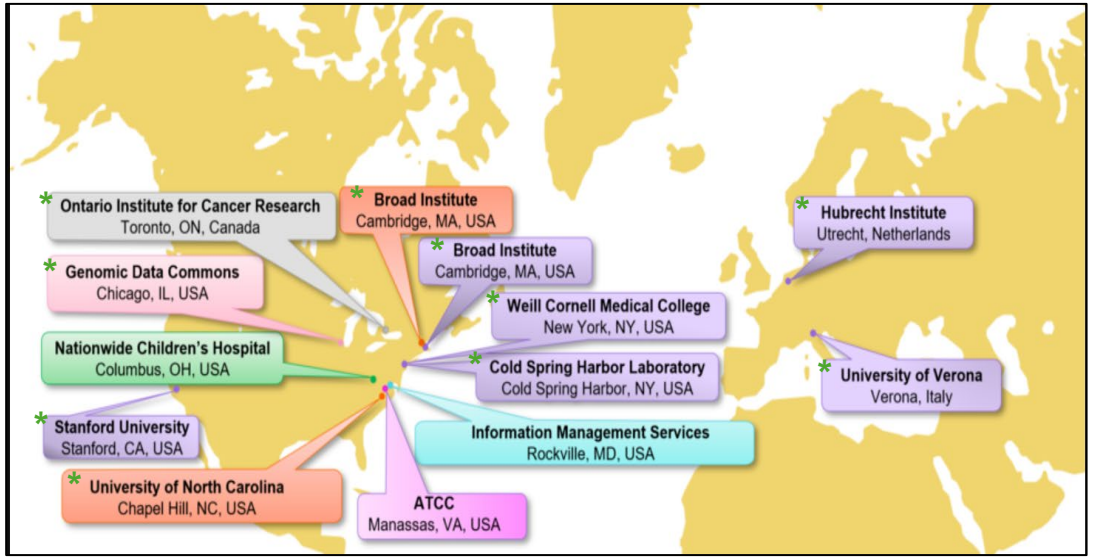
(*monolayer, 2-dimensional (2D) cultures on plastic*)

- Cells plated on flat **plastic surfaces**
- Cells grown in high levels of animal **serum**
- Genetic and transcriptomic **drift**
- Lack stemness, **tumor heterogeneity and plasticity**
- **Overpredict drug efficacy**
- Often lacking clinical information of **parental tumor origin, molecular characterization missing**

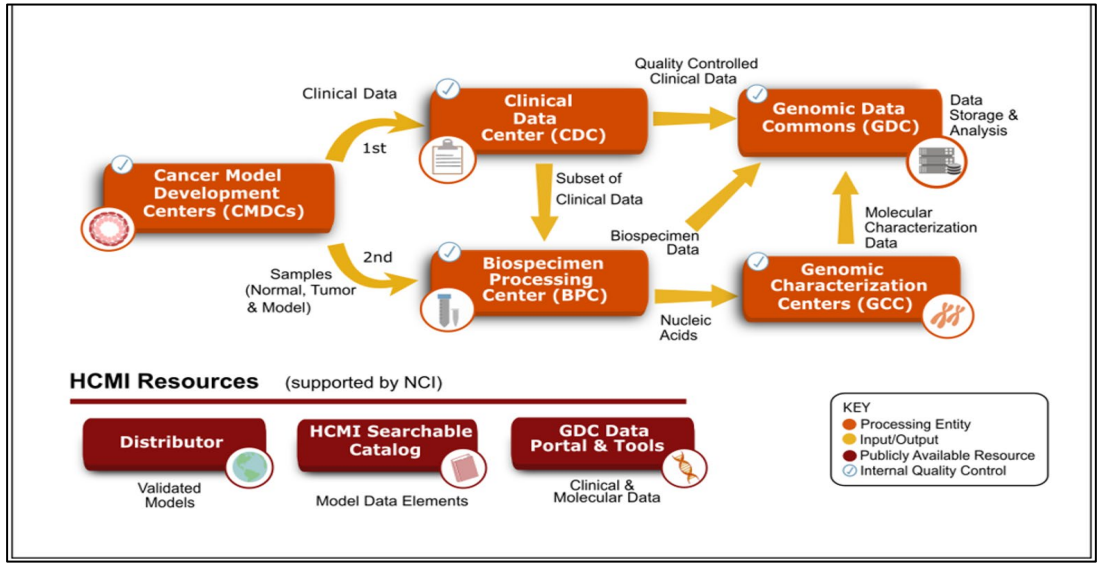
→ Simple, scalable, **low physiological relevance application: e.g., early stages of drug screening**

The Human Cancer Model Initiative (HCMI)

The HCMI is an international consortium founded by the **National Cancer Institute** and dedicated to generating **next-generation, patient-derived cancer models as a community resource** to facilitate cancer research



(Source: https://www.cancer.gov/ccg/research/functional-genomics/hcmi/about/cancer-model-development)

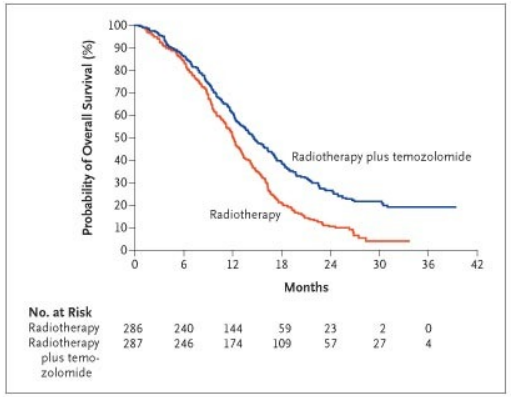


(Source: https://ocg.cancer.gov/programs/hcmi/nci-cancer-model-development)

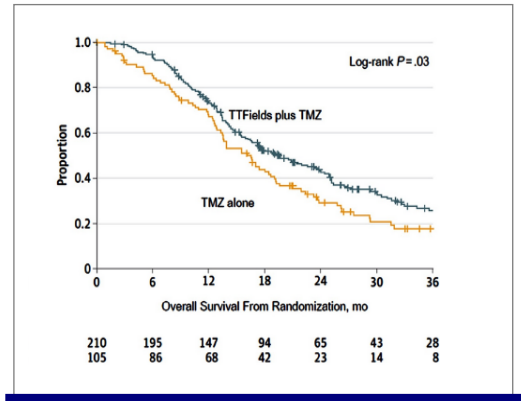
(* Managed by the Frederick National Laboratory for Cancer Research (FNLCR), Leidos Biomedical Research, Inc.)

Glioblastoma: Survival with SoC and Drug Approvals

1 Survival rates with SoC



Stupp *et al*, Lancet 2005, PMID15758009



2 Drug Approvals Lung Cancer vs. Gliomas

Carmustine
Lomustine

Dabrafenib/Trametinib
Tafinlar + Mekinist

Dordaviprone
MODEYSO

Naxitamab
DANYELZA

Tovorafenib
ojemda

Vorasidenib
Voranigo

OPTUNE

Glioblastoma/HGG SoC has minimal effect on survival (med surv = 15.4 mos) with >90% recurrence rate
One drug approval for high-grade gliomas in 10 years
Clinical need for new therapeutics is high

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Challenges for Development of Therapeutics for High-Grade Glioma

1 Key challenges malignant brain tumor treatment

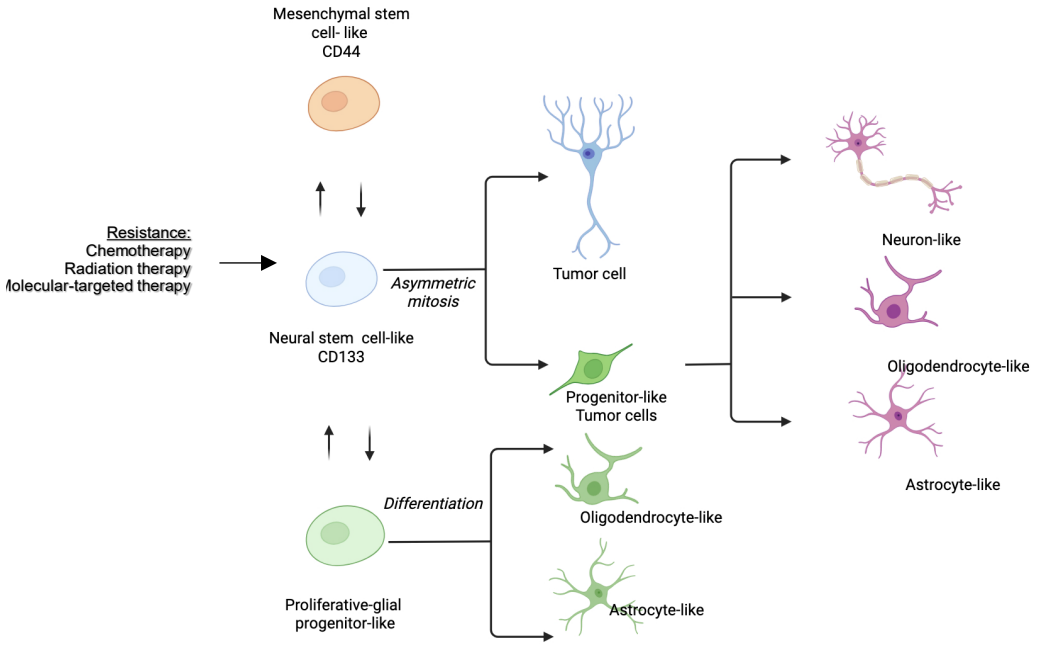
Tumor Heterogeneity

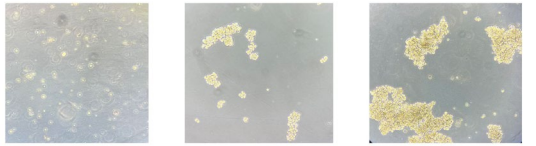
HGG contains multiple genetically distinct clones. Targeting one subpopulation allows resistant clones to repopulate.

Therapy Resistance, Plasticity, Glioma Stem Cells

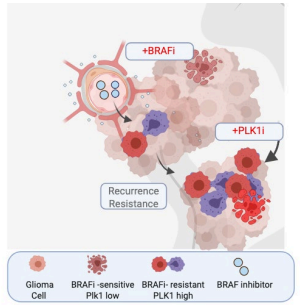
A subpopulation of cells is resistant to radiation and chemotherapy, showing plasticity, driving tumor re-initiation after treatment.

2 Stem cell-like features of malignant brain tumors





Morphology of an epithelioid glioblastoma tumor 3D spheroid cell line HCM-STAN-1297-C71. Morphology. **left**. 2 h after thawing; **center**. at low density; **right**. at high density.



Lerner *et al.* Cancer Res 2015, PMCID:PMC4698003

Several key features of malignant brain tumors contribute to failed efficacy

Next-generation models recapitulate key features of malignant brain tumors

Malignant brain tumors are very diverse and have many subtypes – model panels

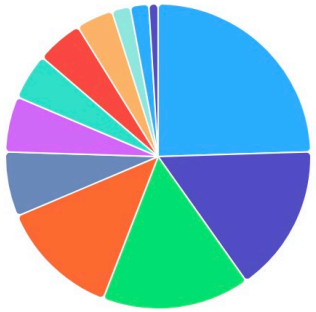
Majority of Current HCMI Models are for Adult Cancers

1 Human Cancer Models Initiative Representation

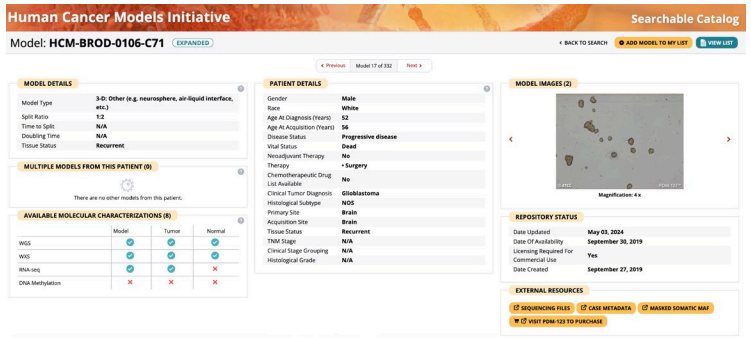
Only 12 pediatric cancer models in the HCMI collection

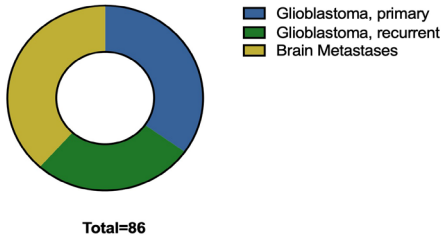
332

Childhood Cancer Incidence, Age 0-19, 2017-2021



2 Models for Brain Malignancies in the HCMI catalogue





- Leukemia: 25%
- Lymphoma: 16%
- Brain and Central Nervous System: 16%
- Epithelial Neoplasms and Melanomas: 13%
- Rhabdomyosarcoma (Soft Tissue Tumors): 7%
- Germ Cell Tumors: 6%
- Neuroblastoma and Other Peripheral Nerve Cell Tumor: 5%
- Bone Tumors: 5%
- Kidney Tumors (including Wilms Tumor): 4%
- Liver Tumors (including Hepatoblastoma): 2%
- Retinoblastoma: 2%
- Other: 1%

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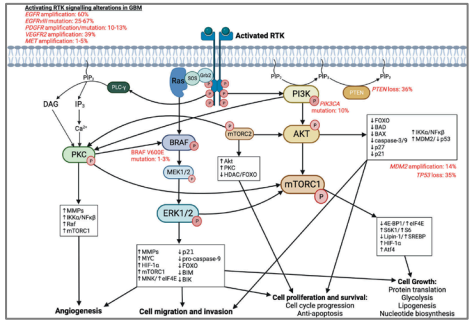
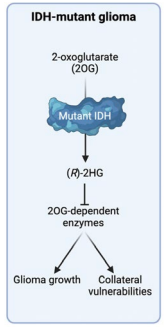
Pediatric and Adult Brain Cancers Are Fundamentally Different

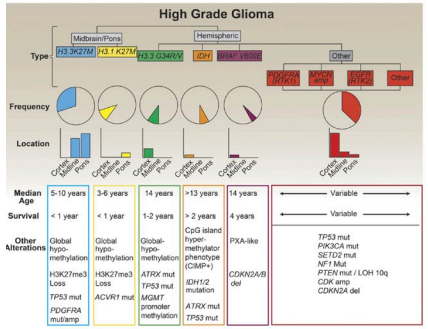
1 Pediatric brain cancer characteristics

- Rare (4-5 x lower incidences than in adults)
- *Different* treatment strategies (minimize long-term sequelae)
- Different pathogenetic mechanisms (developmental, Gliomas: MAPK pathway vs. RTK, IDH1 mut vs BRAF mutations)

2 Common oncogenic pathways of adult glioma

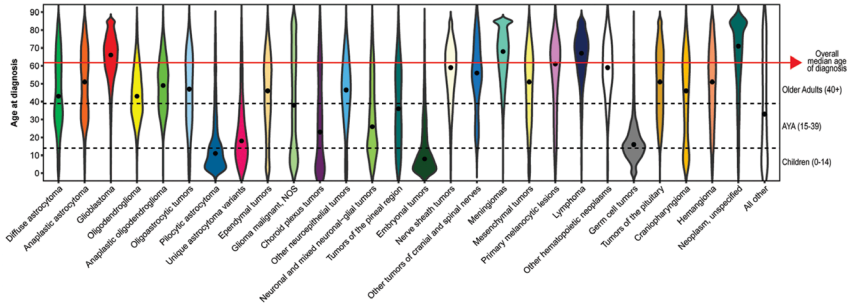


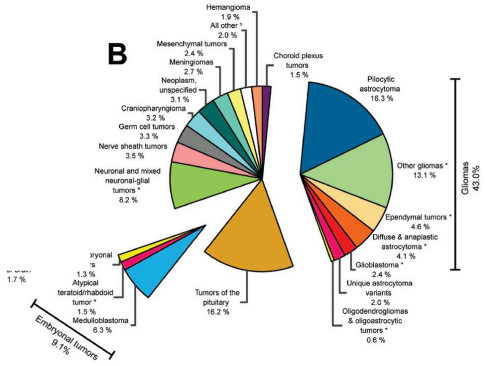




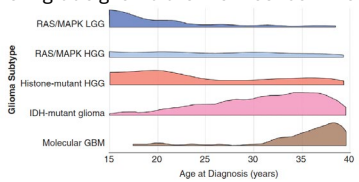
Dewdney *et al*, Signal Transduct Target Ther 2023
PMCID:PMC10587102

3 Distribution of age at diagnosis by selected primary brain tumors





Low-grade glioma are the most common



Price *et al*; Neuro-oncology 2024; https://doin.org/10.1093/neuonc/noae145

Lim-Fat *et al*; Neuro-oncology 2025, PMCID:PMC11726256

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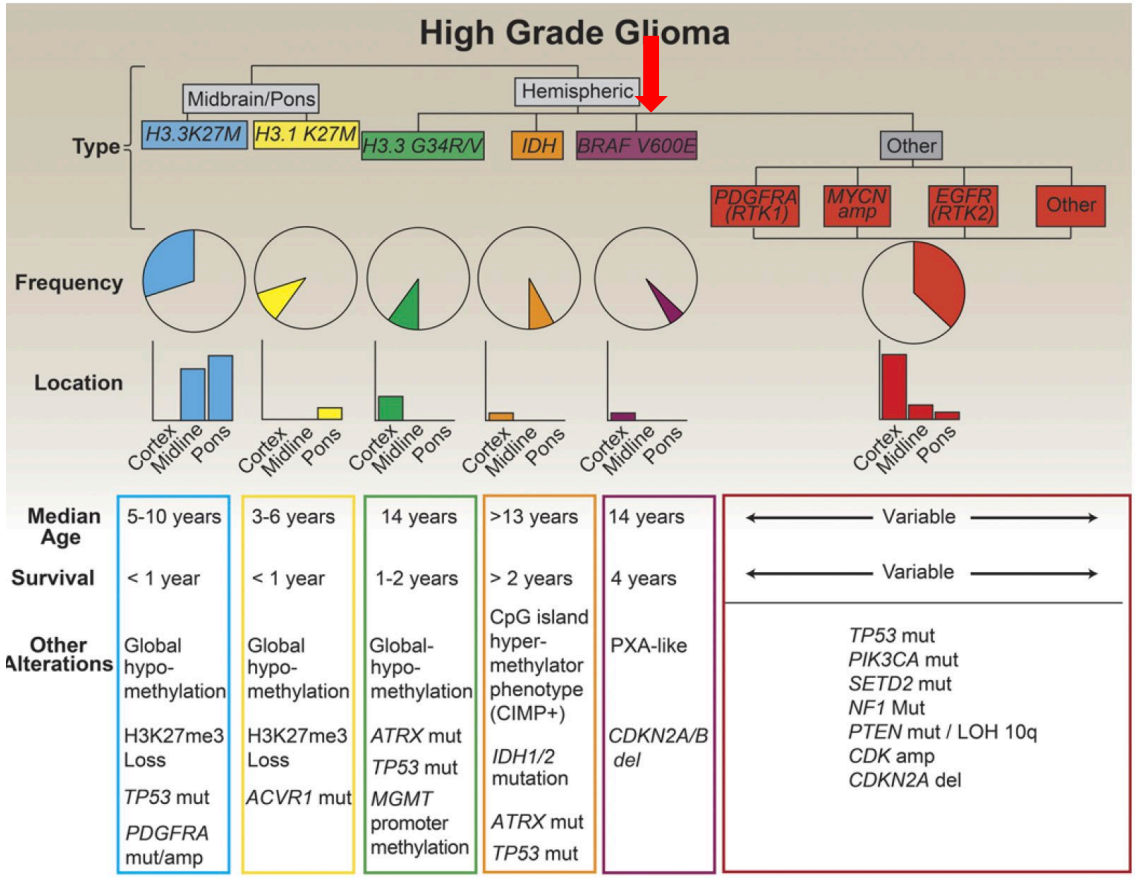
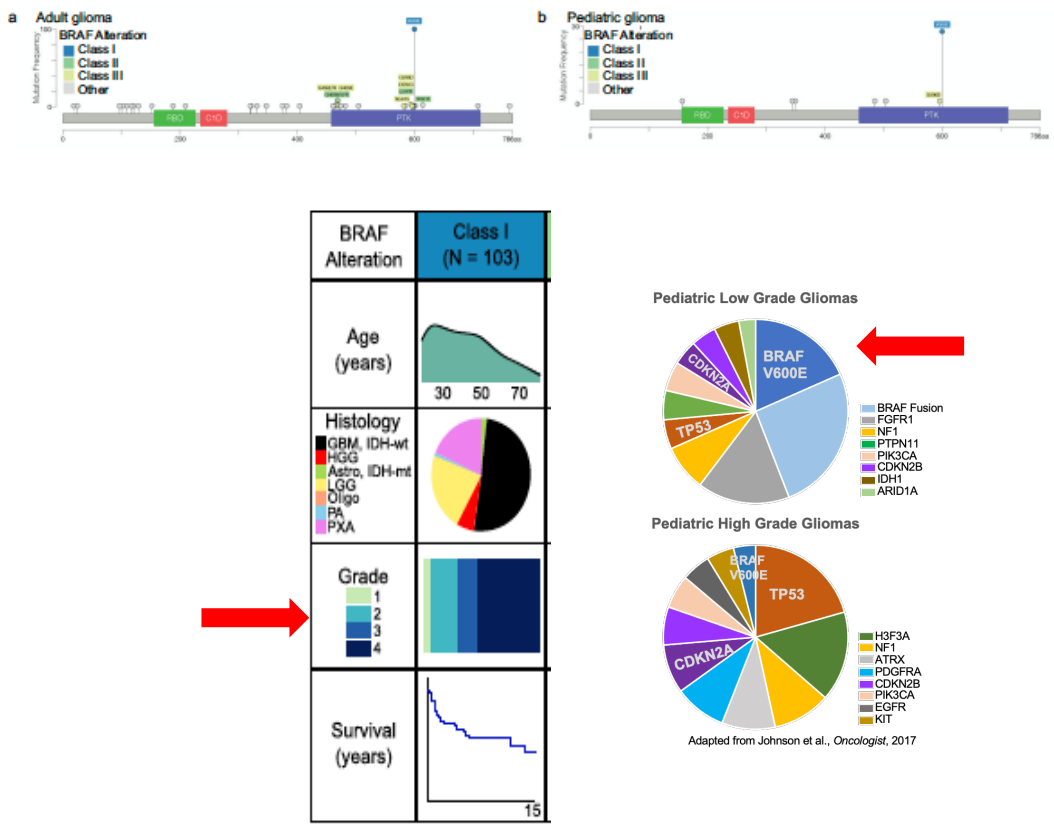
BRAF V600E-mutant glioblastoma as a paradigm for precision medicine in brain tumors

1. BRAF V600E-altered glioma occur in pediatric + adult patients but in different types



Pollack *et al*, J Neurosurg Pediatr 2019, PMCID:PMC6823600



Schreck *et al*, NPJ Precis Oncol 2023, PMCID:PMC9975216

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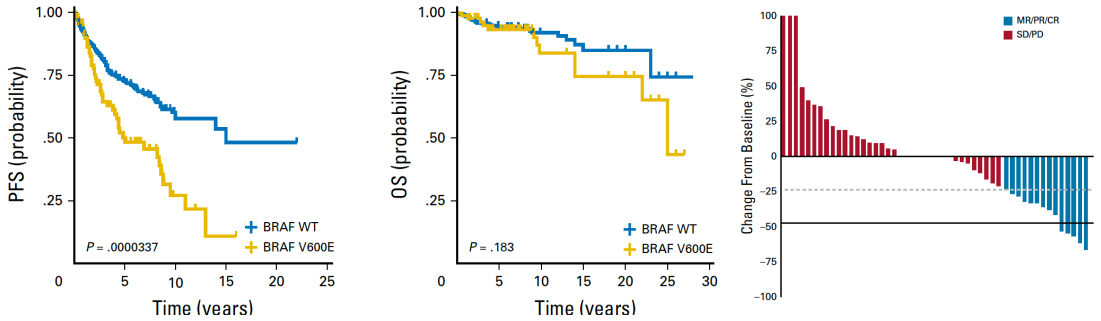
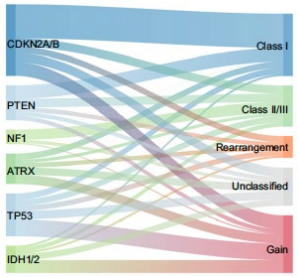
The BRAF V600E-mutant glioma frequency, types, and prognosis

1 BRAF V600E-altered glioma are chemotherapy-resistant

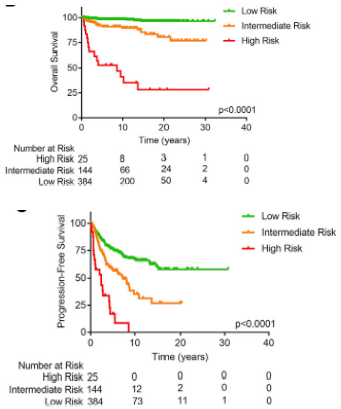


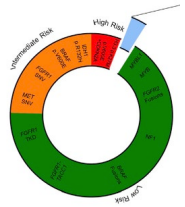
Lassaletta *et al*., JCO 2017, PMCID:PMC5791837

2 BRAF V600E co-mutations affect risk for progression



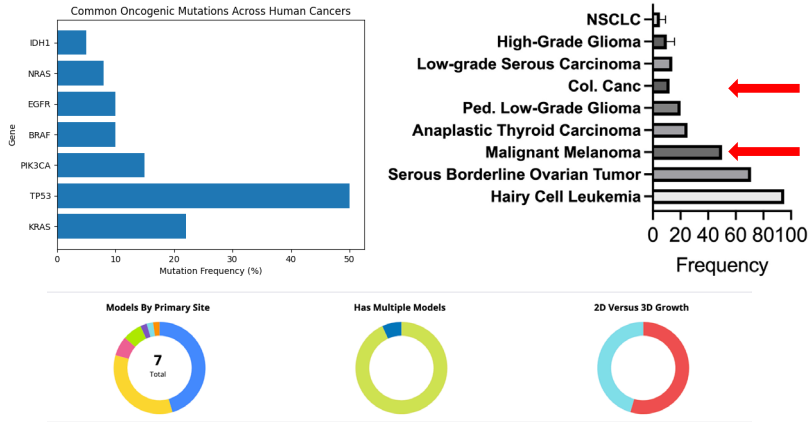
Schreck *et al*, NPJ Precis Oncol 2023, PMCID:PMC9975216





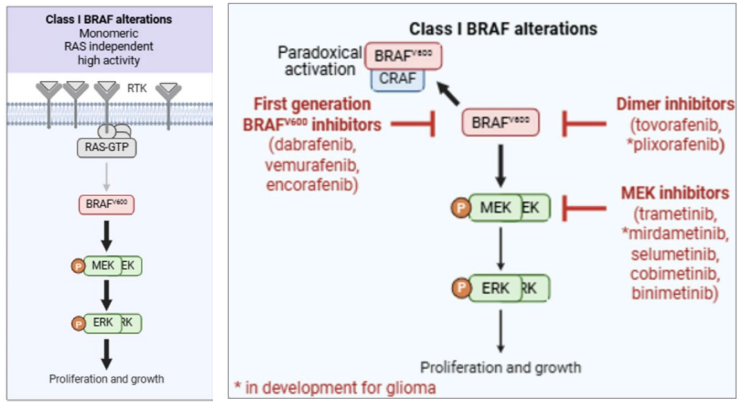
Ryall *et al*. Cancer Cell 2020, PMCID:PMC7169997

3 BRAF V600E mutations found in various cancer



BRAF V600E models in HCMI catalogue

4 BRAF/MEK inhibitors in the clinical arena



Courtesy of Karisa Schreck

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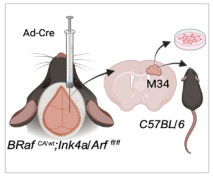
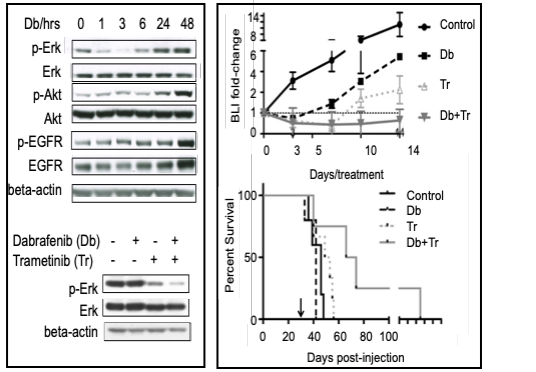
Research Objectives Based on Low-Response Rates

1 MAPK pathway reactivation after BRAF inhibition

Syngeneic Mouse Models For BRAF V600E-mutant High-Grade Glioma:



Grossauer *et al*. Oncotarget, 2016, PMCID:PMC5342782



Dabrafenib (DB)=BRAF inhibitor
Trametinib (Tr)=MEK inhibitor

Understand how BRAF V600E mutated high-grade gliomas respond to clinically relevant, molecular-targeted inhibition (BRAF V600E and MEK inhibition)

Identified potential mechanisms of therapy escape

Find novel therapeutic opportunities to combine with BRAF V600E- targeted therapy to overcome resistance

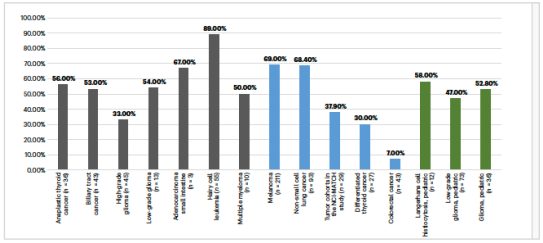
2 Combined BRAF and MAPK inhibition response rates

	Grade III (n=13)	Glioblastoma (n=31)	Age 18–39 years (n=22)	Age ≥40 years (n=23)
Objective response rate by investigator, % (95% CI)	38 (13·9–68·4)	32 (16·7–51·4)	50 (28·2–71·8)	17 (5·0–38·8)
Patients responding at 12 months by investigator assessment, % (95% CI)	100	67 (28·2–87·8)	89 (43·3–98·4)	50 (5·8–84·5)
Median progression-free survival by investigator, months (95% CI)	3·8 (1·7–NR)	2·8 (1·8–13·7)	18·5 (5·5–41·4)	1·7 (0·9–2·5)
Median overall survival, months (95% CI)	45·2 (6·3–NR)*	13·7 (8·4–25·6)	45·2 (17·9–NR)†	8·7 (3·7–11·7)

NR=not reached. *Six deaths among 13 patients. †Eight deaths among 22 patients.

Table 3: Post-hoc subgroup analysis of the high-grade glioma cohort

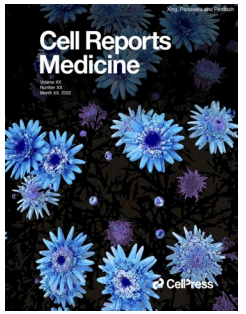
Wen *et al*, The Lancet Oncology 2022, PMID:34838156

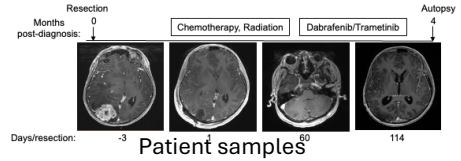


Subbiah *et al*, Nat Med 2023, PMCID:PMC10202803

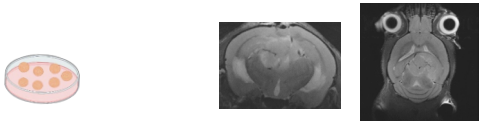
3 Model Development for BRAF V600E-mutant high-grade gliomas

Lerner *et al*. Cancer Res 2015, PMCID:PMC4698003



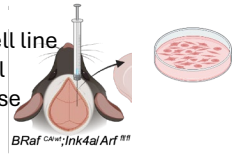


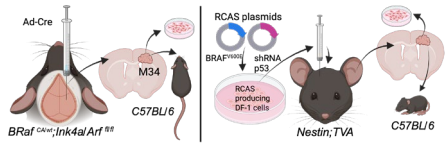
Patient samples



3 patient-derived spheroid lines
+ matched patient-derived xenografts

Patient samples
1 conventional cell line
1 xenograft model
1 orthotopic mouse model





3 syngeneic mouse models-fully immunocompetent

Xing, Panovska et al, Cell Reports Med 2025, PMCID:PMC12208339

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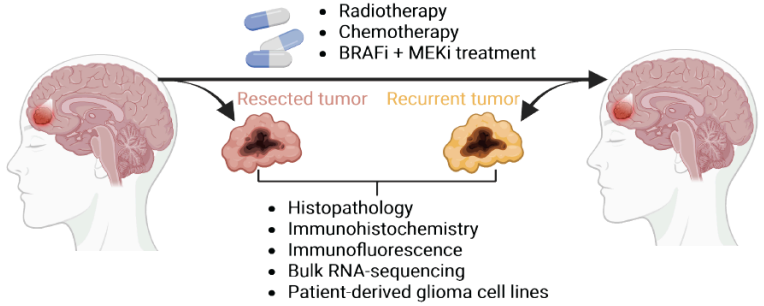
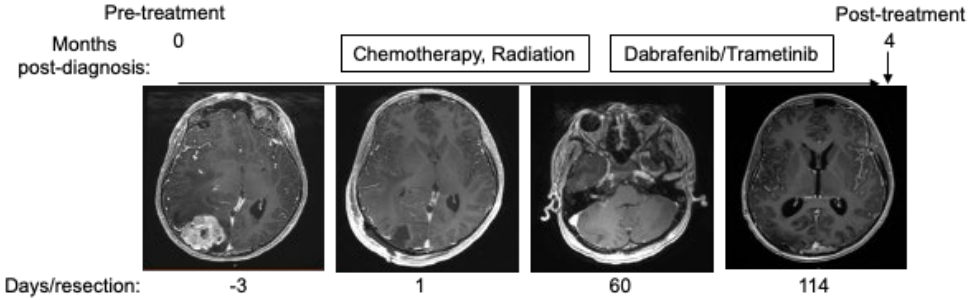
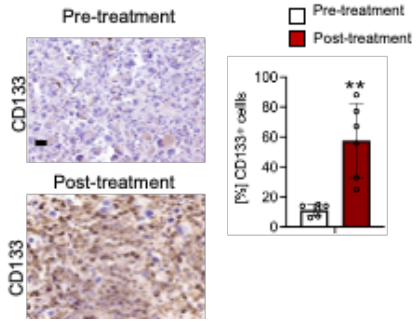
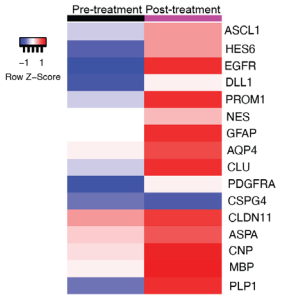
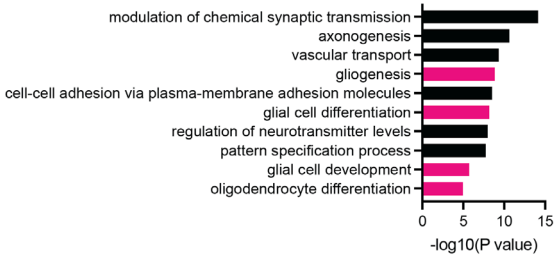
Analyses of Pre- and Post-treatment BRAF V600E-mutant Glioblastoma Samples

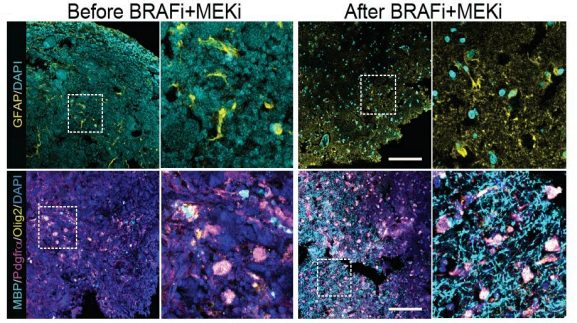


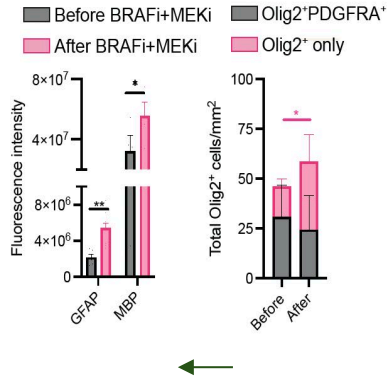












BRAF/MEK inhibition in patients upregulated not only stem-cell markers but also glial differentiation, indicative of therapy-induced increases in cell plasticity and differentiation

Xing, Panovska et al, Cell Reports Med 2025, PMCID:PMC12208339

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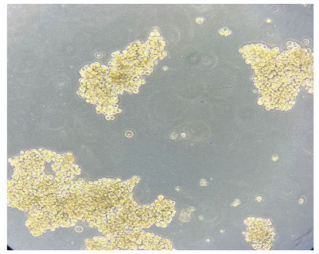
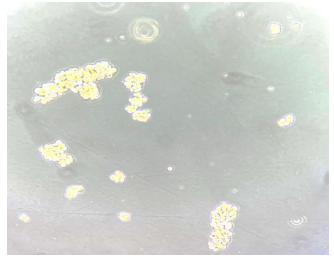
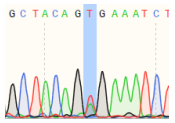
Patient-Derived 3D Models Recapitulate Therapy-Induced Cell State Changes

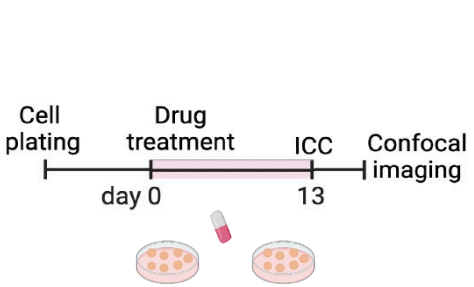
HCM-STAN-1297-C71_A
BRAF V600E

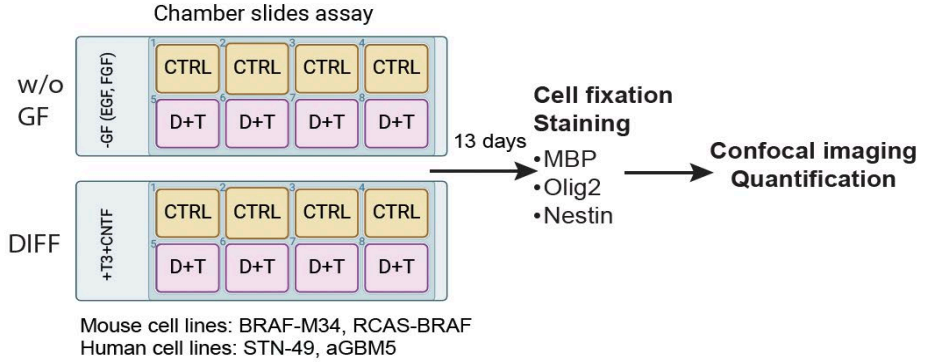
Patient-derived cell line
(PDCL) BRAF V600E #2





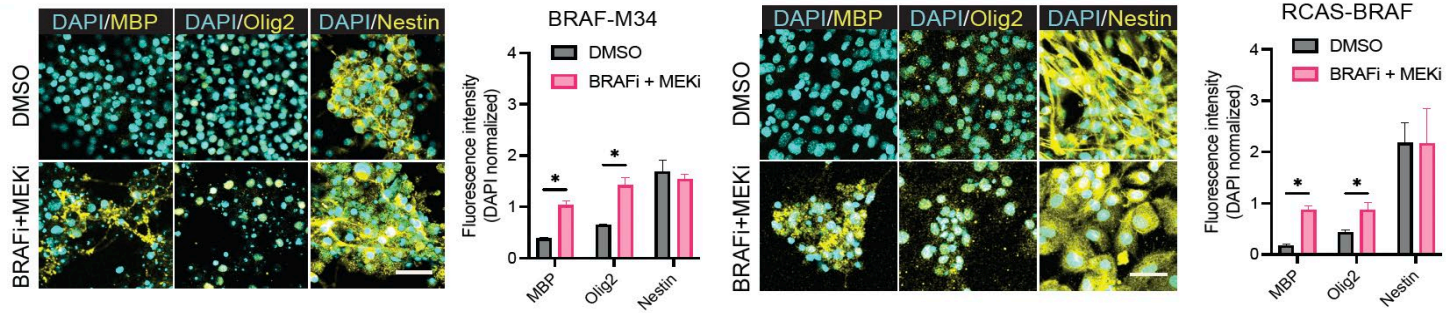


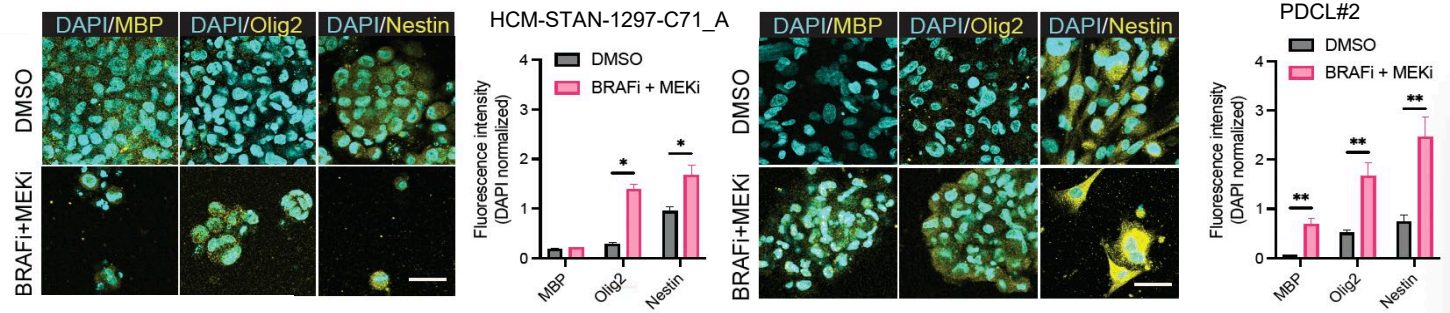




Mouse cell lines: BRAF-M34, RCAS-BRAF
Human cell lines: STN-49, aGBM5

BRAFi+MEKi
Induces
Stem Cells and
Glial Differentiation
States





BRAF/MEK inhibition upregulated not only stem-cell markers but also glial differentiation, in cell culture-based assays, indicative of direct effects on tumor cell plasticity and differentiation

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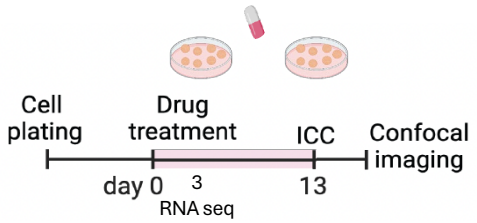
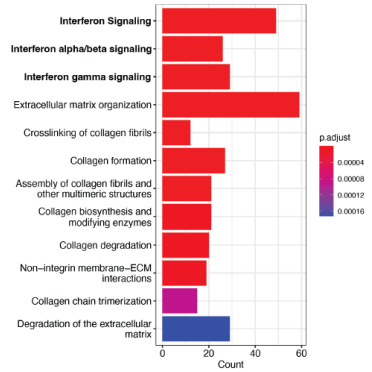
Xing, Panovska et al, Cell Reports Med
2025, PMCID:PMC12208339

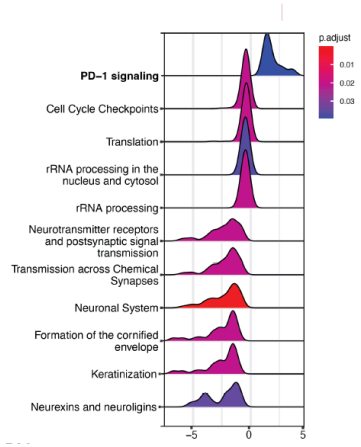
Immune-modulatory Effects of BRAF/MEK inhibitors?

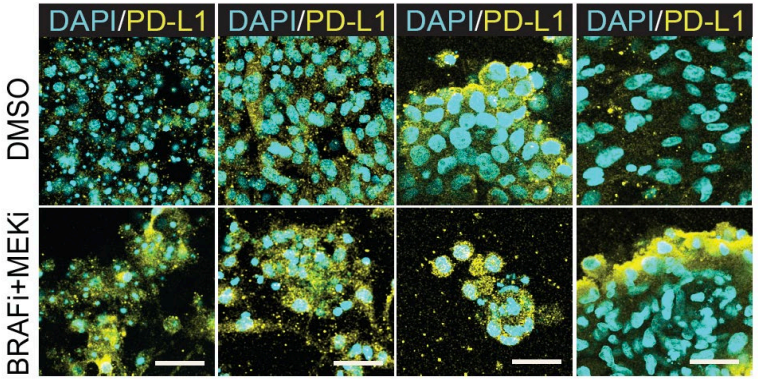


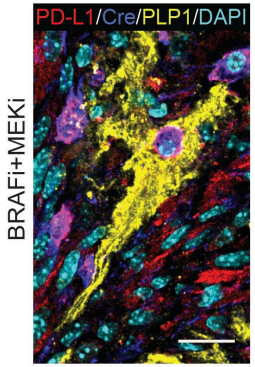
Dabrafenib (DB)=BRAF inhibitor
Trametinib (Tr)=MEK inhibitor

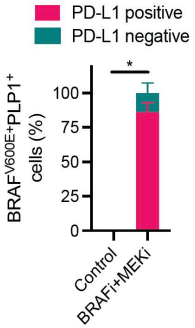
Xing, Panovska et al, Cell Reports Med 2025, PMCID:PMC12208339

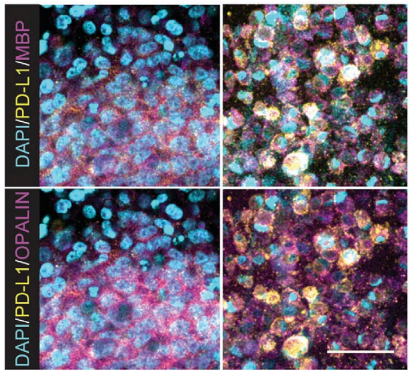


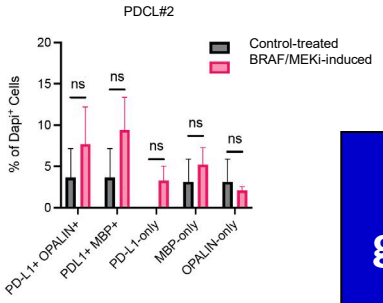




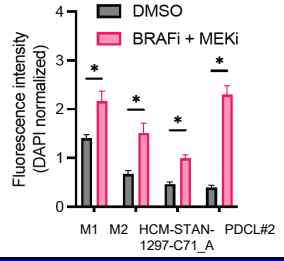








Petritsch Lab, Unpublished , Do not post



BRAF+MEK inhibitors induced **glioma cell differentiation** along with upregulated **antigen presentation** & up-expression of **immune checkpoint inhibitors**

Xing, Panovska et al, Cell Reports Med 2025, PMCID:PMC12208339

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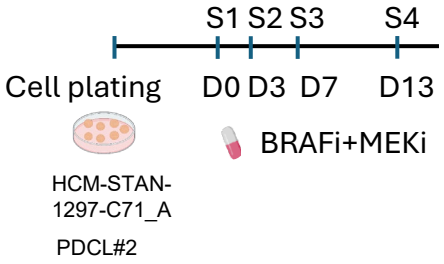
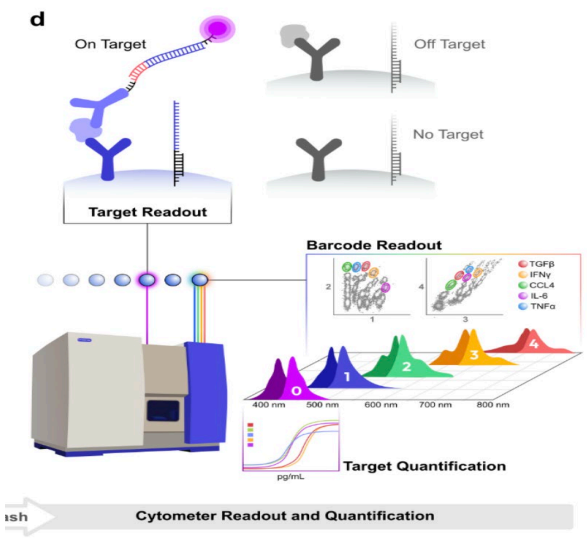
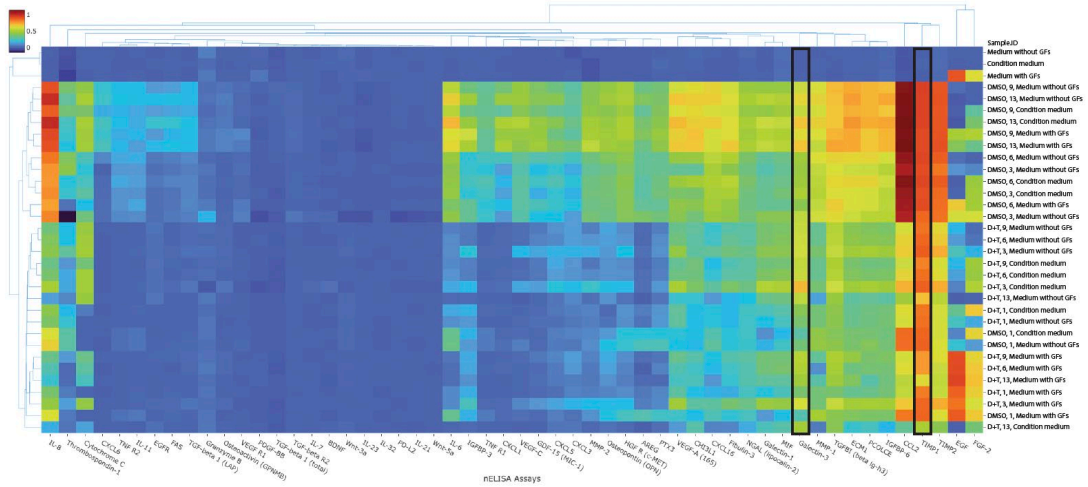
Multi-plex ELISA to Investigate BRAF/MEK inhibitor-Induced Immunoregulation

Nomic - ELISA

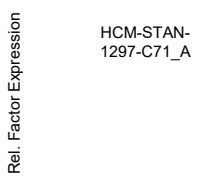


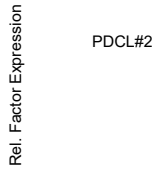


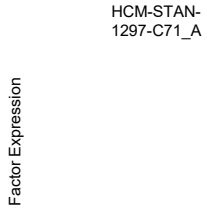


Xing, Panovska et al, Cell Reports Med 2025, PMCID:PMC12208339

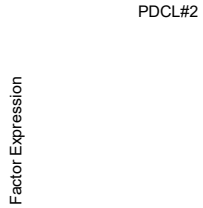
Nomic - ELISA







RNA-seq



BRAF+MEK inhibitors elevate secretion of pro-inflammatory cytokines and T cell inhibitory factors

Summary

MAIN FINDINGS

- BRAF/MEK inhibition induces **glioma cell state** increases differentiation and simultaneously immune evasion
- BRAFi+MEKi activates the interferon response and anti-tumor immunity, while simultaneously **suppressing T cells via PD-L1 upregulation in glial cells**
- Glial differentiation and immune evasion could be mediated by therapy-induced **immune modulatory secretome**
- High PD-L1 expression in BRAF-mutant GBM provides a criterion for anti-PD-1 therapy
- **Concurrent BRAF/MEK and checkpoint inhibition** enhances anti-tumor immunity and survival

CLINICAL IMPLICATION

Our preclinical findings highlight the potential of integrating BRAFi+MEKi treatment with ICI, with emphasis on concurrent treatment

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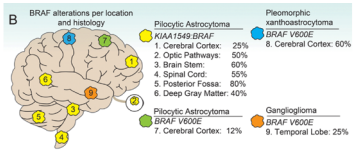
Common Pediatric Brain Tumor Subtypes

>100 subtypes of pediatric solid tumors

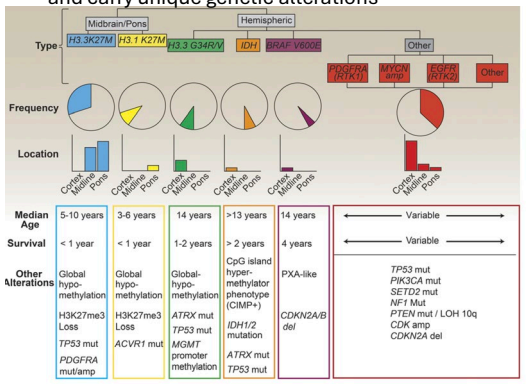
Pfister, SM et al; Cancer Discov. 2022; PMCID:PMC9401511

Subtypes of most common primary pediatric brain tumors



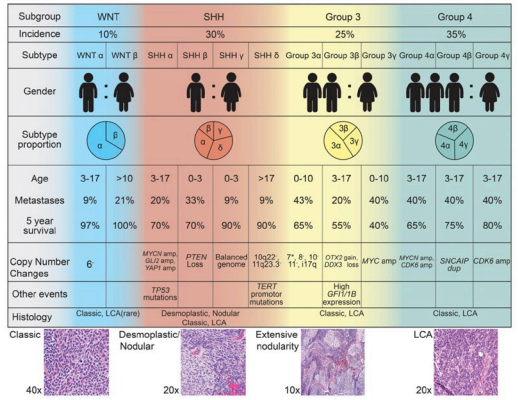
Glioma

Pediatric high-grade glioma are rare but devastating and carry unique genetic alterations

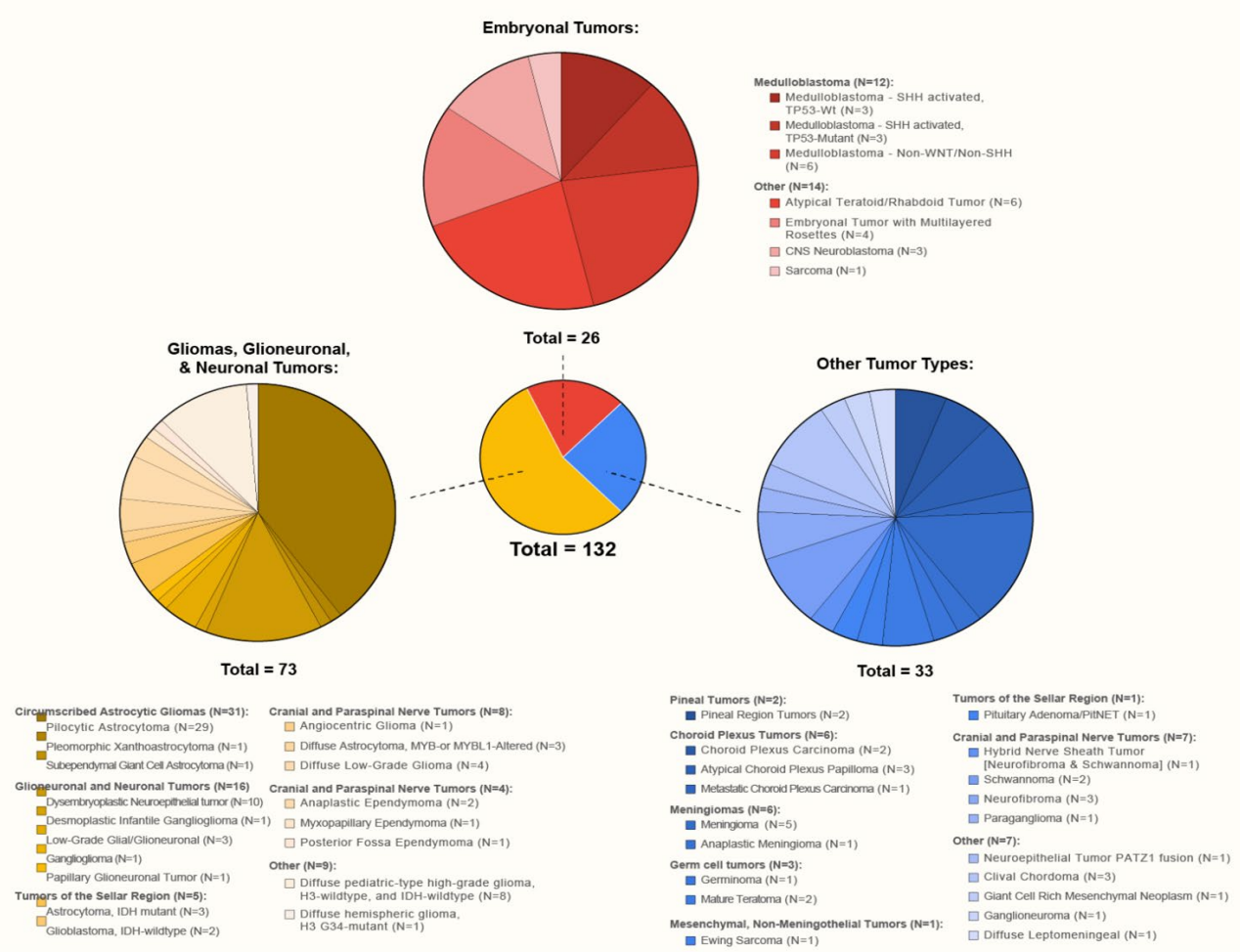


Embryonal Tumors

Medulloblastoma



Representative Cohort of Pediatric Brain Cancer Types at Stanford Pediatrics CMDC



No. of Banked Cases	Tumor Type	Genes/Molecular Profiles Characteristically Altered
3	Astrocytoma, IDH-mutant	IDH1, IDH2, ATRX, TP53, CDKN2A/B
2	Glioblastoma, IDH-wildtype	IDH-wildtype, TERT promoter, chromosomes 7/10, EGFR
2	Diffuse astrocytoma, MYB- or MYBL1-altered	MYB, MYBL1
1	Angiocentric glioma	MYB
4	Diffuse low-grade glioma, MAPK pathway-altered	FGFR1, BRAF
1	Diffuse hemispheric glioma, H3 G34-mutant	H3 G34, TP53, ATRX
9	Diffuse pediatric-type high-grade glioma, H3-wildtype and IDH-wildtype	IDH-wildtype, H3-wildtype, PDGFRA, MYCN, EGFR (methylome)
30	Pilocytic astrocytoma	KIAA1549-BRAF, BRAF, NF1
1	Pleomorphic xanthoastrocytoma	BRAF, CDKN2A/B
1	Subependymal giant cell astrocytoma	TSC1, TSC2
2	Chordoid glioma	PRKCA
1	Ganglion cell tumors	BRAF
10	Dysembryoplastic neuroepithelial tumor	FGFR1
1	Papillary glioneuronal tumor	PRKCA
1	Diffuse leptomeningeal glioneuronal tumor	KIAA1549-BRAF fusion, 1p (methylome)
3	Supratentorial ependymomas	ZFTA, RELA, YAP1, MAML2
1	Posterior fossa ependymomas	H3 K27me3, EZHIP (methylome)
6	Medulloblastoma, SHH-activated	TP53, PTCH1, SUFU, SMO, MYCN, GLI2 (methylome)
6	Medulloblastoma, non-WNT/non-SHH	MYC, MYCN, PRDM6, KDM6A (methylome)
6	Atypical teratoid/rhabdoid tumor	SMARCB1, SMARCA4
5	Embryonal tumor with multilayered rosettes	C19MC, DICER1
3	CNS neuroblastoma, FOXR2-activated	FOXR2
6	Meningiomas	NF2, AKT1, TRAF7, SMO, PIK3CA; KLF4, SMARCE1, BAP1 in subtypes; H3K27me3; TERT promoter, CDKN2A/B in CNS WHO grade 3

- ~90% of cases yield tissue for research
- 45 subtypes of brain cancer and 12 subtypes of non-CNS cancers captured over a 4-year period
- Longitudinal collection is crucial to capture diverse, rare cancers

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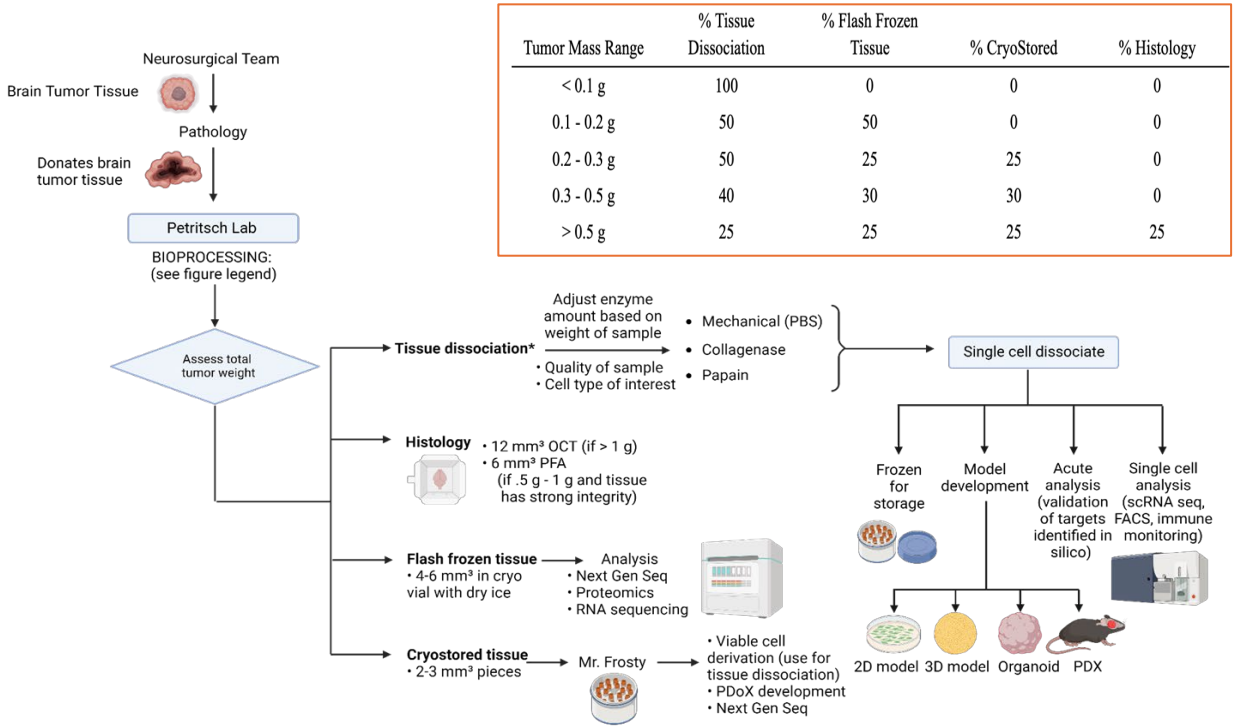
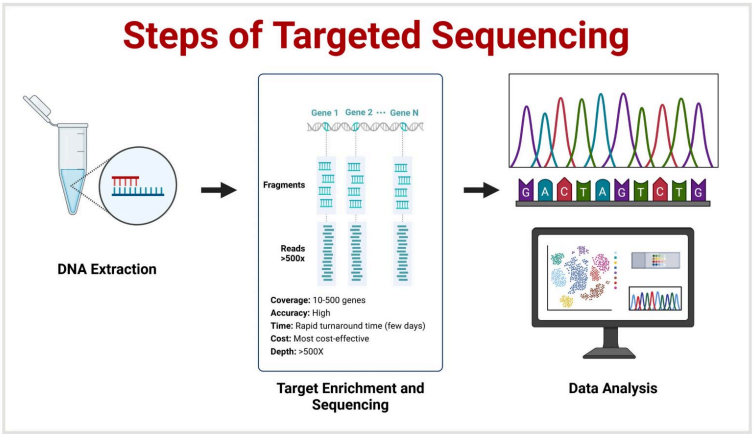
Pediatric Cancer Model Development - Workflow

Established Bioprocessing Workflow in our laboratory



Tumor Mass Range	% Tissue Dissociation	% Flash Frozen Tissue	% CryoStored	% Histology
< 0.1 g	100	0	0	0
0.1 - 0.2 g	50	50	0	0
0.2 - 0.3 g	50	25	25	0
0.3 - 0.5 g	40	30	30	0
> 0.5 g	25	25	25	25

Established Mutation Identification



Stanford Actionable Mutation Panel ~250 genes, UCSF 500 Caris (WES)

An established biobanking workflow with standardized targeted sequencing of patient Tumors to identify recurrent mutations, including oncofusions

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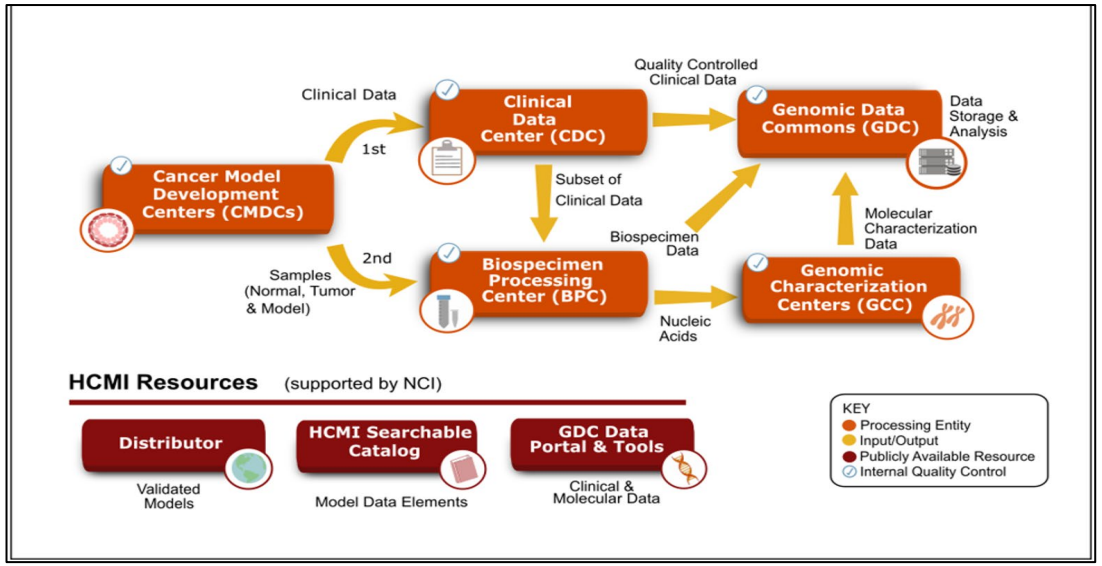
Pediatric Cancer Types for HCMI

- **Pediatric central nervous system (CNS) solid tumors**
  - Diffuse Midline Glioma (DMG), H3-K27-mutant
  - Diffuse High-Grade Glioma (HGG), MAPK activated
  - HGG, H3 and IDH wildtype
  - Glioblastoma
  - Anaplastic Astrocytoma, BRAF V600E-mutant
  - Astrocytoma, IDH-mutant
  - Medulloblastoma, ATRT
  - Ependymoma
  - Ganglioneuroblastoma
  - ETMR
  - Choroid plexus carcinoma
  - Pineoblastoma
- Non-CNS pediatric solid tumors
  - **Malignant Rare Soft Tissue and Bone sarcoma**
  - **Wilms' Tumor (Primary/Metastasis Pair**
  - **Neuroblastoma**
  - Hepatoblastoma

60-70% success rate for model development
27 subtypes of brain cancer and 4 of non-CNS cancer were captured in models
WES/WGS/RNAseq/DNA Methylation



(Source: https://ocg.cancer.gov/programs/hcmi/nci-cancer-model-development)

Planning and management oversight by the team at Frederick National Laboratory for Cancer Research, Leidos Biomedical Research, Inc.
This project has been funded in part with federal funds from the Childhood Cancer Data Initiative (CCDI), National Cancer Institute, National Institutes of Health, Task Order numbers 75N91020F00035, under contract no. 75N91019D00024.

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Poster: Apr 21 @2PM_PO.TB05.02 Pediatric Cancer Models_Section 30 6171/30

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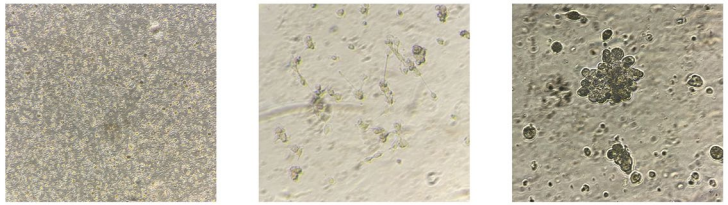
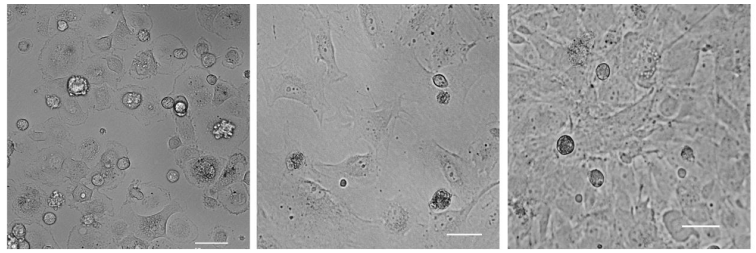
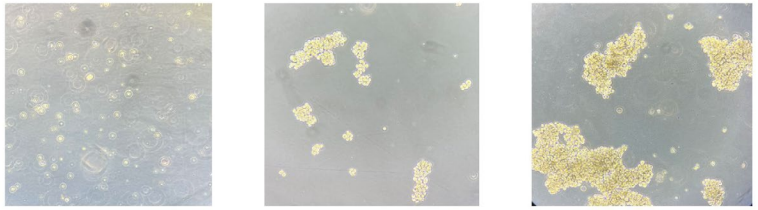
Examples next-generation models of pediatric solid cancer



Morphology of a Giant cell tumor line HCM-STAN-1408-C71. Morphology. **left.** 2 h after thawing; **center.** at low density; **right.** at high-density.

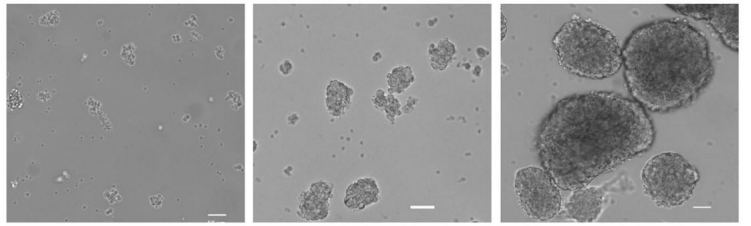


Morphology of a pediatric diffuse anaplastic Wilms tumor 2D adherent cell line HCM-STAN-1353-C64. (A). Morphology. **A**. 2 h after thawing; **B**. at low density; **C**. at high density. Scale bars are 50 µM.



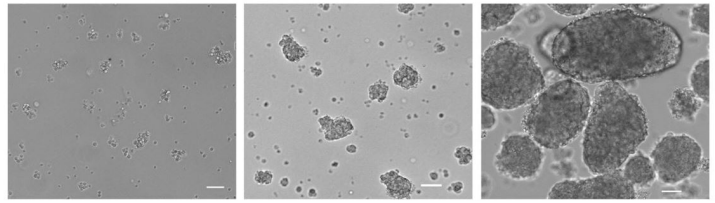
Morphology of an epithelioid glioblastoma tumor 3D spheroid cell line HCM-STAN-1297-C71. Morphology. **left**. 2 h after thawing; **center**. at low density; **right**. at high density.

HCM-STAN-1351-C71-A



Morphology of Neuroblastoma 3D spheroid cell lines generated from bone marrow infiltrated tumor cells of a pediatric MYCN-amplified neuroblastoma patient at diagnosis (HCM-STAN-1351-C71-A) left. 2 h after thawing**; center.** at low density; **right**. at high density

HCM-STAN-1351-C71-B



Morphology of Neuroblastoma 3D spheroid cell lines generated from bone marrow infiltrated tumor cells of a pediatric MYCN-amplified neuroblastoma patient at relapse (HCM-STAN-1351-C71-B). left. 2 h after thaw; **center**. at low density; **right.** at high density. Scale bars are 50 µM.

Morphology of diffuse intrinsic pontine glioma 3D spheroid cell line HCM-STAN-1420-C71. **A**. 2 h after thawing; **B**. at low density; **C**. at high density. Scale bars are 5000 µM.

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Stanford Pediatric CMDC Team and Partners _Acknowledgements

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The HCMI Network

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Stanford Cancer Institute
NCI Designated Comprehensive Cancer Center

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Poster: Apr 22 @9A_PO.MCB04.01._Section 22 7297/9

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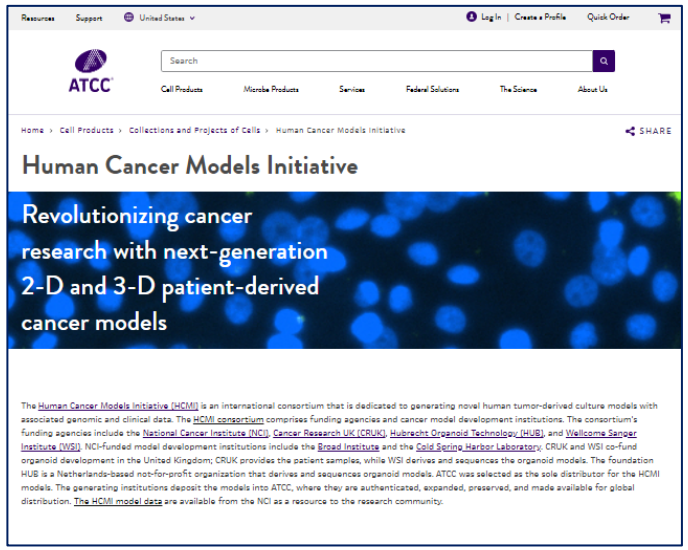
Resources to learn more about ATCC and the HCMI

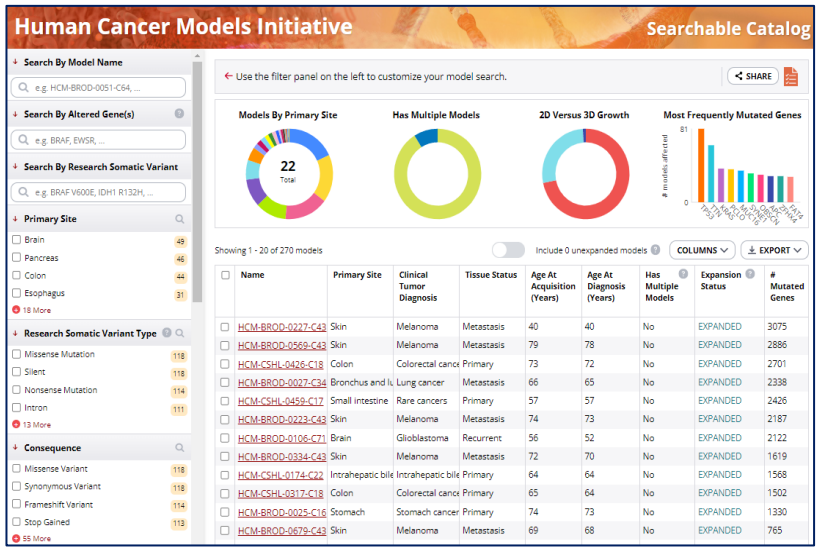
Browse and search unreleased HCMI models at ATCC

- Use the “Submit your Input” button on the HCMI Landing page

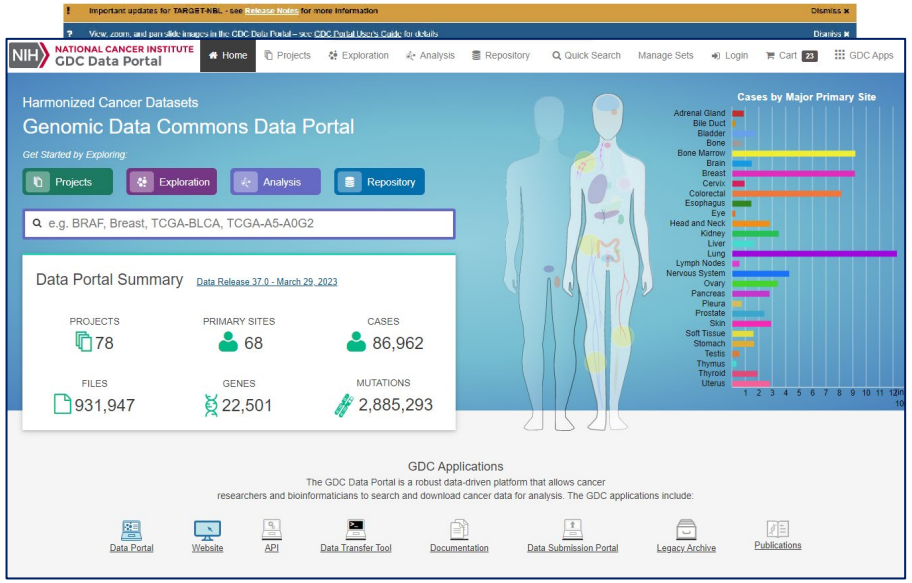
www.atcc.org/hcmi-input



HCMI Landing page
atcc.org/hcmi



HCMI Searchable Catalog
https://hcmi-searchable catalog.nci.nih.gov



NCI Genomic Data Commons
https://portal.gdc.cancer.gov/projects/HCMI-CMDC

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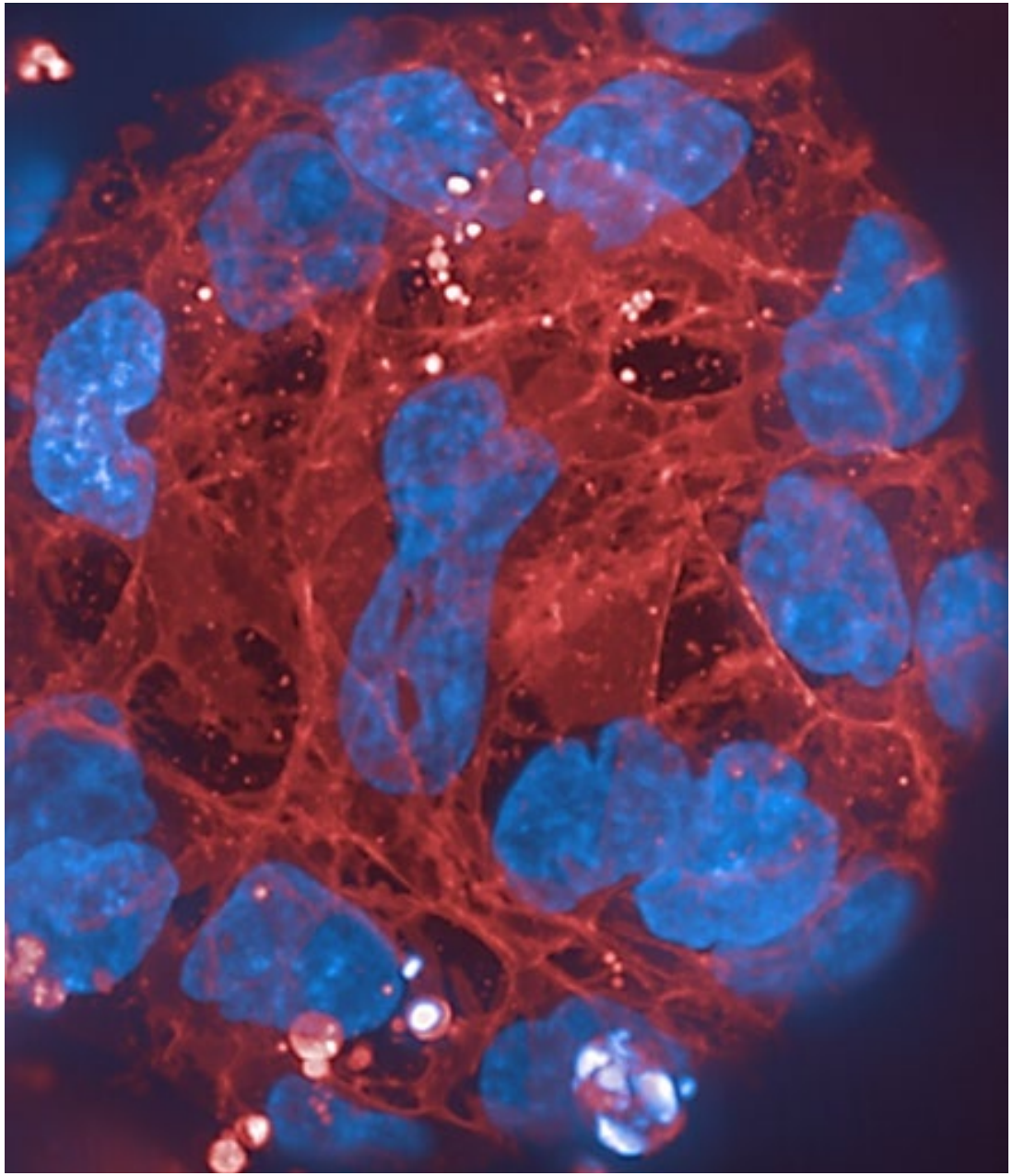

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Patient derived tumor organoid for therapeutic modeling in cancer

Benjamin D Hopkins, PhD

Director Tumor Organoid Platform
Englander Institute
for Precision Medicine

Disclosure Information

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Englander Institute
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Benjamin D. Hopkins

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Stockholder in: Faeth Therapeutics/Sensei Biotherapeutics

Organoid Pipeline Entry Points

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EIPM Organoid Pipeline:

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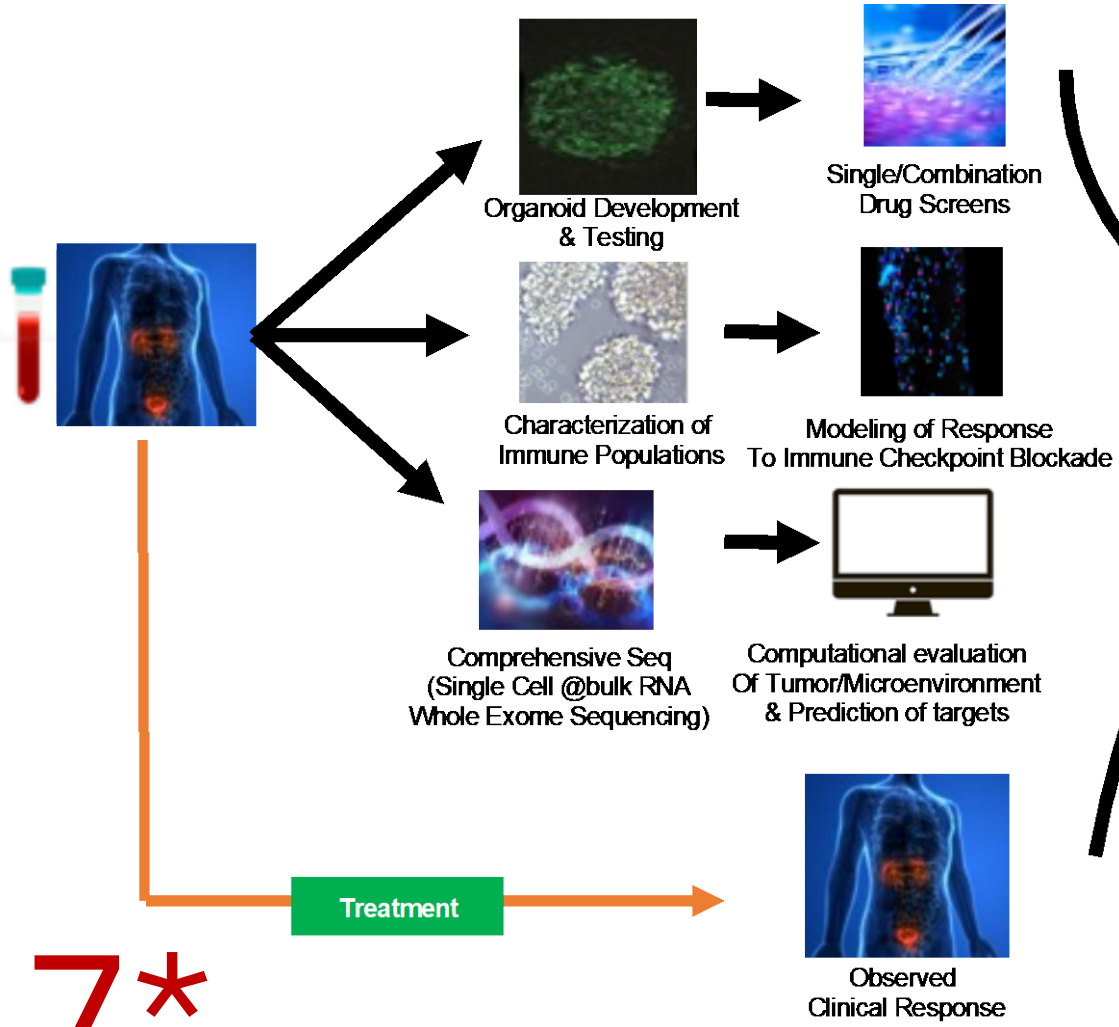

Targeted
Clinical Interventions

~17*

people/case

Starting Point: EXaCT1 (WES)

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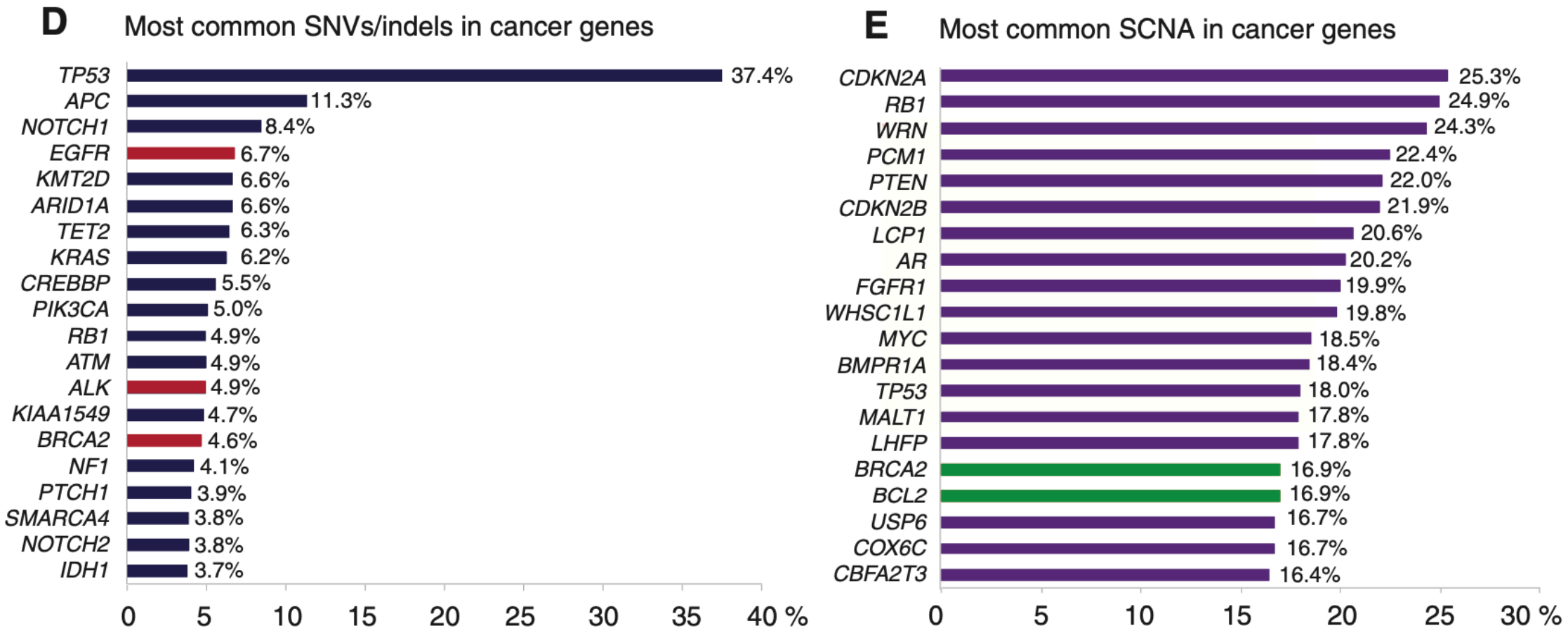

Pauli et al, Cancer Discover 2017
PMID: 28331002

Therapeutic Options based on WES

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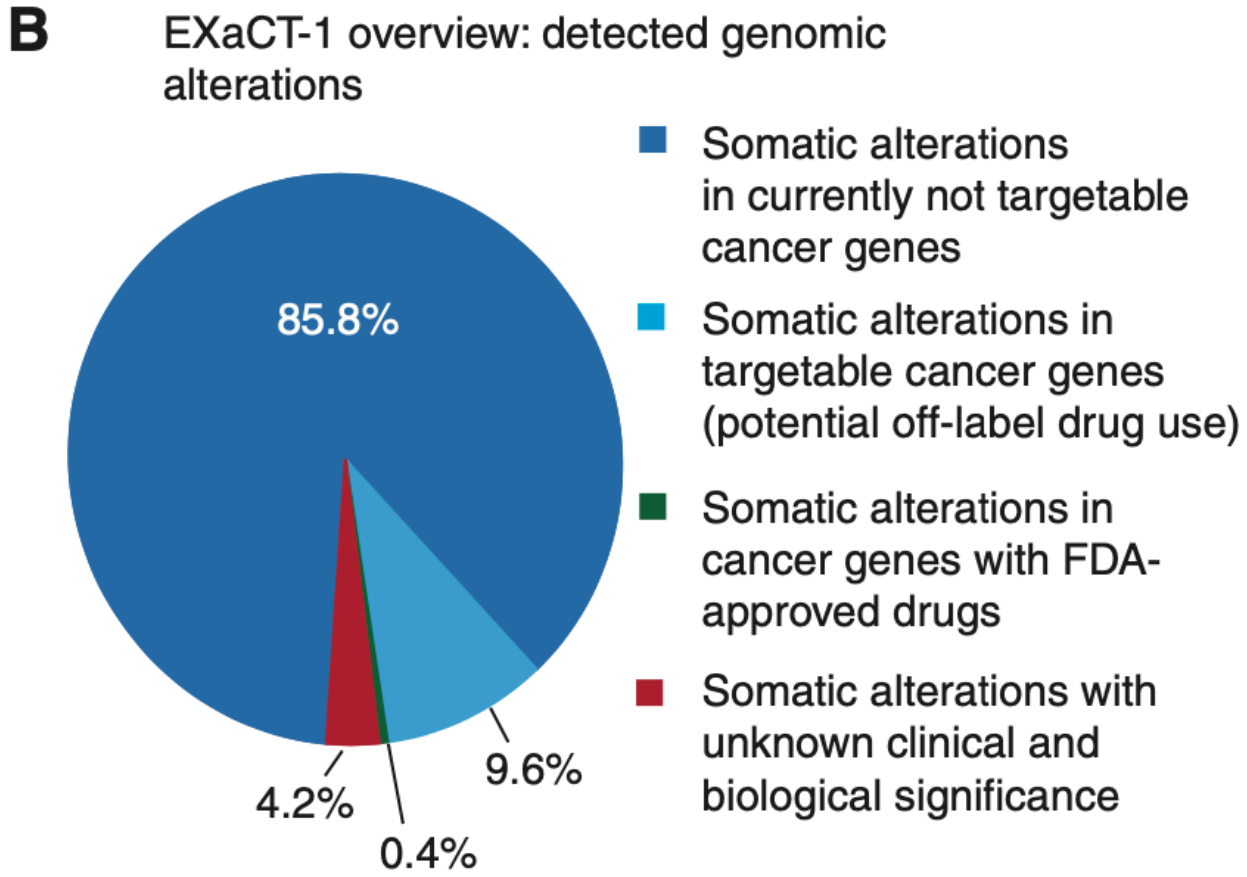

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PMID: 28331002

Patient Derived Organoids

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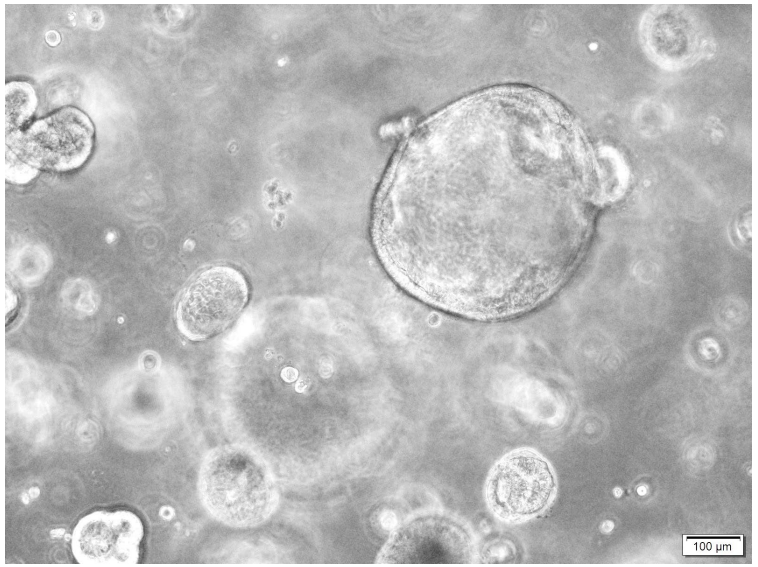
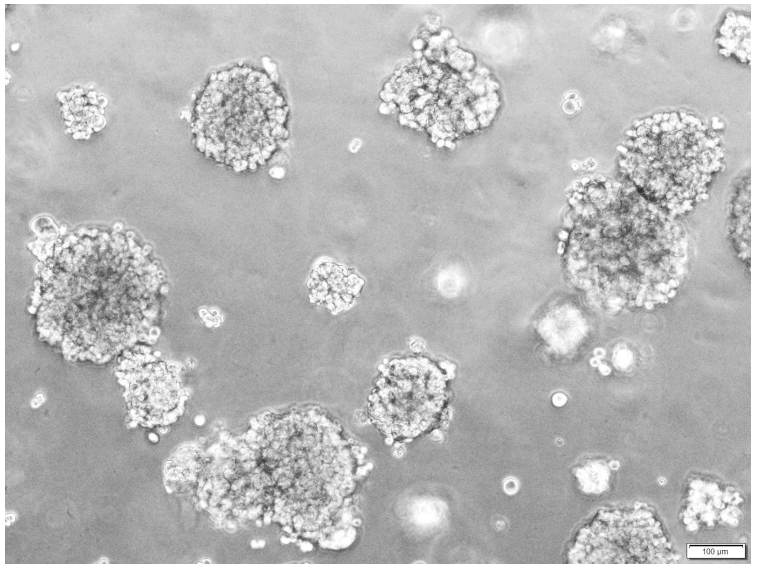
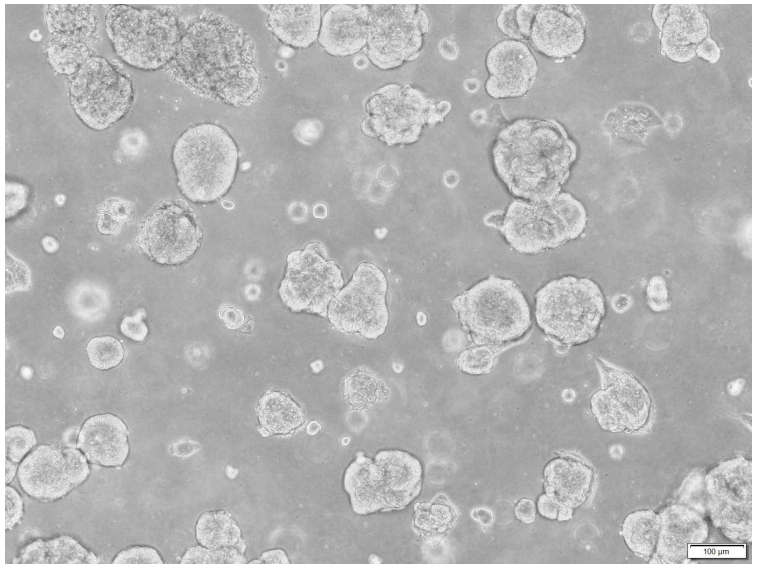
Bladder Tumors



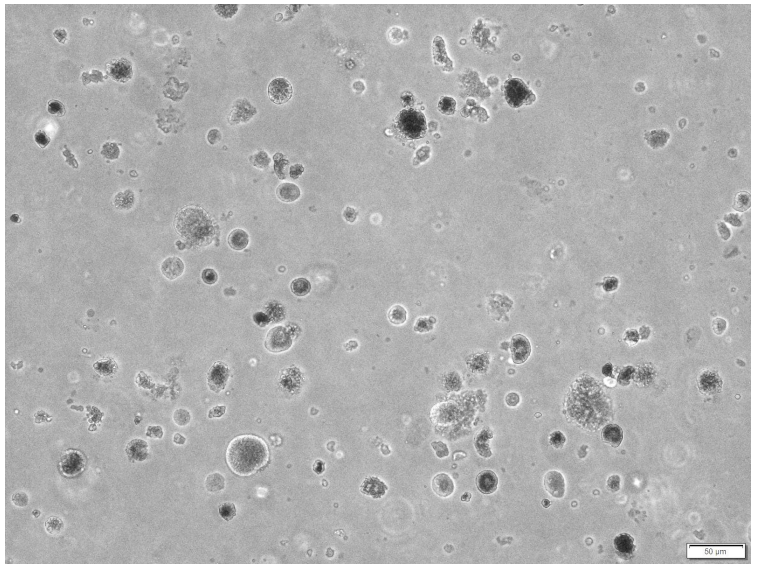
Stomach Tumors



Kidney Tumors



Melanoma Tumors



Pathology of Organoid Models

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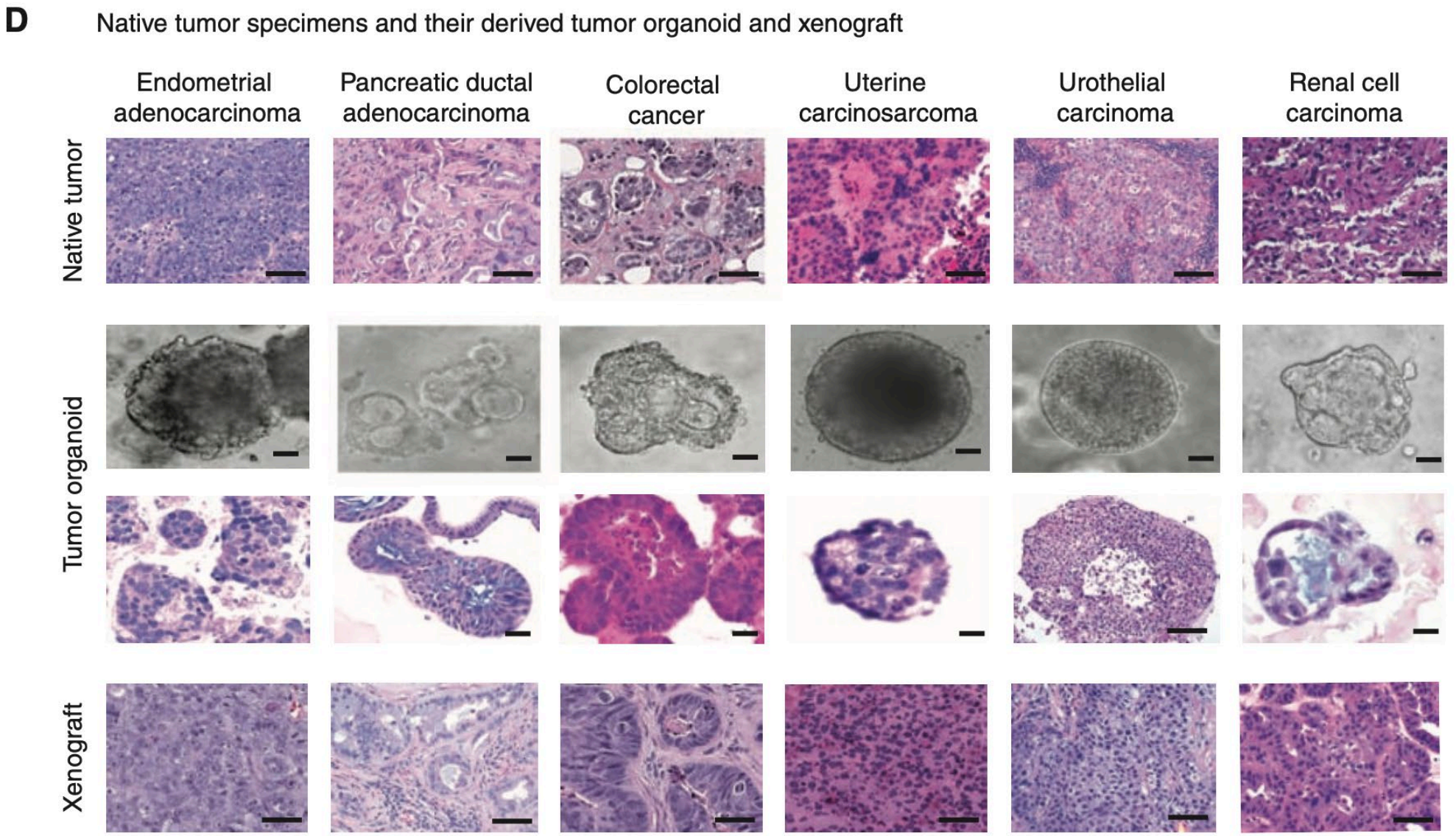

Pauli et. al. Cancer Discovery 2017

Genomic Fidelity of Models:

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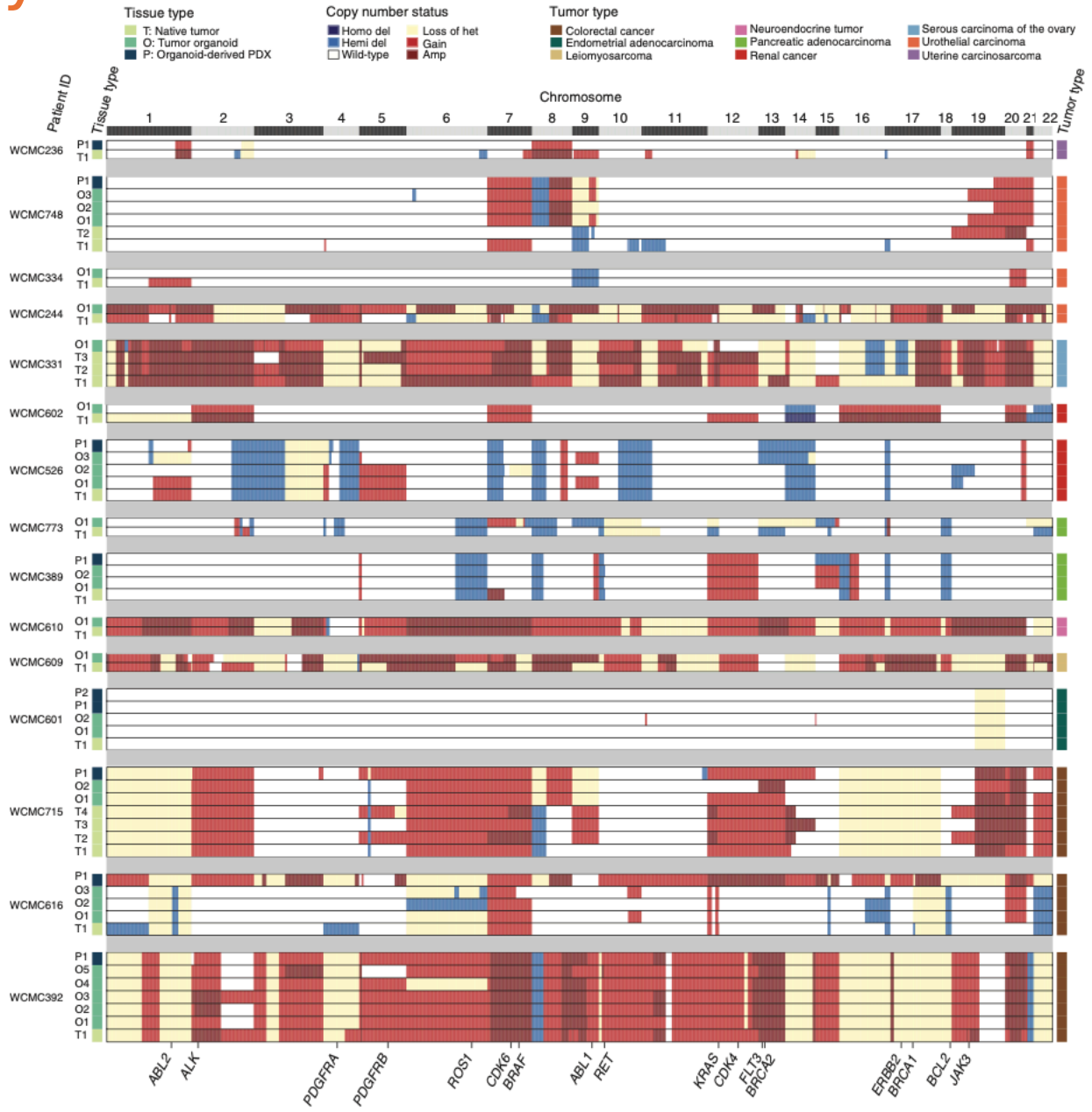

Pauli et al, Cancer Discover 2017
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Pipeline Questions

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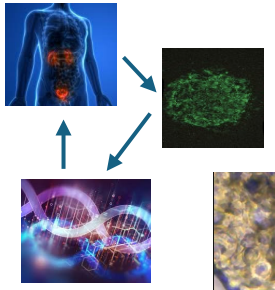
What is the best drug for the patient?

What is the best patient for each drug?



Automated Drug Screening

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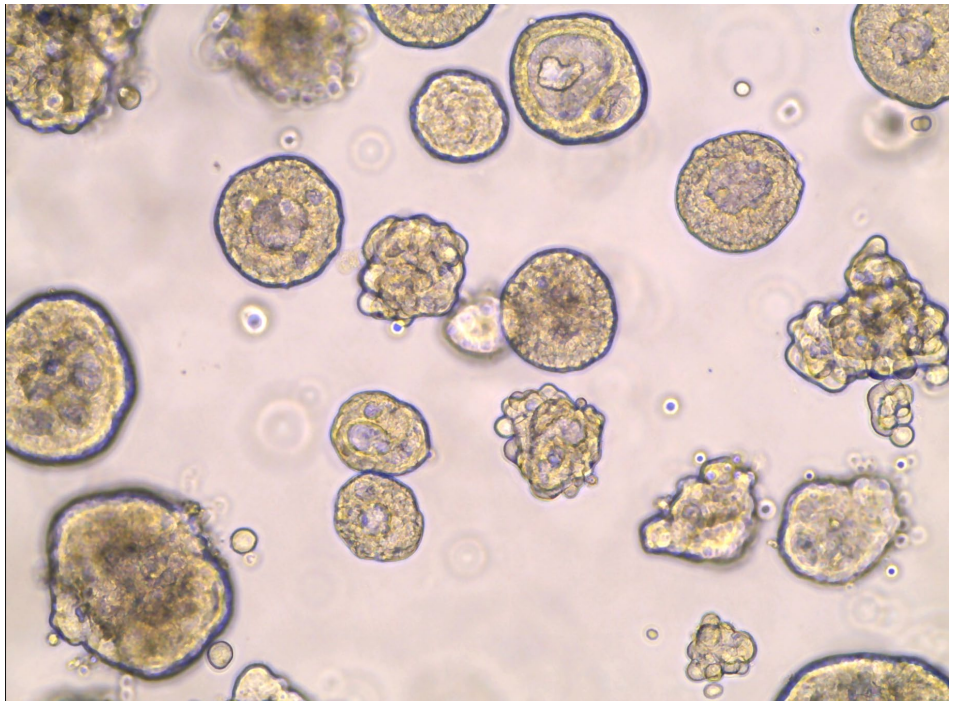

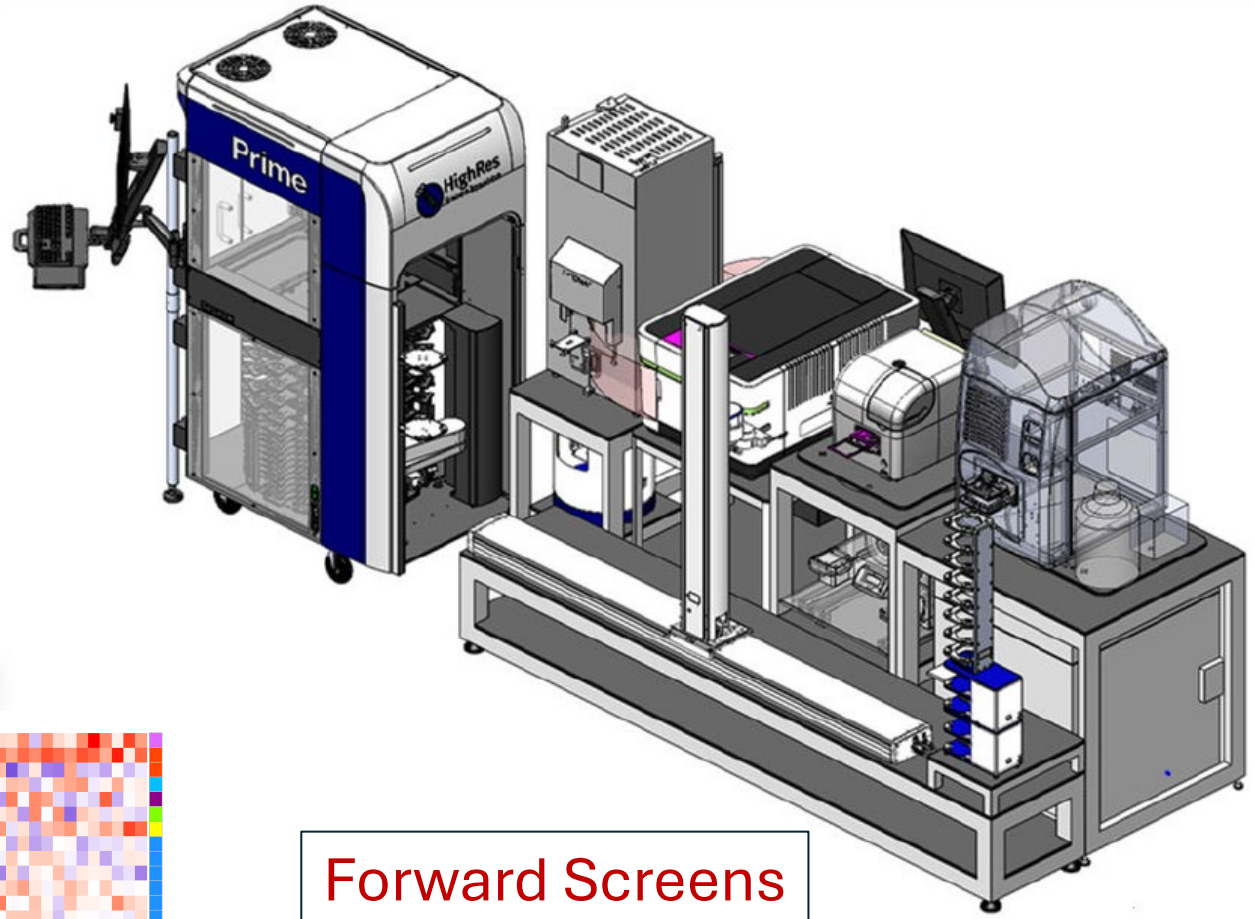

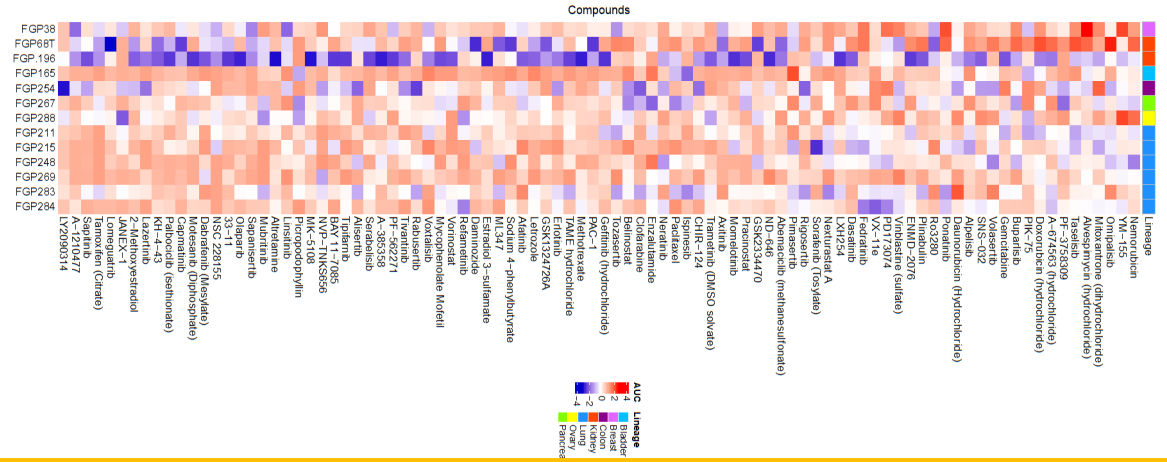

Forward Screens
Inverse Screens

Identification of Tumor Specific Drug Sensitivities

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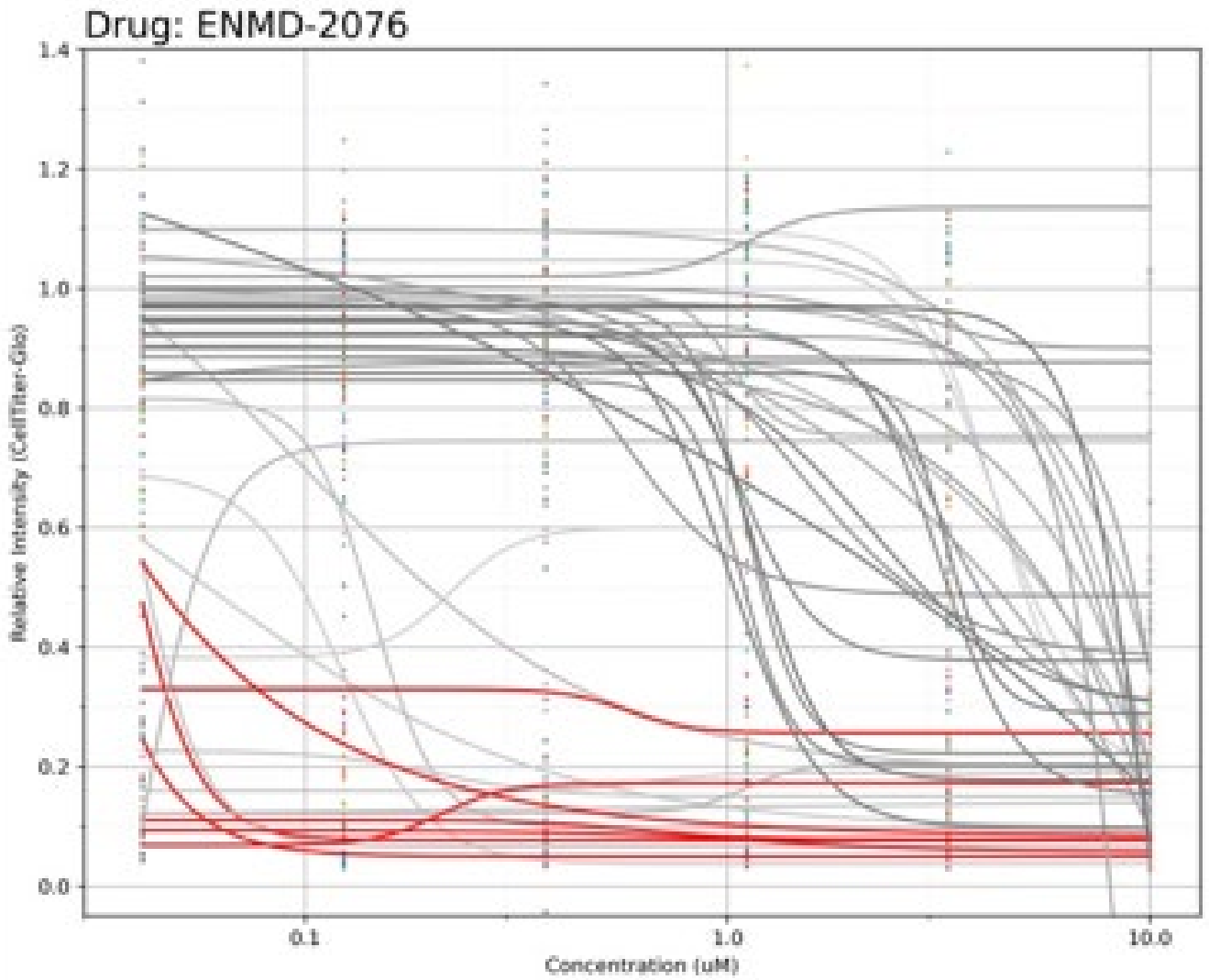

Co-Clinical Modeling

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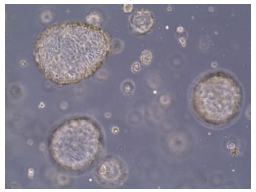

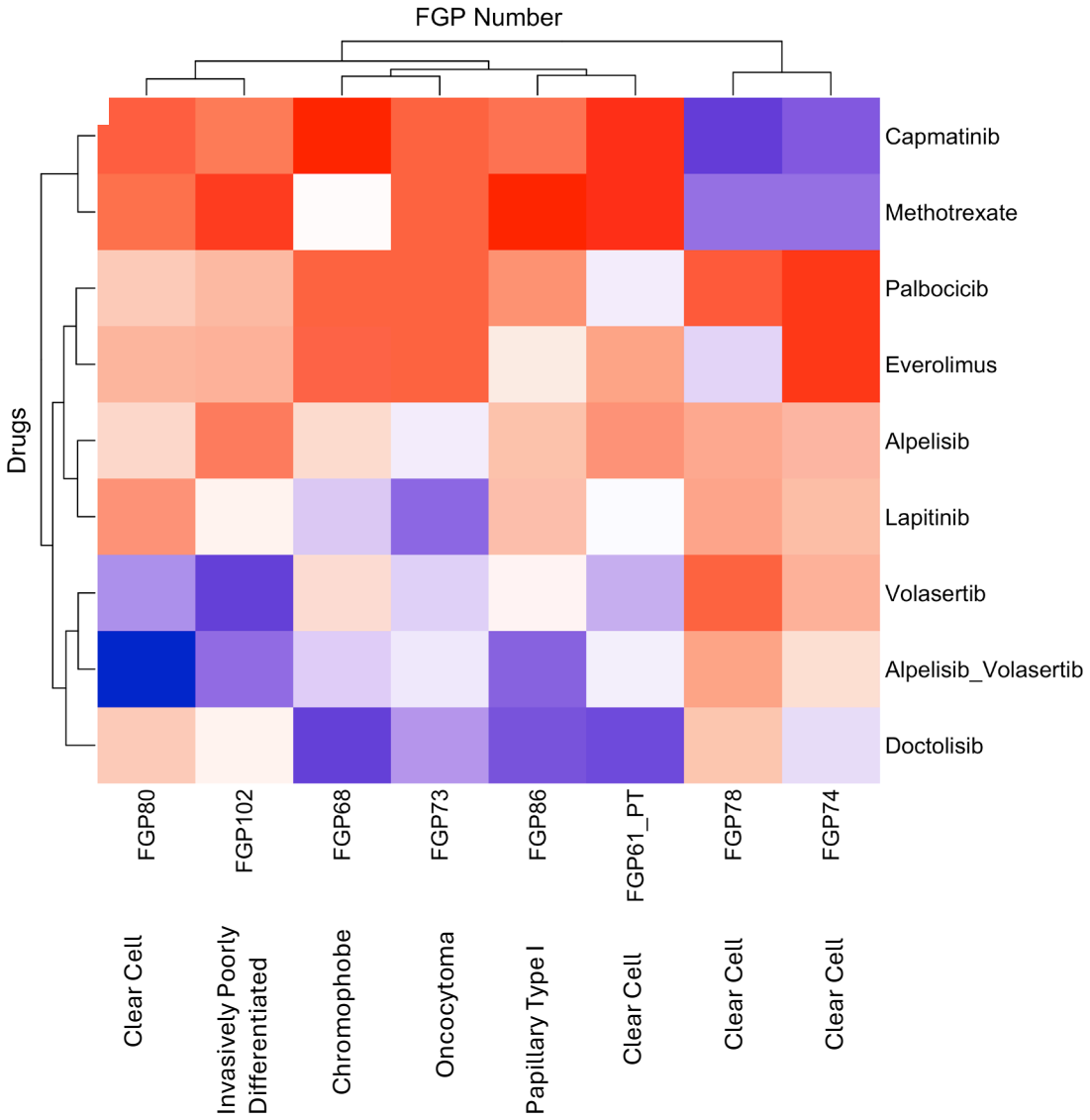

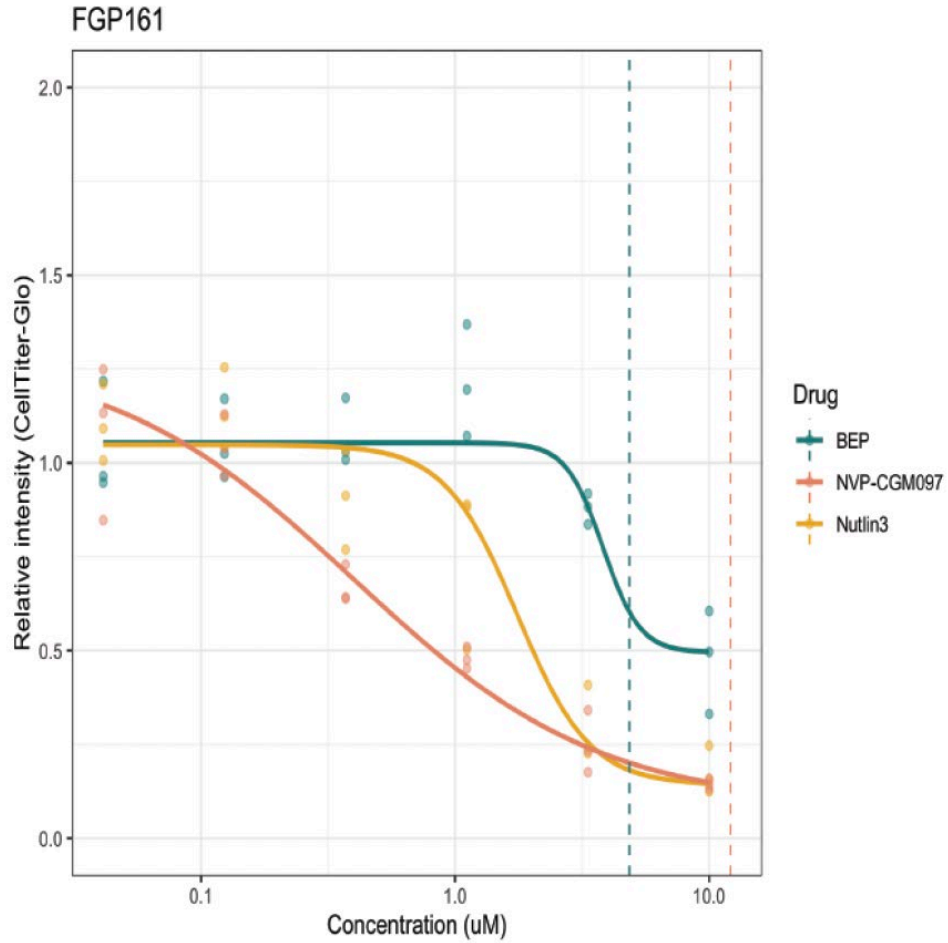

Inverse Screening

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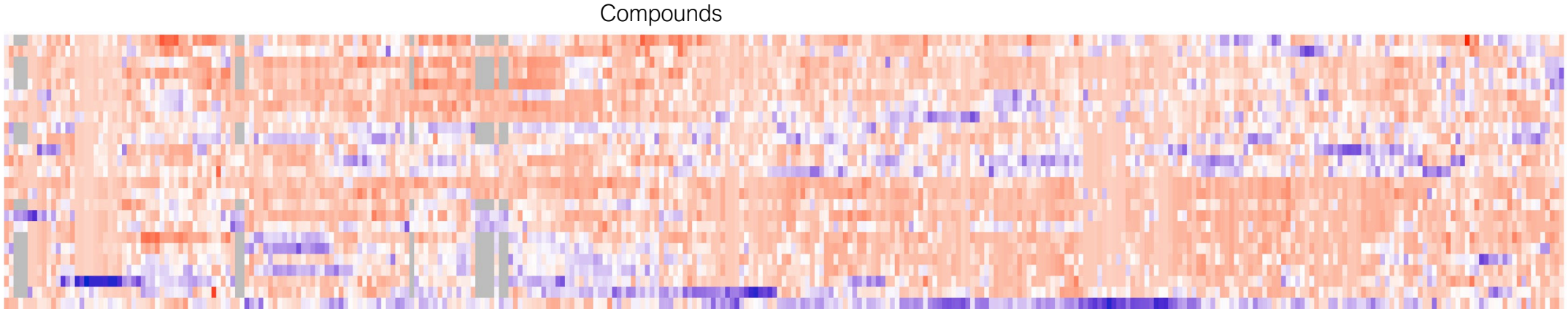
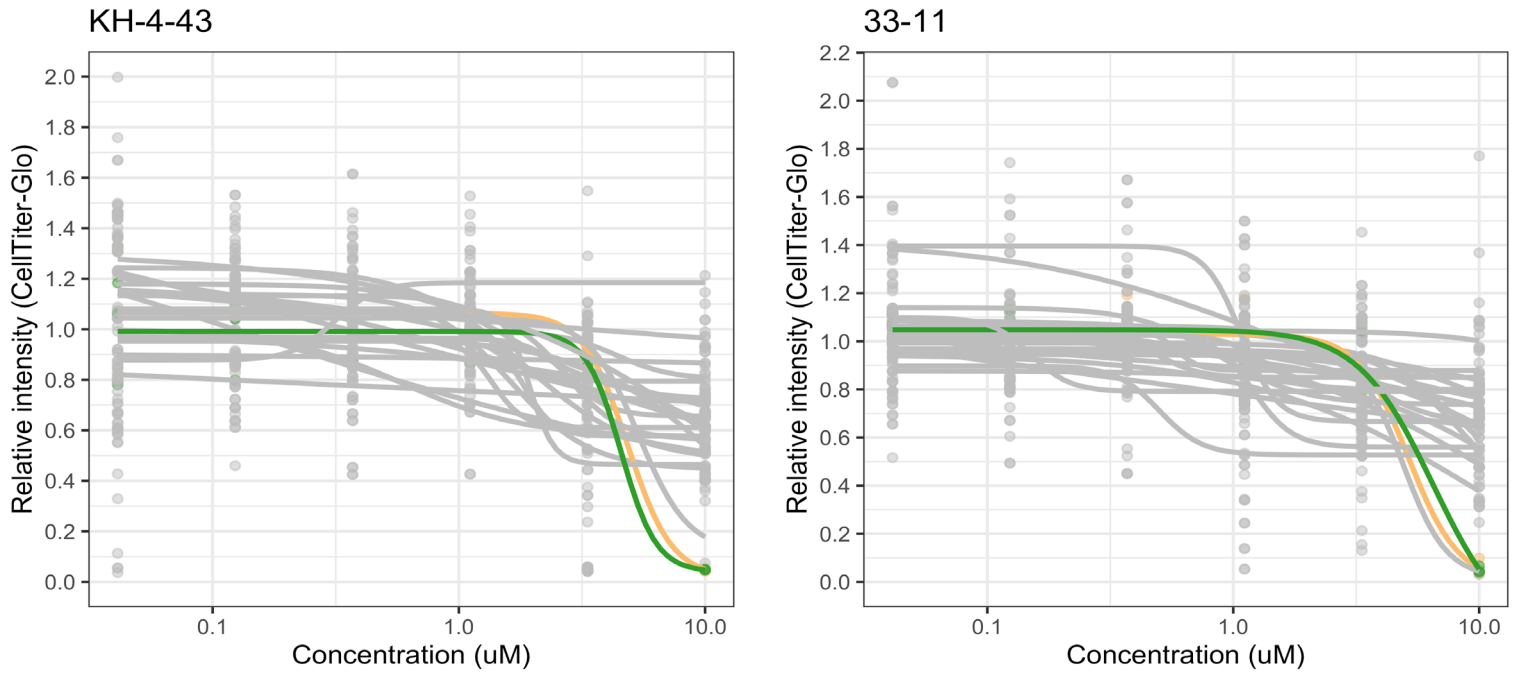
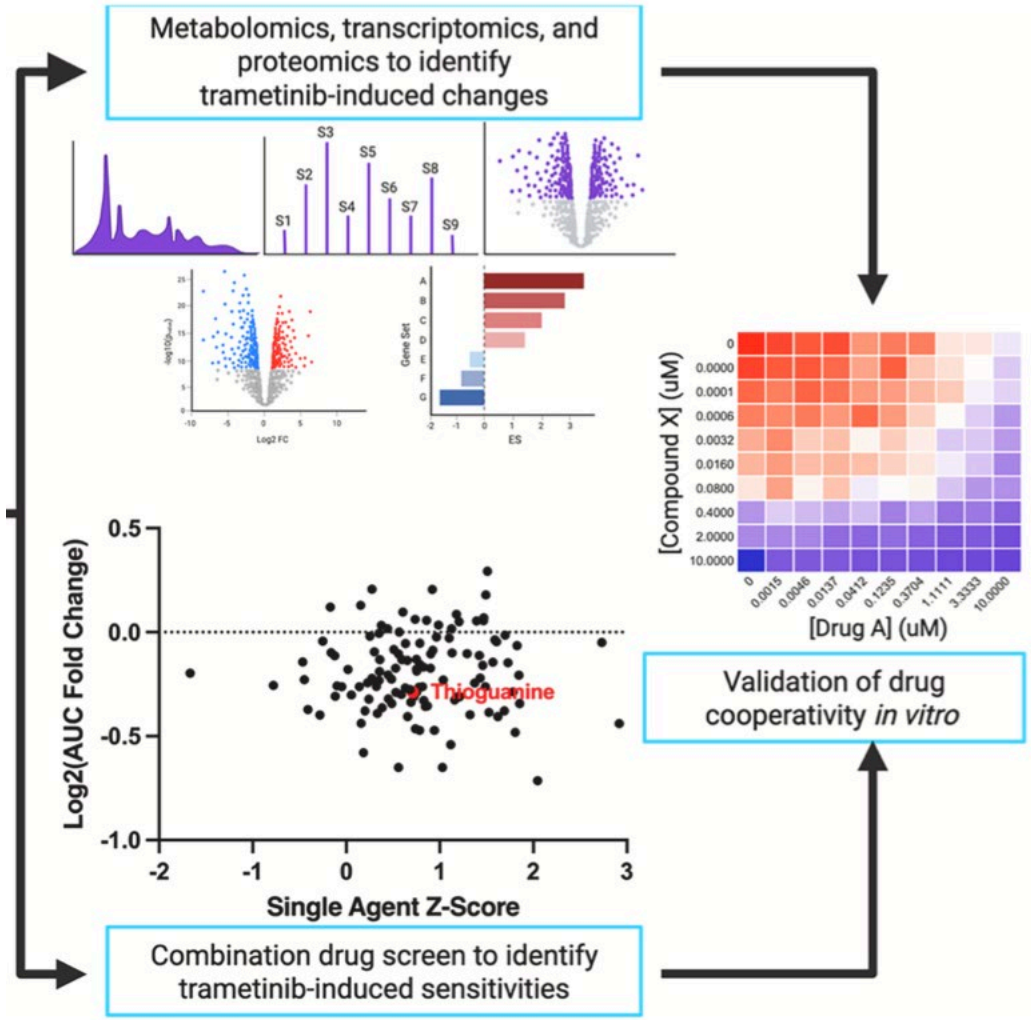




Claridge et al 2025

Organoids facilitate the integration of multi-omics with functional modeling providing platform to explore the effects of combinations in clinically representative models.



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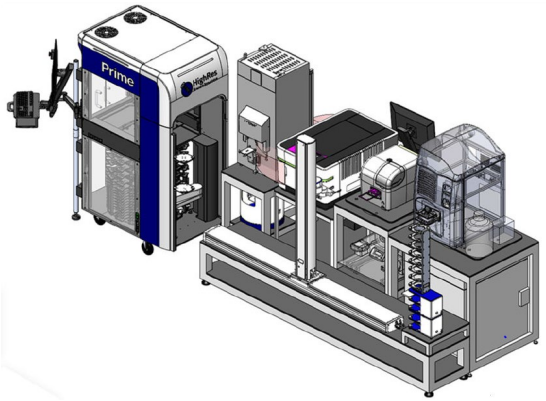

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Rachel Brody
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Organoid biobank

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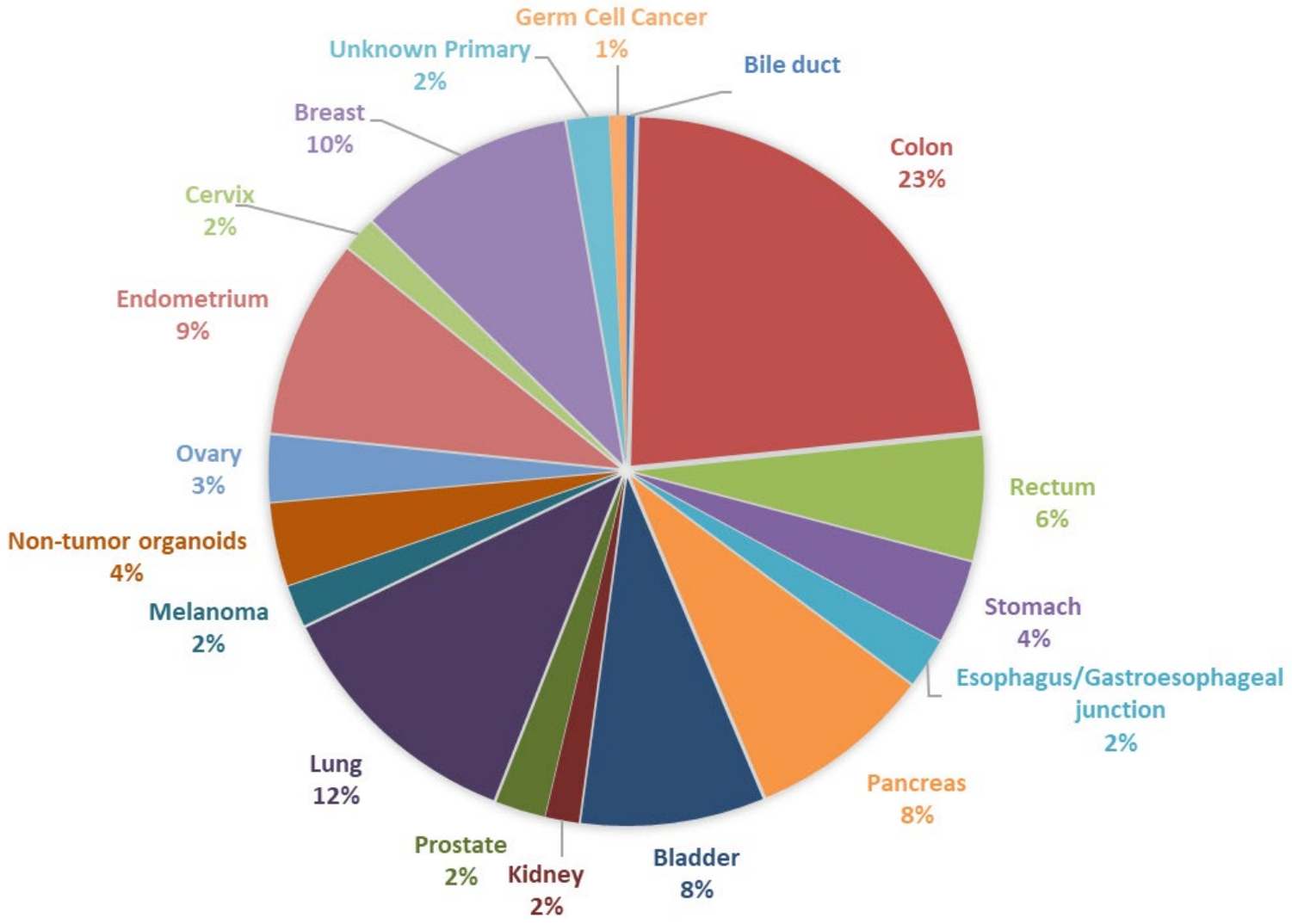

Model accessibility

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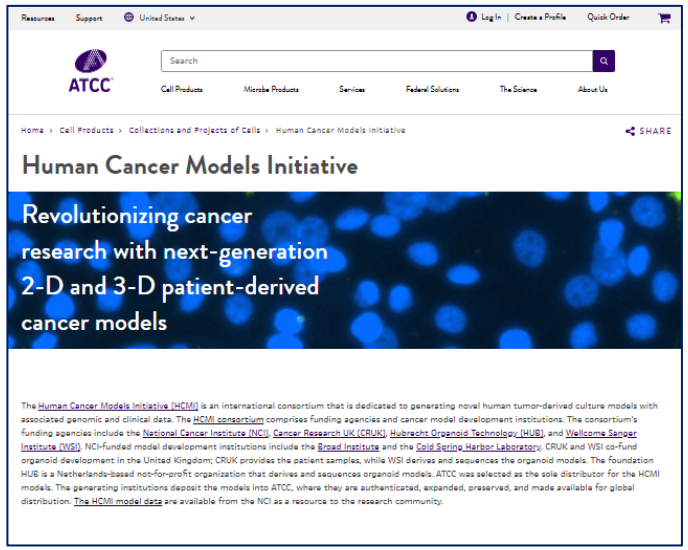
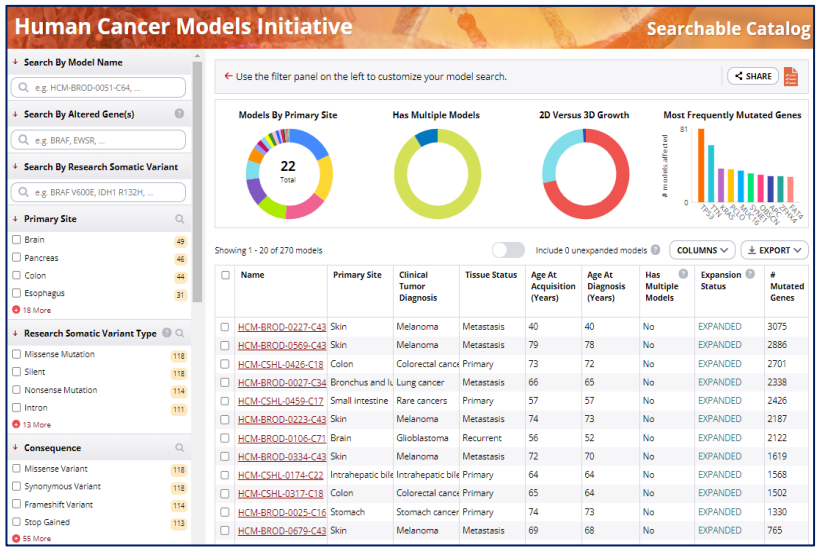
- Use the “Submit your Input” button on the HCMI Landing page

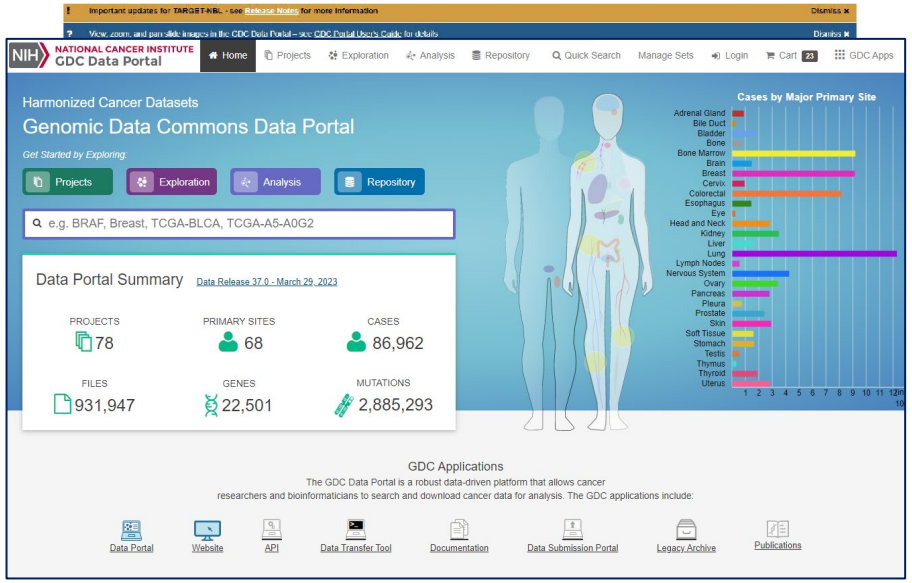
www.atcc.org/hcmi-input



HCMI Landing page
atcc.org/hcmi



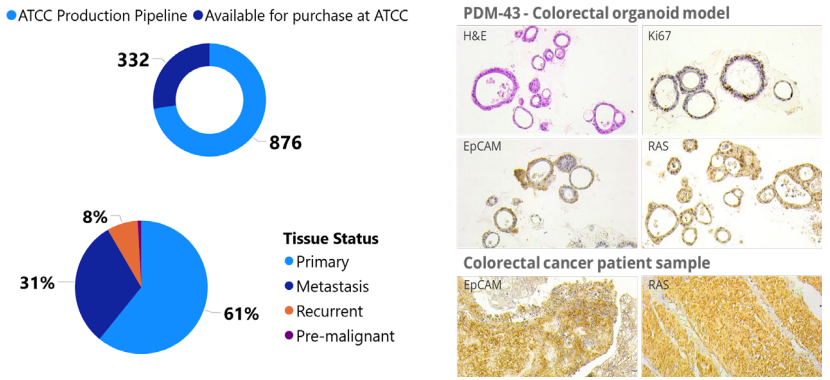
HCMI Searchable Catalog
https://hcmi-searchable catalog.nci.nih.gov



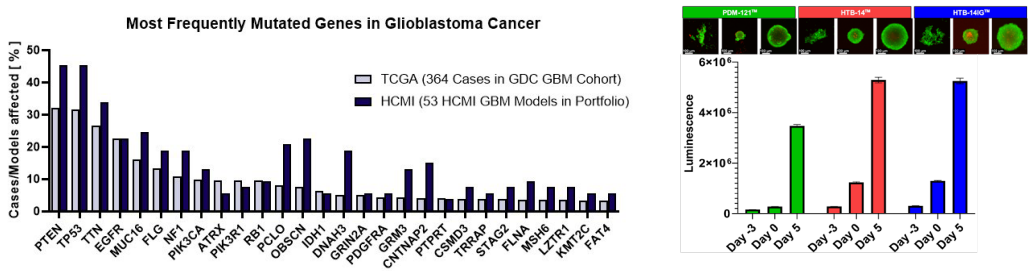
NCI Genomic Data Commons
https://portal.gdc.cancer.gov/projects/HCMI-CMDC

Posters

ATCC's patient-derived 2-D & 3-D cancer models make translational oncology a reality for the scientific community
4/20/2026 2:00:00 PM
Location: Poster Section 28
Poster Board Number: 10
Presentation Number: 3405



Patient-derived pediatric glioblastoma models provide key insights into IDH1-driven drug resistance
4/21/2026 2:00:00 PM
Location: Poster Section 30
Poster Board Number: 7
Presentation Number: 6171



Transcriptomic and therapeutic insights from patient-derived colorectal cancer organoids
4/21/2026 2:00:00 PM
Location: Poster Section 1
Poster Board Number: 24
Presentation Number: 5457