

# Integrative Clinical and Molecular Characterization of 665 Next- Generation In Vitro Models from the Human Cancer Models Initiative

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# ATCC's Human Cancer Models Initiative (HCMI) Portfolio

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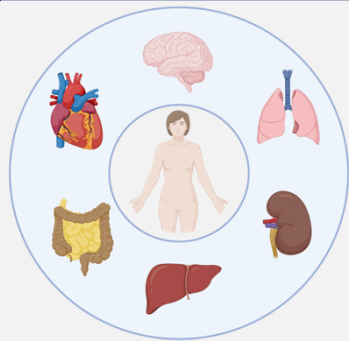
# About ATCC®



- Founded in 1925, ATCC is a non-profit organization with HQ in Manassas, VA, and an R&D and Services center in Gaithersburg, MD
- World's largest, most diverse biological materials and information resource for cell culture – the “gold standard”
- Innovative R&D company featuring new product formats that support drug development and microphysiological systems
- Partner with government, industry, and academia
- Leading global supplier of authenticated cell lines, viral and microbial standards
- Sales and distribution in 150 countries, 20 international distributors
- Talented team of 550+ employees, over one-third with advanced degrees

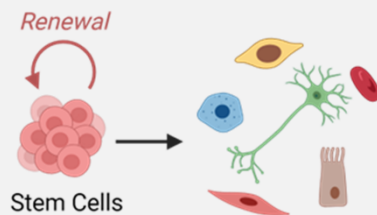


# Microphysiological Systems Program at ATCC



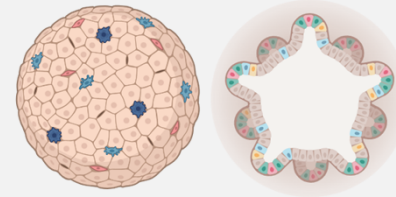
## Primary Cells

- Product development
- Isolation
- Preservation
- Manufacture
- Characterization



## iPSC-derived Cells

- Product development
- Characterization
- Differentiation
- Functional assays



## HCMI Organoids

- Product development
- Manufacture
- Model replenishment
- Characterization



## MPS Systems

- Organ-on-chip
- Microfluidics
- Heterogenous/Co-cultures
- High cost
- Greatest relevance

# ATCC's Human Cancer Models Initiative

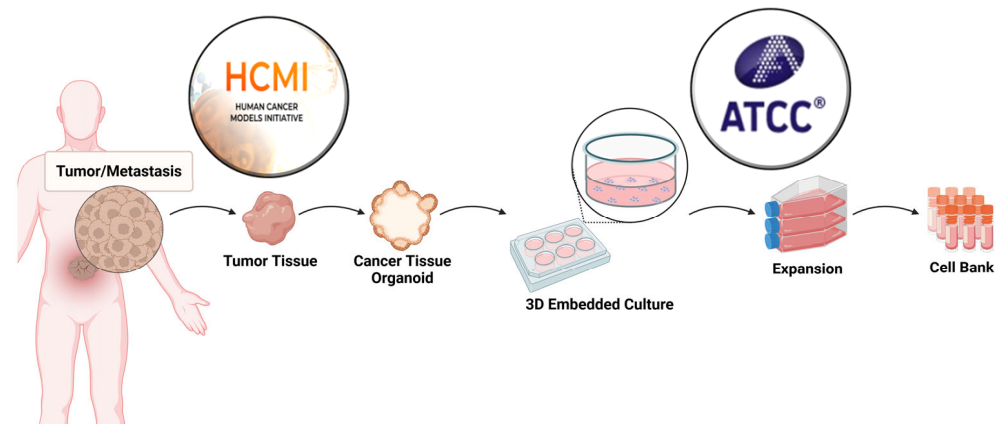


- The Human Cancer Models Initiative (HCMI) is an international consortium dedicated to generating patient-derived cancer models to facilitate cancer research.
- **ATCC is the sole distributor of HCMI models**

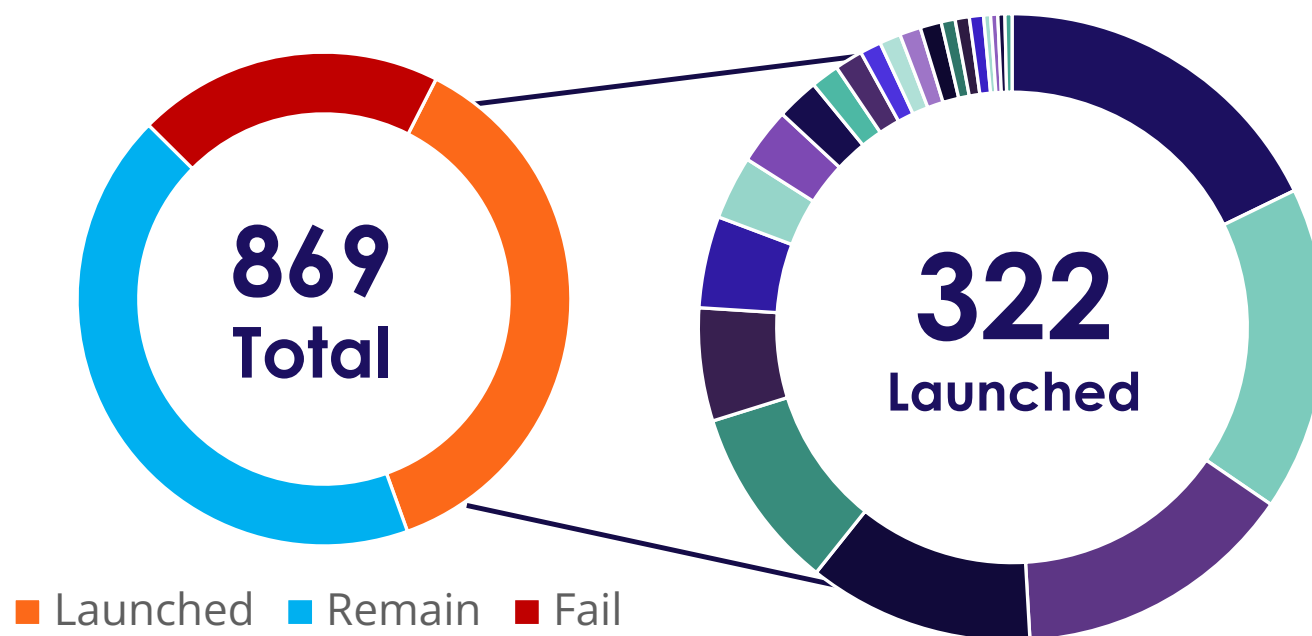
## Founders and Members

- 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



## Diverse Catalogue of Tumor Models



### TOP 10 (80% of total)

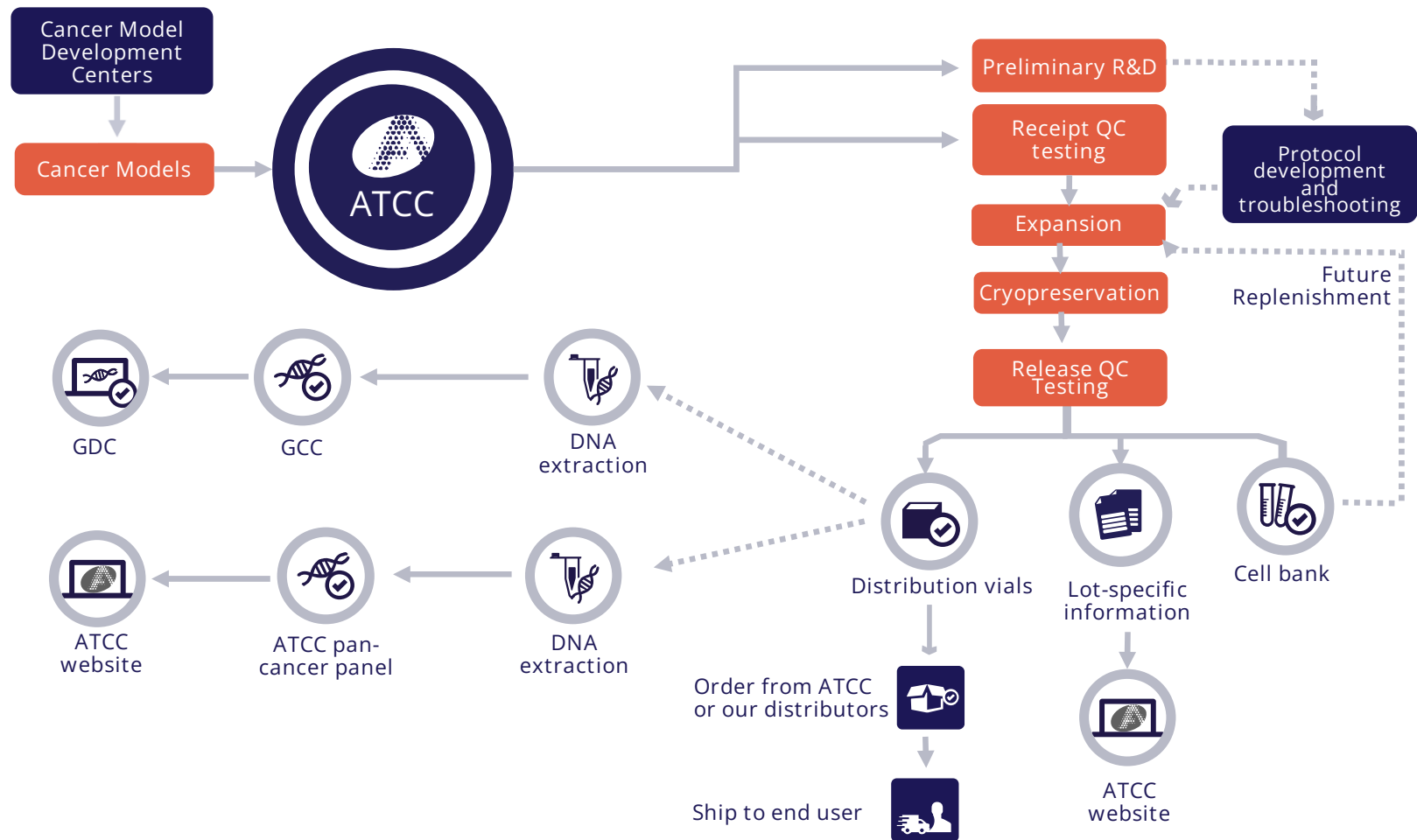
|                   |    |
|-------------------|----|
| Brain             | 50 |
| Pancreas          | 53 |
| Colon             | 59 |
| Esophagus         | 36 |
| Skin              | 30 |
| Rectum            | 21 |
| Stomach           | 18 |
| Connective tissue | 9  |
| Biliary tract     | 8  |
| Breast            | 7  |
| Lung              | 6  |

*Collection includes models derived from rare adult and pediatric cancers such as rhabdomyosarcoma, leiomyosarcoma, Ewing sarcoma and Wilms tumor.*

Complete list available at  
[atcc.org/hcml](https://atcc.org/hcml)

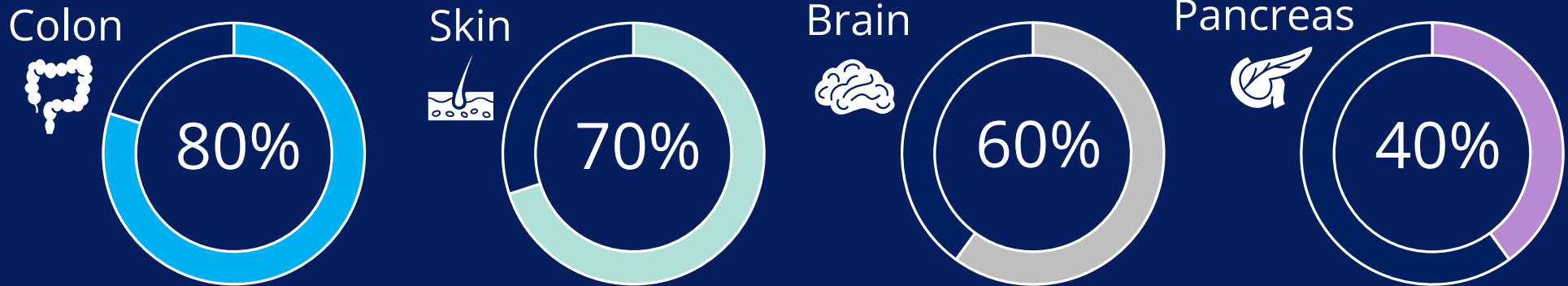


# ATCC's Model Manufacturing Workflow



# Concordance with TCGA Genomic Landscape

Top 10 frequently mutated genes vs. The Cancer Genome Atlas:



Top 10 genes captured in the HCM1 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   |

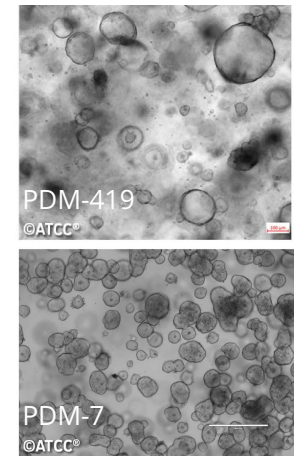
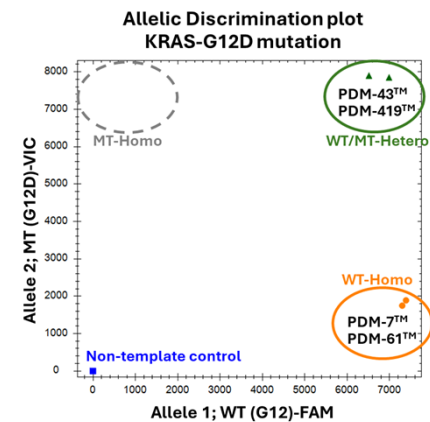
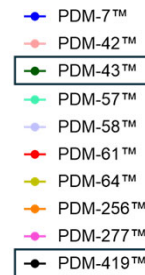
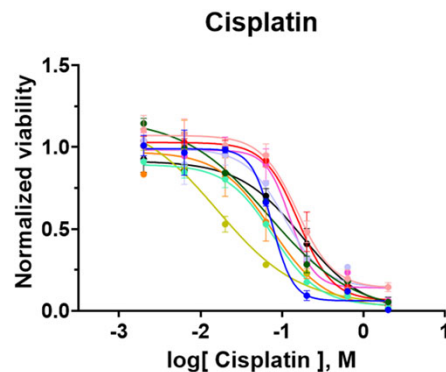
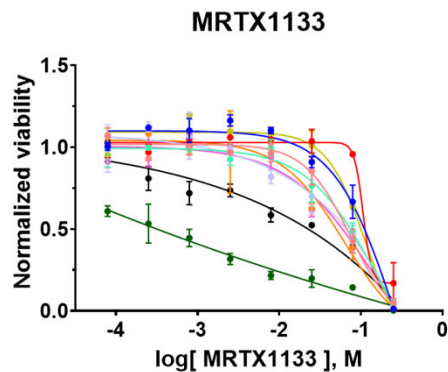
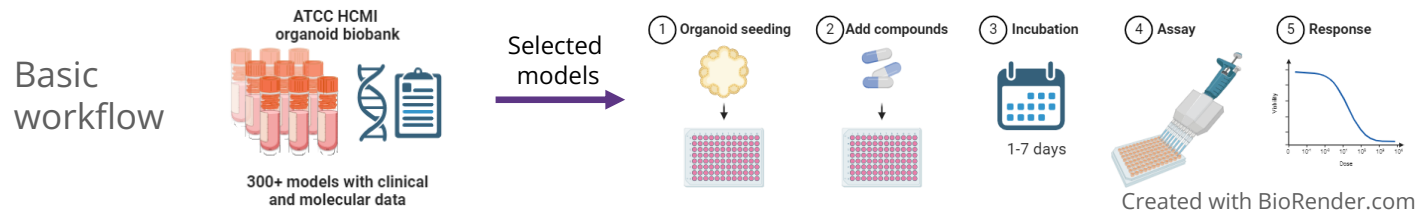
|        |       |
|--------|-------|
| KRAS   | MUC4  |
| TP53   | MUC16 |
| SMAD4  | KMT2D |
| CDKN2A | FAT3  |
| ACVR2A | RHOH  |

Canonical mutation

Matching mutation

# Targeting KRAS in Human Colorectal Models

- HCMI portfolio contains over 59 Colorectal cancer models with mutations in disease relevant genes (e.g., APC, KRAS, SMAD4, TP53, etc). Subset were screened across multiple compounds.



- MRTX1133, a non-covalent small molecule inhibitor of KRAS G12D exhibited increased toxicity in PDM-43 and PDM-419

# ATCC's HCMI Model Information Resource



HCMI Landing Page [atcc.org/hcmi](https://atcc.org/hcmi)

## Human Cancer Model Initiative

277 Products  
Results 1-24 of 277

Search

Refine by

Product category

- Human cells 270
- Media 7

Product type

- Cell model 170
- Organoid 100

Product application

- 2D cell culture 245
- Cancer research 235
- Neuroscience 44
- Cell culture 7
- Cell growth and viability 7

Search

Product format

- Frozen 277

Biosafety level

- BSL 1 205
- Not BSL 7
- BSL 2 2

Organism

- Human explants 270

Tissue

Search

Filtering options

HCMI model-specific product page

HCM-CSHL-0065-C20  
PDM-9™

Product category: Human cells  
Product type: Cell model  
Organism: Homo sapiens, human  
Morphology: Organoid  
Tissue: Large Intestine, Rectum  
Disease: Colorectal cancer, Primary metastatic  
Applications: 2D cell culture, Cancer research  
Product format: Frozen  
Storage conditions: Vapor phase of liquid nitrogen

SEE ADDITIONAL PRODUCT INFORMATION

Detailed product information

General

Characteristics

Handling information

Quality control specifications

History

Legal disclaimer

Permits & Restrictions

Material Transfer Agreement Addendum for Organoid Products

For every order of this item, you must provide a signed [Material Transfer Agreement Addendum for Organoid Products](#) via email with the item and we will mail it to you. Email the signed addendum to [atcc@atcc.org](mailto:atcc@atcc.org) with a reference to both your account and order numbers. Once received, your addendum will be reviewed and the item will be released for shipment if all requirements are met. If you need assistance with your order, please contact our Customer Care team or your applicable distributor.

Import Permit for the State of Hawaii

If shipping to the U.S. state of Hawaii, you must provide either an import permit or documentation stating that an import permit is not required. We cannot ship this item until we receive the documentation. Contact the [atcc@atcc.org](mailto:atcc@atcc.org) to determine if an import permit is required.

Images

ATCC Number: PDM-9  
Designation: HCM-CSHL-0065-C20

Purchase model

Model resources and protocols, lot-specific information

QC testing specifications

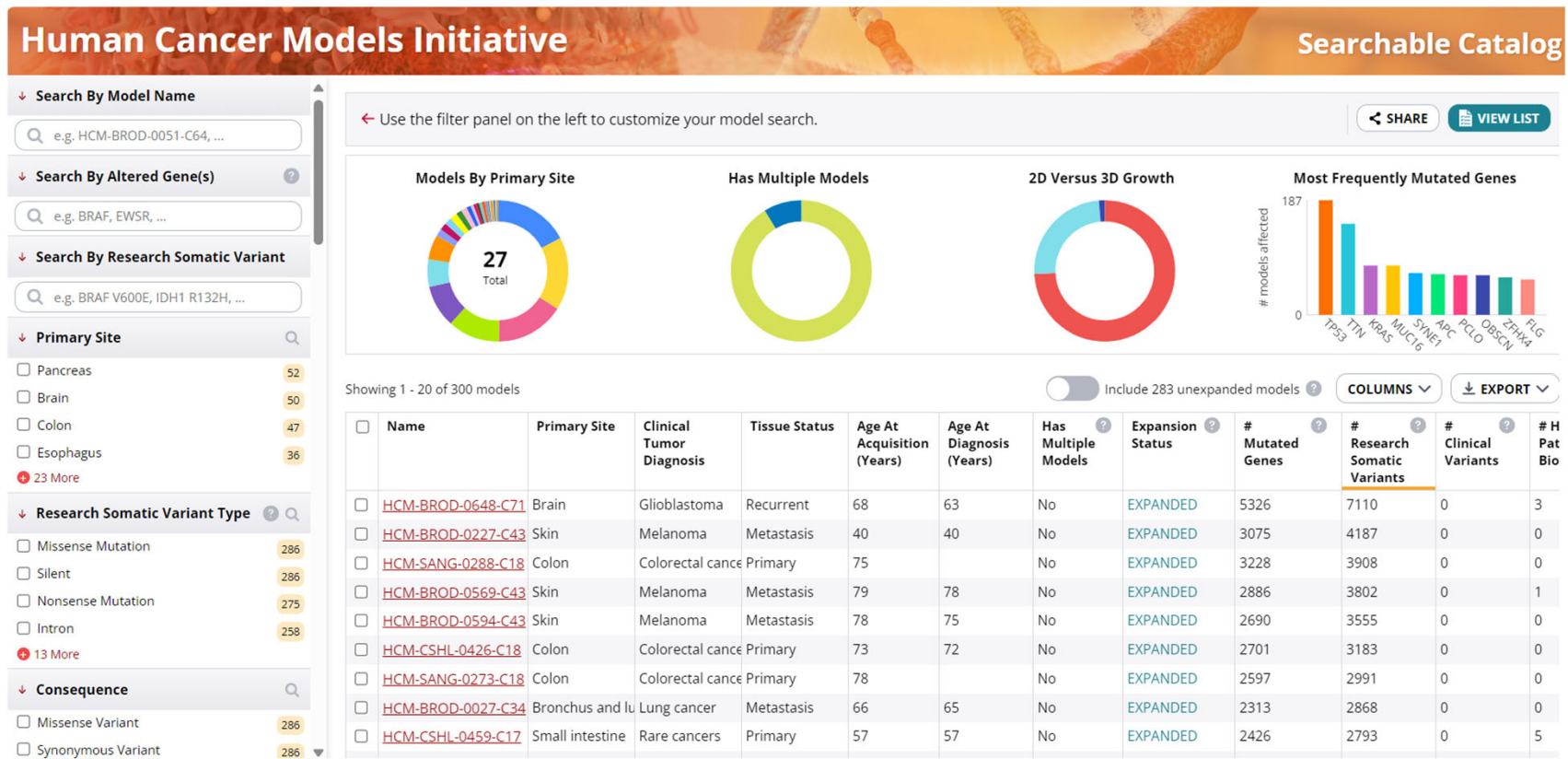
HCMI Searchable Catalog

GDC

Model images and other information

# NCI - ATCC Model Information Resource

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





# Shaping the Next Wave of HCMI Models: Your Feedback Matters



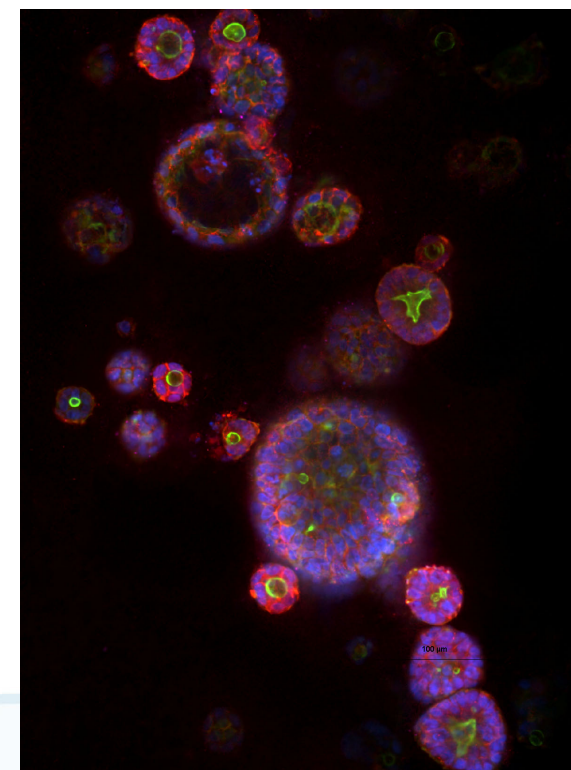
2026 Pipeline: Breast (TNBC), Ovarian (6 models from a single donor), Pediatric Glioblastoma, Pediatric Leiomyosarcoma models

Browse and search unreleased HCMI models at ATCC

- Listed as “HCMI unmanufactured models”
- Use the “Submit your Input” button on the HCMI Landing page
- Direct link: [www.atcc.org/hcml-input](http://www.atcc.org/hcml-input)

Email us which HCMI models are most relevant for your research

- Contact us at: [hcml@atcc.org](mailto:hcml@atcc.org)



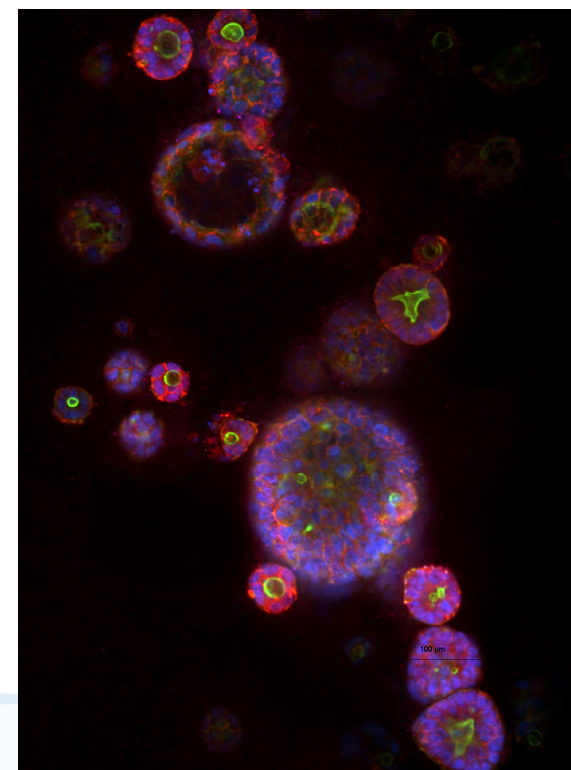
## Resources



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Email us which HCMI models are most relevant for your research

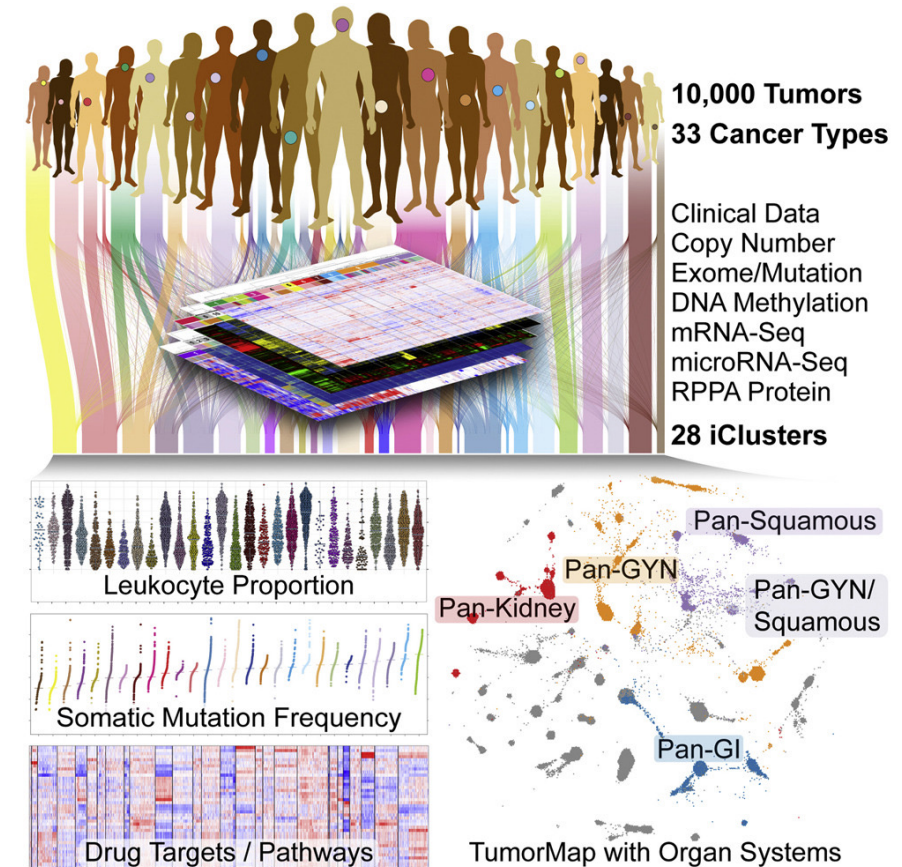
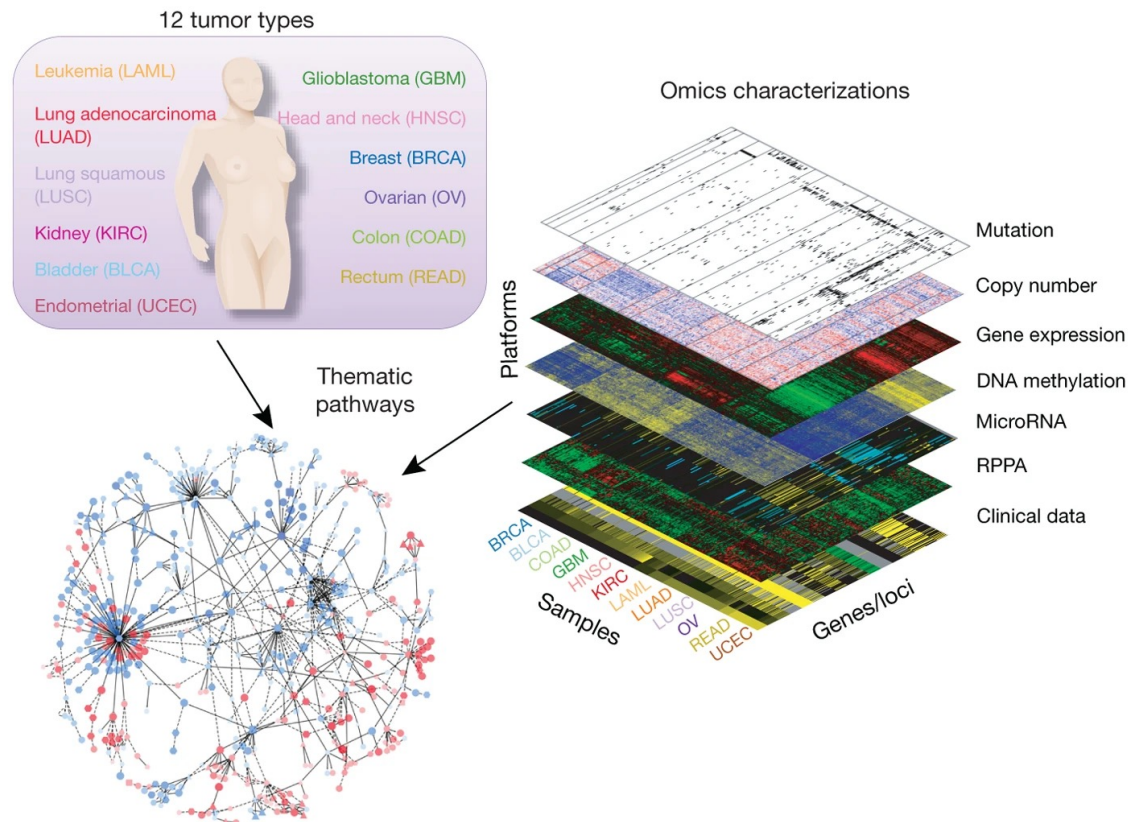
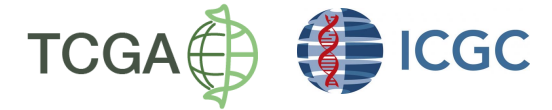
- Contact us at: [hcml@atcc.org](mailto:hcml@atcc.org)
- HCMI Searchable Catalog <https://hcml-searchable-catalog.nci.nih.gov/>



**Integrative Clinical and Molecular Characterization of  
665 Next-Generation In Vitro Cancer Models from the  
Human Cancer Models Initiative**

## Background: Genomic cancer atlases

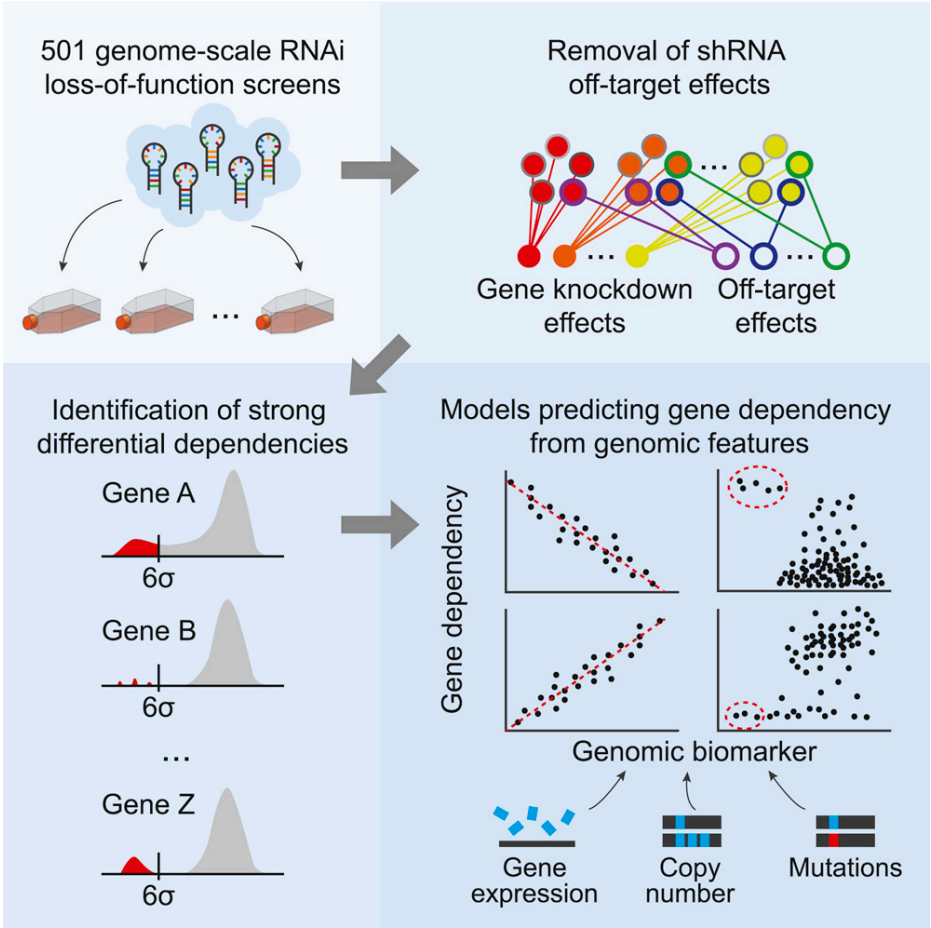
- International consortia have played an essential role in generating extensive **cancer atlases** which have redefined **molecular drivers**, cancer **subtypes** and prompted hypotheses for **preclinical validation**.



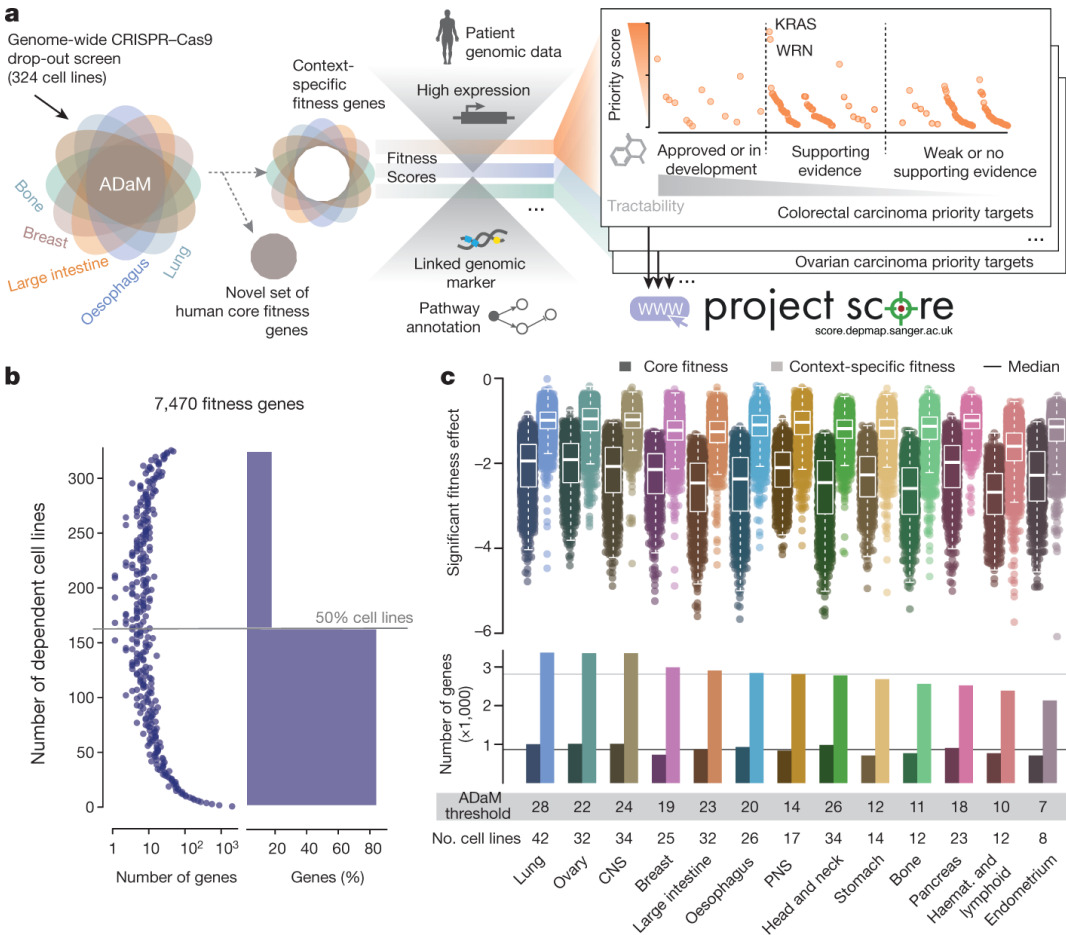


# Background: Functional translation

- **Project Achilles (Broad)** and **Project Score (Sanger)**: large-scale efforts that identify genetic dependencies in cancer using RNAi and CRISPR-based loss-of-function screens



Tsherniak et al, Cell. 2018



Behan F, et al, Nature. 2019



## Why historical cell lines fall short?

- Many historical cell lines may have extensive **genomic drift**, lineage marker loss, and limited **transcriptional and epigenetic fidelity**.
- **Functional discoveries** using historical cell lines have reached a **plateau**.
- Lack of clinical context: few **treatment histories, clinical outcomes, or resistance profiles** makes them unsuitable for modeling therapy response.
- Rare cancer types, metastatic samples, and **non-European ancestries** are underrepresented.



**Human Cancer Models Initiative (HCMI)** was launched (2014)  
to build a global resource of **high-fidelity, clinically-annotated**  
cancer **next generation models**

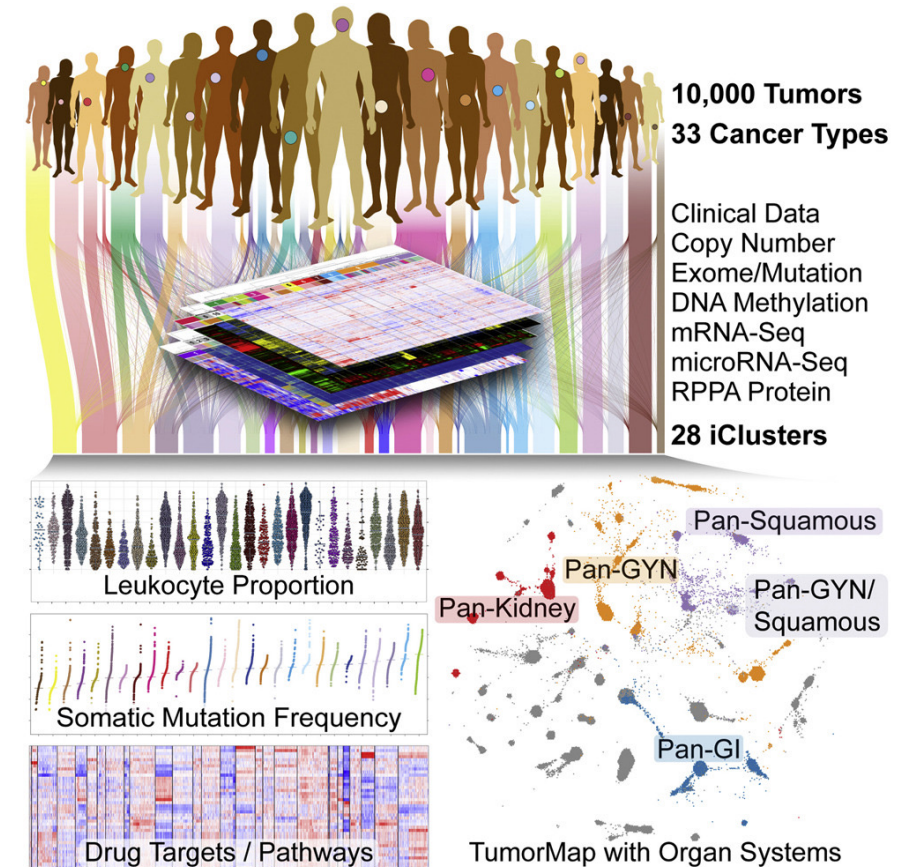
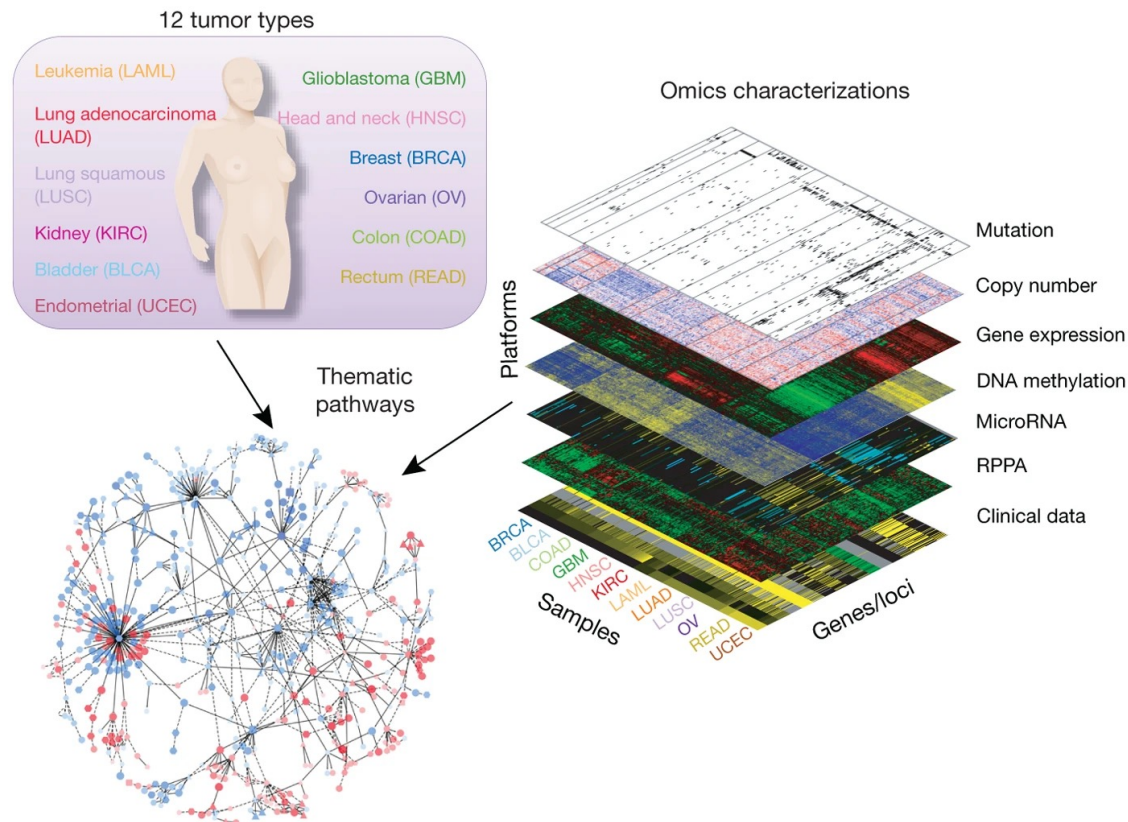
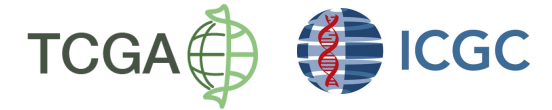
Cancer Model Derivation Centers and partners



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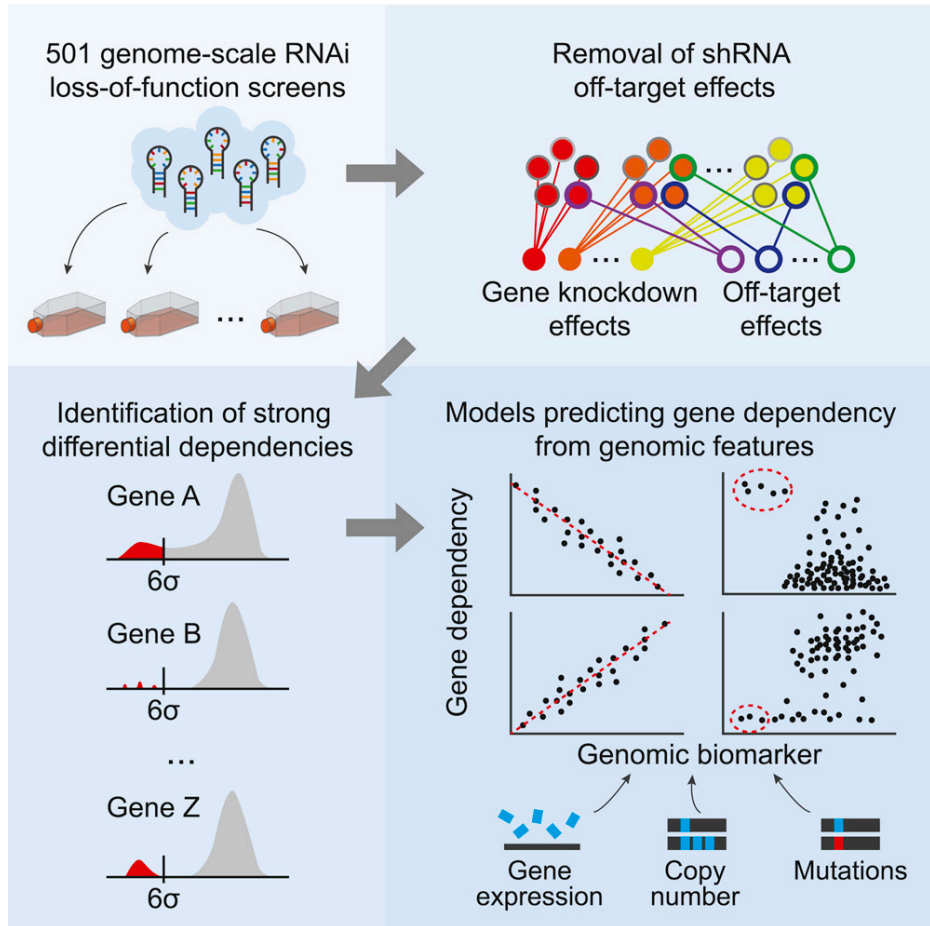
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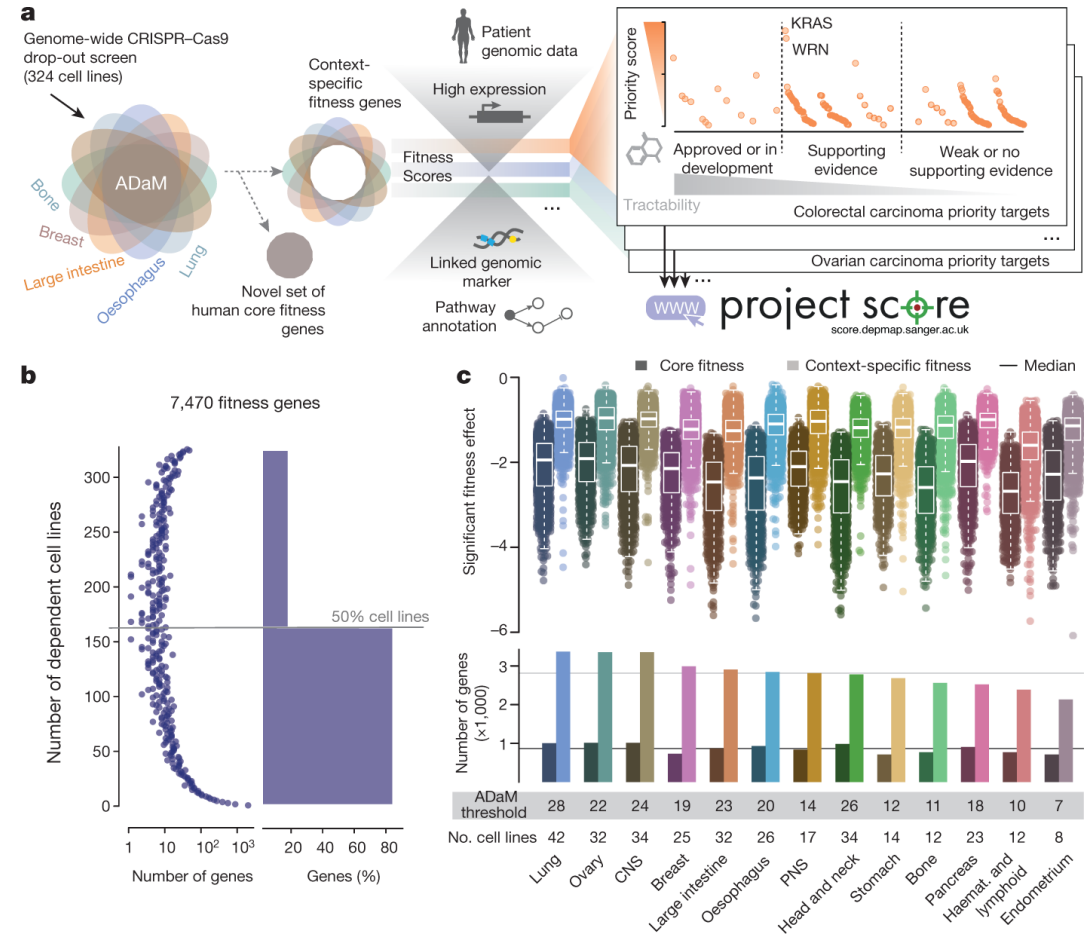


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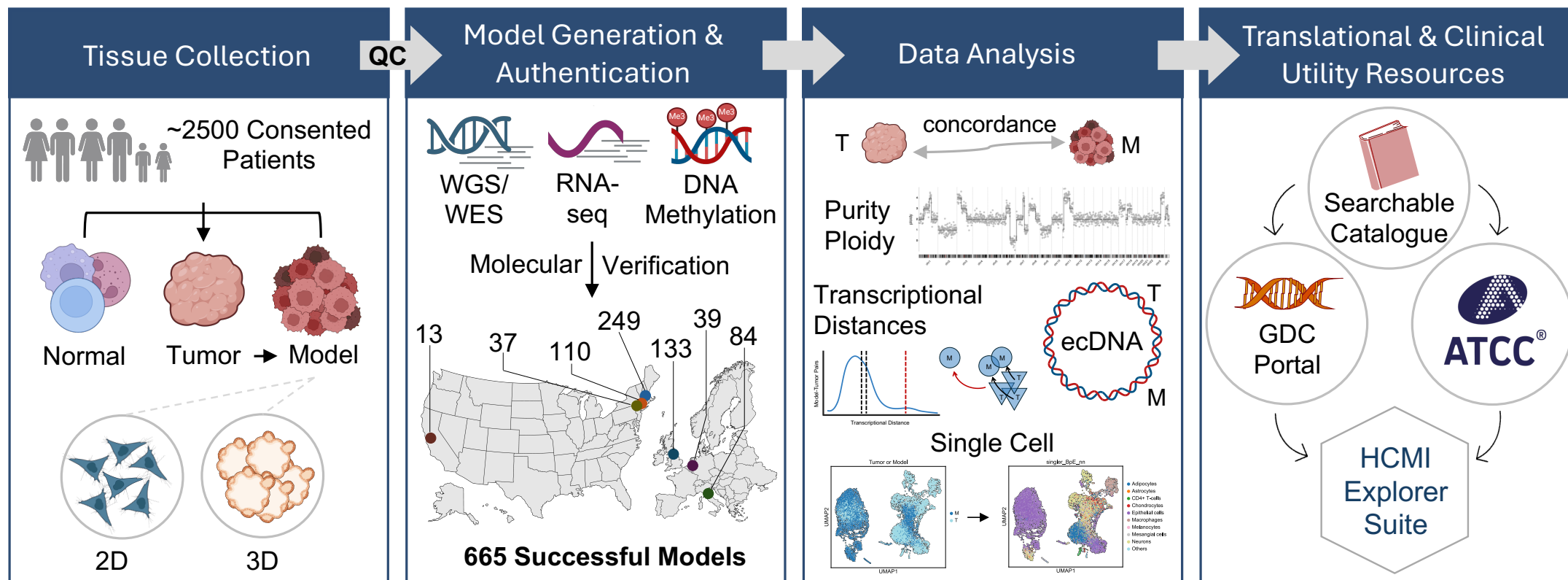
Cancer Model Derivation Centers and partners





# Generation of 665 next-generation cancer models

- A compendium of next generation patient derived models across 25 tumor types as a public resource.



## Flagship aims of the Human Cancer Model Initiative

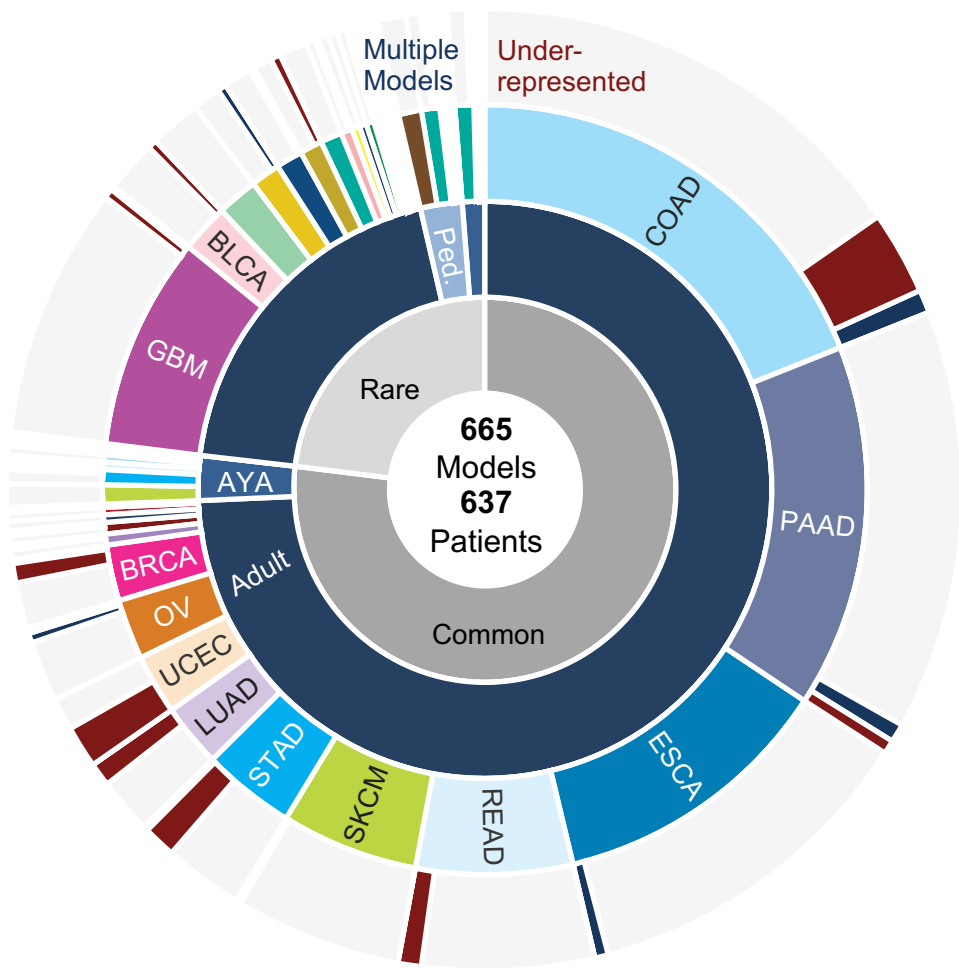
- 1 Establish a diverse and distributable compendium of next generation patient derived models.
- 2 Systematically evaluate molecular fidelity of tumor–model pairs.
- 3 Identify the translational utility of the HCMI resource.

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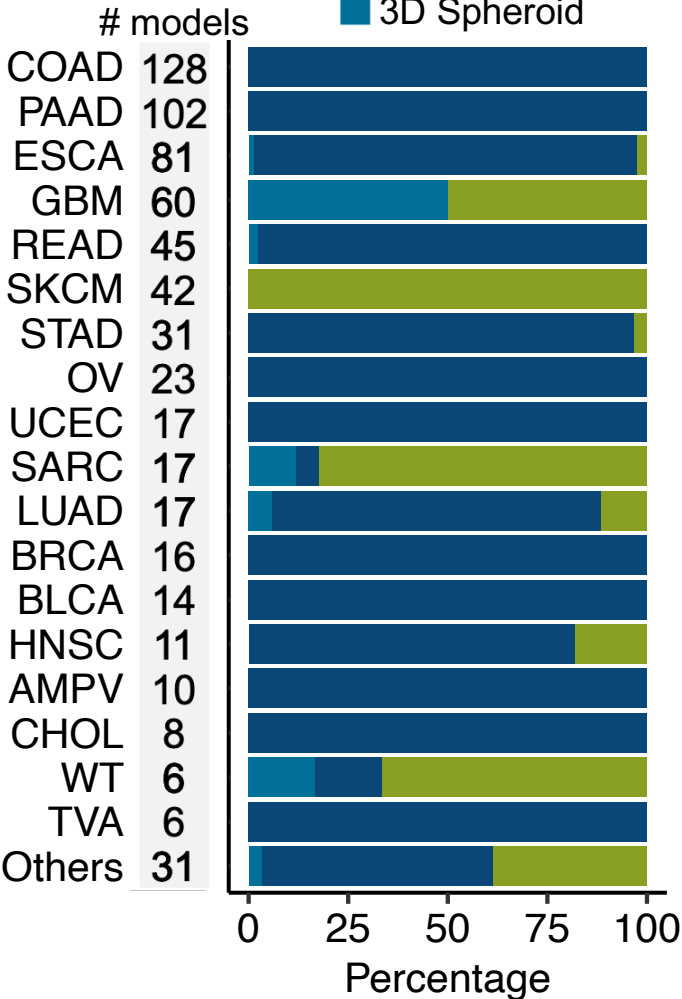
# Distribution of HCMI models by cancer type, patient demographics and growth patterns

- Rare cancer types (n = 153)
- Adult donors (n = 622)
- Pediatric and adolescent (AYA) donors (n = 43)
- Non-European populations (n = 71)
  
- 3D organoids (n = 519)
- 3D spheroids (n = 37)
- 2D adherent cell lines (n = 109)



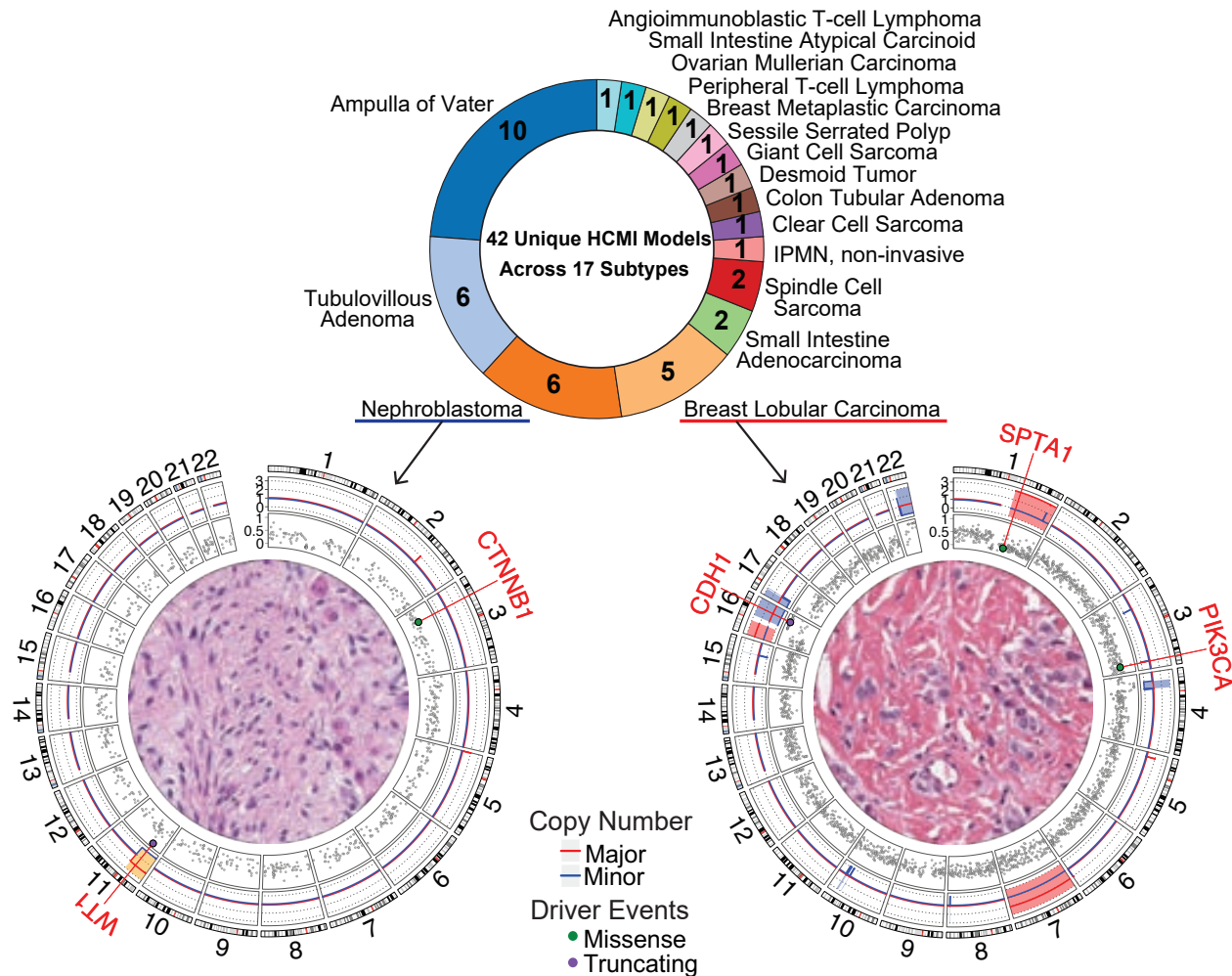
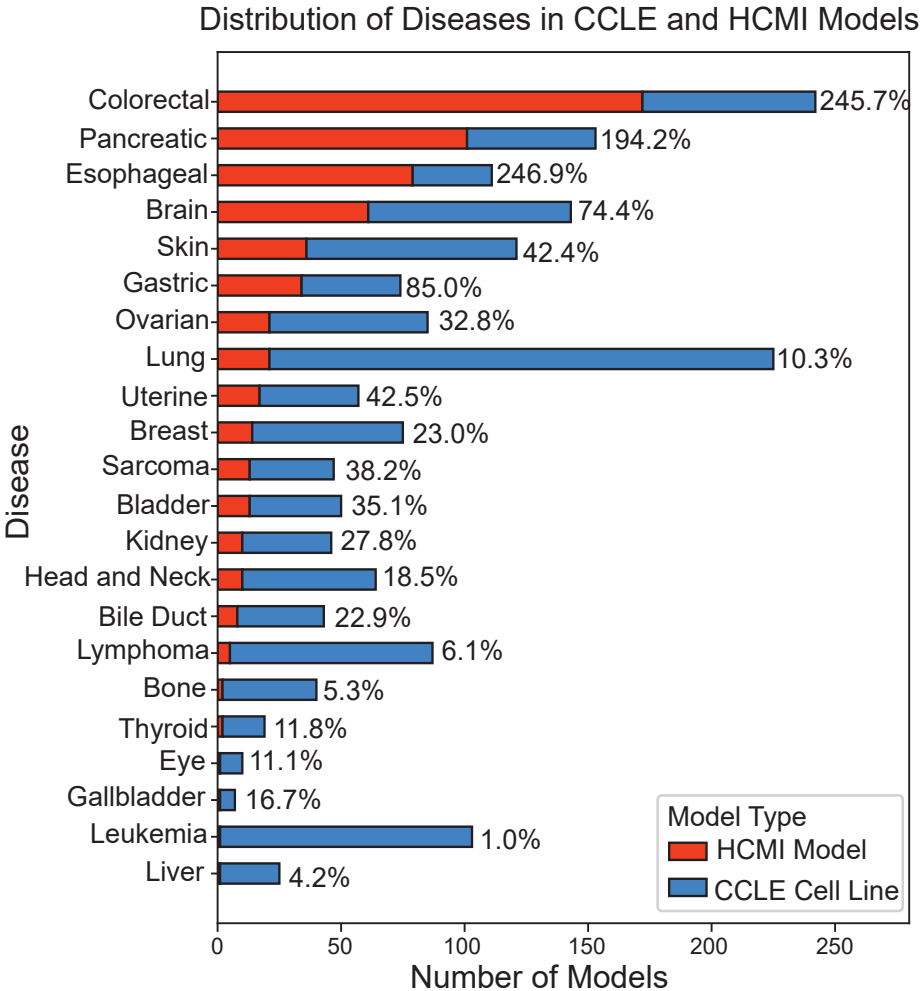
Growth Pattern

- 2D Cell Line
- 3D Organoid
- 3D Spheroid



# HCMI expands cancer model diversity

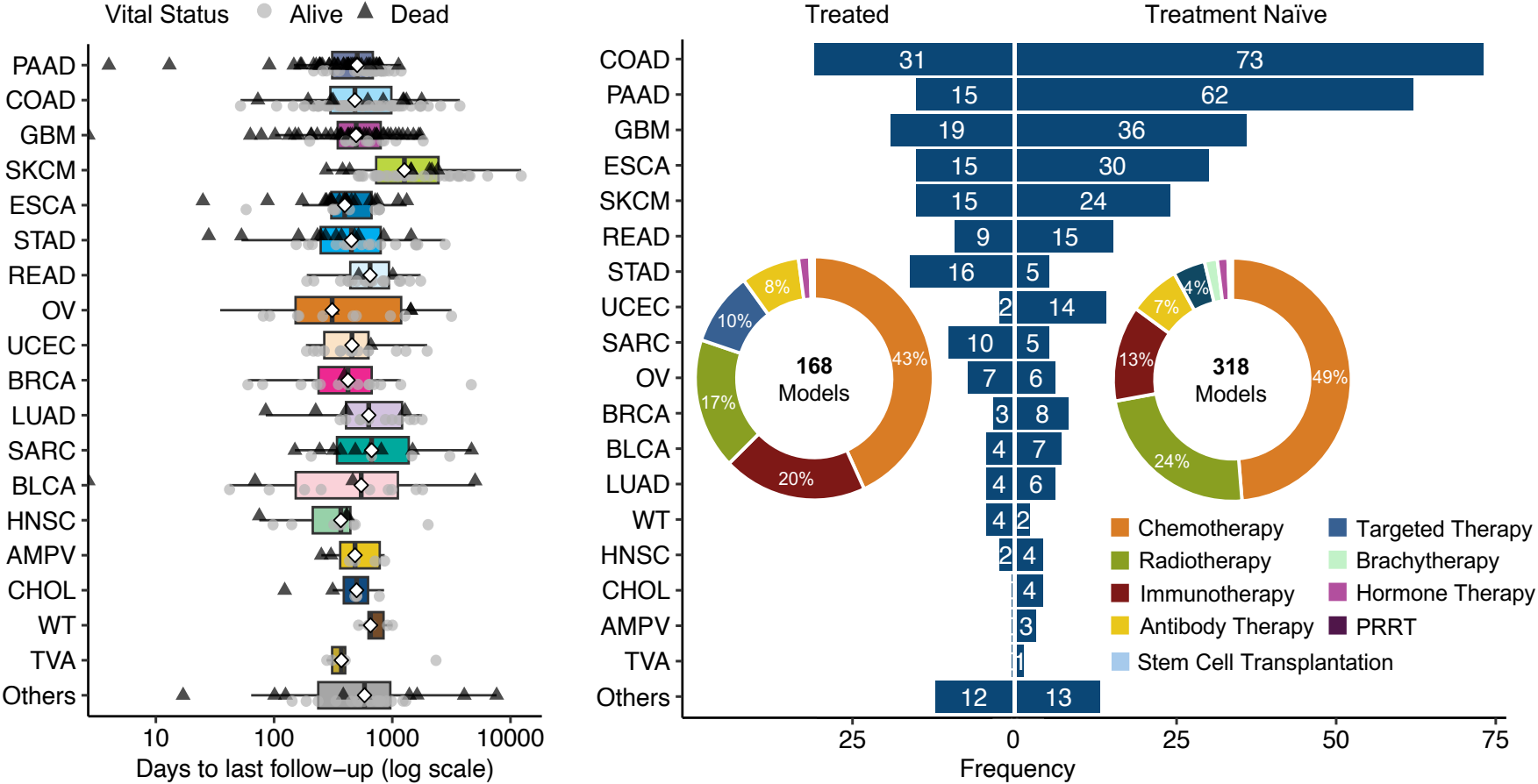
- The HCMI resource expands cancer model diversity, more than doubling models for colorectal, pancreatic, and esophageal cancers, and increasing others by a median of 25.4%.





# Comprehensive clinical outcomes and modern treatment histories of cancer models

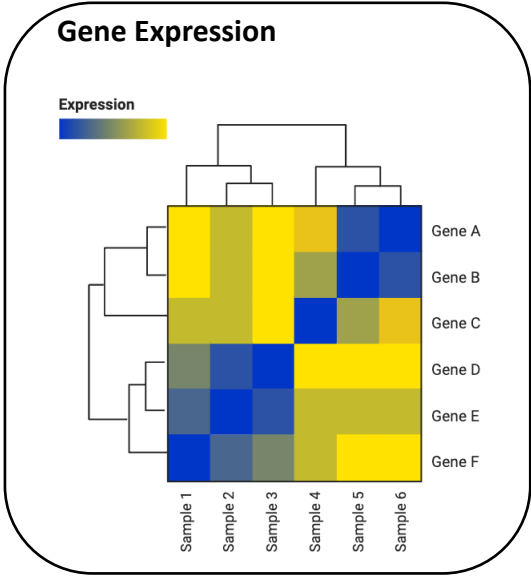
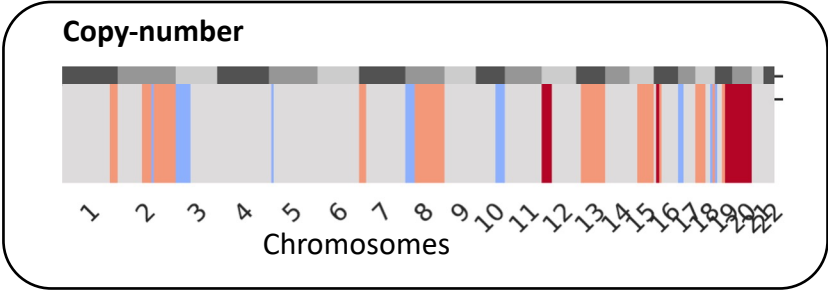
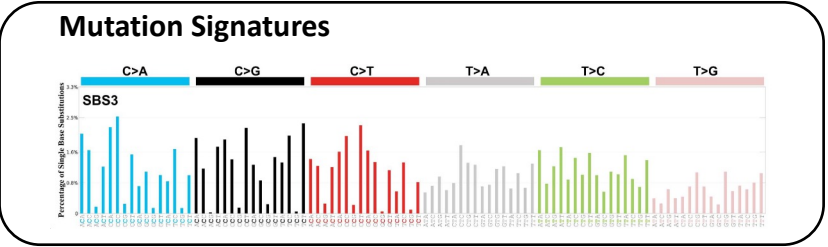
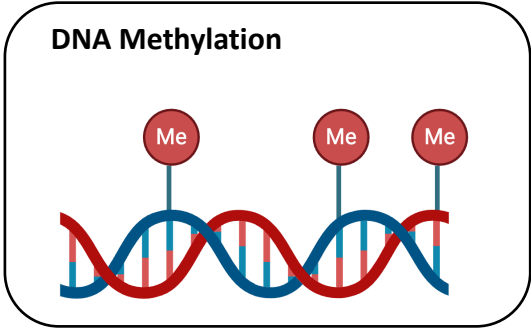
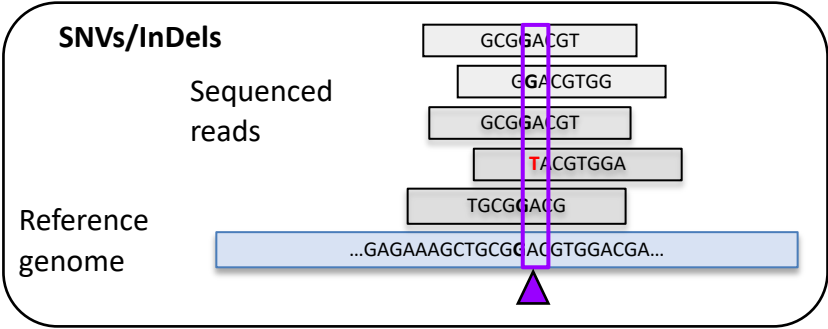
- **Comprehensive clinical data** was collected for 78% (n = 519) of models.
- Patient data includes survival outcomes (range: 0–34 years), **treatment history**, and duration of response to post-biopsy therapy (median follow-up: 1.4 years).



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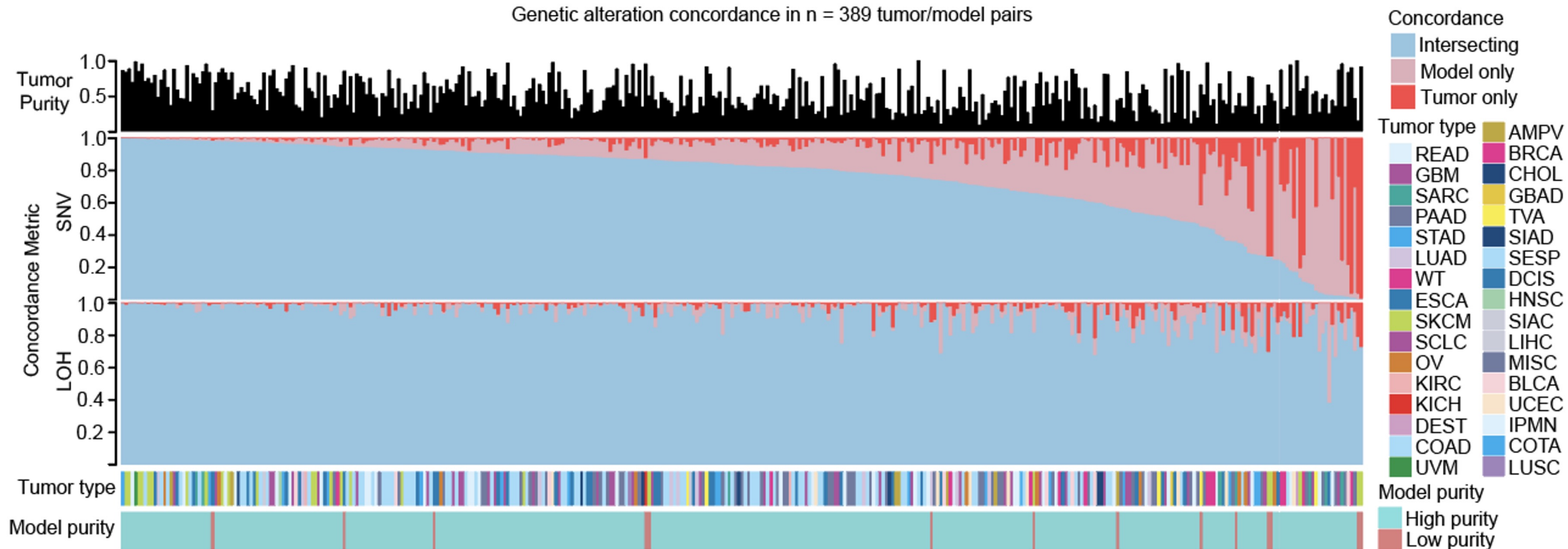
# Genomic, Epigenetic and Transcriptional Validation of Model Fidelity



# Concordance of SNVs and Loss of Heterozygosity across 389 tumor / model pairs

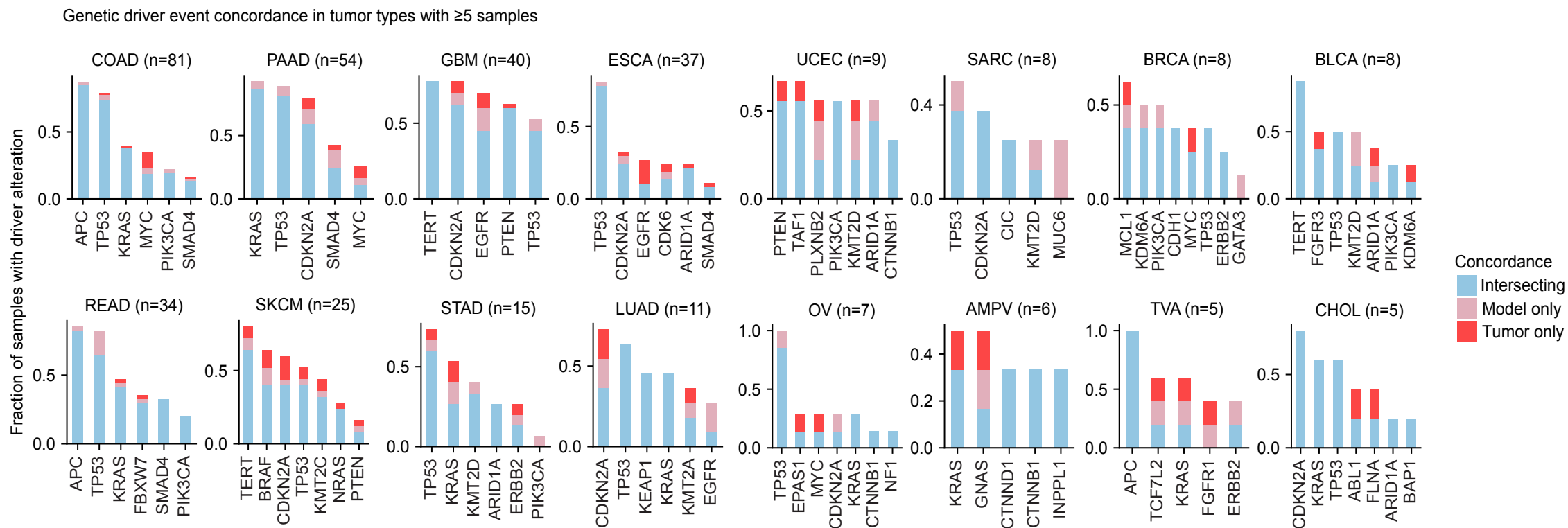
- Median of 82.4% of SNV/indels were shared by tumors and models
- Average LOH concordance 94%, 361 exceeding 90% concordance

Genetic alteration concordance in n = 389 tumor/model pairs



# Do HCMI models retain driver mutations of their matching patient tumors?

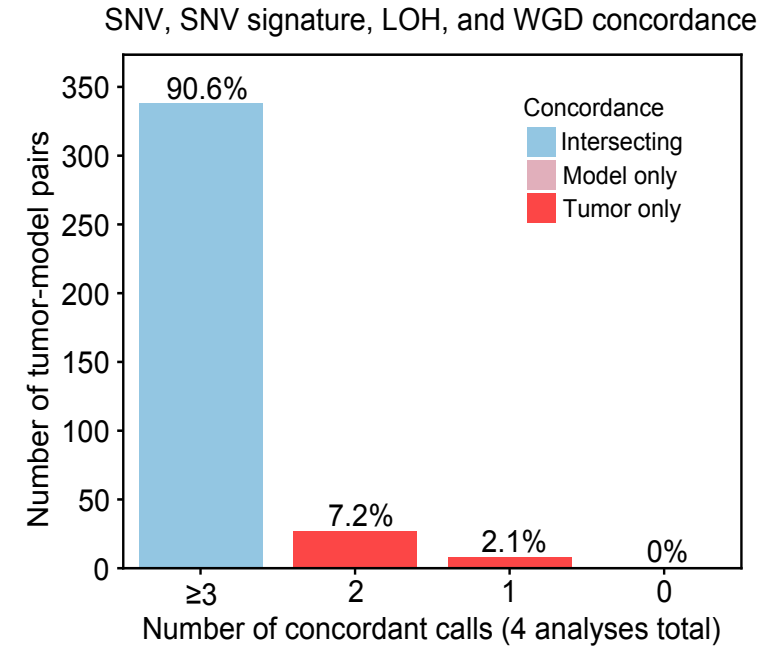
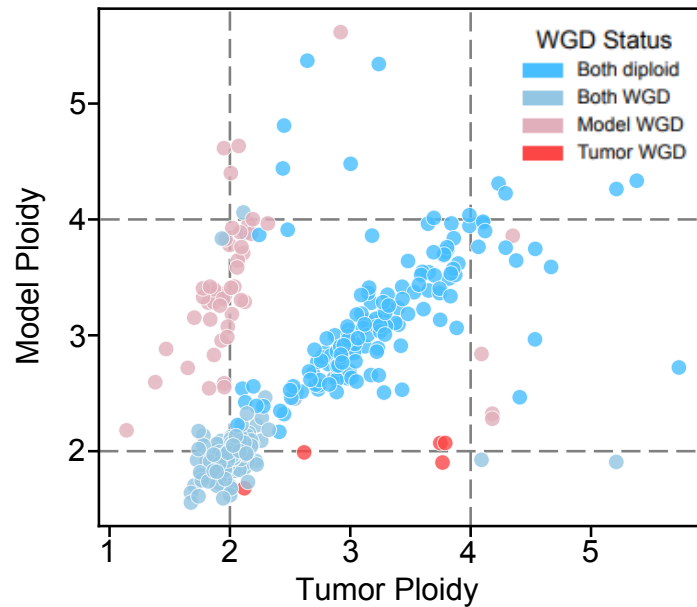
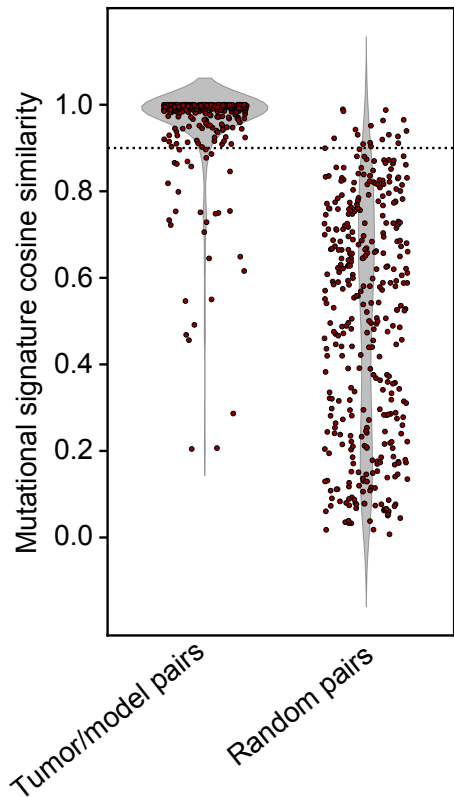
- **81%** of oncogenic drivers were retained in the models.
- The highest levels of concordance (>90% concordance between tumor and model) were observed for mutations in **BRAF**(SKCM), **KRAS** (PAAD), **APC** (COAD and READ), and **TP53** (ESCA).





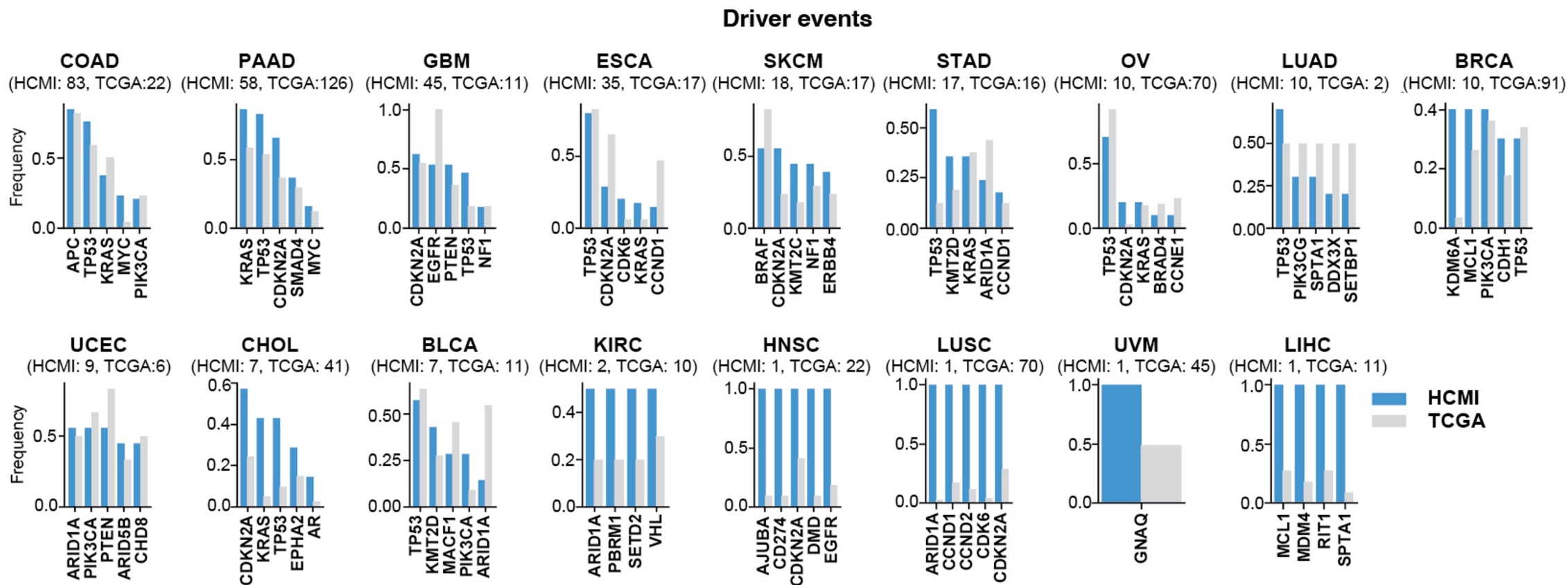
## Do HCMI models retain the genomic features of their matching patient tumors?

- Evaluation of 4 DNA-based features: **driver mutations**, **mutational signature**, Loss of Heterozygosity (**LOH**) and Whole Genome Doubling (**WGD**) (n = 388).
- SNV mutational signatures were conserved between tumor/model pairs. No specific signature enriched in models
- Despite being passaged in culture for at least one year, the majority of HCMI models (97.8%) retained at least two and often all four of the DNA-based features.



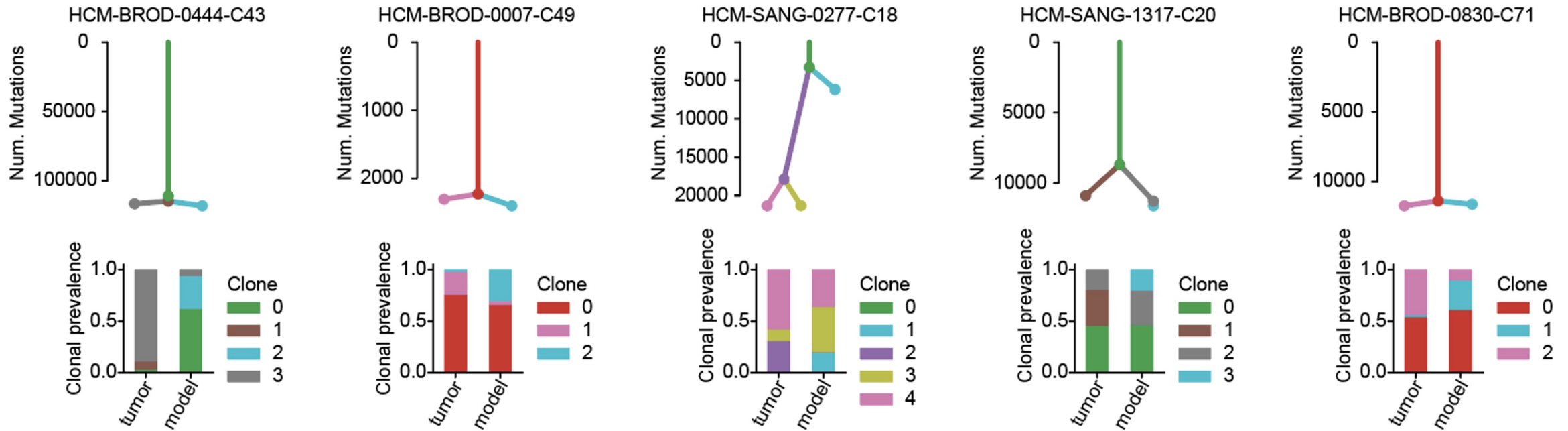
# Are HCMI models consistent with TCGA drivers by cancer type?

- Distribution of driver mutations within each cancer type consistent with TCGA, especially for cancers types with better representation including COAD, PAAD, ESCA and BRCA



# Phylogenetic analysis of Tumor/Model Clones show generally low divergence

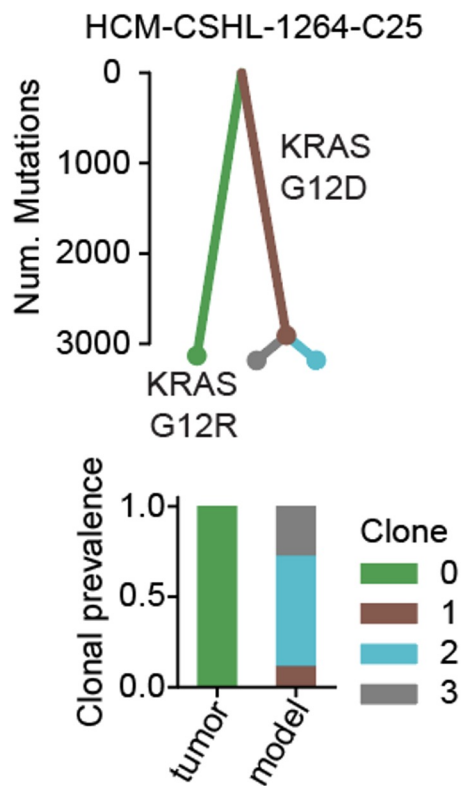
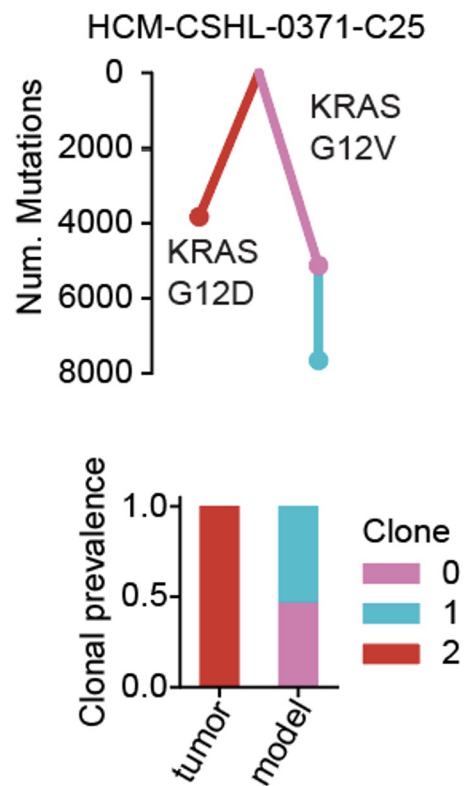
- Predicted phylogenies show a long truncal branch, followed by a small amount of divergence between model specific clones and tumor specific clones.



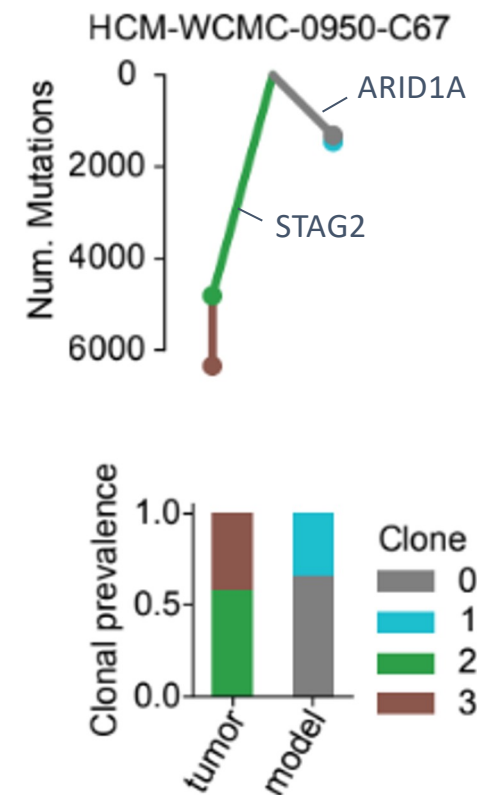
## Divergence occurs in a small subset of tumors due to tumor heterogeneity and clone specific outgrowth in the model

- Two Pancreatic Adenocarcinoma Driven by Selection of Distinct KRAS Clones during Model Development
- Selection of an Independent Tumor in a Bladder Cancer Model

PAAD

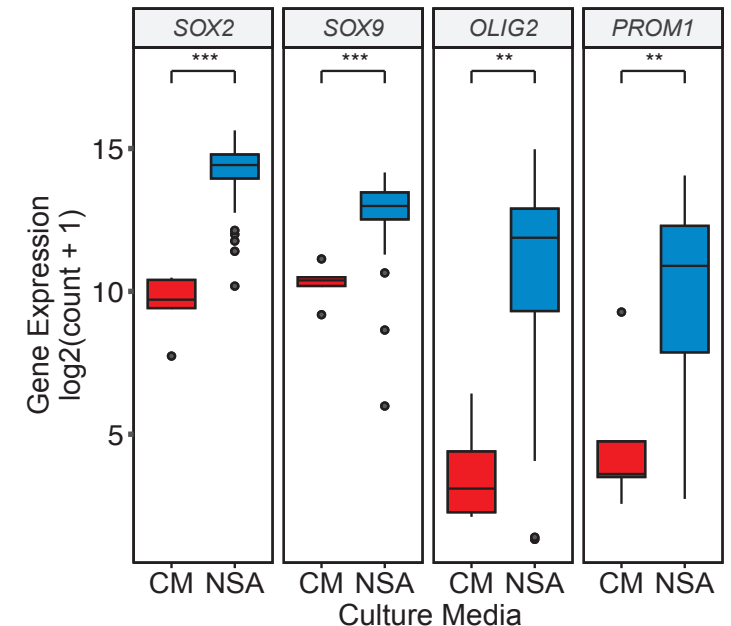
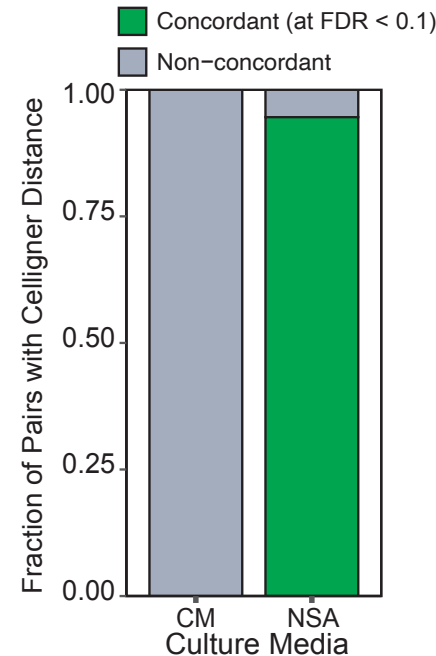
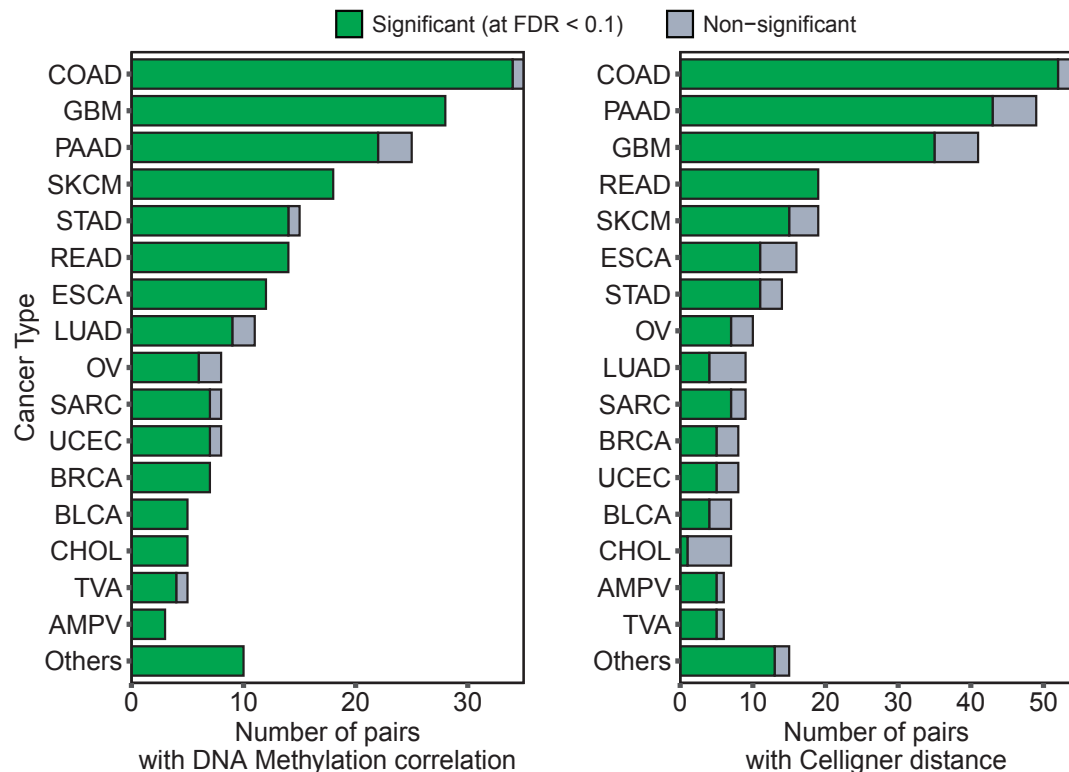


BLCA

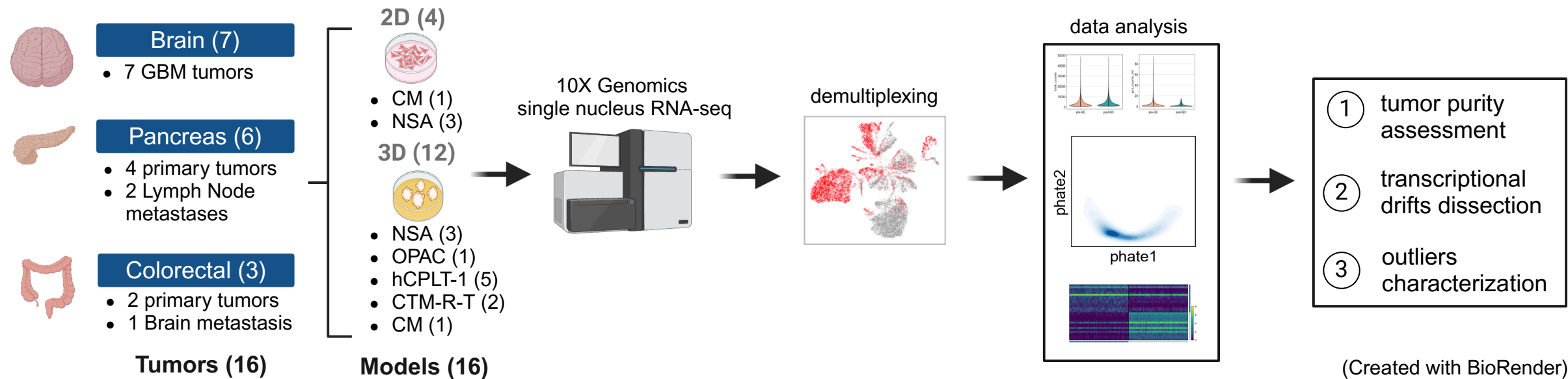


# Do HCMI models recapitulate epigenomic and transcriptomic profiles of matching tumors?

- **95% (190/201)** showed **statistically significant concordance**, meaning the model closely matched the tumor's DNA methylation landscape.
- **>80% of tumor–model pairs** showed **significant transcriptional similarity** compared to random pairs.
- GBM models that propagated in NeuroCult NS-A basal (**NSA**) medium were significantly more similar to their matching tumors than those cultured in Conditioned Medium (**CM**).



# Single-cell analysis of tumor/model pairs





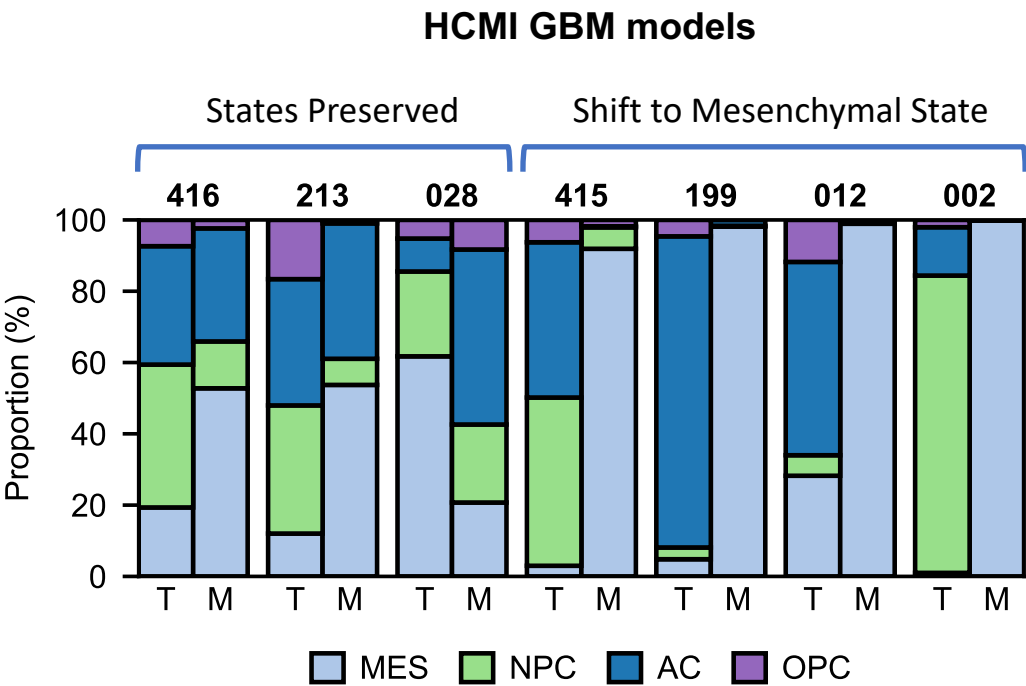
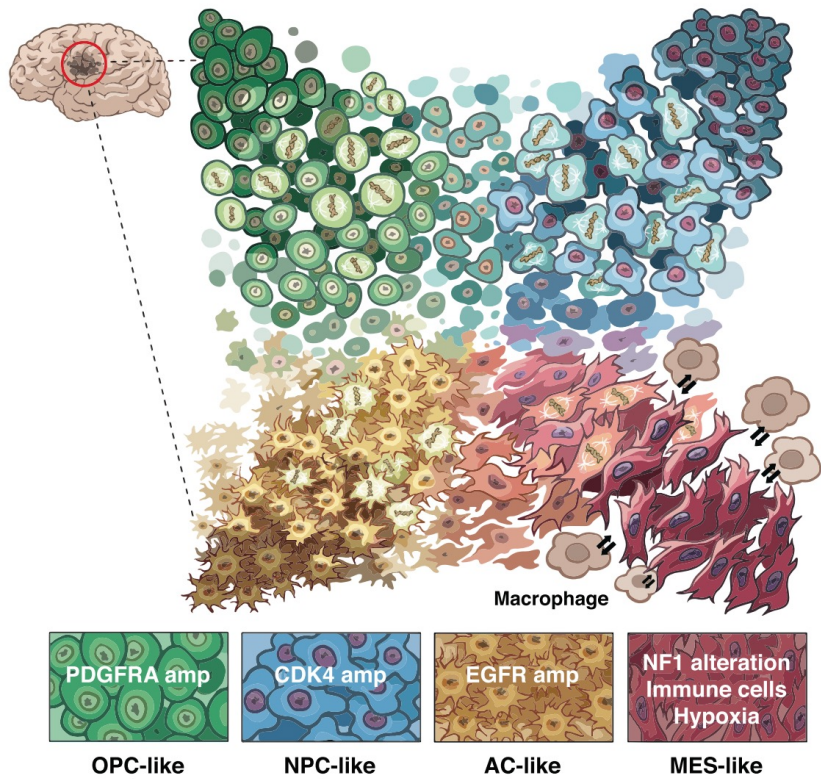
# Single-cell analysis of tumor/model pairs: GBM

## Cell

### An Integrative Model of Cellular States, Plasticity, and Genetics for Glioblastoma

Authors

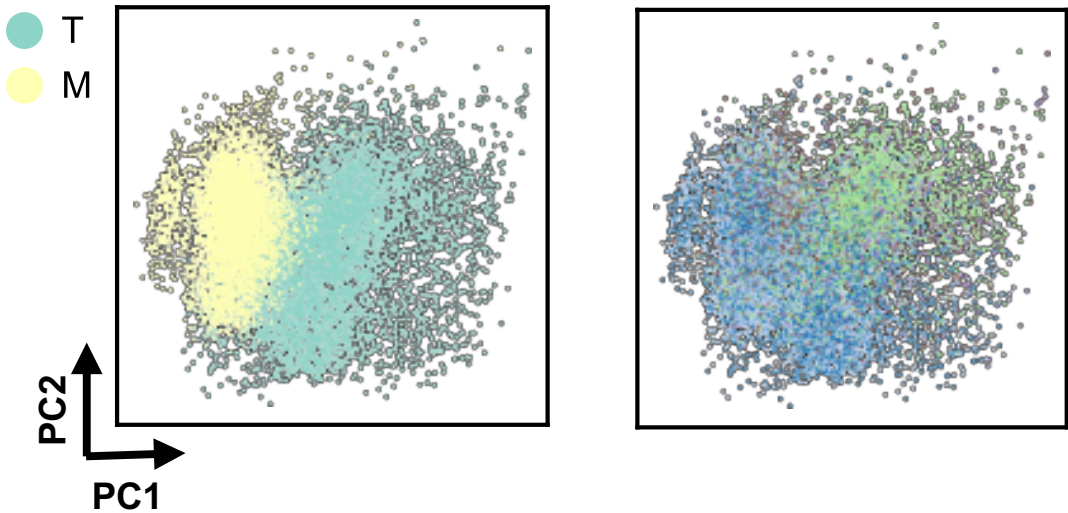
Cyril Neftel, Julie Laffy,  
Mariella G. Filbin, ..., Bradley E. Bernstein,  
Itay Tirosh, Mario L. Suvà



# Single-cell analysis of tumor/model pairs

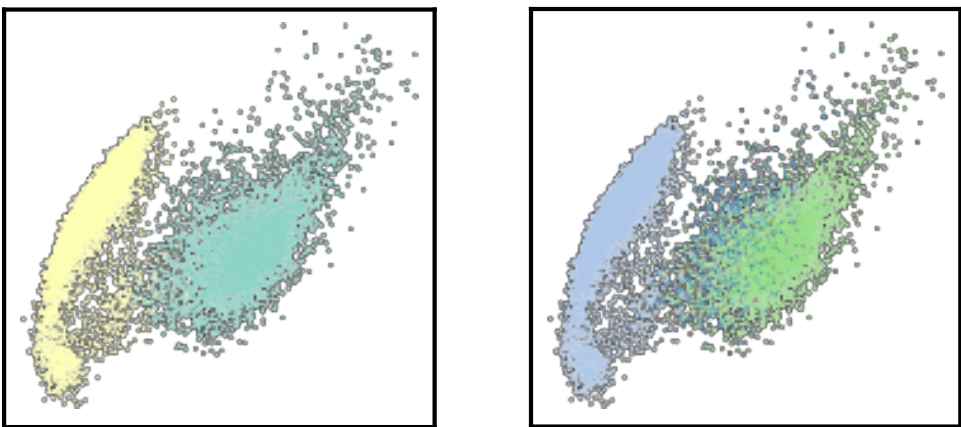
HCM1 GBM Model (NSA – Serum free)

416 - convergent



HCM1 GBM Model (CM – Serum)

002 - divergent



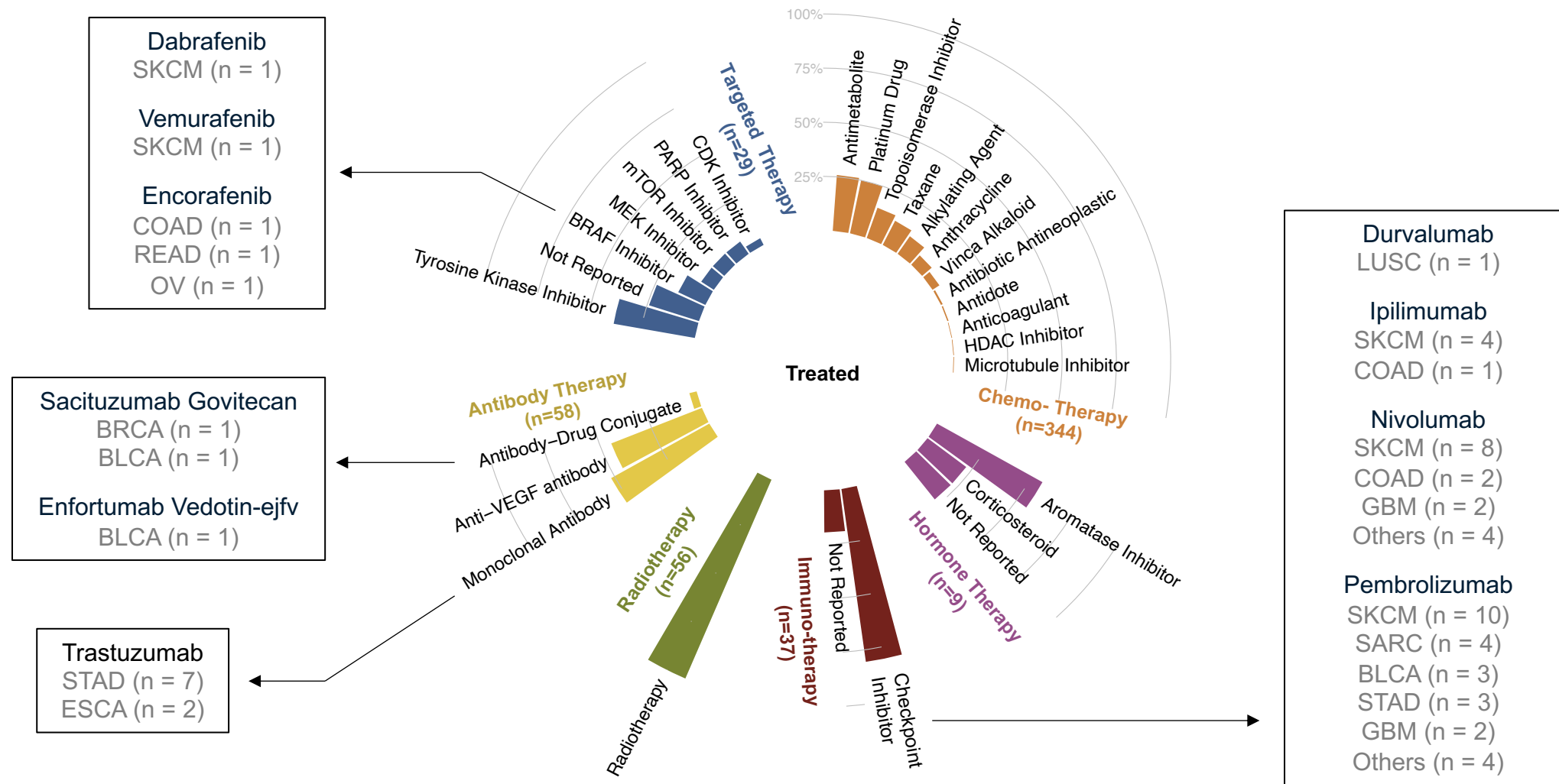
- AC
- MES
- NPC
- OPC
- Unknown

## Flagship aims of the Human Cancer Model Initiative

- 1 Establish a diverse and distributable compendium of next generation patient derived models.
- 2 Systematically evaluate molecular fidelity of tumor–model pairs.
- 3 Identify the translational utility of the HCMI resource.

# Translational utility of HCMI models

- The HCMI collection includes models that were derived from patients with **533 treatment events** prior to model generation representing **6 therapeutic classes**, encompassing **33 distinct mechanisms of action**.



# Resistance mutations across the HCMI resource

- Matched tumor–model pairs evaluated for resistance mutations using: Sanger resistance compendium, COSMIC, and known oncogenic driver hotspots
- TP53 emerged as the most frequently altered resistance-associated gene across both treatment-naïve and treated models, followed by RAS family mutations.
- **234 models** harboring resistance-associated variants documented in the COSMIC reference set or known driver gene hotspots.

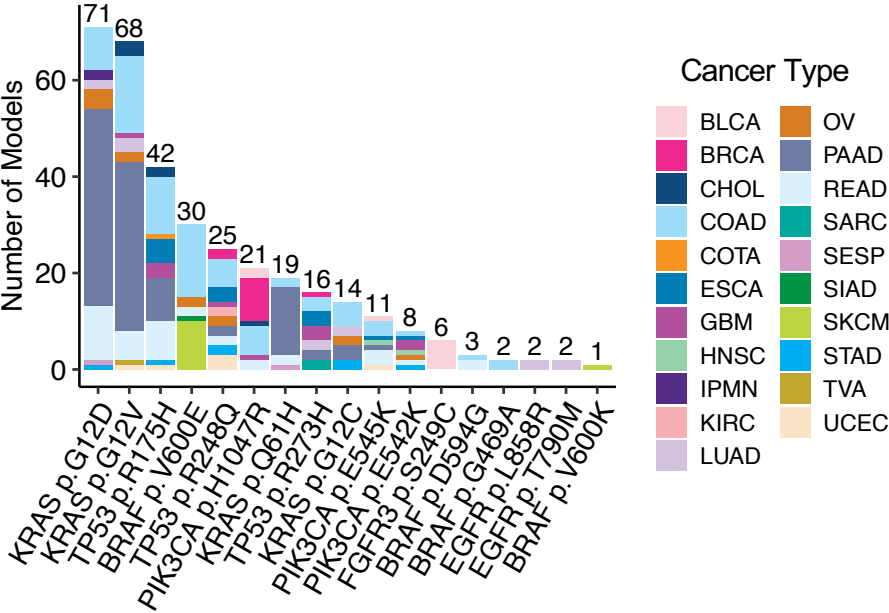
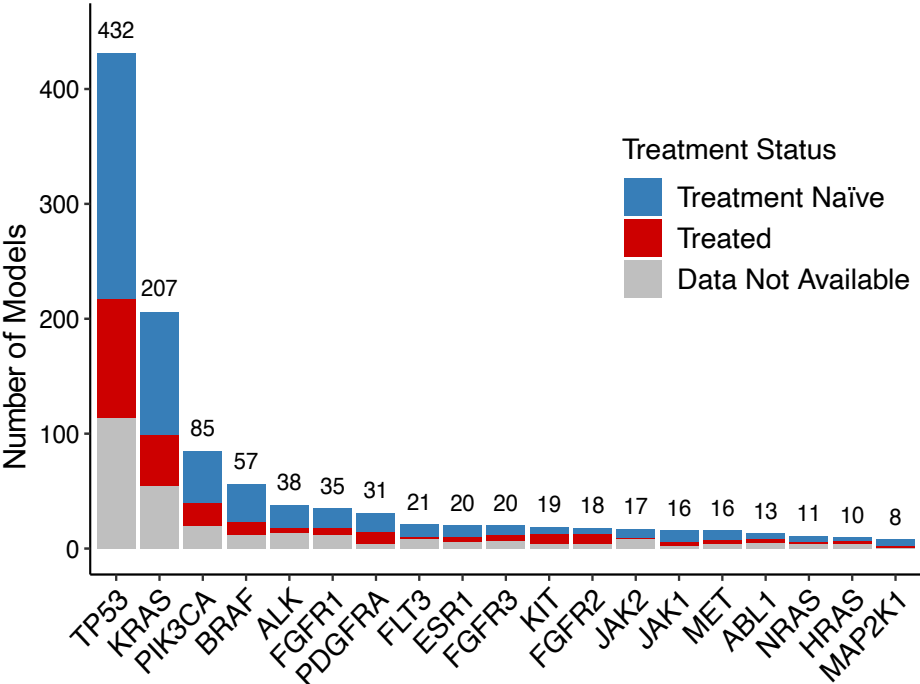


Article | [Open access](#) | Published: 18 October 2024

**Base editing screens define the genetic landscape of cancer drug resistance mechanisms**

[Matthew A. Coelho](#) , [Magdalena E. Strauss](#), [Alex Watterson](#), [Sarah Cooper](#), [Shriram Bhosle](#), [Giuditta Illuzzi](#), [Emre Karakoc](#), [Cansu Dincer](#), [Sara F. Vieira](#), [Mamta Sharma](#), [Marie Moullet](#), [Daniela Conticelli](#), [Jonas Koeppel](#), [Katrina McCarten](#), [Chiara M. Cattaneo](#), [Vivien Veninga](#), [Gabriele Picco](#), [Leopold Parts](#), [Josep V. Forment](#), [Emile E. Voest](#), [John C. Marioni](#), [Andrew Bassett](#) & [Mathew J. Garnett](#) 

[Nature Genetics](#) **56**, 2479–2492 (2024) | [Cite this article](#)



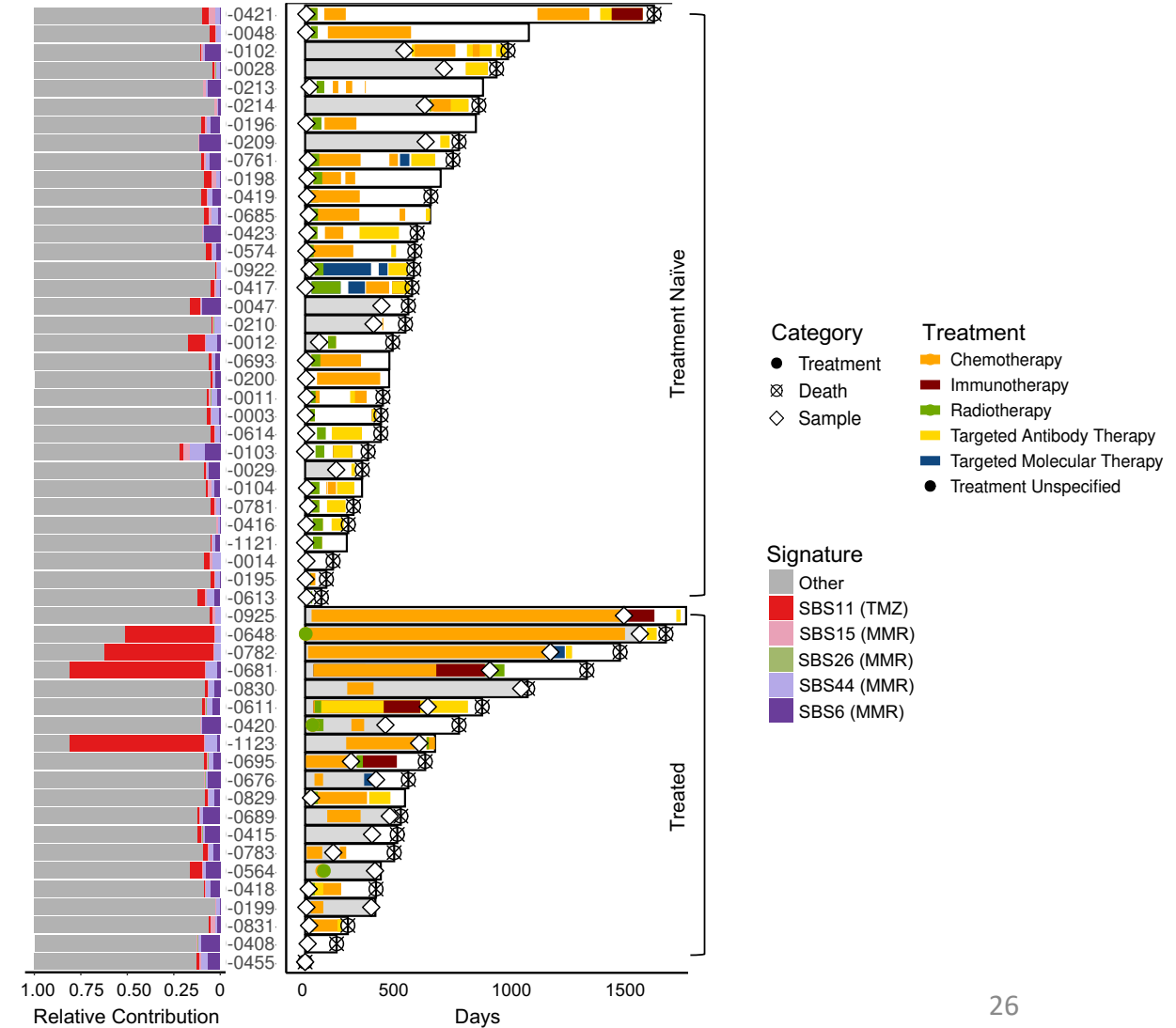
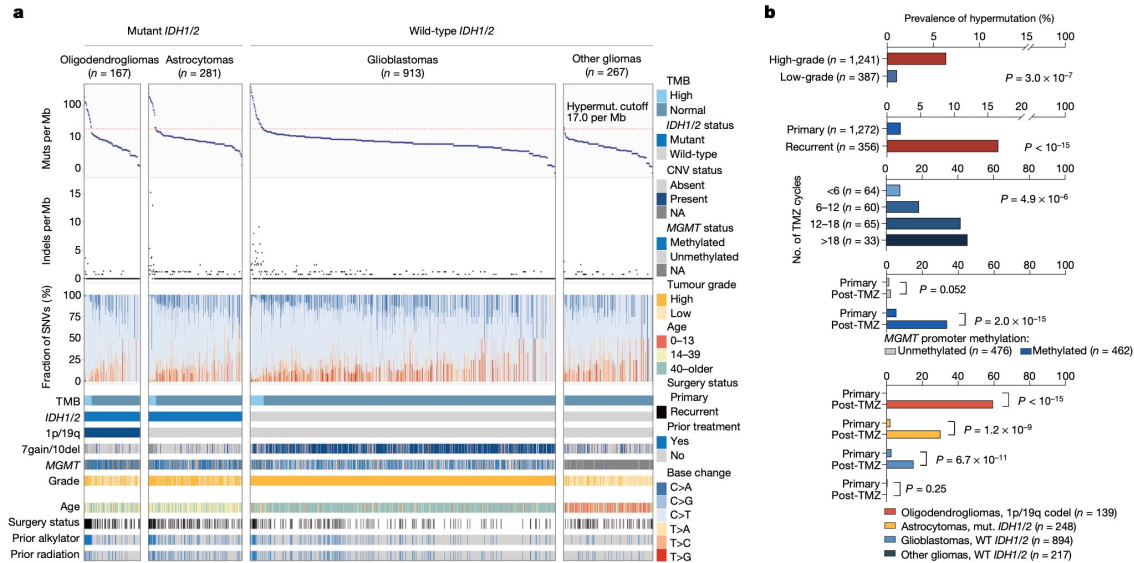
# Intrinsic and acquired TMZ resistance in HCGI GBM models

- GBM models revealed the presence of MMR mutational signatures linked to intrinsic resistance (SBSs 6, 15, 26, and 44) as well as acquired TMZ resistance (SBS 11) within the cohort.

## Mechanisms and therapeutic implications of hypermutation in gliomas

Mehdi Touat , Yvonne Y. Li, Adam N. Boynton, Liam F. Spurr, J. Bryan Iorgulescu, Craig L. Bohrsen, Isidro Cortes-Ciriano, Cristina Birzu, Jack E. Geduldig, Kristine Pelton, Mary Jane Lim-Fat, Sangita Pal, Ruben Ferrer-Luna, Shakti H. Ramkissoon, Frank Dubois, Charlotte Bellamy, Naomi Currimjee, Juliana Bonardi, Kenin Qian, Patricia Ho, Seth Malinowski, Leon Taquet, Robert E. Jones, Aniket Shetty, ... Keith L. Ligon  + Show authors

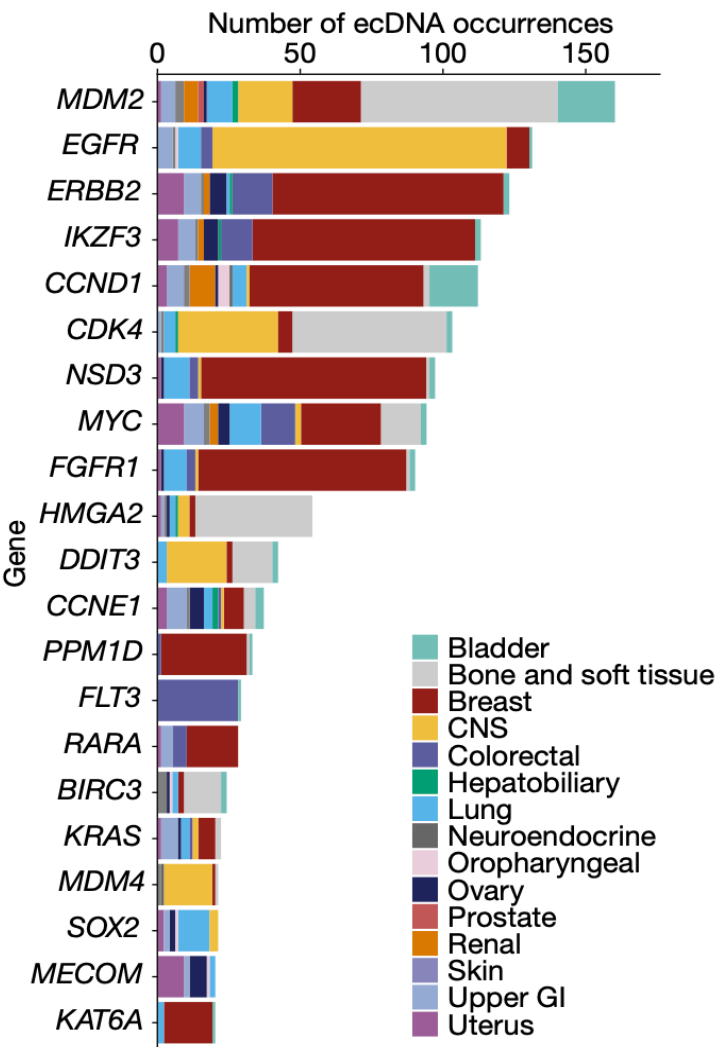
*Nature* 580, 517–523 (2020) | [Cite this article](#)



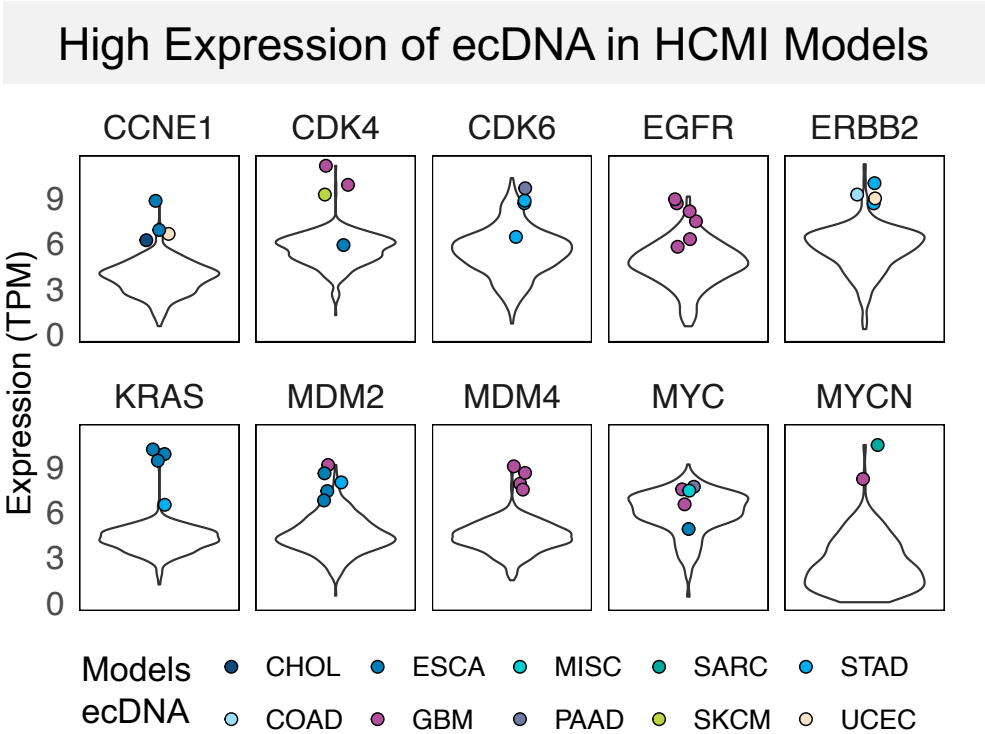
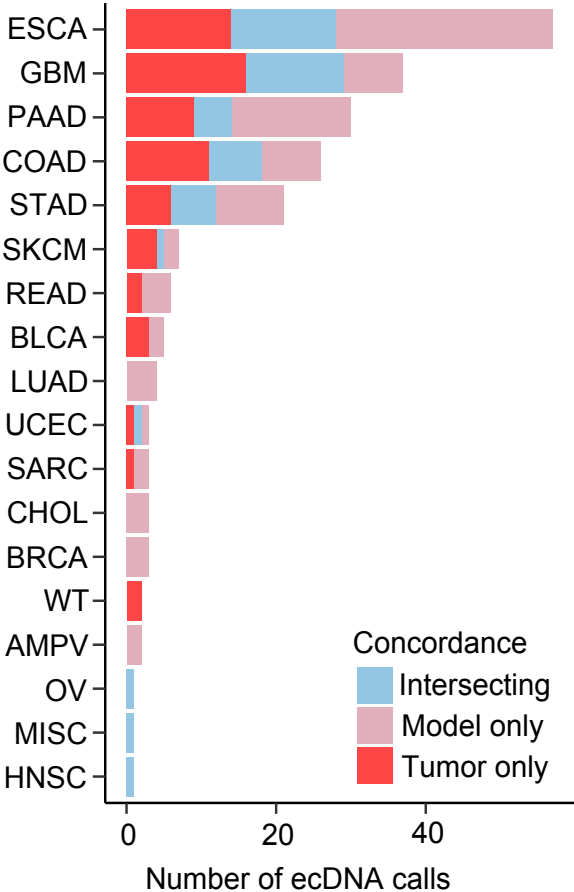


# Extrachromosomal DNA (ecDNA) amplifications in HCMI models

➤ ecDNA is a major contributor to treatment resistance and poor outcomes in cancer



➤ ~24% (n = 50) tumor-model pairs retained ecDNA amplification patterns



# HCMI Access/Data Availability: Searchable Catalog

Human Cancer Models Initiative

Searchable Catalog

Search By Model Name

Q

e.g. HCM-BROD-0051-C64, ...

Search By Altered Gene(s)

Q

e.g. BRAF, EWSR, ...

Search By Research Somatic Variant

Q

e.g. BRAF V600E, IDH1 R132H, ...

Primary Site

☐ Colon

55

☐ Pancreas

52

☐ Brain

50

☐ Esophagus

36

24 More

Research Somatic Variant Type

☐ Missense Mutation

286

☐ Silent

286

☐ Nonsense Mutation

275

☐ Intron

258

13 More

Consequence

☐ Missense Variant

286

☐ Synonymous Variant

286

☐ Frameshift Variant

273

☐ Stop Gained

273

65 More

← Use the filter panel on the left to customize your model search.

SHARE

VIEW LIST

Models By Primary Site

28

Total

Has Multiple Models

2D Versus 3D Growth

Most Frequently Mutated Genes

# models affected

187

0

TP53

TTN

KRAS

SYN

MUC16

APC

PCLO

OBSCN

ZHFX4

ILG

Showing 1 - 20 of 316 models

Include 272 unexpanded models

COLUMNS

EXPORT

| <input type="checkbox"/> | Name                              | Primary Site      | Clinical Tumor Diagnosis | Tissue Status | Age At Acquisition (Years) | Age At Diagnosis (Years) | Has Multiple Models | Expansion Status | # Mutated Genes | # Res. Somatic Variants |
|--------------------------|-----------------------------------|-------------------|--------------------------|---------------|----------------------------|--------------------------|---------------------|------------------|-----------------|-------------------------|
| <input type="checkbox"/> | <a href="#">HCM-BROD-0648-C71</a> | Brain             | Glioblastoma             | Recurrent     | 68                         | 63                       | No                  | EXPANDED         | 5326            | 711                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0227-C43</a> | Skin              | Melanoma                 | Metastasis    | 40                         | 40                       | No                  | EXPANDED         | 3075            | 418                     |
| <input type="checkbox"/> | <a href="#">HCM-SANG-0288-C18</a> | Colon             | Colorectal cancer        | Primary       | 75                         |                          | No                  | EXPANDED         | 3228            | 390                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0569-C43</a> | Skin              | Melanoma                 | Metastasis    | 79                         | 78                       | No                  | EXPANDED         | 2886            | 380                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0594-C43</a> | Skin              | Melanoma                 | Metastasis    | 78                         | 75                       | No                  | EXPANDED         | 2690            | 355                     |
| <input type="checkbox"/> | <a href="#">HCM-CSHL-0426-C18</a> | Colon             | Colorectal cancer        | Primary       | 73                         | 72                       | No                  | EXPANDED         | 2701            | 318                     |
| <input type="checkbox"/> | <a href="#">HCM-SANG-0273-C18</a> | Colon             | Colorectal cancer        | Primary       | 78                         |                          | No                  | EXPANDED         | 2597            | 299                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0027-C34</a> | Bronchus and Lung | Lung cancer              | Metastasis    | 66                         | 65                       | No                  | EXPANDED         | 2313            | 286                     |
| <input type="checkbox"/> | <a href="#">HCM-CSHL-0459-C17</a> | Small intestine   | Rare cancers             | Primary       | 57                         | 57                       | No                  | EXPANDED         | 2426            | 279                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0223-C43</a> | Skin              | Melanoma                 | Metastasis    | 74                         | 73                       | No                  | EXPANDED         | 2187            | 267                     |
| <input type="checkbox"/> | <a href="#">HCM-SANG-0282-C18</a> | Colon             | Colorectal cancer        | Primary       | 85                         |                          | No                  | EXPANDED         | 2313            | 263                     |
| <input type="checkbox"/> | <a href="#">HCM-BROD-0724-C43</a> | Skin              | Melanoma                 | Metastasis    | 74                         | 74                       | No                  | EXPANDED         | 2013            | 253                     |

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