Targeting the Chromatin Regulatory System

Broadening the Impact of Precision Medicines for Oncology and Other Diseases

FOGHORN® THERAPEUTICS

Research & Development Webinar

June 15th, 2021

Agenda

9:00 – 9:10 a.m.	**Welcome and Introduction** Adrian Gottschalk, President and CEO, Foghorn Therapeutics
9:10 – 9:25 a.m.	**Chromatin Regulatory System – Disease Relevance** Cigall Kadoch, Ph.D., Broad Institute, DFCI, Scientific Co-Founder Foghorn Therapeutics
9:25 – 9:45 a.m.	**Gene Traffic Control Platform** Steve Bellon, Ph.D, SVP, Head of Drug Discovery, Foghorn Therapeutics
9:45 – 10:05 a.m.	**FHD-286 – Clinical Applications of Dual BRM/BRG1 Inhibition** Sam Agresta, MP, MPH & TM, CMO, Foghorn Therapeutics
10:05 – 10:20 a.m.	**Acute Myeloid Leukemia** Eytan Stein, M.D., Memorial Sloan Kettering Cancer Center
10:20 – 10:30 a.m.	**Synovial Sarcoma – FHD-609** Sam Agresta, MP, MPH & TM, CMO, Foghorn Therapeutics
10:30 – 10:45 a.m.	**Targeting the Chromatin Regulatory System in Cancer** Howard “Skip” Burris III, MD, FASCO, FACP, Sara Cannon Research Institute
10:45 – 11:00 a.m.	**Q&A**

Welcome and Introduction

Adrian Gottschalk

Chief Executive Officer, Foghorn Therapeutics

Forward-Looking Statements

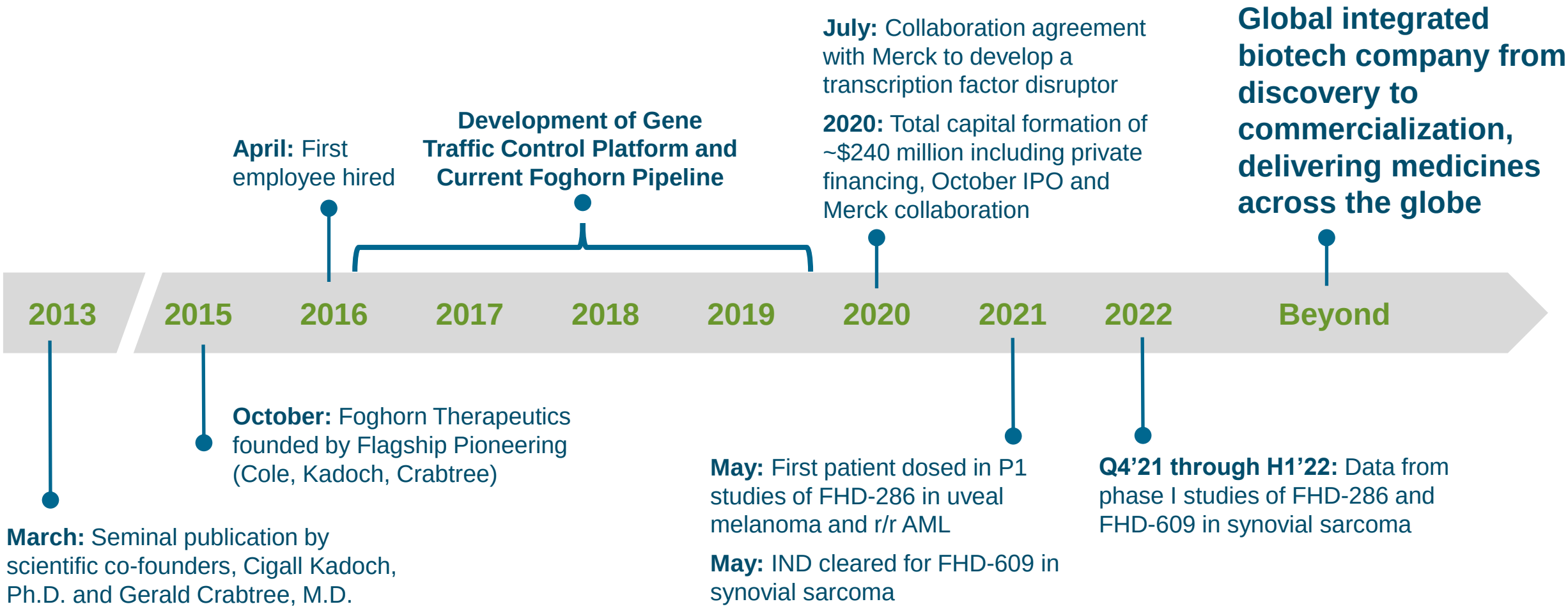
This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplate,” “believe,” “estimate,” “predict,” “potential” or “continue” or the negative of these terms or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements include, but are not limited to, statements concerning: the initiation, timing, progress and results of our research and development programs and preclinical and clinical trials, our ability to advance product candidates that we may develop and successfully complete preclinical and clinical studies; our ability to leverage our initial programs to develop additional product candidates using our Gene Traffic Control Platform; the impact of the COVID-19 pandemic in our and our collaborators’ business operations, including our research and development programs and preclinical studies; developments related to our competitors and our industry; our ability to expand the target populations of our programs and the availability of patients for clinical testing; our ability to obtain regulatory approval for FHD-286, FHD-609 and any future product candidates from the FDA and other regulatory authorities; our ability to identify and enter into future license agreements and collaborations; our ability to continue to rely on our CDMOs and CROs for our manufacturing and research needs; regulatory developments in the United States and foreign countries; our ability to attract and retain key scientific and management personnel; the scope of protection we are able to establish, maintain and enforce for intellectual property rights covering FHD-286 and FHD-609, our future products and our Gene Traffic Control Platform; and our use of proceeds from our initial public offering, estimates of our expenses, capital requirements and needs for additional financing. Any forward-looking statements represent the Company’s views only as of today and should not be relied upon as representing its views as of any subsequent date. The Company explicitly disclaims any obligation to update any forward-looking statements. The Company’s business is subject to substantial risks and uncertainties which are described under the heading "Risk Factors" in our Annual Report on Form 10-K filed with the U.S. Securities and Exchange Commission and are accessible on the SEC's website at www.sec.gov.

The Foghorn Commitment

OUR PLEDGE

At Foghorn, we pledge to partner with patients as we work to discover and develop new, effective therapies for a wide spectrum of diseases, including many types of cancer. Every member of our team is committed to making a difference in the lives of others.

Broadening the Impact of Precision Medicines for Oncology and Other Diseases: Targeting the Chromatin Regulatory System



FOGHORN THERAPEUTICS

Dysregulation of the Chromatin Regulatory System Has Been Implicated in up to 50% of All Cancers

50%

of All Cancers

Based on exome sequencing, the chromatin regulatory system is implicated in ~50% of all cancers

2.5M

People

Potential impact for Foghorn's pipeline of precision oncology programs

$400+

billion

2030 global oncology market opportunity

First Two Programs in the Clinic, Broad Pipeline Advancing

Precision Oncology / Breadth and Depth

Program / Target	Modality	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3	Global Rights
FHD-286 (BRG1 / BRM)	Enzyme inhibitor	AML		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
		Uveal melanoma		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
FHD-609 (BRD9)	Protein degrader	Synovial sarcoma		Early Clinical Data (H1 2022)			FOGHORN THERAPEUTICS
Selective BRM	I) Enzyme inhibitor	BRG1 mutated cancers	IND 2022				FOGHORN THERAPEUTICS
	II) Protein degrader	BRG1 mutated cancers					
Selective ARID1B	Protein degrader	ARID1A mutated cancers					FOGHORN THERAPEUTICS
Synthetic Lethal Targets (multiple)	I) Enzyme inhibitors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Transcription Factors (multiple)	I) Transcription factor disruptors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Partnered program (undisclosed)	Transcription factor disruptor						MERCK

Gene Traffic Control® Platform

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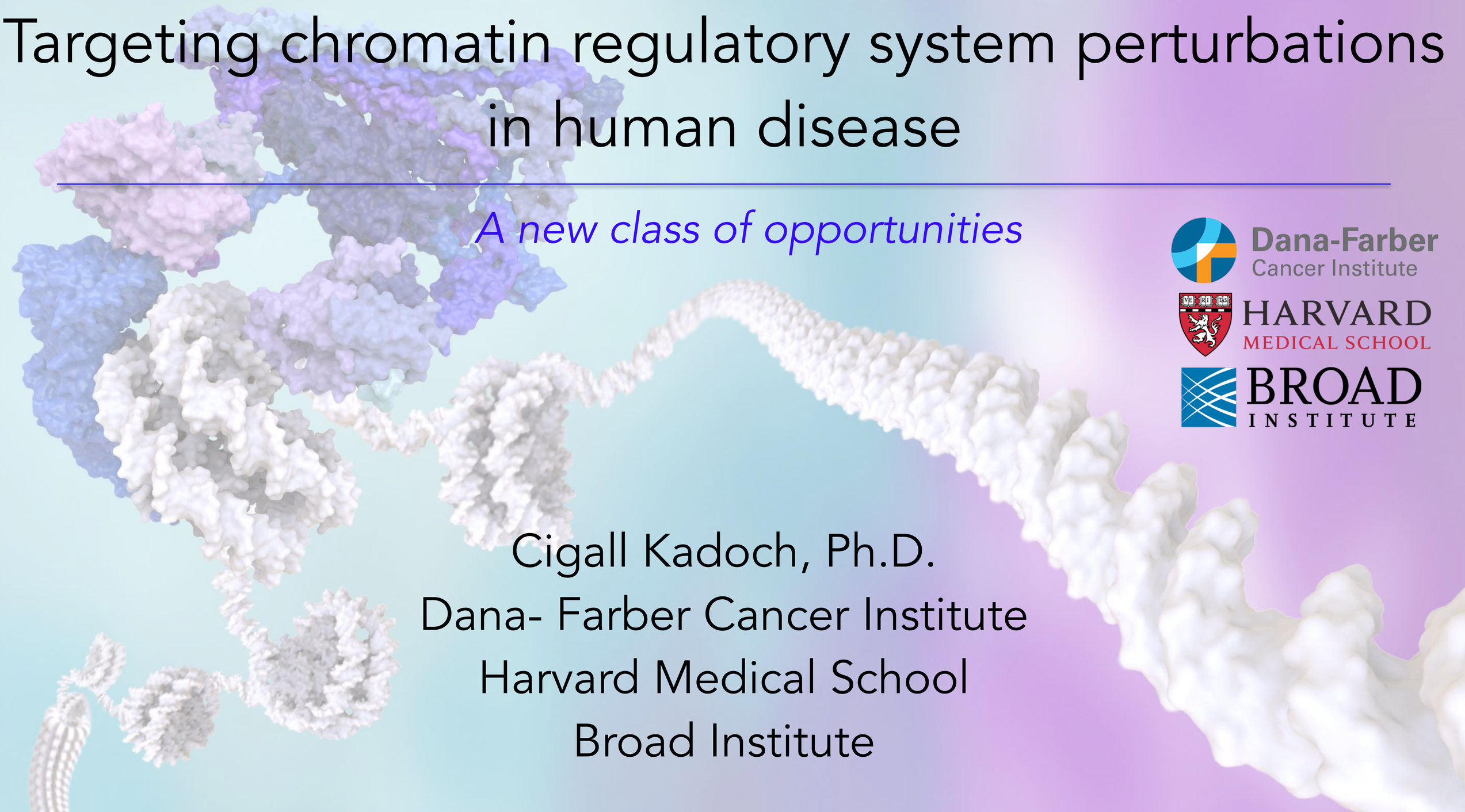
Chromatin Regulatory System – Disease Relevance

Cigall Kadoch, Ph.D.

Broad Institute, DFCI, Scientific Co-Founder of Foghorn Therapeutics

Targeting chromatin regulatory system perturbations in human disease

A new class of opportunities



Cigall Kadoch, Ph.D.

Dana- Farber Cancer Institute

Harvard Medical School

Broad Institute

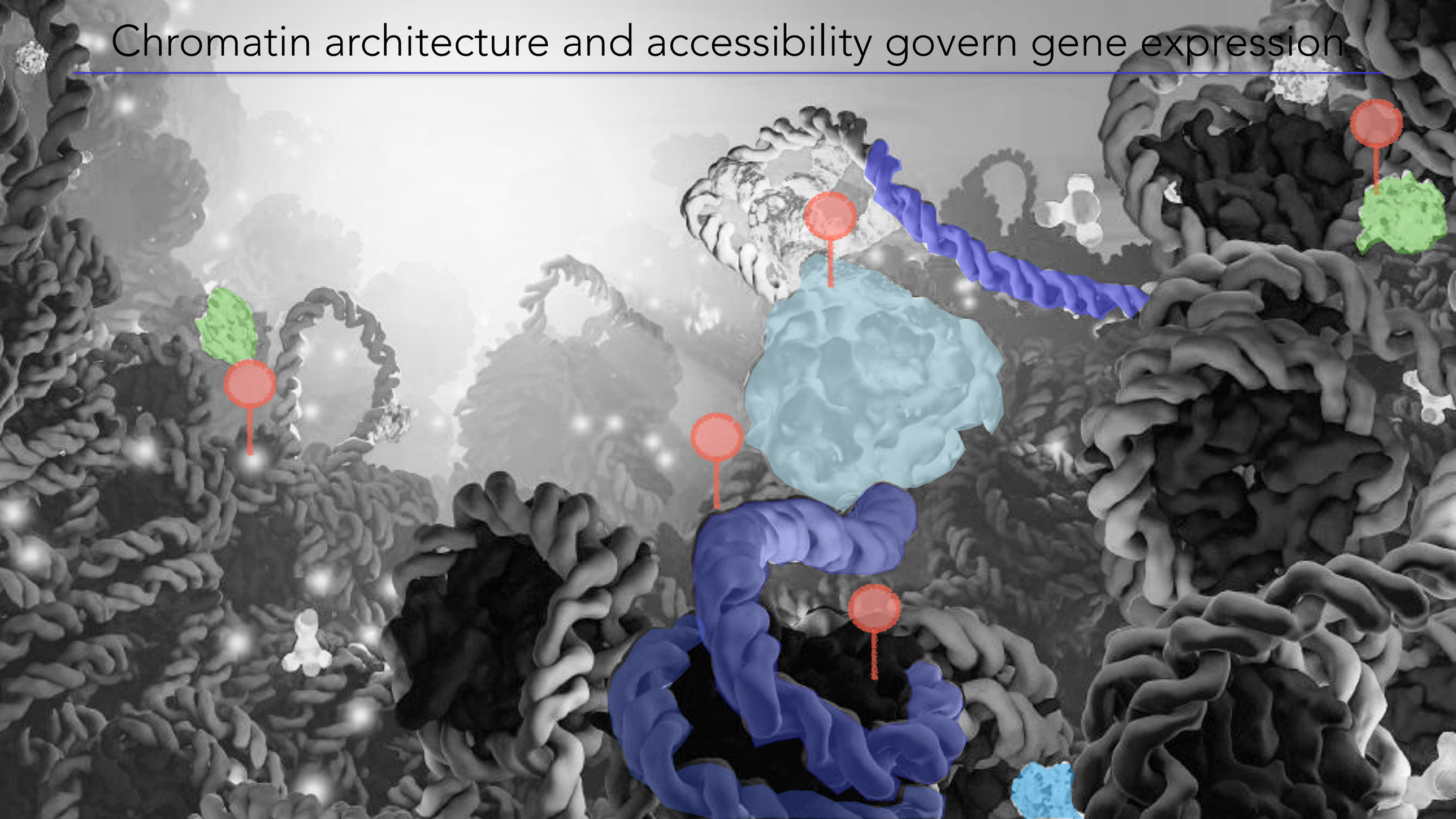
Chromatin architecture and accessibility govern gene expression

V. Attoulian, Science

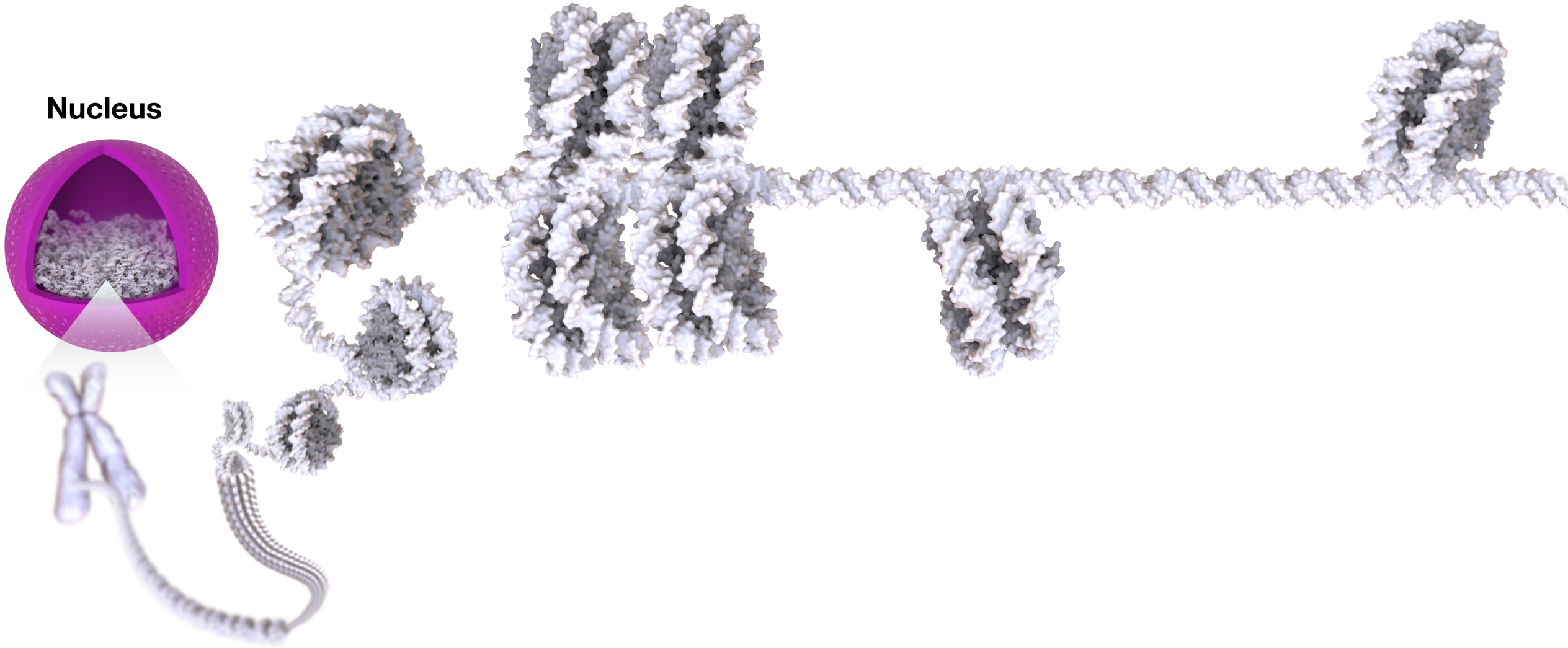
Chromatin architecture and accessibility govern gene expression

Chromatin architecture and accessibility govern gene expression

Chromatin architecture and accessibility govern gene expression



The chromatin regulatory system

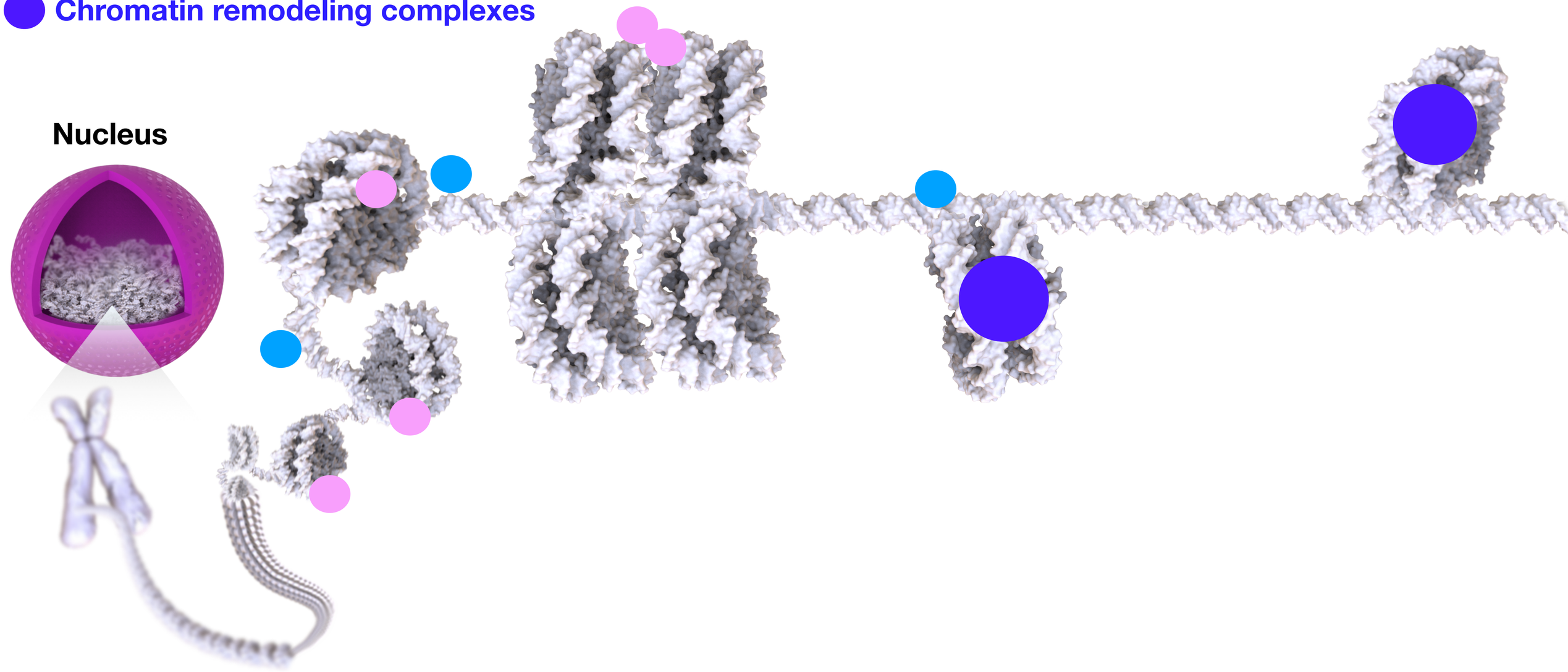


The chromatin regulatory system

DNA modification

Histone modification

Chromatin remodeling complexes



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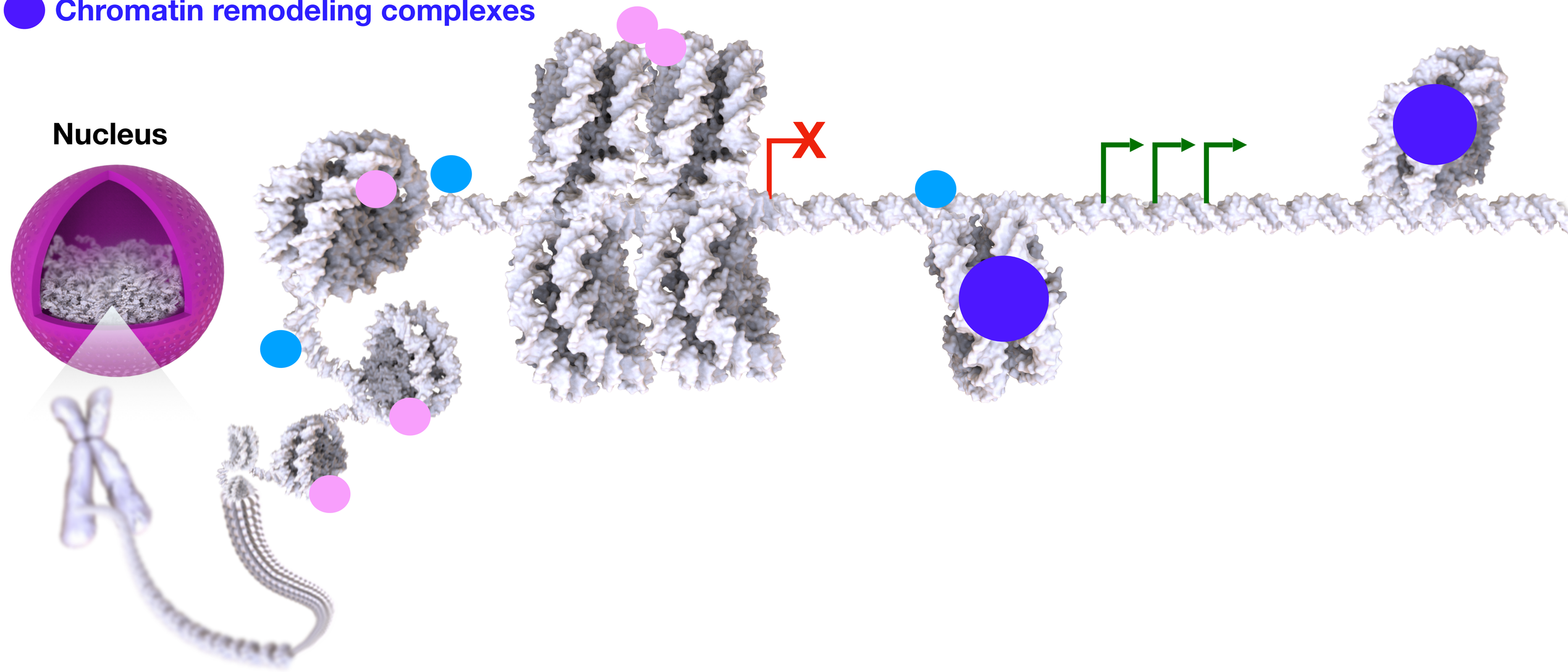
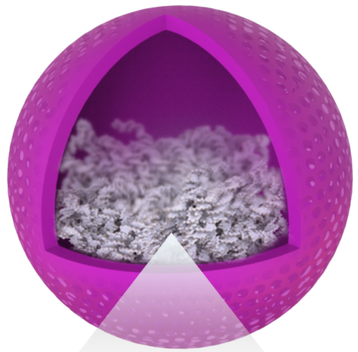


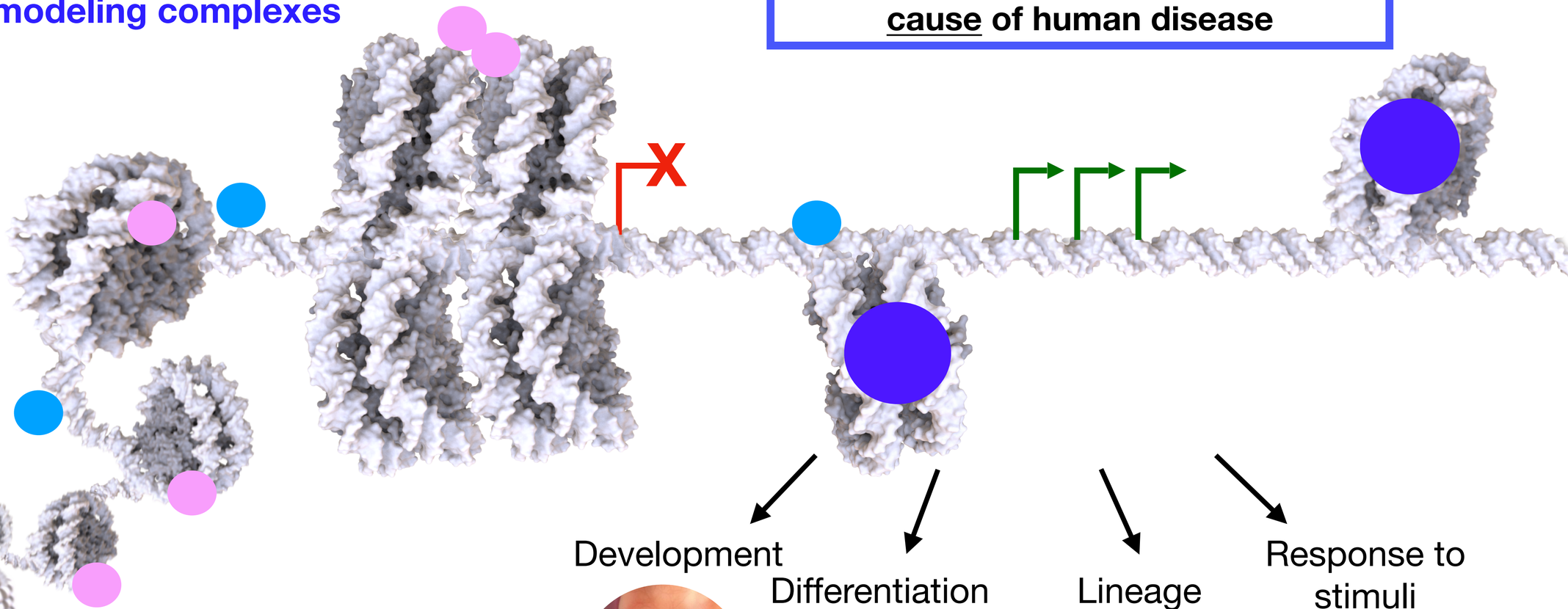
The chromatin regulatory system

- **DNA modification**
- **Histone modification**
- **Chromatin remodeling complexes**

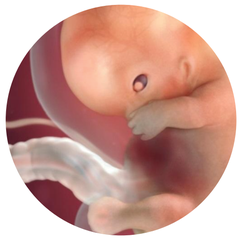
Defects in the control of chromatin structure are a major <u>cause</u> of human disease

Nucleus

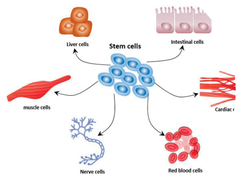




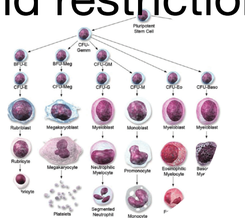
Development



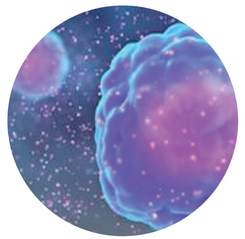
Differentiation



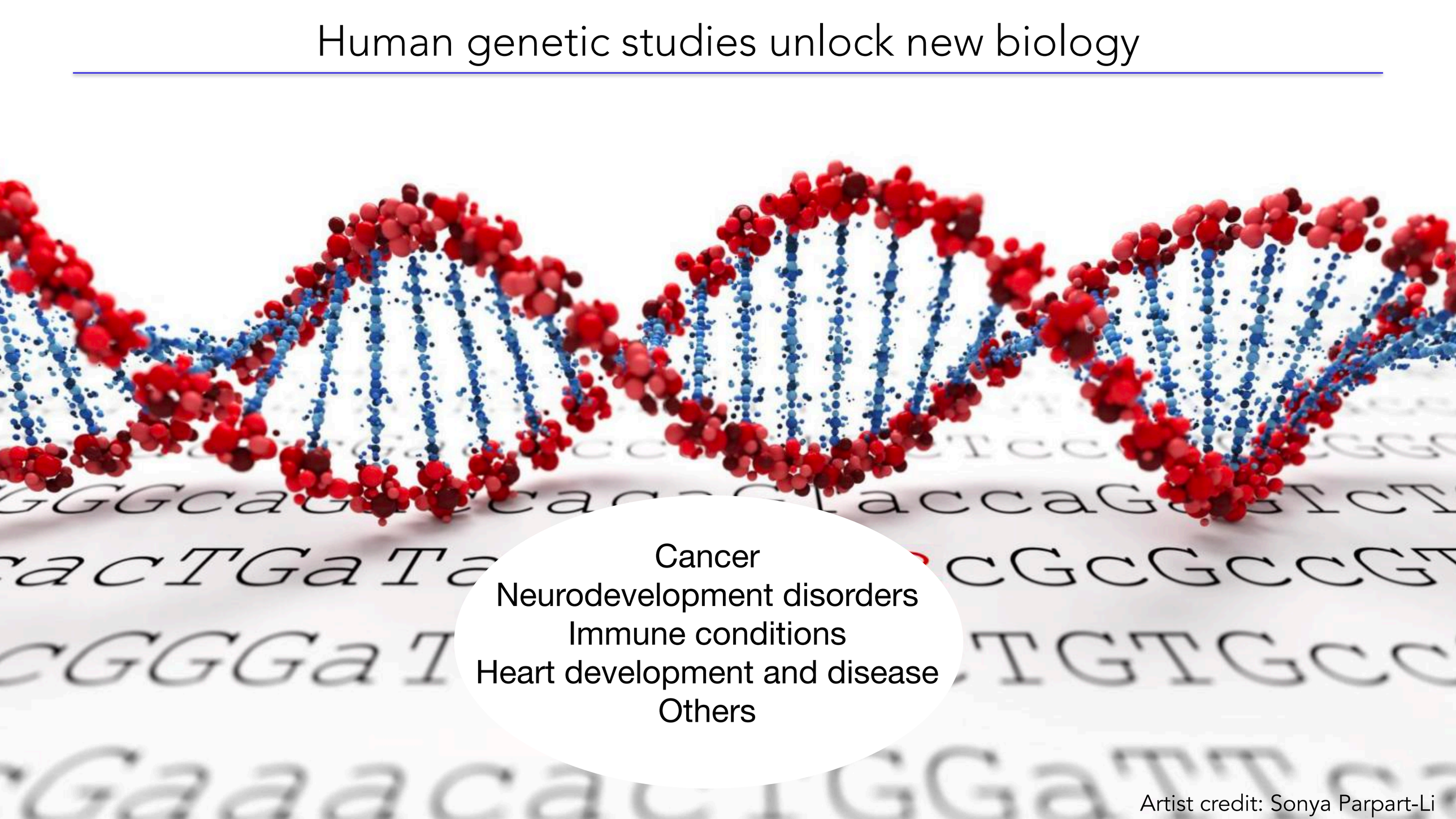
Lineage commitment and restriction



Response to stimuli



Human genetic studies unlock new biology



Cancer
Neurodevelopment disorders
Immune conditions
Heart development and disease
Others

Artist credit: Sonya Parpart-Li

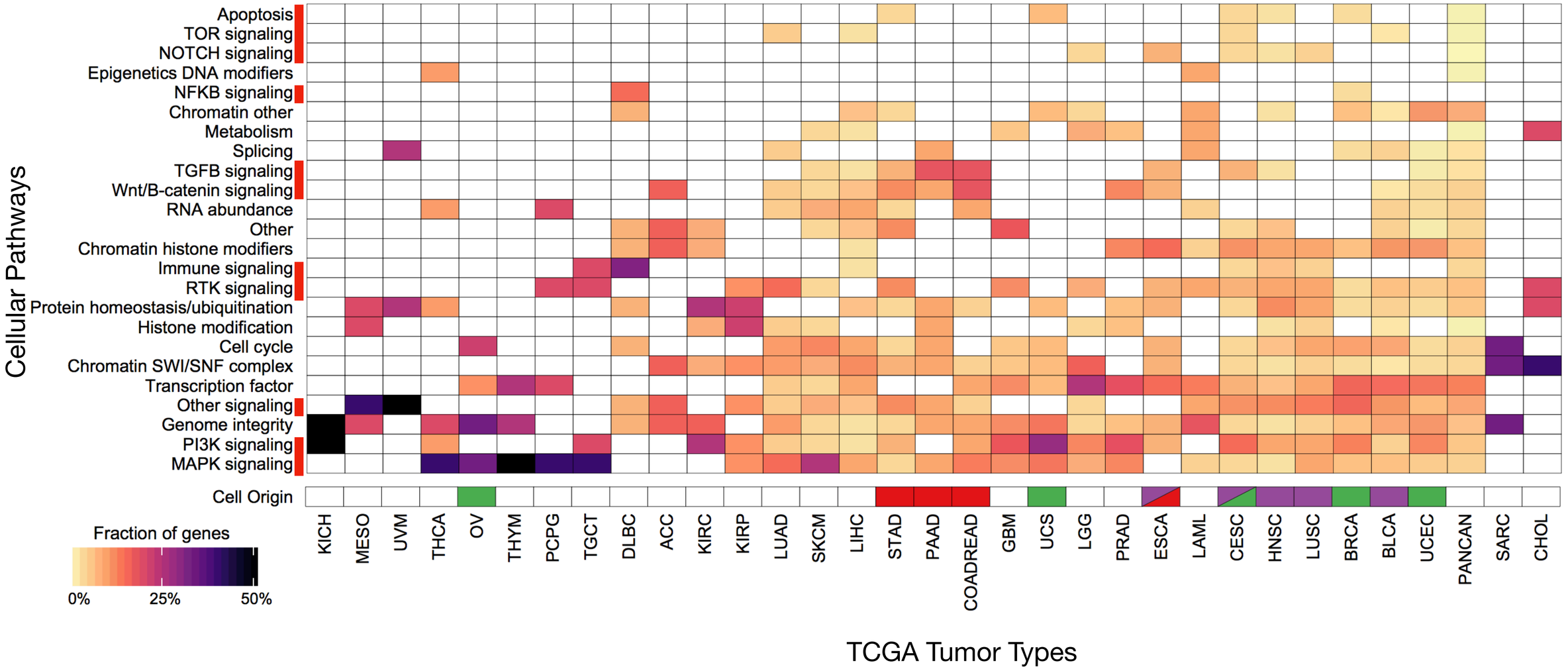
Cancer mutations span multiple cellular pathways

30+ Cancer Types

>10,000 tumor samples sequenced

~300 driver genes

>3,400 putative missense driver mutations



Adapted from Bailey et al., *Cell* 2018

Chromatin regulatory system disruptions are extensive

30+ Cancer Types

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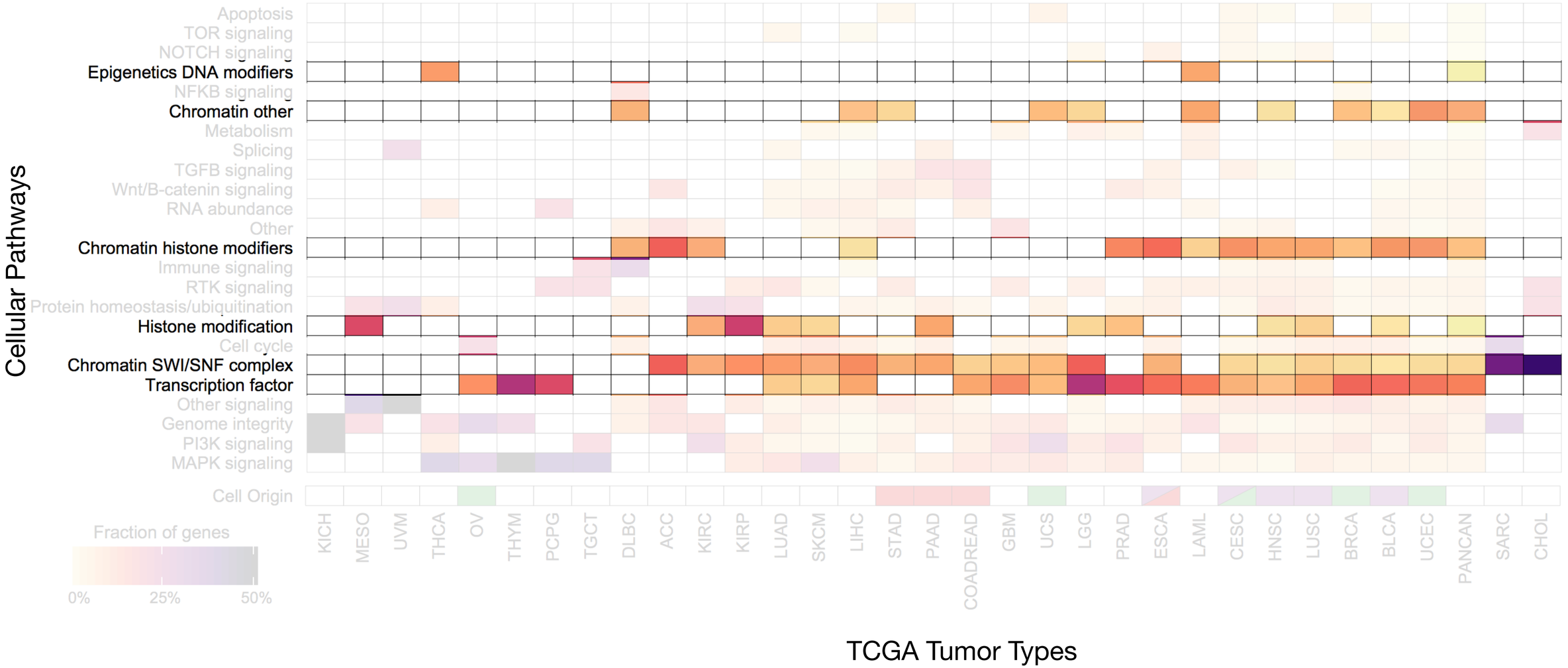

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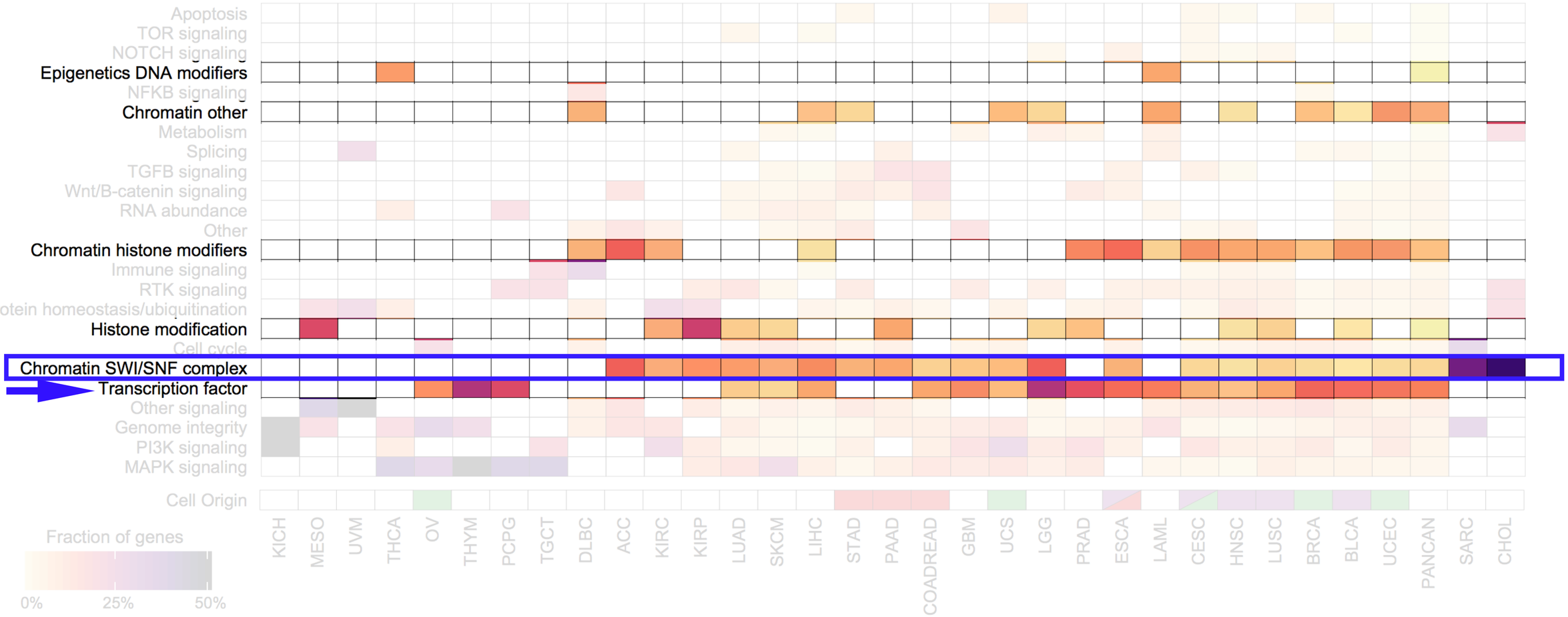
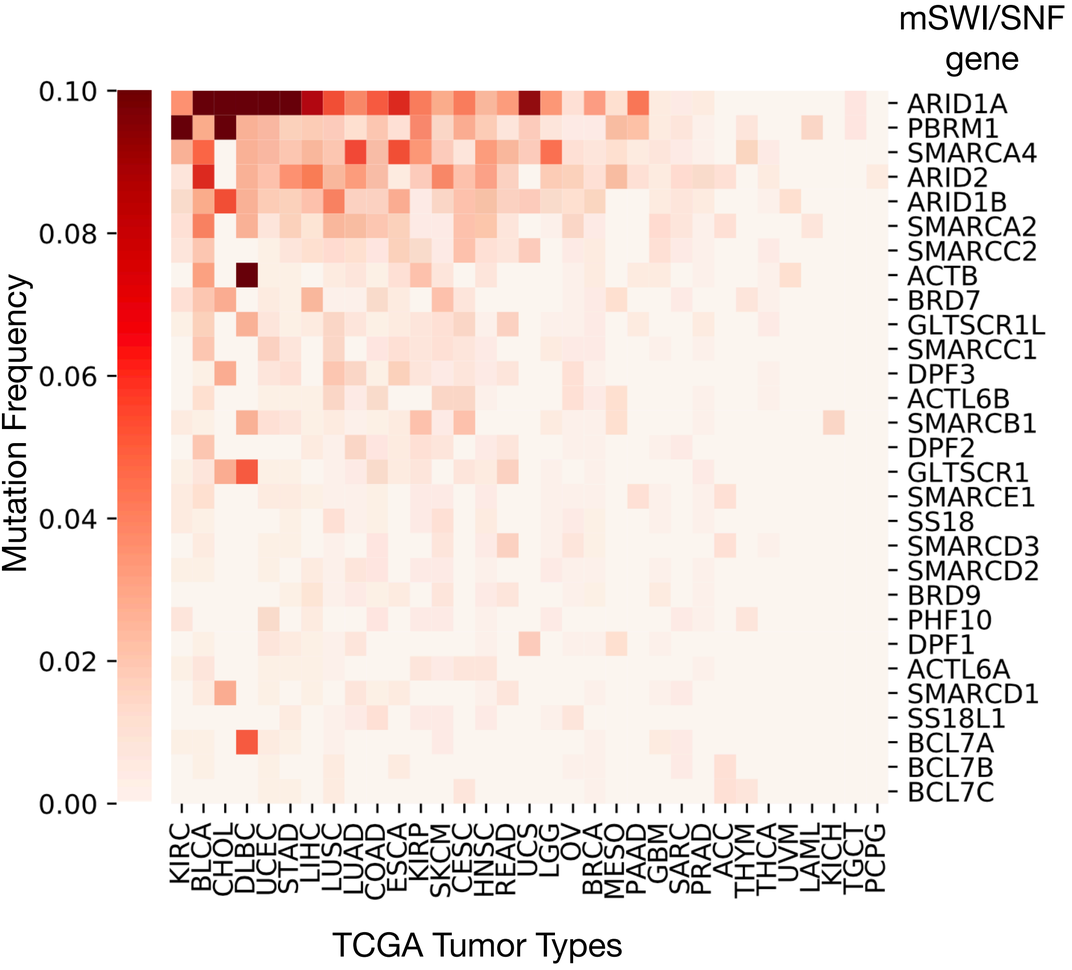


SWI/SNF complexes, other ATP-dependent remodelers, helicases, etc.

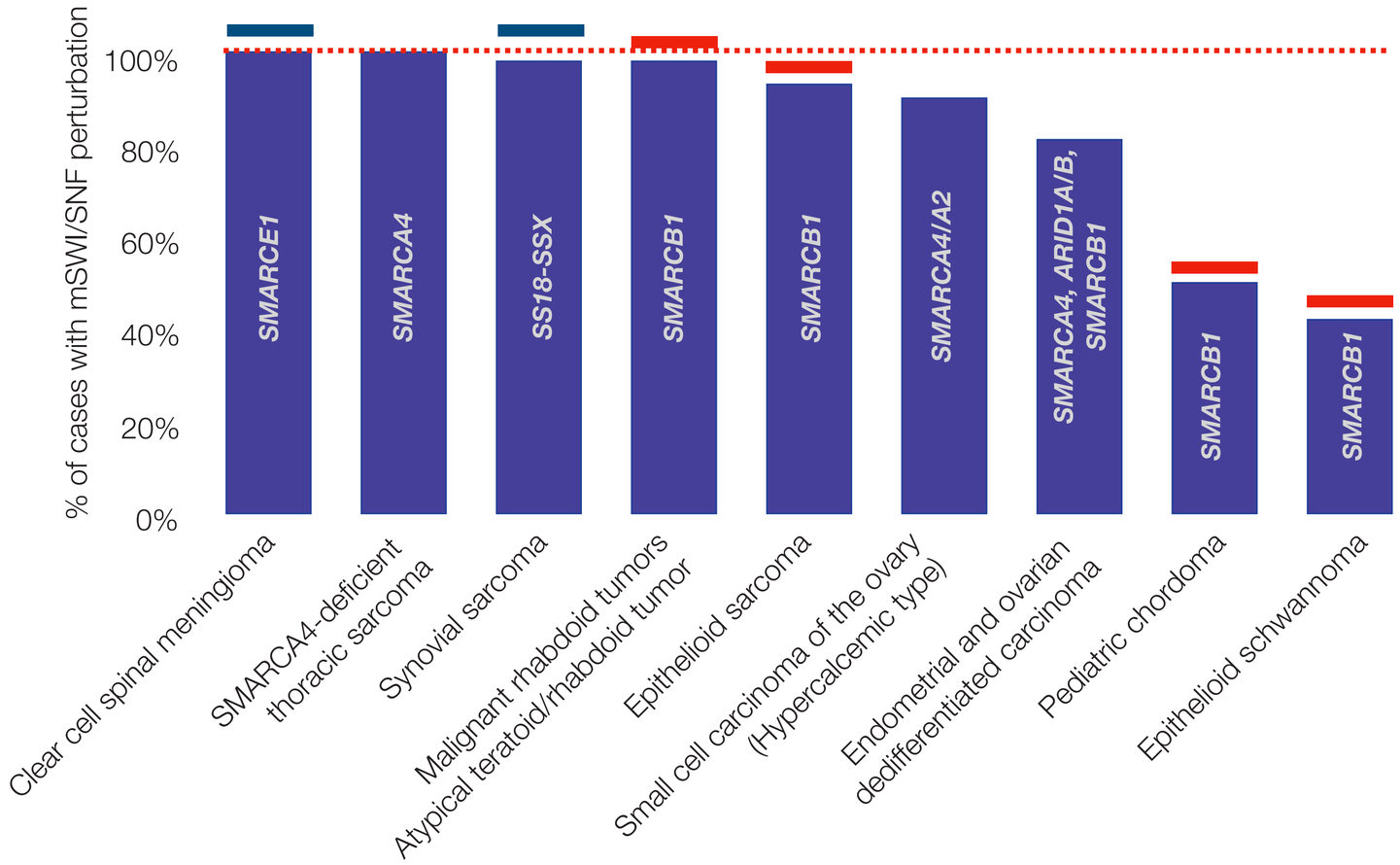
Adapted from Bailey et al., *Cell* 2018

An example: mutational landscape of mSWI/SNF genes in human cancer

~20+% of human cancers



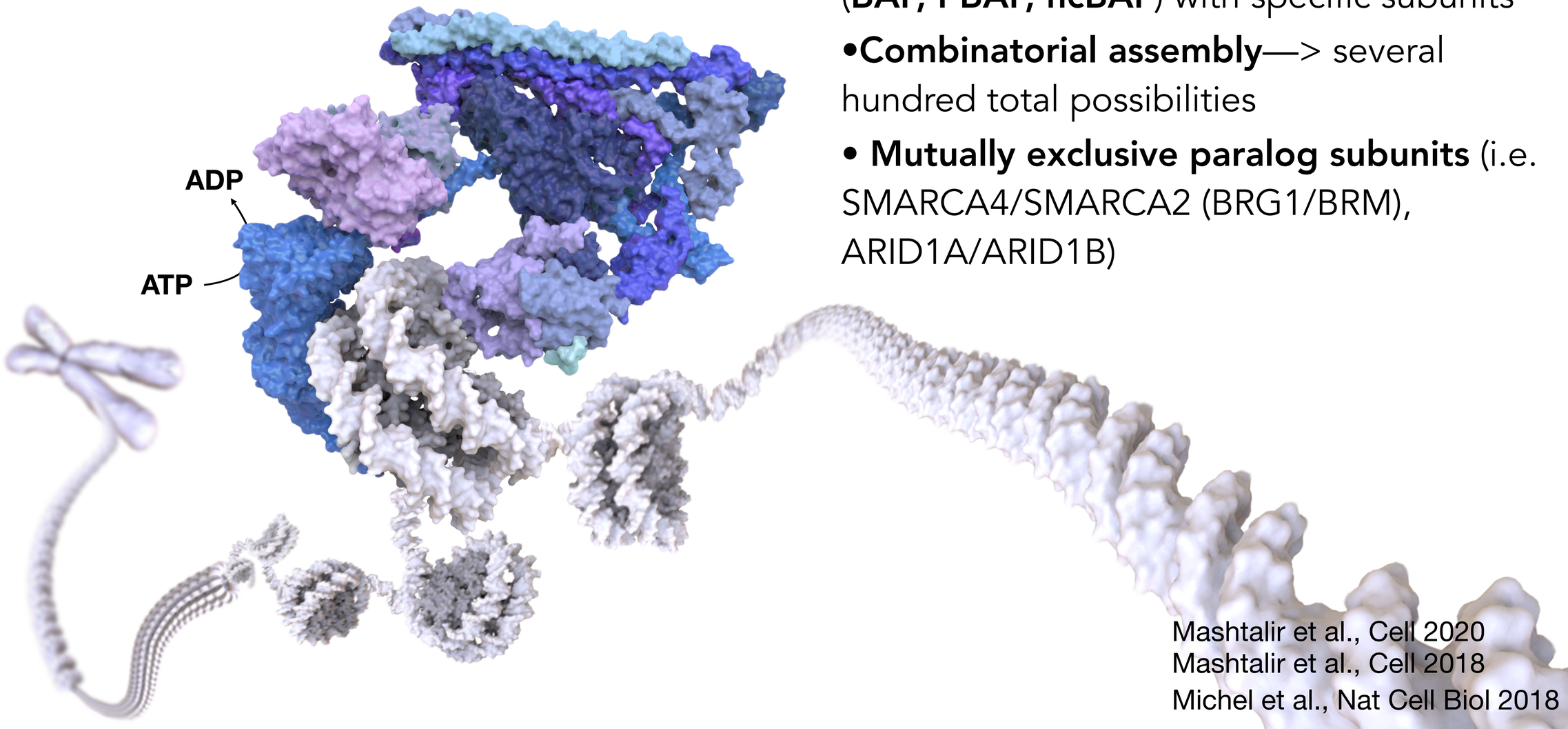
Drivers of rare cancers (many 90-100% of cases)



Mammalian SWI/SNF complexes: chromatin remodeling machines

ARID1A
ARID1B
ARID2
SMARCA4
SMARCA2
SMARCC1
SMARCC2
SMARCD1
SMARCD2
SMARCD3
SMARCE1
SMARCB1
PBRM1
DPF1
DPF2
DPF3
PHF10
BRD7
BRD9
SS18
SS18L1
ACTL6A
ACTL6B
ACTB
GLTSCR1
GLTSCR1L
BCL7A
BCL7B
BCL7C

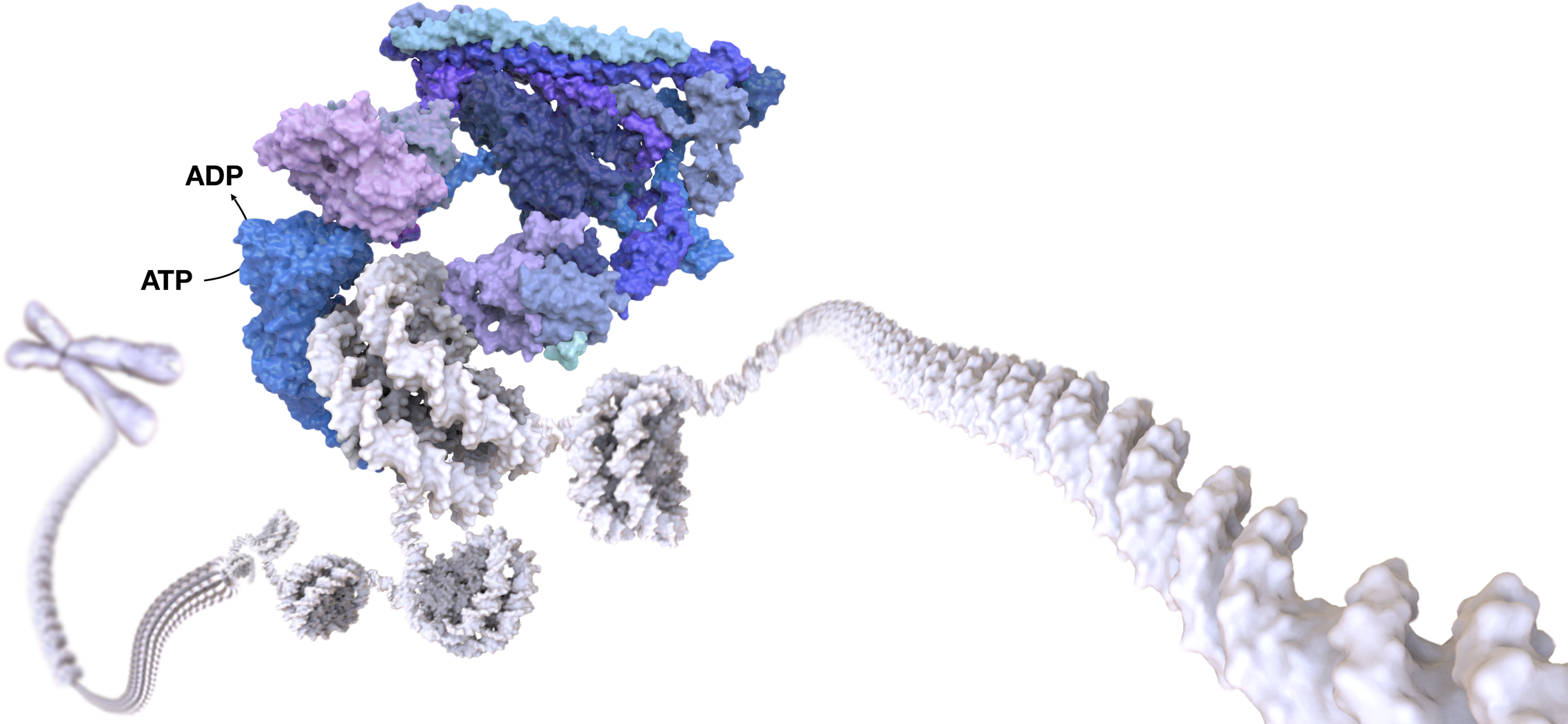
mSWI/SNF (BAF) complex



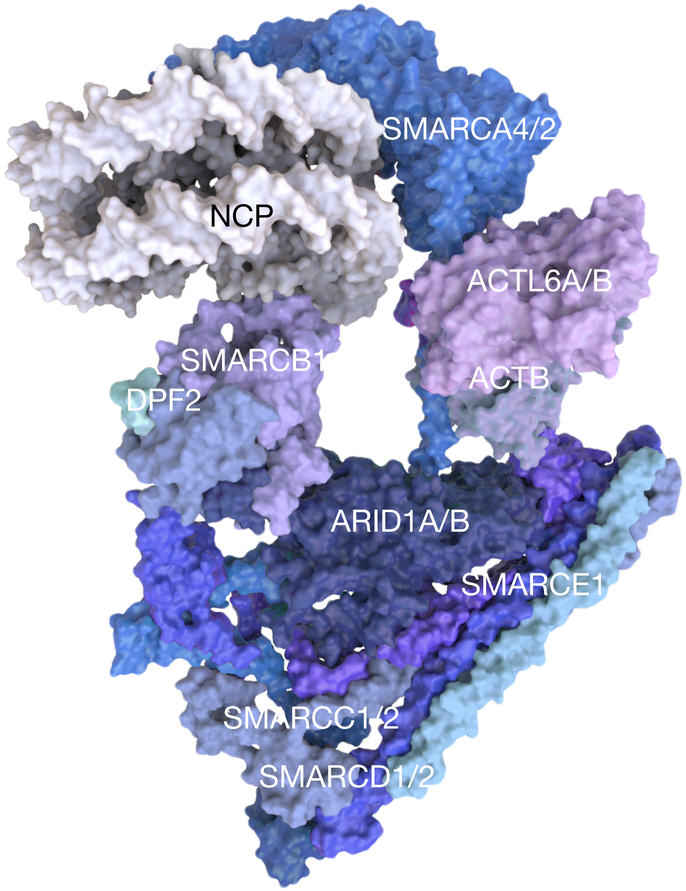
- Three distinct final forms of complexes (**BAF, PBAF, ncBAF**) with specific subunits
- **Combinatorial assembly**—> several hundred total possibilities
- **Mutually exclusive paralog subunits** (i.e. SMARCA4/SMARCA2 (BRG1/BRM), ARID1A/ARID1B)

Mashtalir et al., Cell 2020
Mashtalir et al., Cell 2018
Michel et al., Nat Cell Biol 2018

Mammalian SWI/SNF complexes: chromatin remodeling machines



mSWI/SNF family complexes: cBAF, PBAF, and ncBAF types



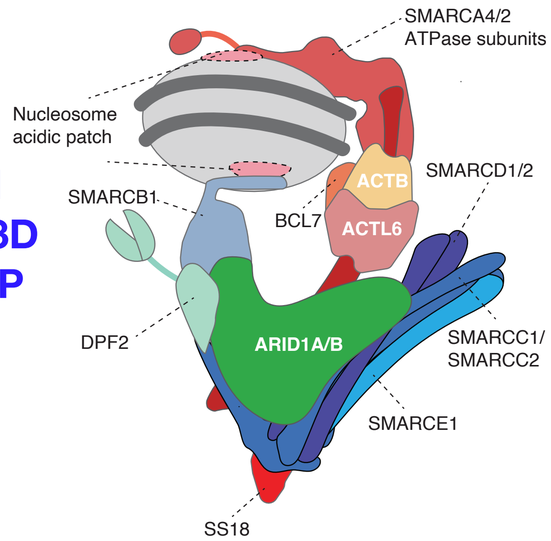
Mashtalir et al., *Cell* 2018
Michel et al., *Nat Cell Biol* 2018

mSWI/SNF: diverse assemblies yield diverse opportunities for targeting

Biochemical composition, 3D structure, NCP interactions

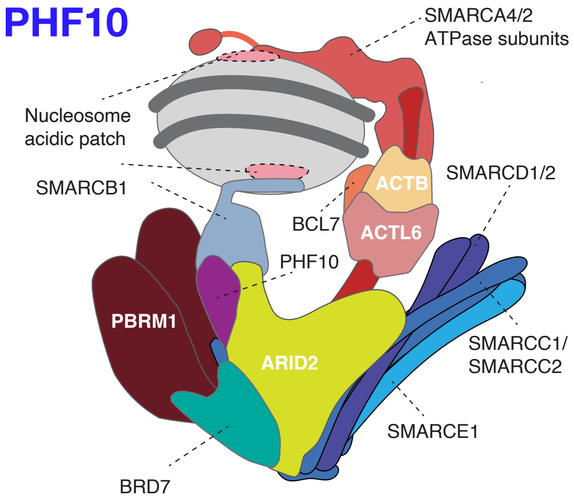
ARID1/B
DPF2

cBAF
1.04 MDa



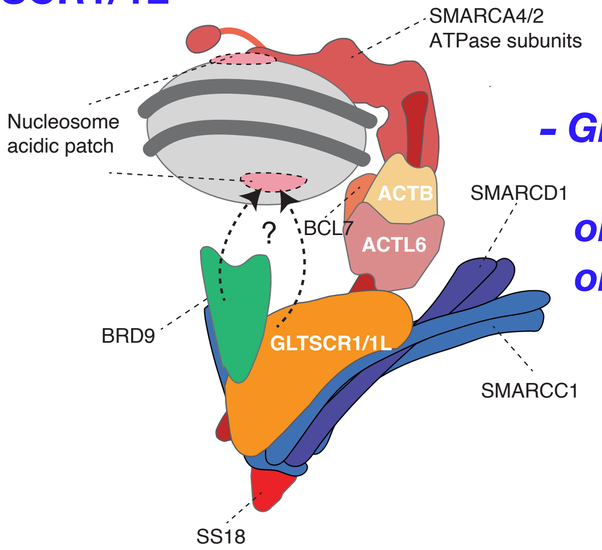
ARID2
PBRM1
PHF10

PBAF
1.41 MDa



BRD9
GLTSCR1/1L

ncBAF
0.87MDa



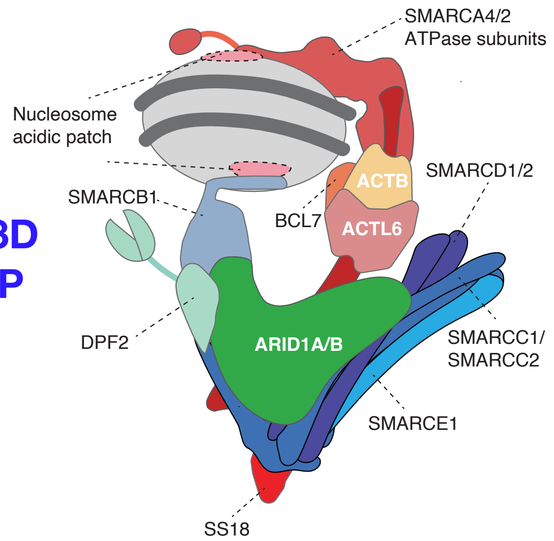
-SMARCB1
-SMARCE1
- GLTSCR1 instead of ARID
only SMARCD1
only SMARCC1

Mashtalir et al., *Cell* 2018
Michel et al., *Nat Cell Biol* 2018

Compositional and functional diversity of mSWI/SNF complexes

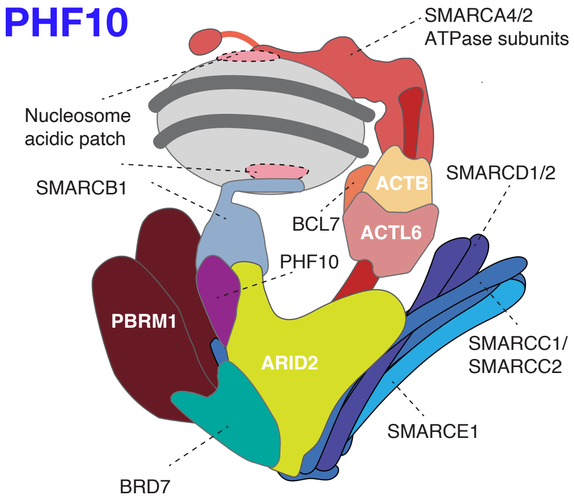
ARID1/B
DPF2

cBAF
1.04 MDa



ARID2
PBRM1
PHF10

PBAF
1.41 MDa



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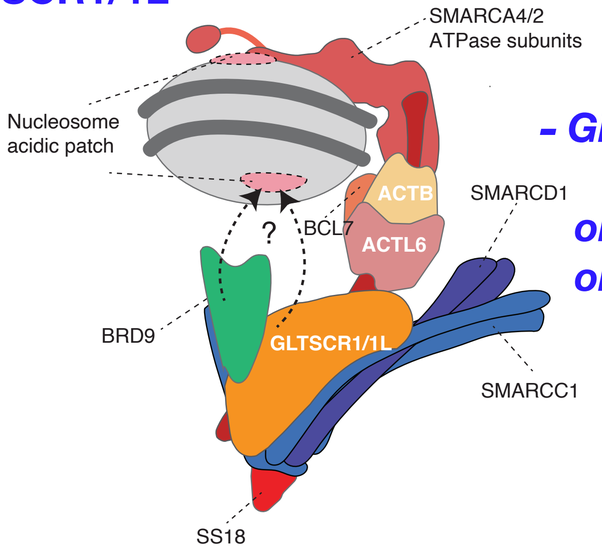

-SMARCB1
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- GLTSCR1 instead of ARID
only SMARCD1
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Biochemical composition, 3D structure, NCP interactions

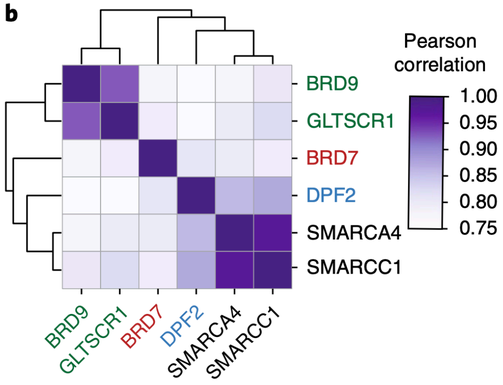
Diversity

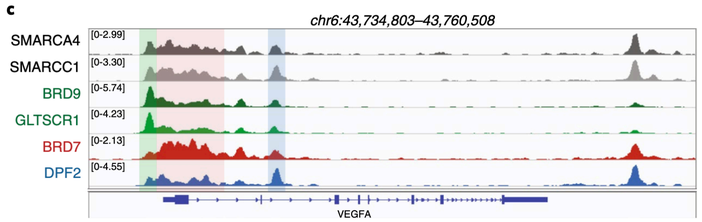
$$\binom{n}{r} = {}_nC_r = \frac{n!}{r!\,(n-r)!}$$

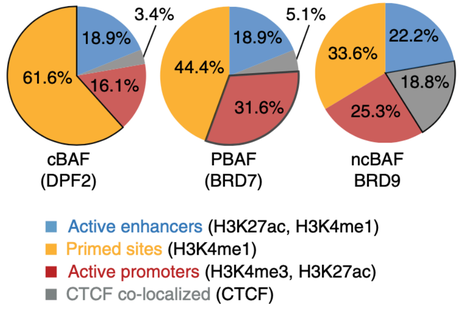
1296	108	48
108	54	12

Across cell types
In one cell

Targeting on chromatin







Multi-subunit/multi-cancer studies potentiate identification of new mechanisms

SMARCA4/ SMARCA2

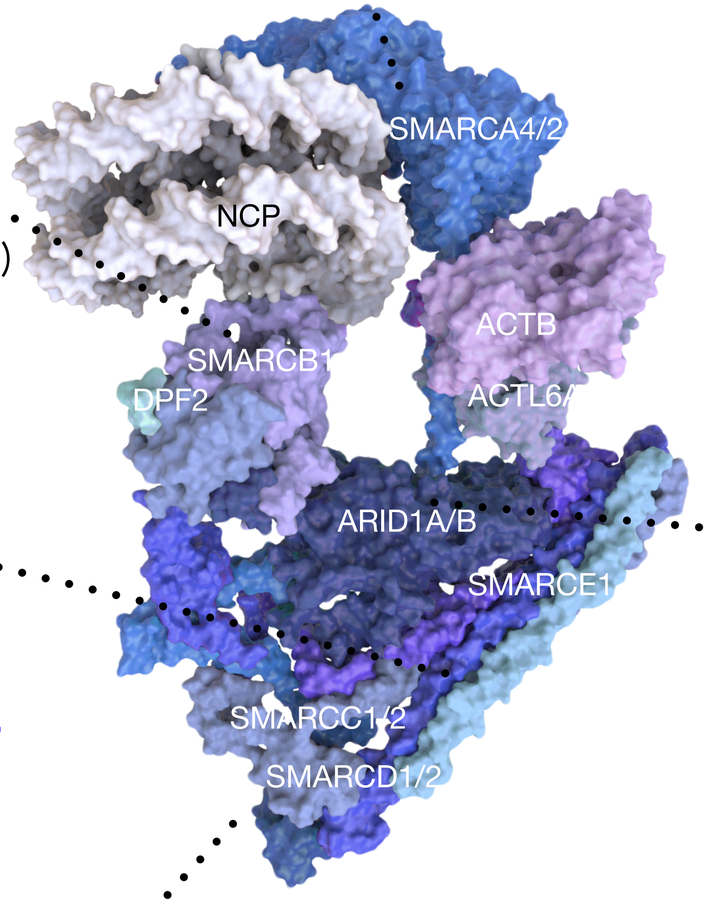
Non-small cell lung cancer (12%
SCCOHT (~100%)
SA4-deficient thoracic sarcomas (~100%)

Pan et al.,
Nature Genetics 2019

SMARCB1

Malignant rhabdoid tumor (>98%)
Atypical teratoid/rhabdoid tumor (>98%)
Epithelial sarcoma (>90%)
Pediatric chordoma (50%)

Valencia et al., *Cell,* 2019
Nakayama et al., *Nature Genetics* 2017



Clear cell meningioma (~100%) **SMARCE1**

Kadoch and Crabtree,
Cell 2013

McBride, Pulice et al.,
Cancer Cell 2018

Michel, D'Avino et al.,
Nat Cell Biol 2018

McBride, Mashtalir et al.,
Nat Struct Mol Bio 2020

ARID1A/ ARID1B

Ovarian clear cell carcinoma (68%)
Dedifferentiated endometrial (~100%)
Colorectal cancer
Breast cancer
Hepatocellular carcinoma
Pancreatic cancer
Neuroblastoma

SS18

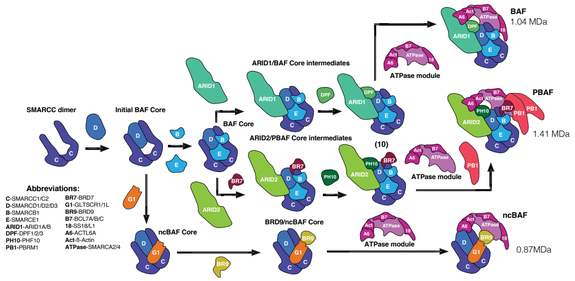
Synovial sarcoma
SS18-SSX (100%)

(Fusion oncoprotein)

Multi-subunit/multi-cancer studies potentiate identification of new mechanisms

Biochemical assembly and modularity

Mashtalir et al., *Cell* 2018



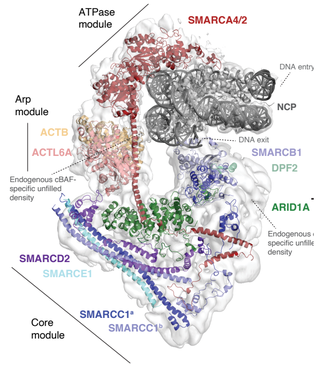
SMARCA4/ SMARCA2

SCCOHT (~100%)
SA4-deficient thoracic sarcomas (~100%)
Non-small cell lung cancer (12%)

Pan et al., *Nature Genetics* 2019

3D structural studies

Mashtalir et al., *Cell* 2020



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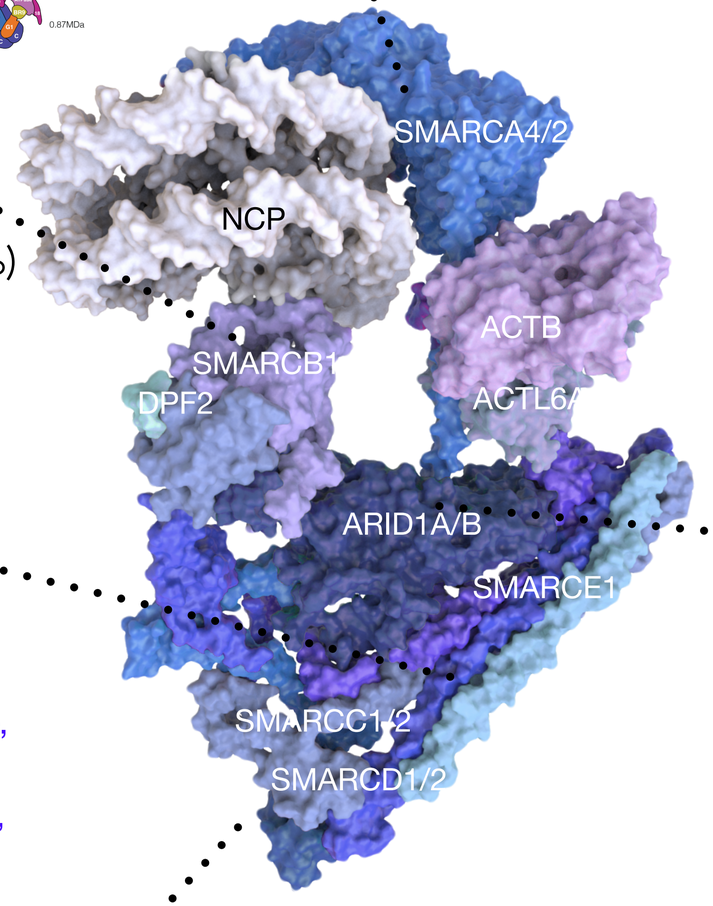
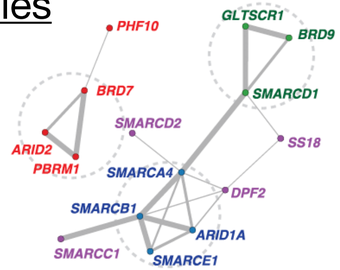


Functional modules

Pan, Meyers et al., *Cell Systems* 2018



ARID1A/ ARID1B

Ovarian clear cell carcinoma (68%)
Dedifferentiated endometrial (~100%)
Neuroblastoma

Clear cell meningioma (~100%) **SMARCE1**

Kadoch and Crabtree, *Cell* 2013

McBride, Pulice et al., *Cancer Cell* 2018

Michel, D'Avino et al., *Nat Cell Biol* 2018

McBride, Mashtalir et al., *Nat Struct Mol Bio* 2020

SS18

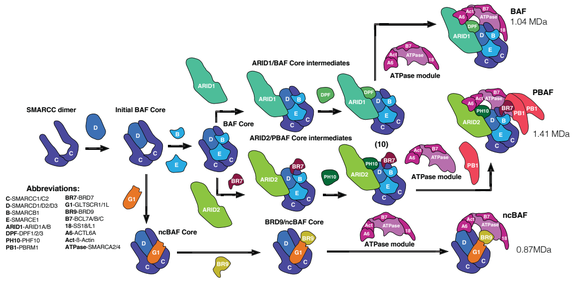
Synovial sarcoma
SS18-SSX (100%)

(Fusion oncoprotein)

Multi-subunit/multi-cancer studies potentiate identification of new mechanisms

Biochemical assembly and modularity

Mashtalir et al., *Cell* 2018



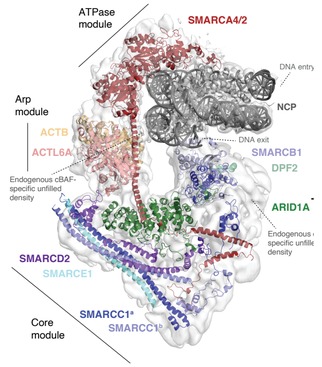
SMARCA4/ SMARCA2

SCCOHT (~100%)
SA4-deficient thoracic sarcomas (~100%)
Non-small cell lung cancer (12%)

Pan et al., *Nature Genetics* 2019

3D structural studies

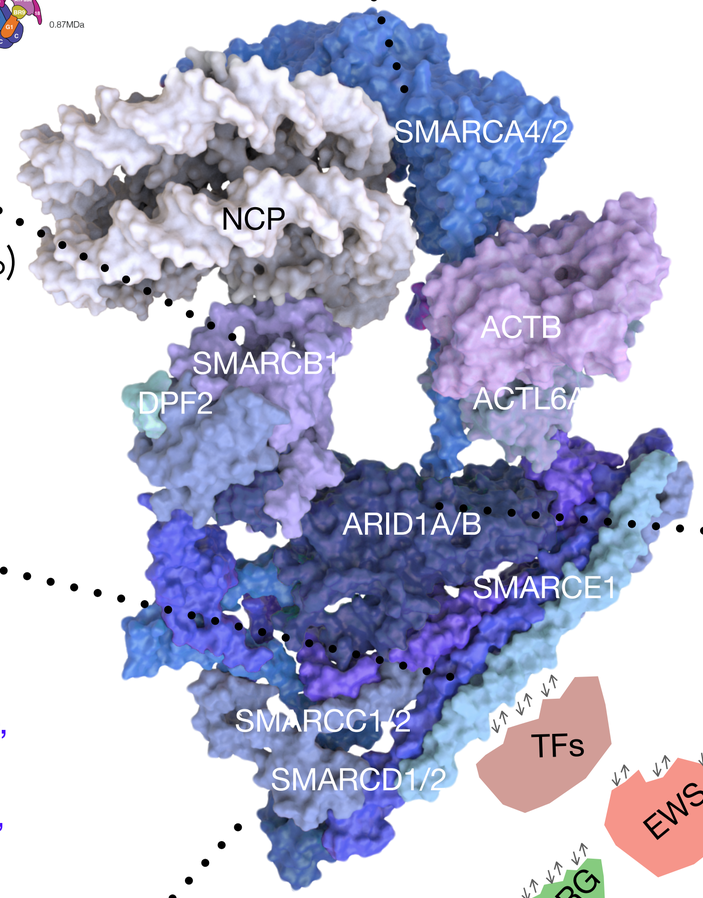
Mashtalir et al., *Cell* 2020



SMARCB1

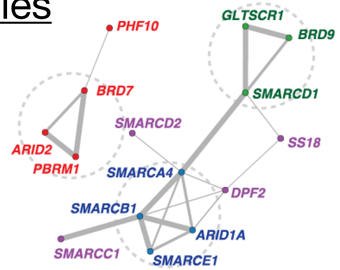
Malignant rhabdoid tumor (>98%)
Atypical teratoid/rhabdoid tumor (>98%)
Epithelial sarcoma (>90%)
Pediatric chordoma (50%)

Valencia et al., *Cell,* 2019
Nakayama et al., *Nature Genetics* 2017



Functional modules

Pan, Meyers et al., *Cell Systems* 2018



Clear cell meningioma (~100%) **SMARCE1**

ARID1A/ ARID1B

Ovarian clear cell carcinoma (68%)
Dedifferentiated endometrial (~100%)
Neuroblastoma

Boulay et al. *Cell* 2017

Kadoch and Crabtree, *Cell* 2013

McBride, Pulice et al., *Cancer Cell* 2018

Michel, D'Avino et al., *Nat Cell Biol* 2018

McBride, Mashtalir et al., *Nat Struct Mol Bio* 2020

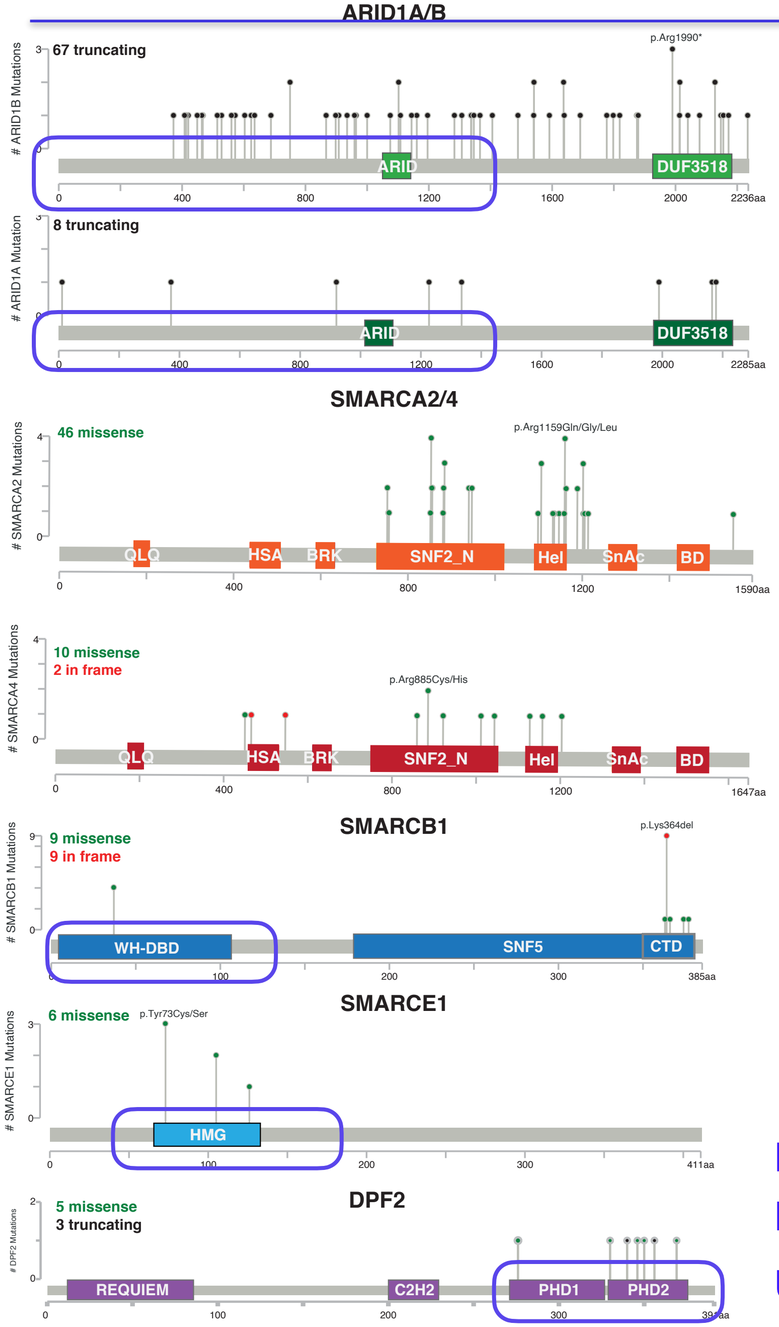
SS18

Synovial sarcoma
SS18-SSX (100%)

(Fusion oncoprotein)

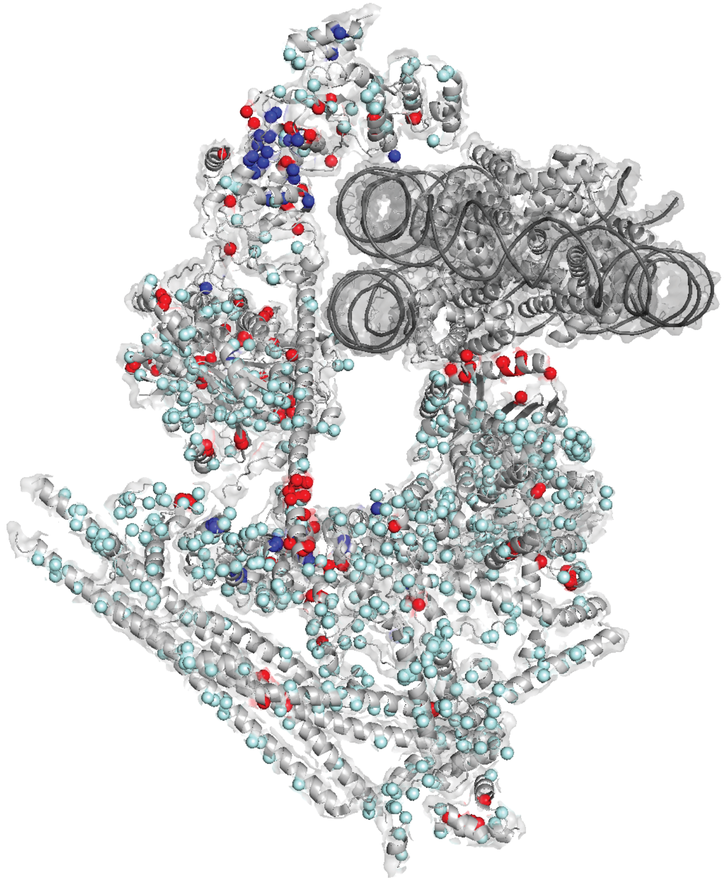
Sandoval et al. *Mol Cell* 2018

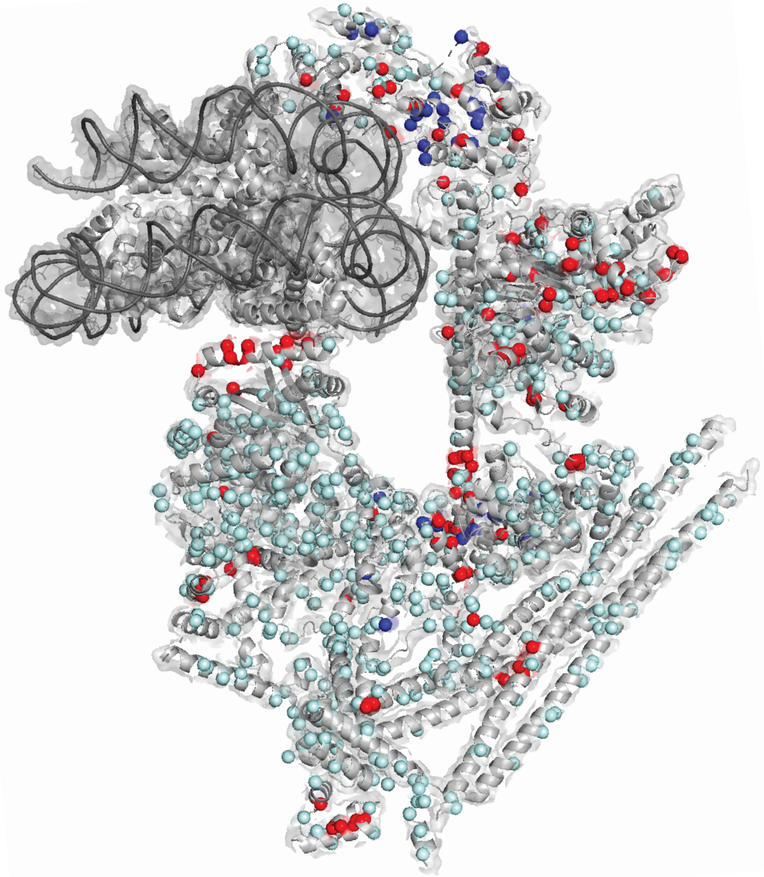
Much more to do: chromatin remodeler complex mutations are widespread



cBAF Non-truncating Neurodevelopmental Disorder (NDD) Mutations

- NDD-subunit specific
- NDD-mapped from paralog
- gnomAD (general population)





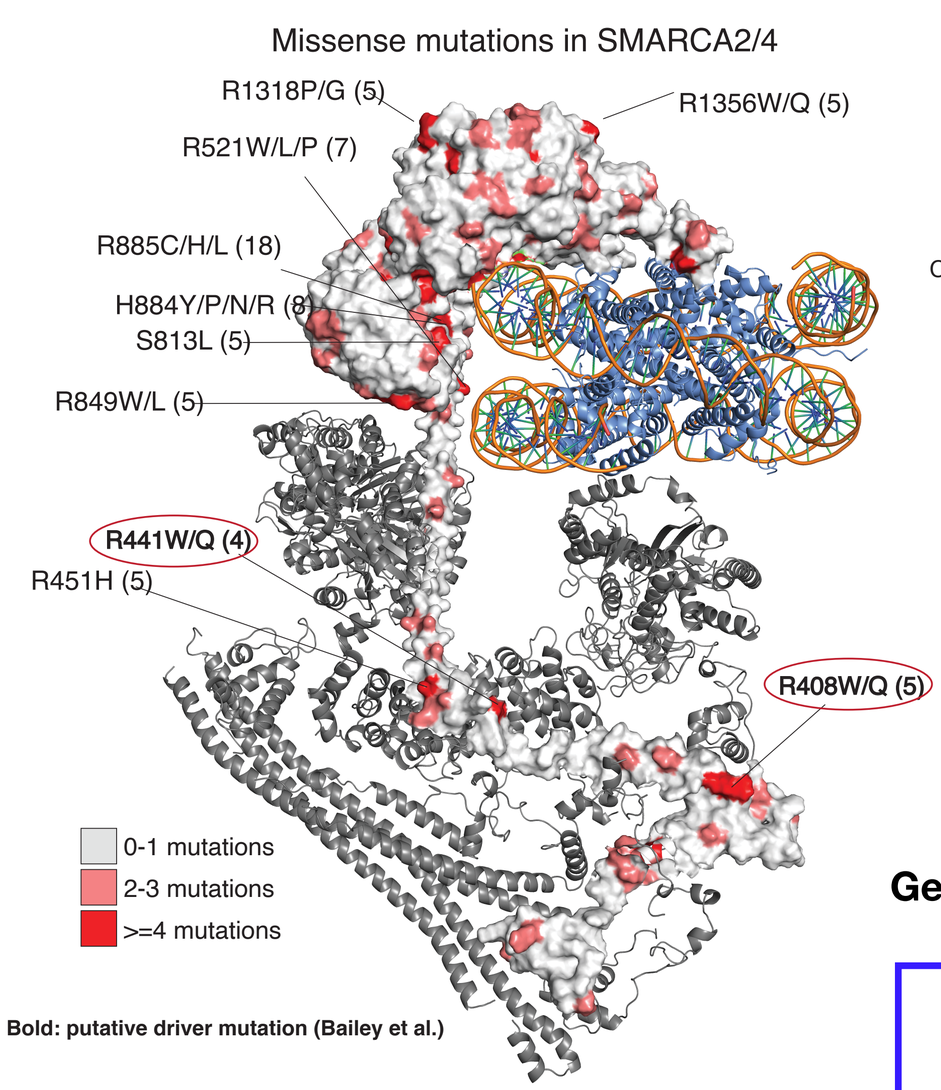
DNA-binding domains

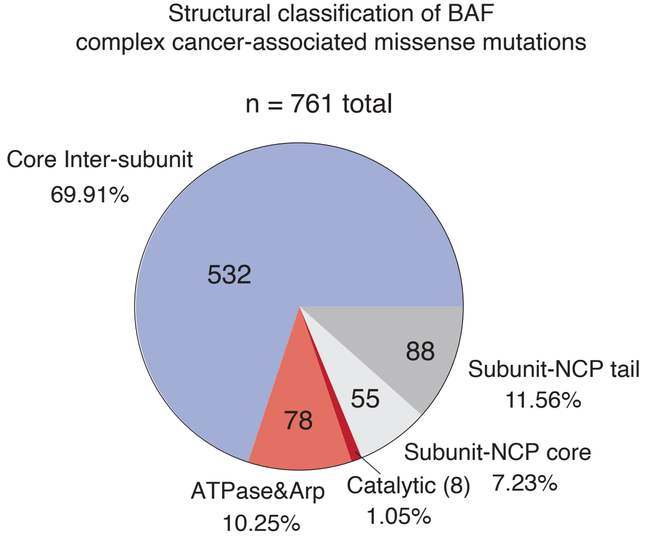
Histone reader domains

Unstructured regions

Valencia et al., *In Preparation*

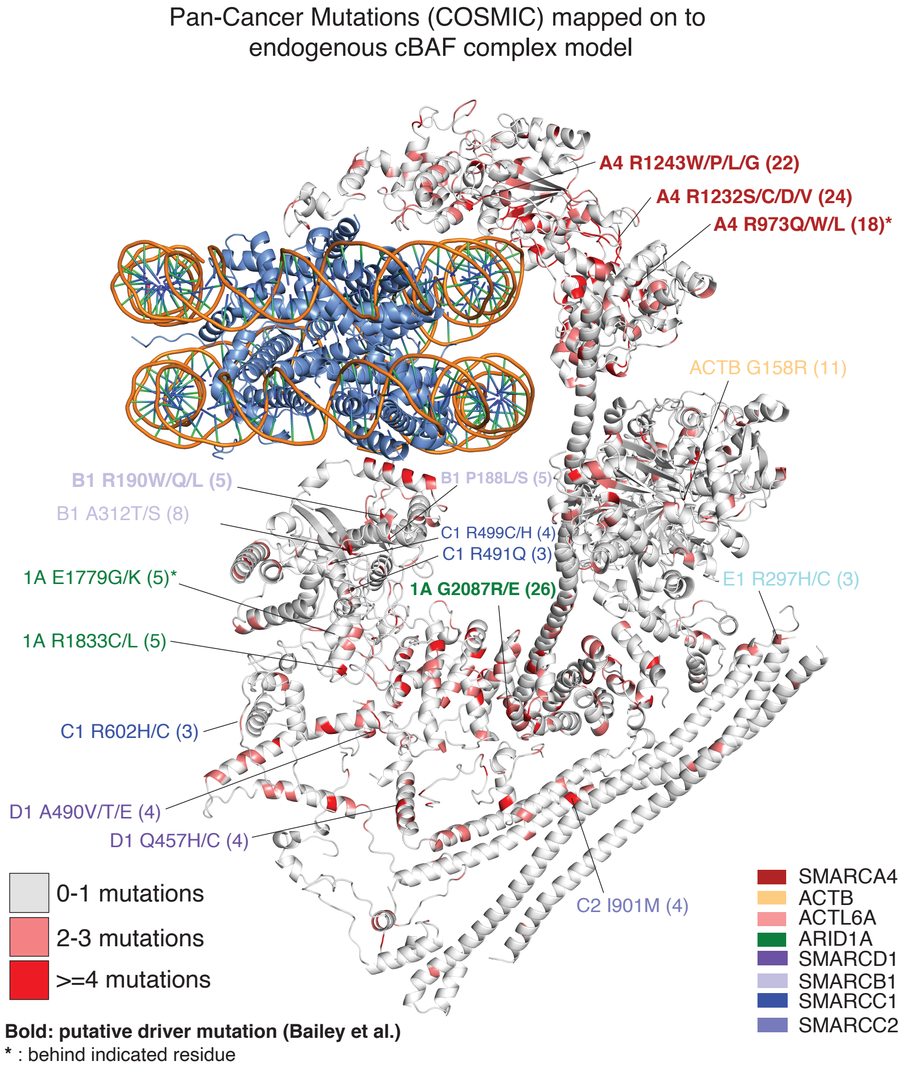
Recurrent cancer-associated mutations highlight broad opportunity for precision medicine





Generate functional classes of mutations

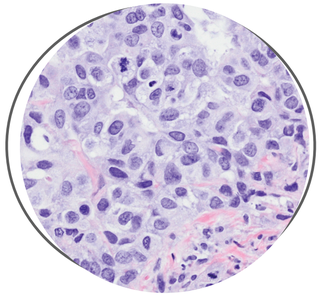
Provide foundation for therapeutic stratification



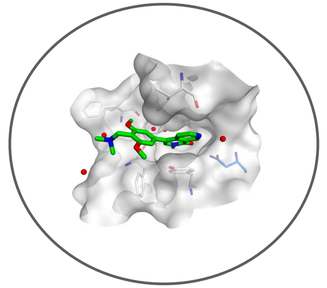
Chromatin remodeling complexes are *major determinants* of chromatin architecture and gene expression

- **Actionable dependencies arise from:**
  - Paralog deletions/mutations
    - SMARCA4/SMARCA2, ARID1A/B, others
  - **Shifted assembly and activities of complexes**
    - **cBAF-perturbed cancers—> BRD9/ncBAF**
  - Transcription factor network across cancers
    - SMARCA4/2 dual ATPase inhibition
  - **Locus-specific/process-specific activity**
    - **Additional targets in development (see pipeline)**
  - Chromatin remodelers with other functions (DNA repair, DNA damage, loop architecture, others)

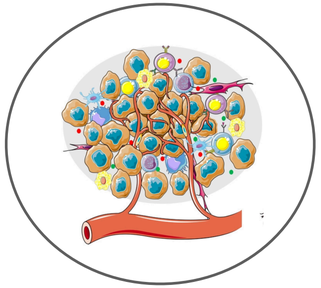
Tissue-specific context



Therapeutic approaches



Cell extrinsic sequelae



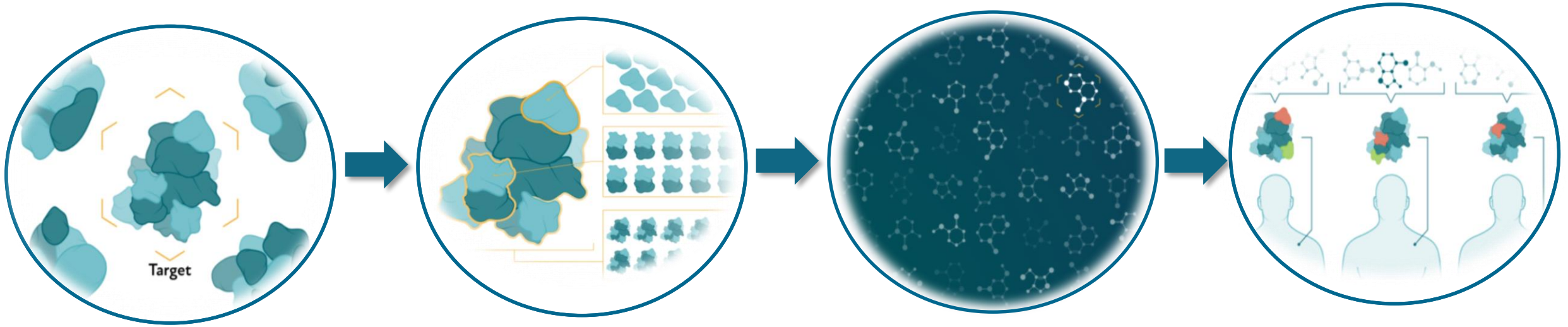
Gene Traffic Control Platform

Steve Bellon, Ph.D.

SVP of Drug Discovery, Foghorn Therapeutics

Our Gene Traffic Control Platform Makes It Possible to Understand and Drug Genetic Dependencies within the Chromatin Regulatory System

Integrated, Scalable, Efficient – Repeatable Paradigm



Target Identification & Validation

Determine dependencies (e.g., synthetic lethal)

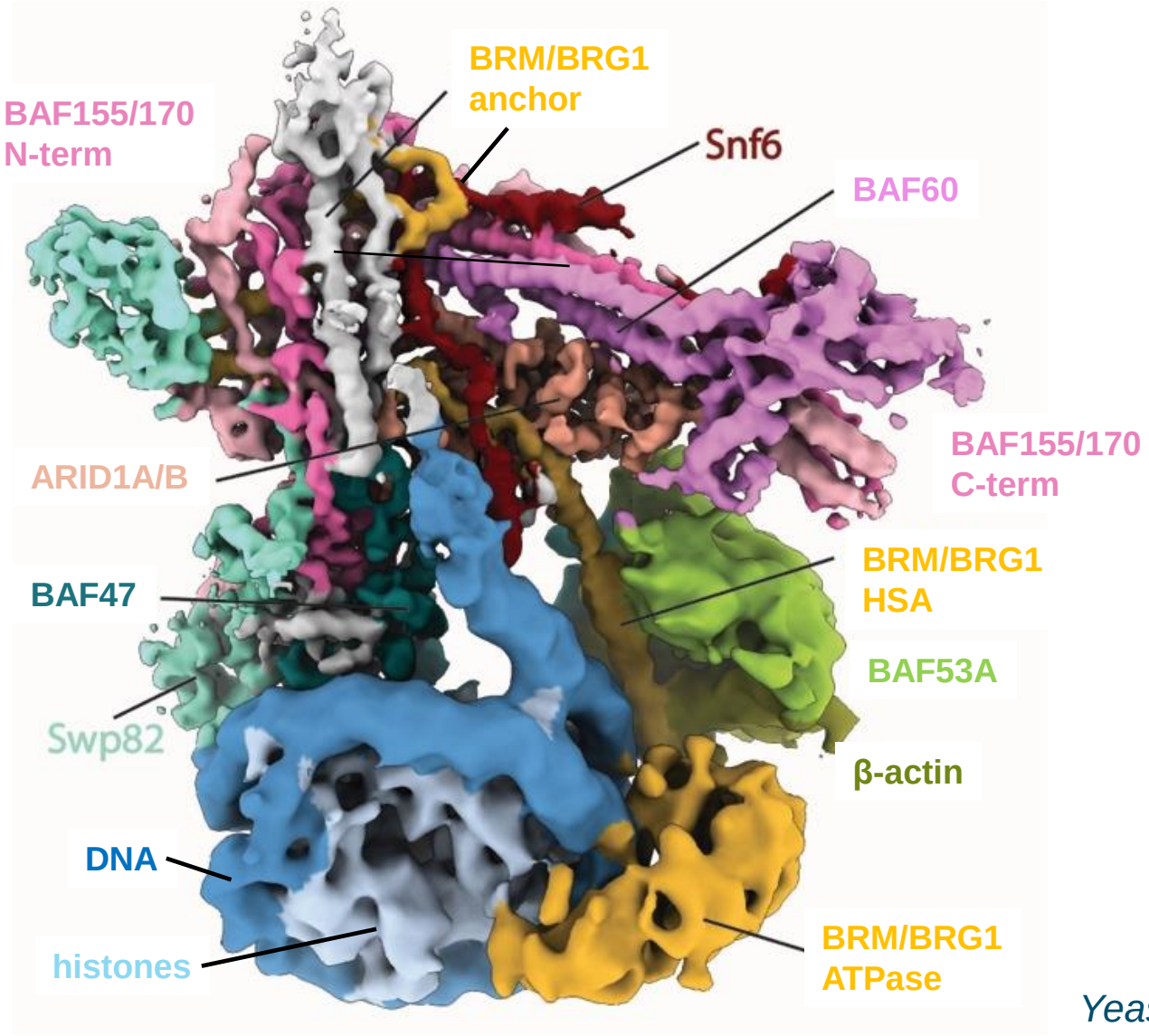
Production of Chromatin Regulatory System Components at Scale & Proprietary Assays

Discovery and Optimization of Chemical Matter

Targeted Protein Degraders
Enzymatic Inhibitors
Transcription Factor Disruptors

Translation to Clinic and Identification of Biomarkers

Rational for BAF Targets: BRM/BRG1 Threads Through Entire Complex



Allostery!

Yeast Swi/Snf cryo EM structure

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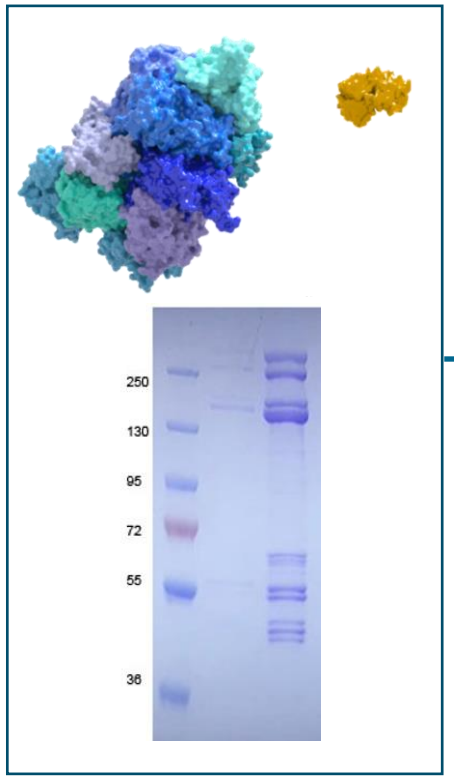
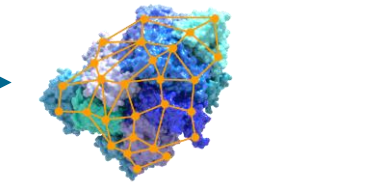
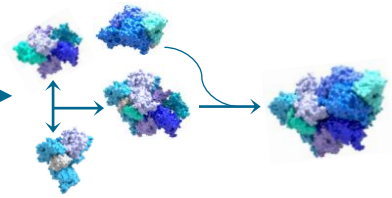
Platform is Powered by Ability to Produce Components at Scale

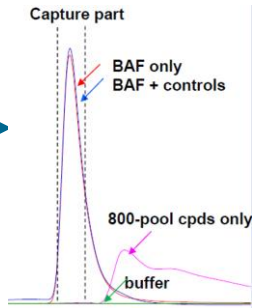
Drives Drug Discovery Pipeline with Cutting Edge Technology

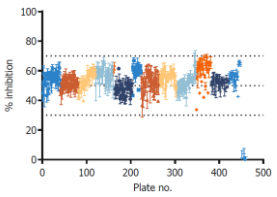
Production of Chromatin Regulatory System Components

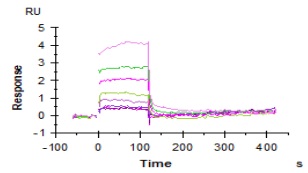












Features	Benefits
Surface Mapping	Characterize TF/BAF Binding Sites
Assembly	Synthesize subcomplexes to enable drug discovery
Affinity Screening and Validation	ASMS on full complex to yield novel degraders
HTS	Multiple screening options with full complex
Biophysics/ SPR	Validation of novel small molecule binders

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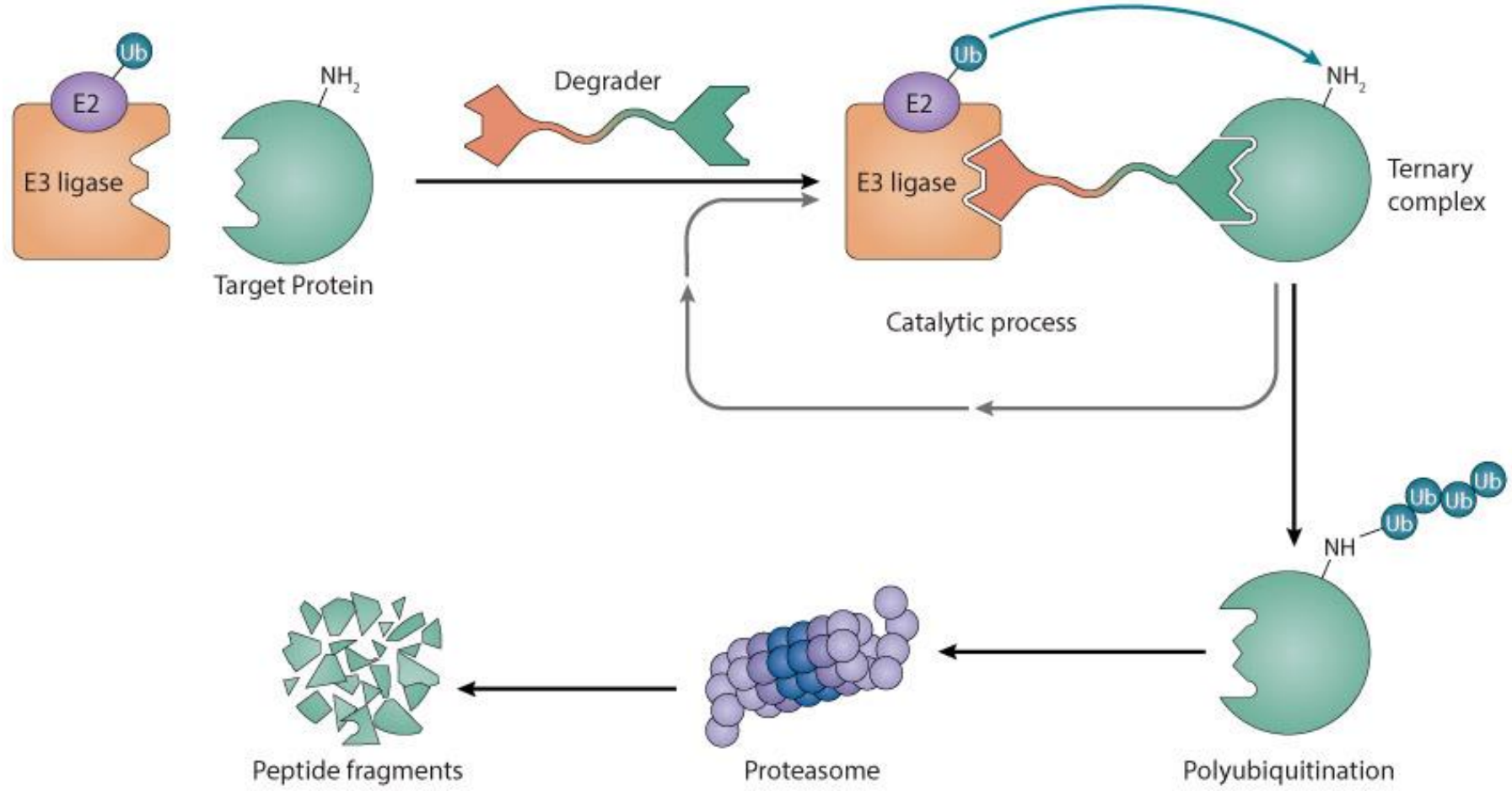
Protein Degradation – Evolving Therapeutic Modality



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Heterobifunctional Degrader Platform

Foghorn Pursuing >8 Targeted Protein Degraders

Bioinformatics	• Optimal E3 ligase target pairing • Proteomics
Screening and Characterization	• Proprietary chromatin remodeling assays • Protein degradation kinetics
Chemical Toolbox	• Proprietary library of drug-like linkers and E3 ligase binders • Chemistry to rapidly identify and optimize degraders
Structural and Computational Approaches to Degrader Design	• Structure based optimization of binders • Ternary complex crystal structures and modeling approaches for degrader optimization
Optimization of Degrader Drug Properties	• Guidelines for both of oral and IV administered degraders • PKPD/efficacy and safety modeling to optimize dosing and scheduling

Targeting ARID1A Mutated Cancers: ARID1B Protein Degrader

Advantaged by Gene Traffic Control Platform and Protein Degrader Capabilities

Gene Traffic Control Platform

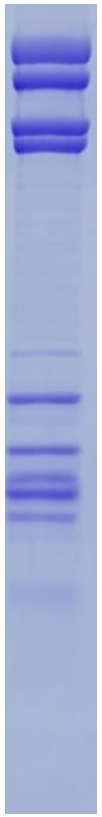
- Platform produces BAF complexes and subcomplexes containing either ARID1A or ARID1B at scale
- Enables proprietary screens against ARID1B

Protein Degrader Capabilities

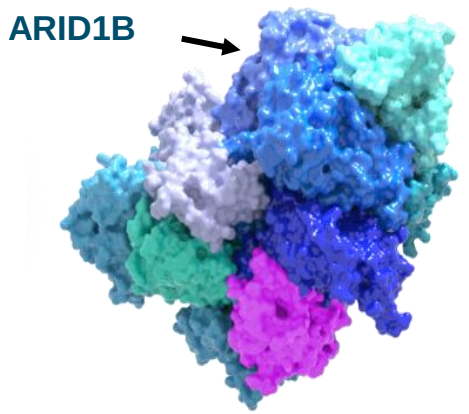
- Utilize protein degrader toolbox to create ARID1B hetero-bifunctional degraders

Biology & Opportunity

- ARID1A is the most highly mutated subunit of the BAF complex in cancer
- ~5% of all tumors harbor ARID1A mutations including; endometrial cancer (~40%), bladder cancer (~25%) and ovarian (~15%)
- Synthetic lethal relationship with ARID1B



Highly purified ARID1B / BAF complex



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First Two Programs in the Clinic, Broad Pipeline Advancing

Precision Oncology / Breadth and Depth

Program / Target	Modality	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3	Global Rights
FHD-286 (BRG1 / BRM)	Enzyme inhibitor	AML		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
		Uveal melanoma		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
FHD-609 (BRD9)	Protein degrader	Synovial sarcoma		Early Clinical Data (H1 2022)			FOGHORN THERAPEUTICS
Selective BRM	I) Enzyme inhibitor	BRG1 mutated cancers	IND 2022				FOGHORN THERAPEUTICS
	II) Protein degrader	BRG1 mutated cancers					
Selective ARID1B	Protein degrader	ARID1A mutated cancers					FOGHORN THERAPEUTICS
Synthetic Lethal Targets (multiple)	I) Enzyme inhibitors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Transcription Factors (multiple)	I) Transcription factor disruptors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Partnered program (undisclosed)	Transcription factor disruptor						MERCK

Gene Traffic Control® Platform

FOGHORN® THERAPEUTICS

Selective BRM Modulators for BRG1 Mutated Cancers

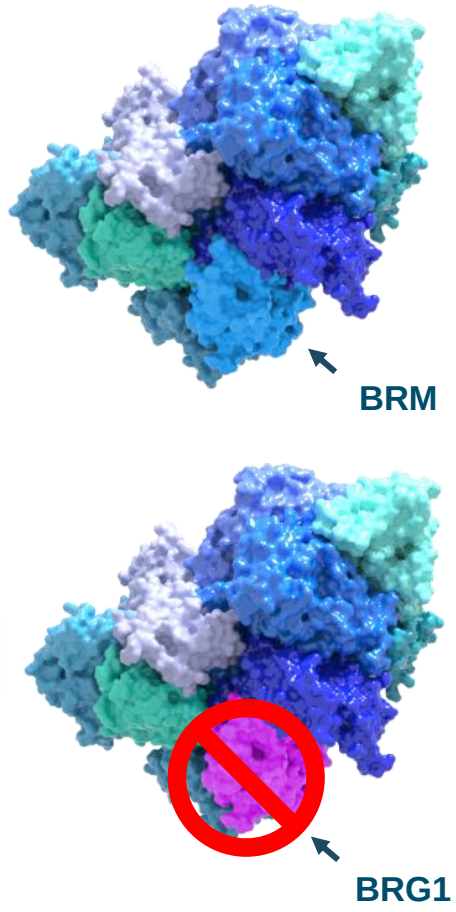
Enzymatic Inhibitor and Protein Degrader Programs

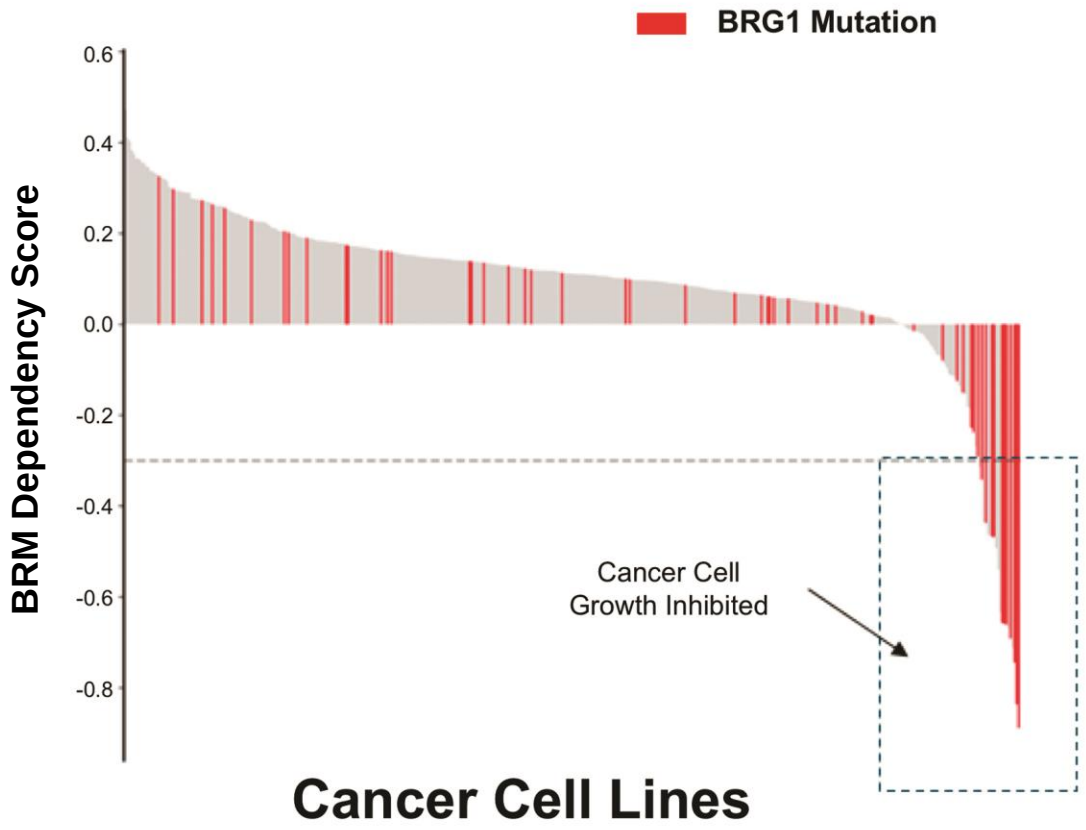
BRG1 Mutations Create a Genetic Dependency on BRM

Selective BRM Modulators Overview

Target / Approach	• BRM • Enzymatic inhibitor • Targeted protein degrader
Indication	• BRG1 mutated cancers (e.g., NSCLC), 30+ cancers with BRG1 mutations
Mutation / Aberration	• BRG1
Stage	• Pre-clinical
New Patients Impacted / year*	• **> 100,000**

* US, EU5, Japan





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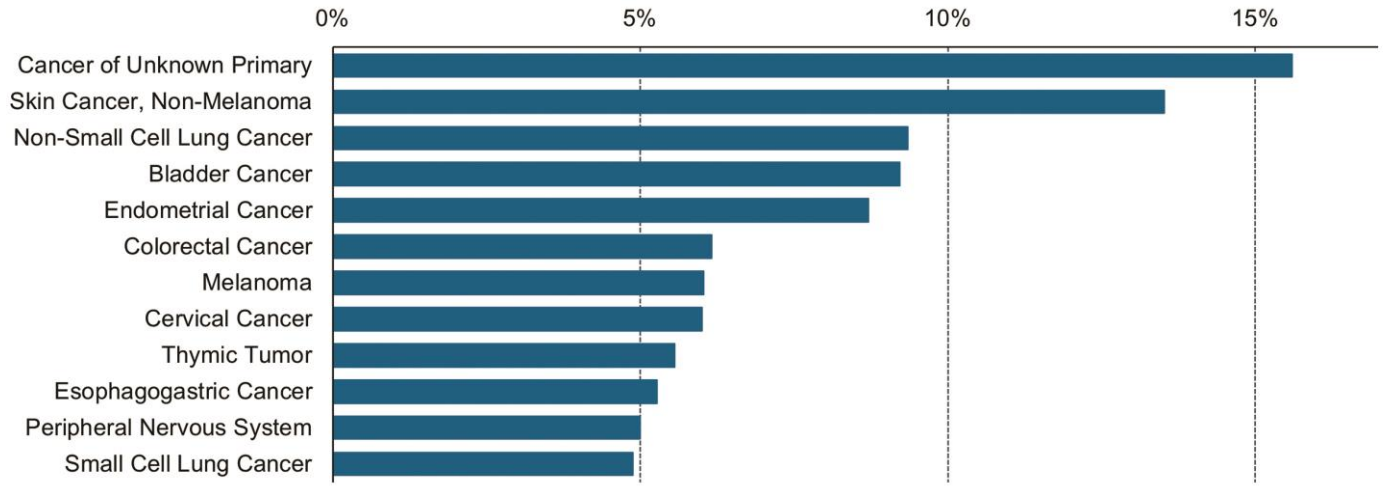
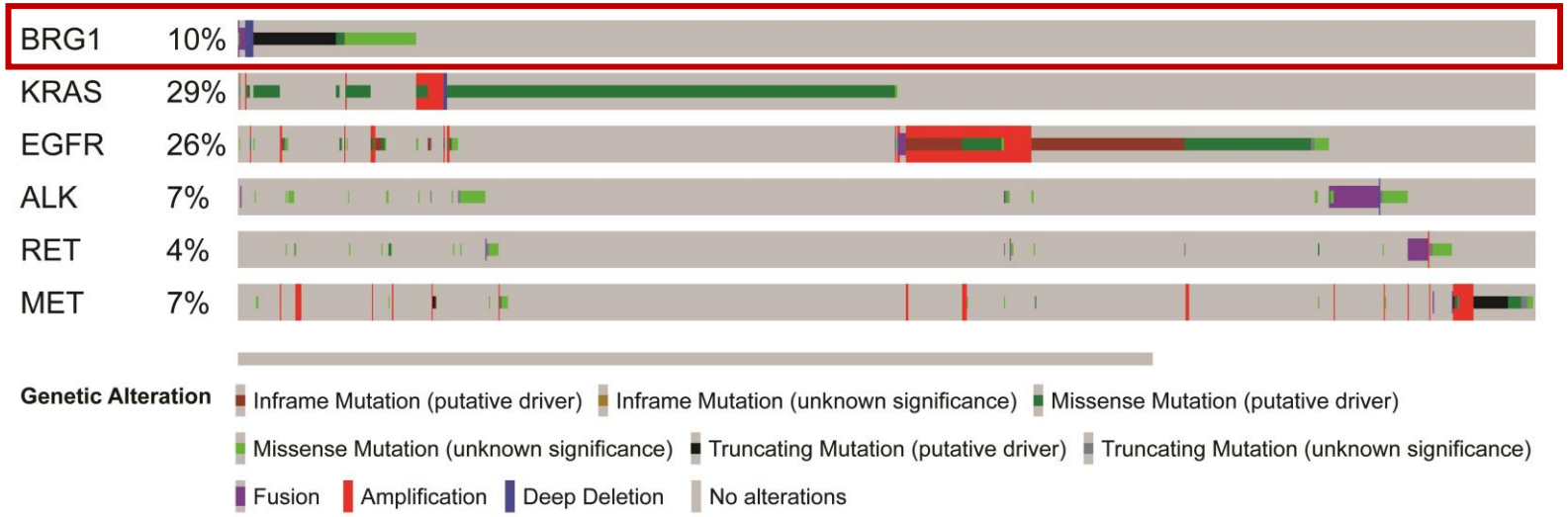
BRG1 Mutated in ~5% of All Tumors

Broad Addressable Patient Population



BRG1 mutated across range of tumors

Accounts for ~5% of all tumors



BRG1 mutated in up to 10% of NSCLC tumors, minimal overlap with other mutations

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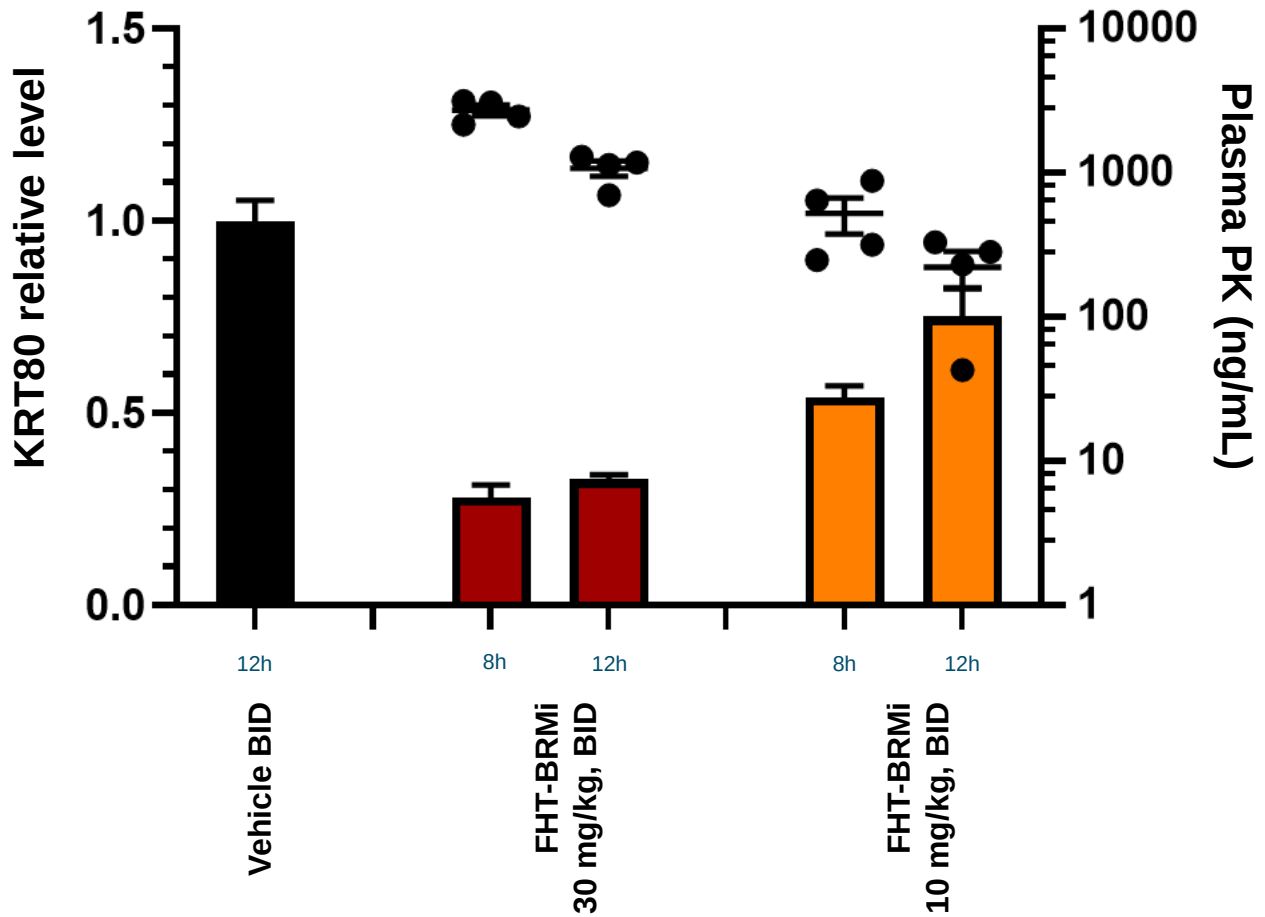
In Vivo Target Engagement With ~20X Selective BRM Selective Inhibitor

PD Modulation in the H1299 BRG1-Null NSCLC PD Model Establishing a Direct Exposure-Response Relationship

Targeting BRM: KRT80 PD Biomarker



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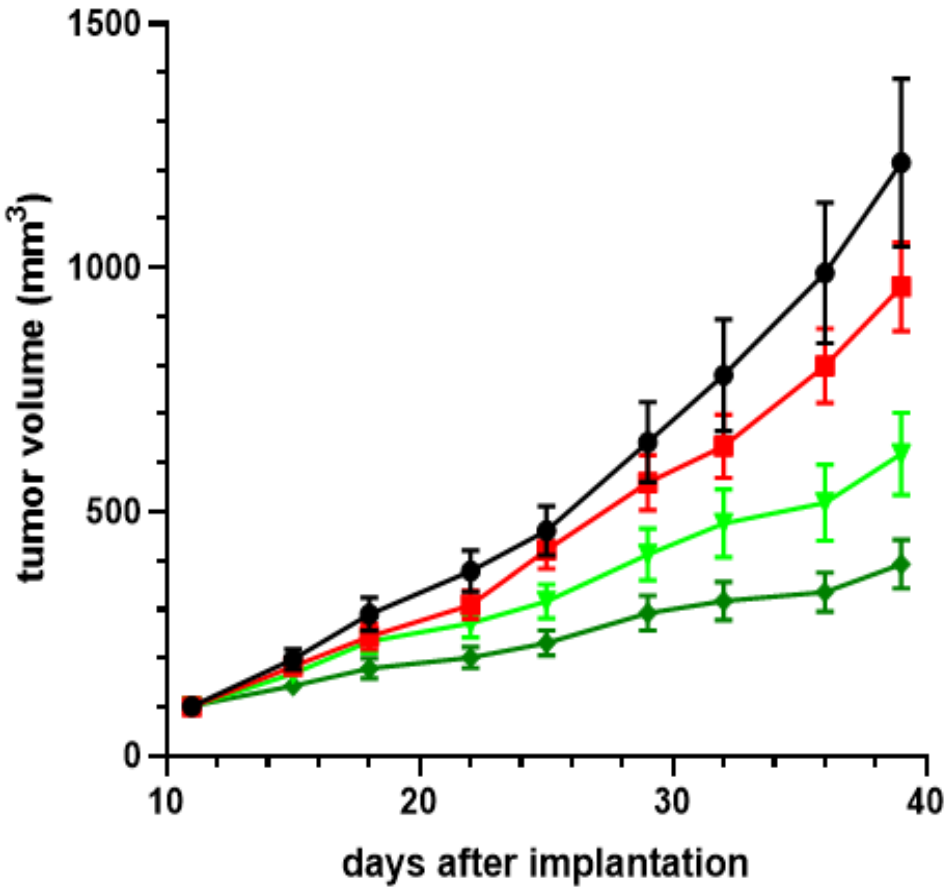
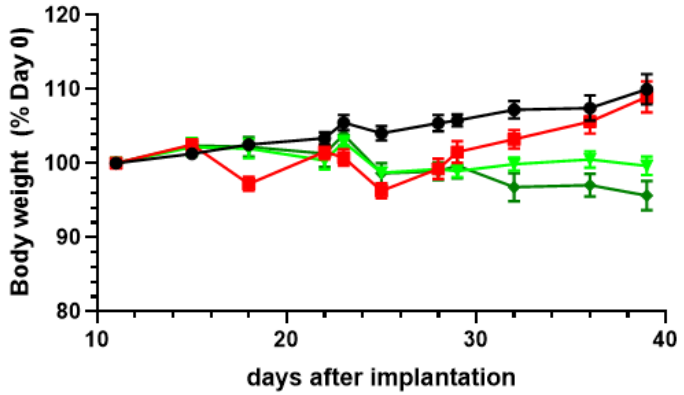
BRM Selective Inhibitor In vivo Efficacy

Demonstrates PK/PD and In vivo Efficacy in a BRG1 Mutant Lung CDX Model

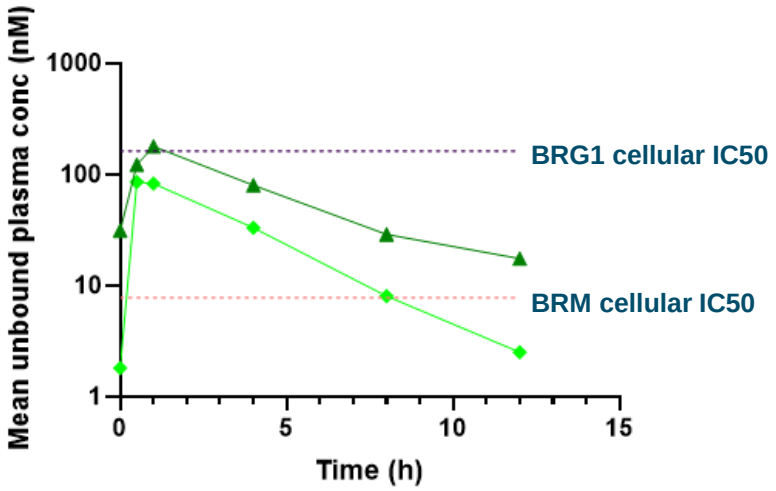
A549-BRG1 Mutant NSCLC Model



Body Weight



Plasma Exposure



- Vehicle Control (BID)
- Cisplatin 4 mg/kg (IP)
- FHT-BRMi 15 mg/kg (BID)
- FHT-BRMi 30 mg/kg (BID)

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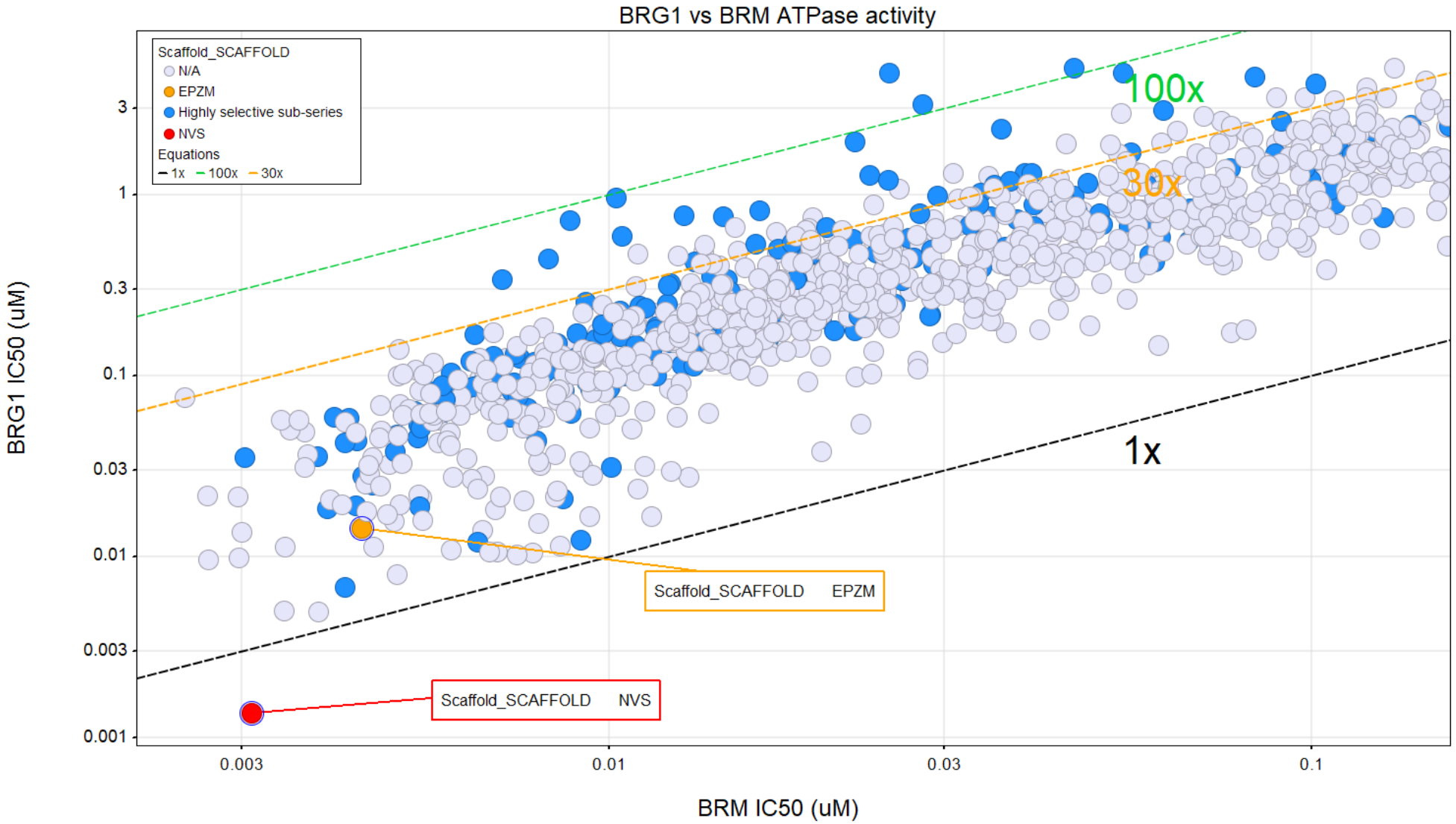
Scaffolds With Enzymatic Selectivity > 100X



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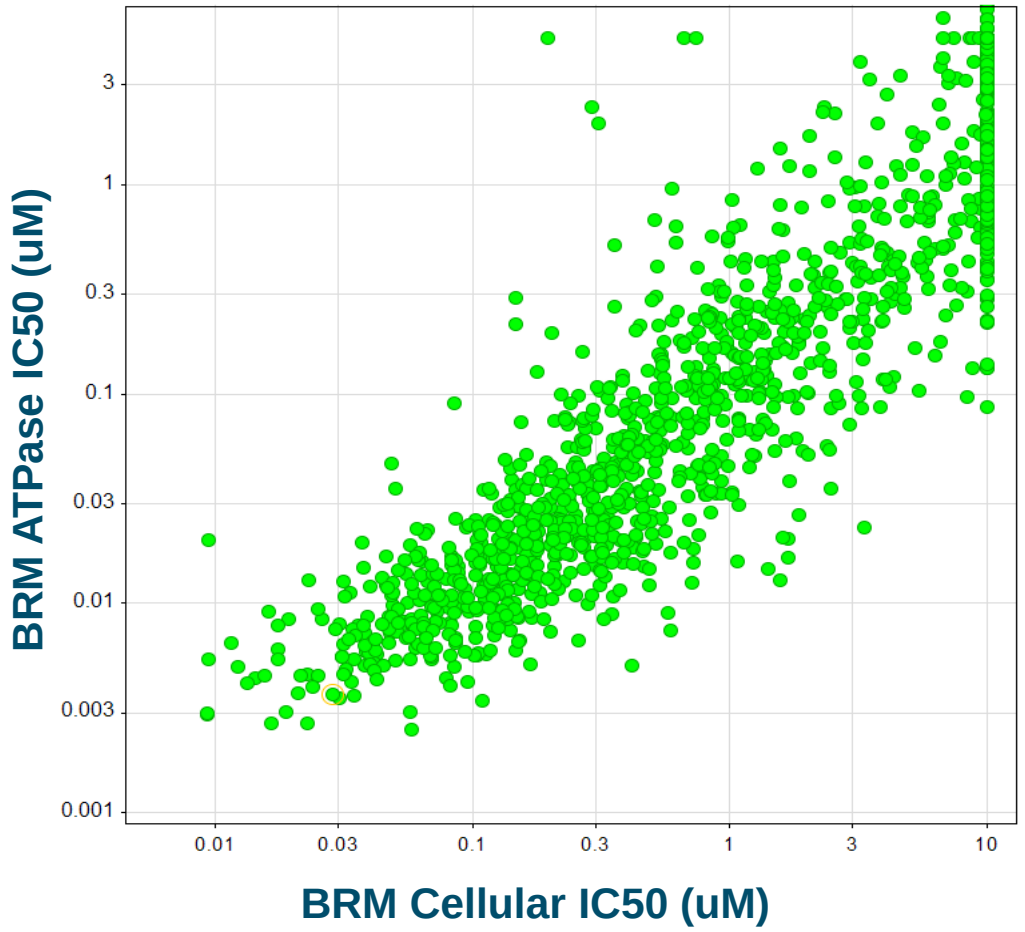
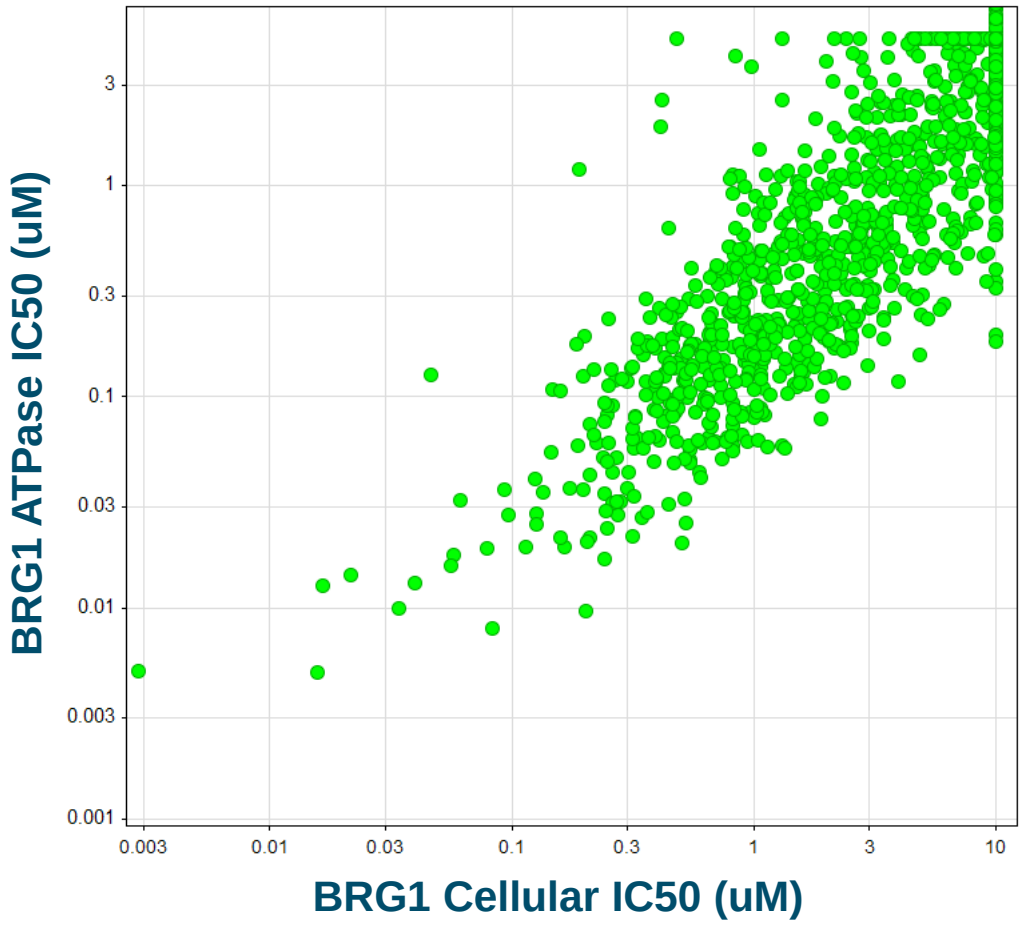
Strong Correlation from Enzyme to Cellular Reporter Assays





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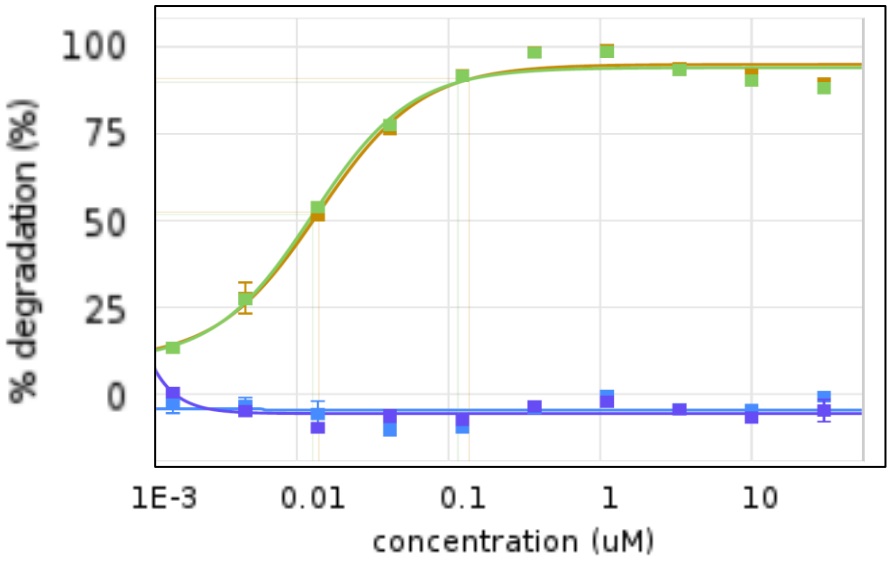
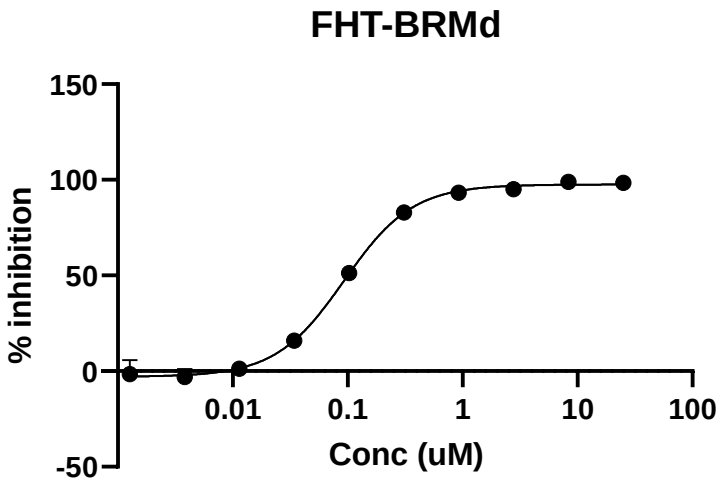
Advancing BRM Selective Degraders

Achieving Complete BRM Degradation

BRM/BRG1 HiBit Data



A549 Ten-Day Proliferation Assay



Degraders cause time- and dose-dependent BRM degradation, antiproliferative effects in A549 BRG1 mutant NSCLC lung model

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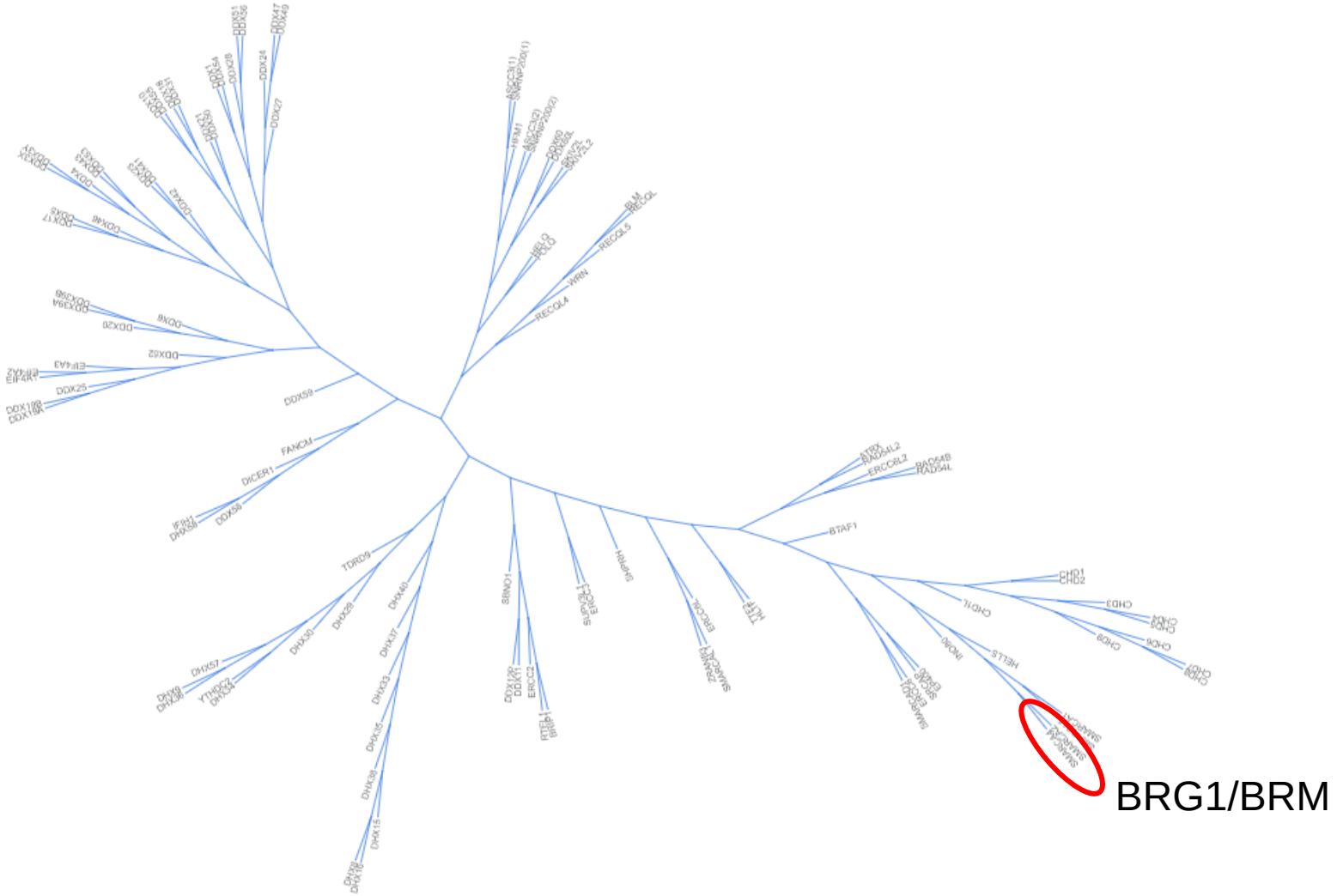
Target Hopping to Related Helicase Targets

ATP Dependent Helicases

Potential to Broaden Pipeline and Further Validate Platform Breadth



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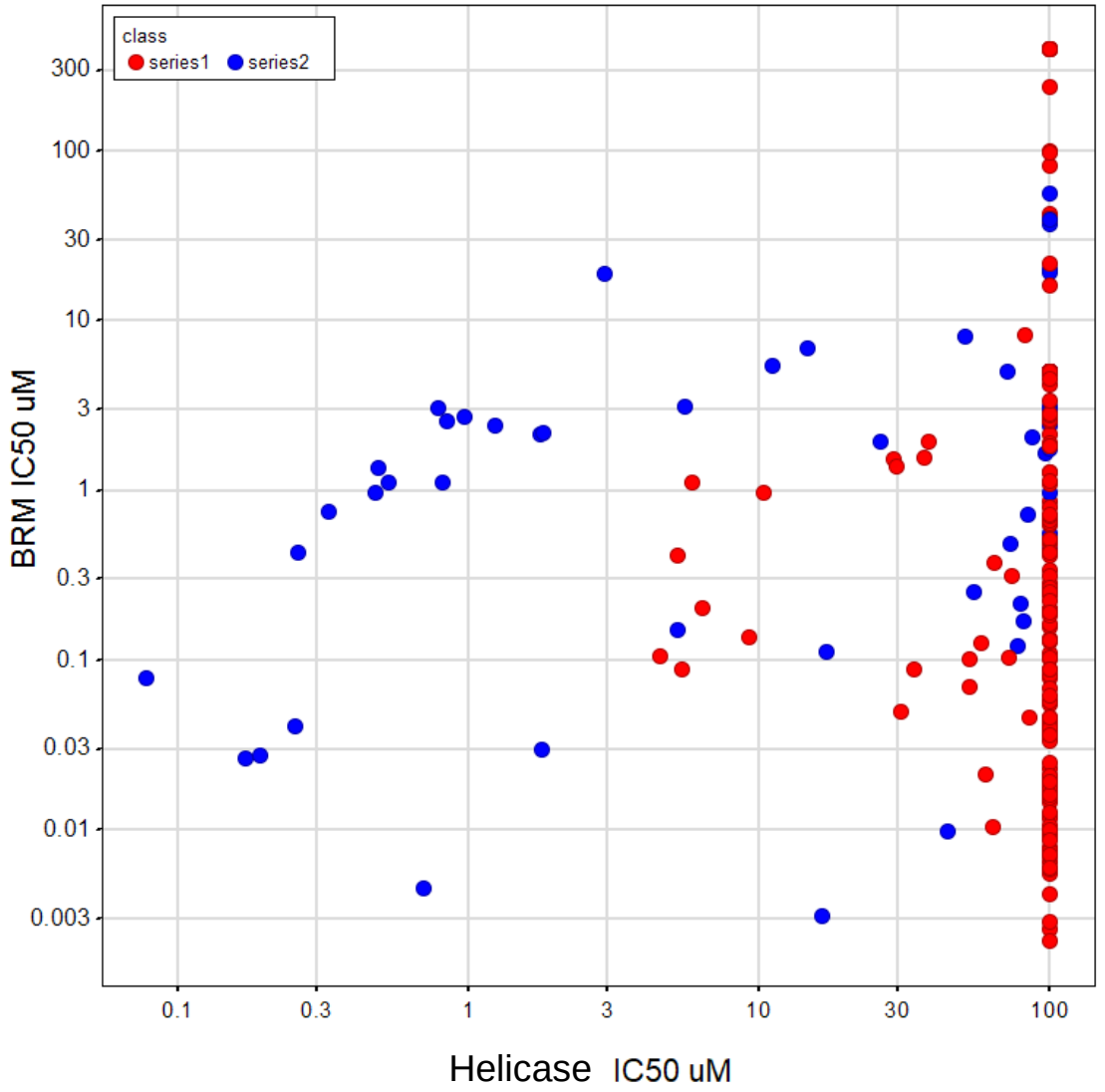
Target Hopping to Related Helicase Targets



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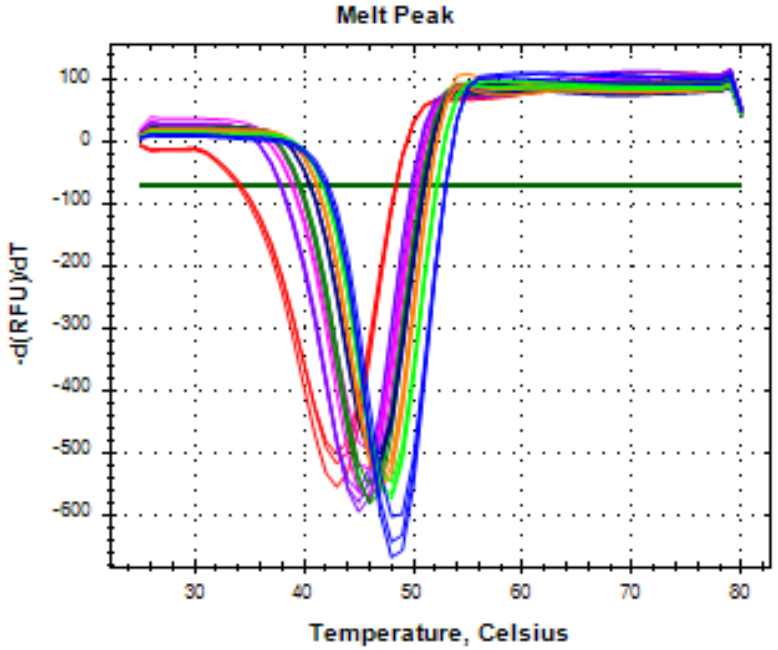
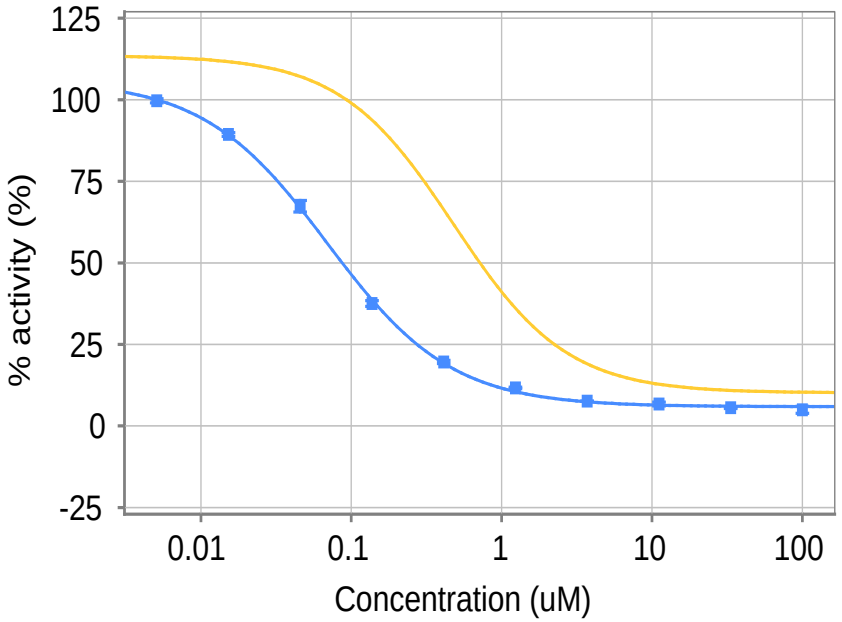
Potency Improved to Sub 100 nM

Target Hopping Hits → **Validation by ASMS and DMF** → **Medicinal Chemistry** → **IC50 = 70 nM**





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FOGHORN® THERAPEUTICS

Novel Approach to Targeting Transcription Factors

Disrupting Transcription Factor – Chromatin Remodeling Complex Interactions

A New Approach to Drugging Transcription Factors

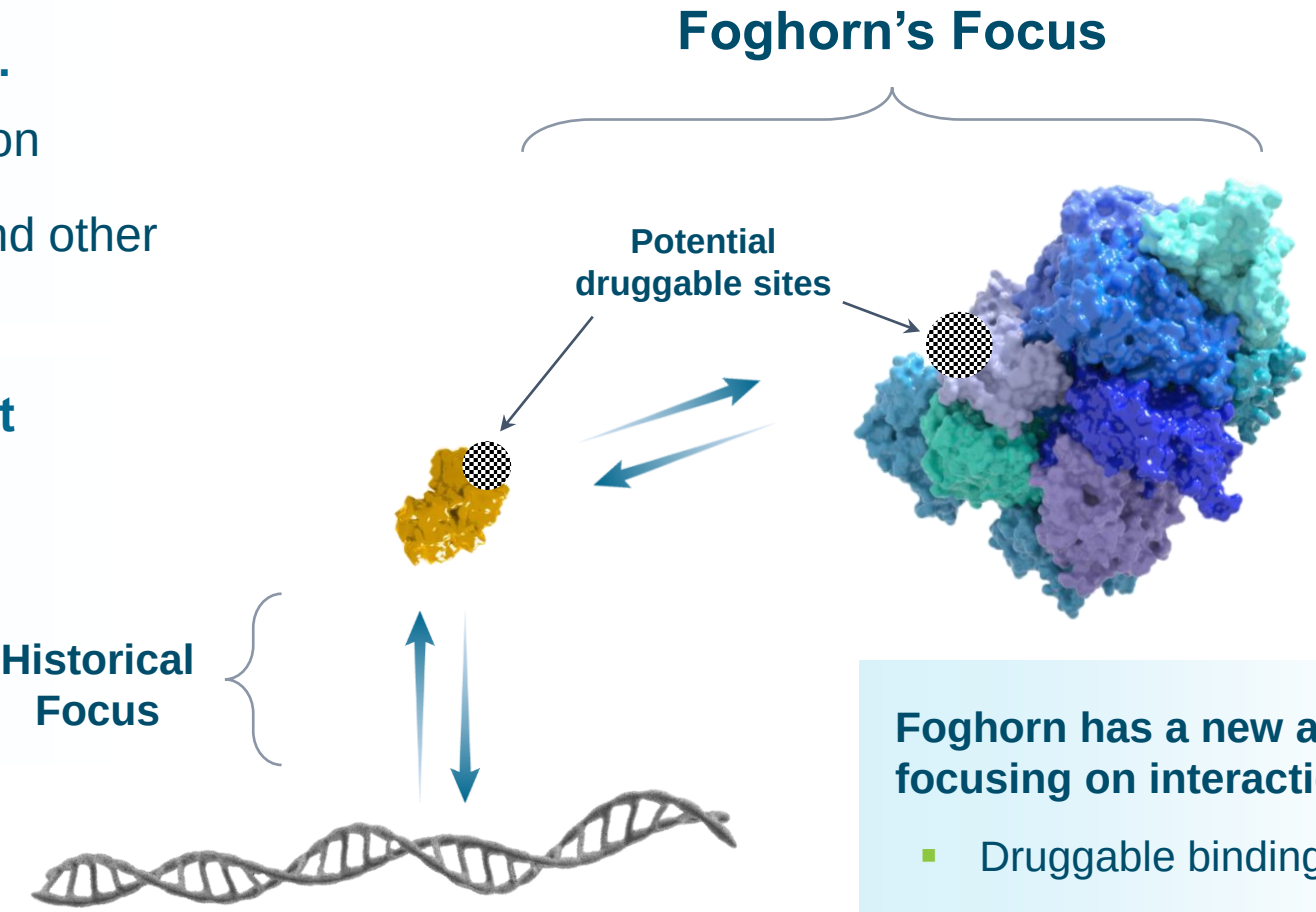
Enabled by Proprietary Ability to Purify and Synthesize Chromatin Regulatory System Components

TFs are compelling drug targets…

- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…but historically difficult to target

- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities



Foghorn has a new approach focusing on interaction with BAF

- Druggable binding pockets
- Druggable affinities

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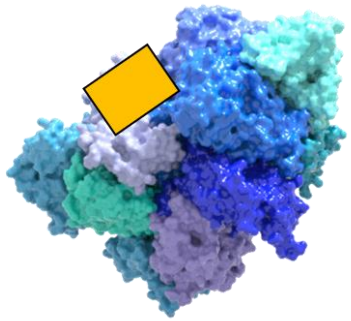
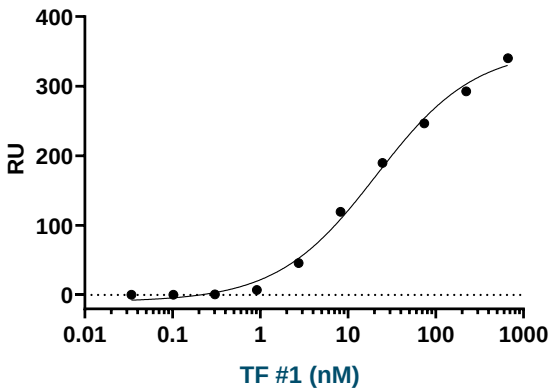
Transcription Factor-Chromatin Remodeling Complex Interactions

Unique Insights in Where and How Transcription Factors Bind

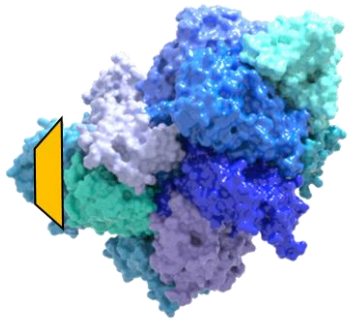
TF #1



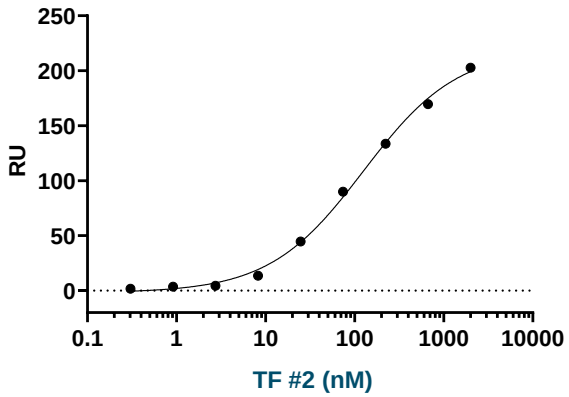
KD = 21 nM



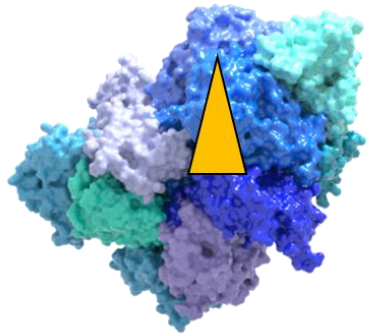
TF #2



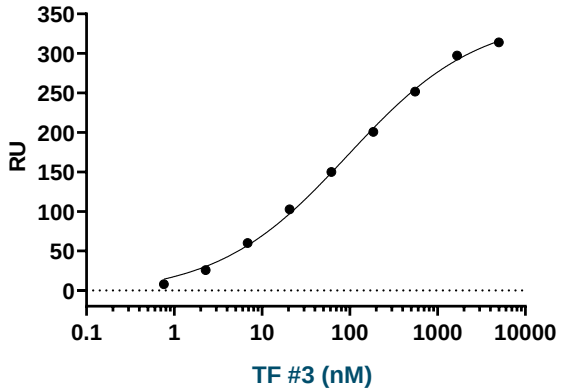
KD = 125 nM



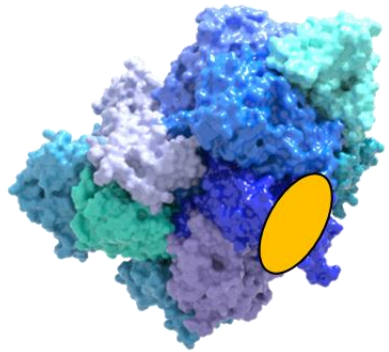
TF #3



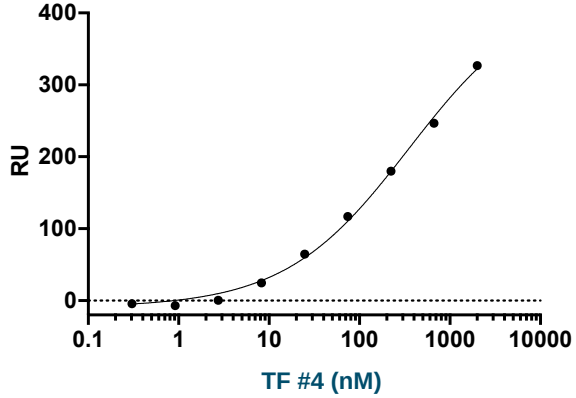
KD = 94 nM



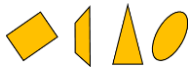
TF #4



KD = 351 nM



Transcription Factors (TF):



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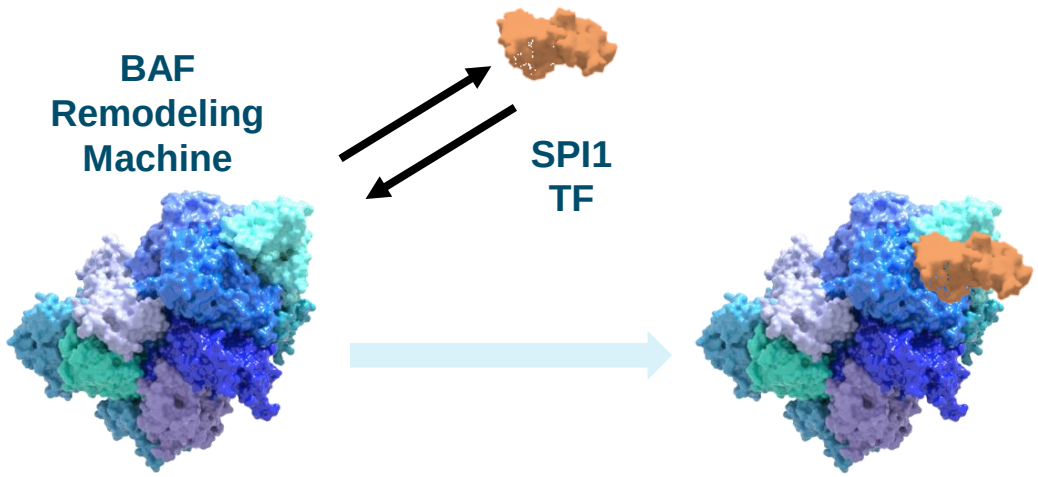
SPI1: Transcription Factor SPI1 Dependency in AML

Aberrant SPI1 overexpression

BAF Remodeling Machine



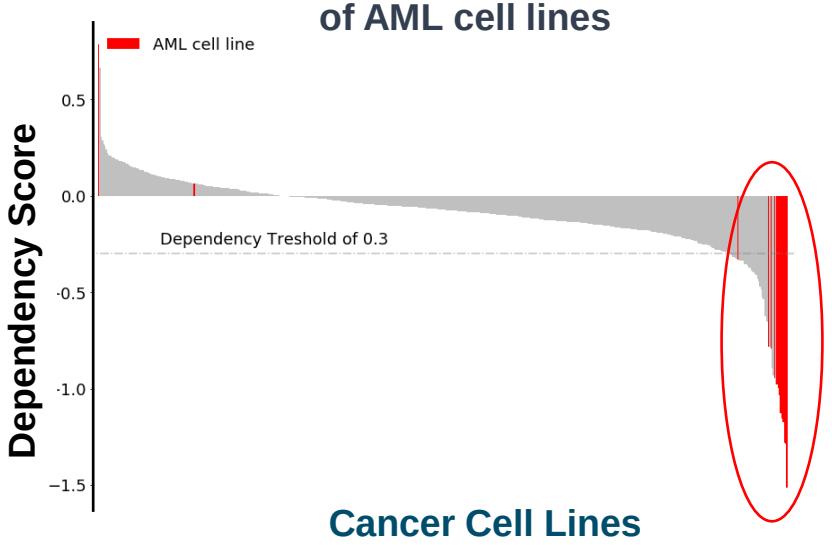
SPI1 TF

SPI1 Target Gene Sites

Overexpression of Genes Regulated by SPI1

AML

SPI1 inhibition selectively inhibits the growth of AML cell lines



Cancer Cell Lines

CRISPR Screening data by Achilles (>400 cancer cell lines)

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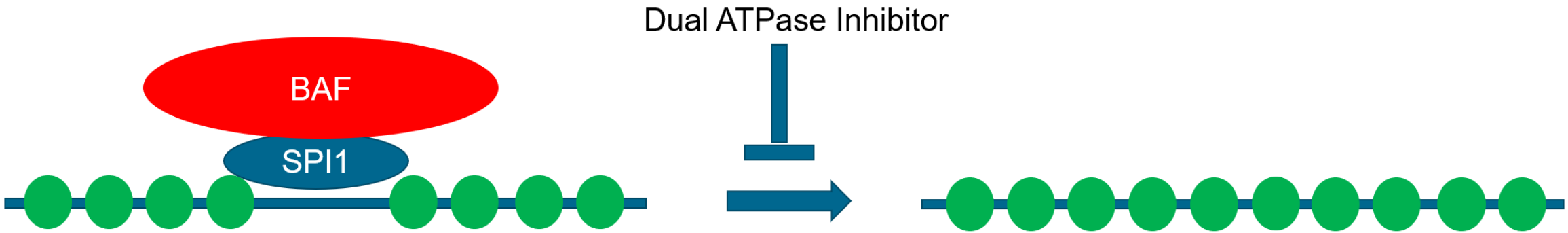
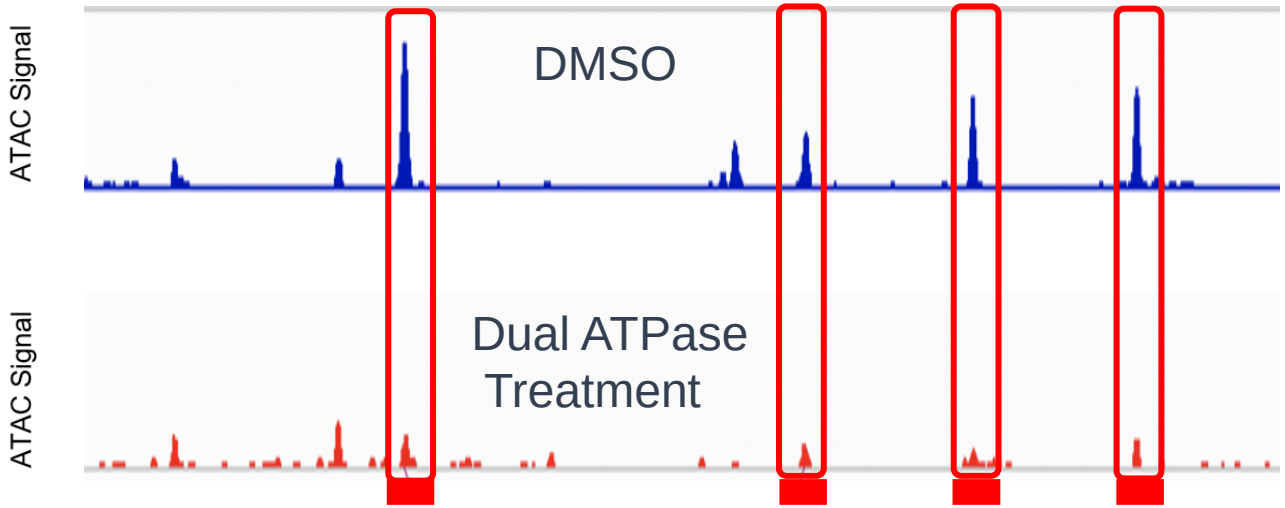
ATAC-seq Shows SPI1 Regulatory Elements are BAF Dependent



ATAC-Seq measures open regions of genome



Rank	Motif	Name	P-value	log P-pvalue
1		PU.1(ETS)/ThioMac-PU.1-ChIP-Seq(GSE21512)/Homer	1e-95	-2.209e+02
2		ELF5(ETS)/T47D-ELF5-ChIP-Seq(GSE30407)/Homer	1e-77	-1.792e+02
3		SpiB(ETS)/OCILY3-SPIB-ChIP-Seq(GSE56857)/Homer	1e-63	-1.471e+02
4		PU.1-IRF(ETS:IRF)/Bcell-PU.1-ChIP-Seq(GSE21512)/Homer	1e-61	-1.412e+02
5		EHF(ETS)/LoVo-EHF-ChIP-Seq(GSE49402)/Homer	1e-57	-1.329e+02
6		ELF3(ETS)/PDAC-ELF3-ChIP-Seq(GSE64557)/Homer	1e-56	-1.295e+02
7		PU.1:IRF8(ETS:IRF)/pDC-Irf8-ChIP-Seq(GSE66899)/Homer	1e-55	-1.268e+02

What motifs exist at newly closed chromatin?

All SPI1 Binding Signature

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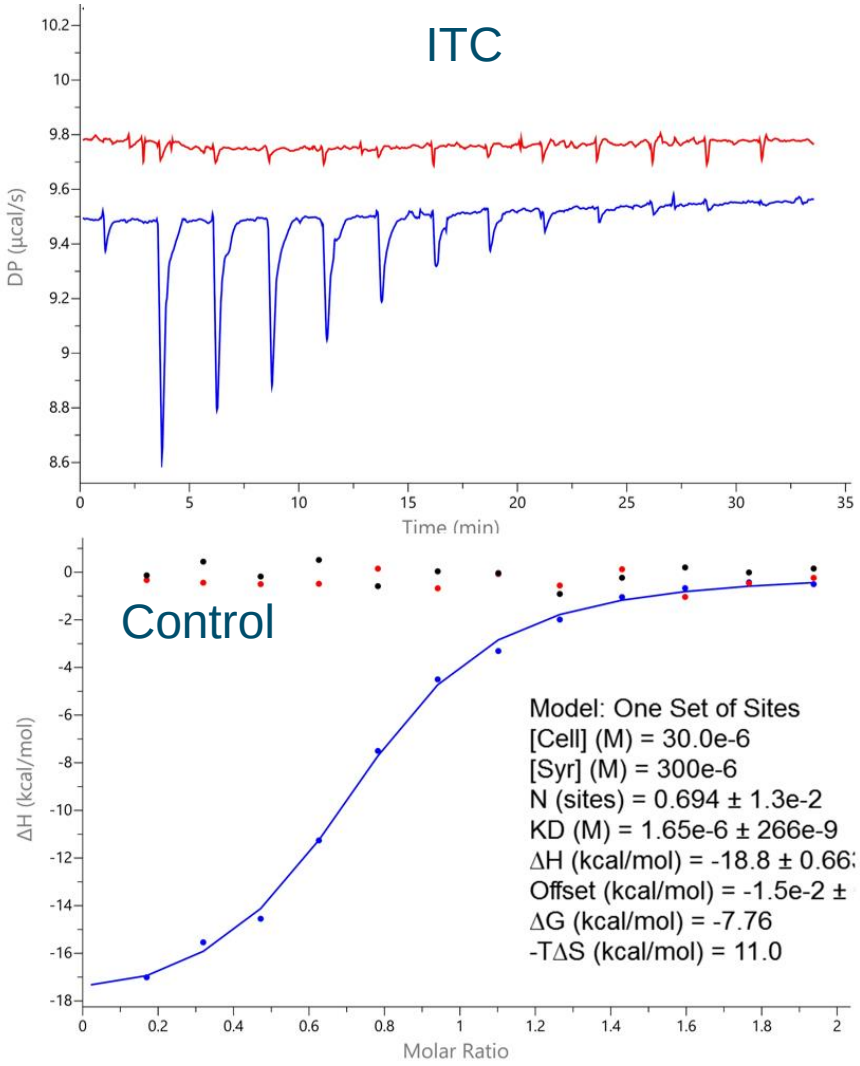
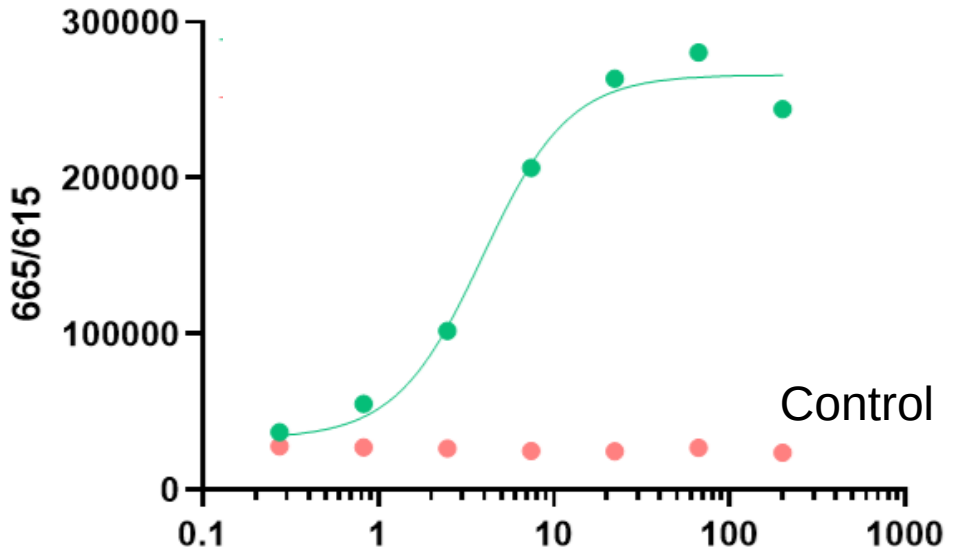
Full BAF Mapping Identifies Key Interactions With SPI1

TR-FRET Based Screening Assays Constructed

ITC



TR-FRET



Control curves have key interaction residues deleted from BAF subunit

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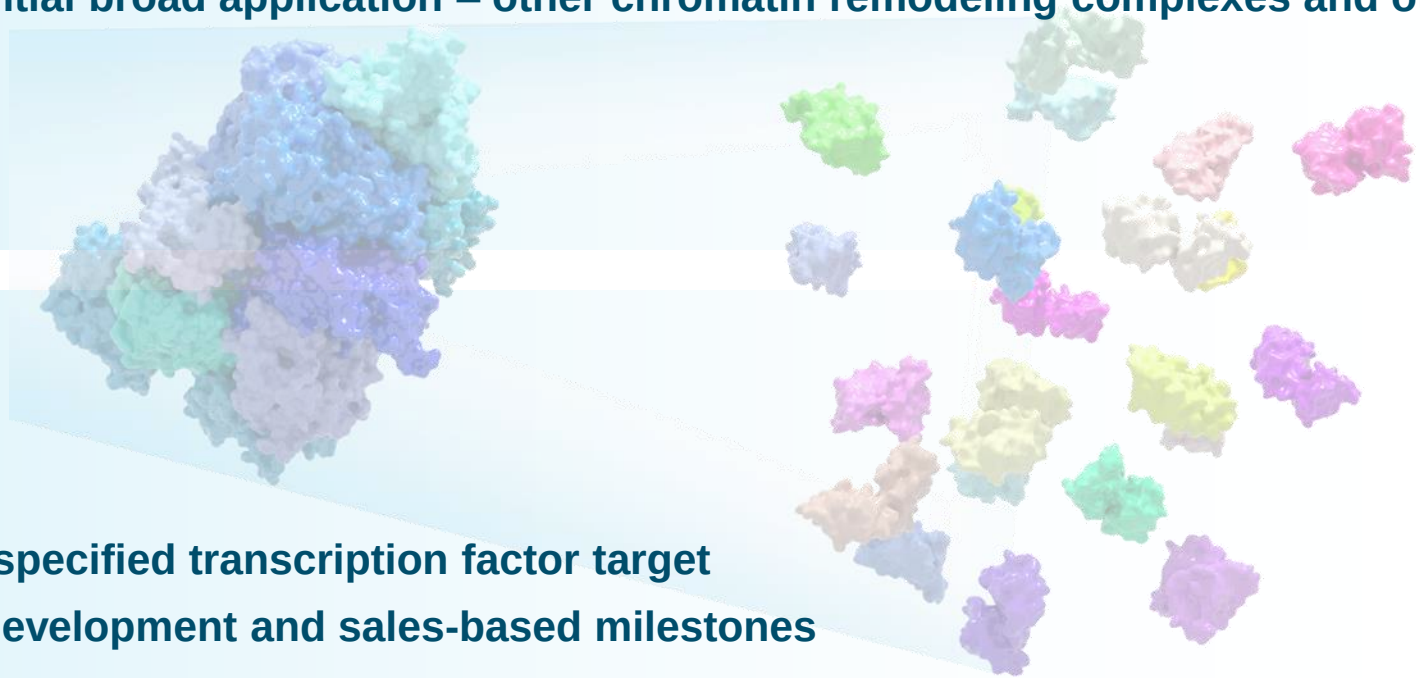
Highly Scalable Approach and Significant Unmet Medical Need

Potential to Drug > 100 TFs Associated with BAF

- **>100 TFs estimated associated with BAF**
- **Foghorn pursuing multiple TFs in parallel**
- **Approach highly scalable and potential broad application – other chromatin remodeling complexes and other diseases**



MERCK

- **Merck collaboration to drug single specified transcription factor target**
- **$425 million in up-front, research, development and sales-based milestones**
- **Up to low double-digit royalties on product sales**

FHD-286: Clinical Entry Point - AML and Uveal Melanoma

Sam Agresta, MP, MPH & TM

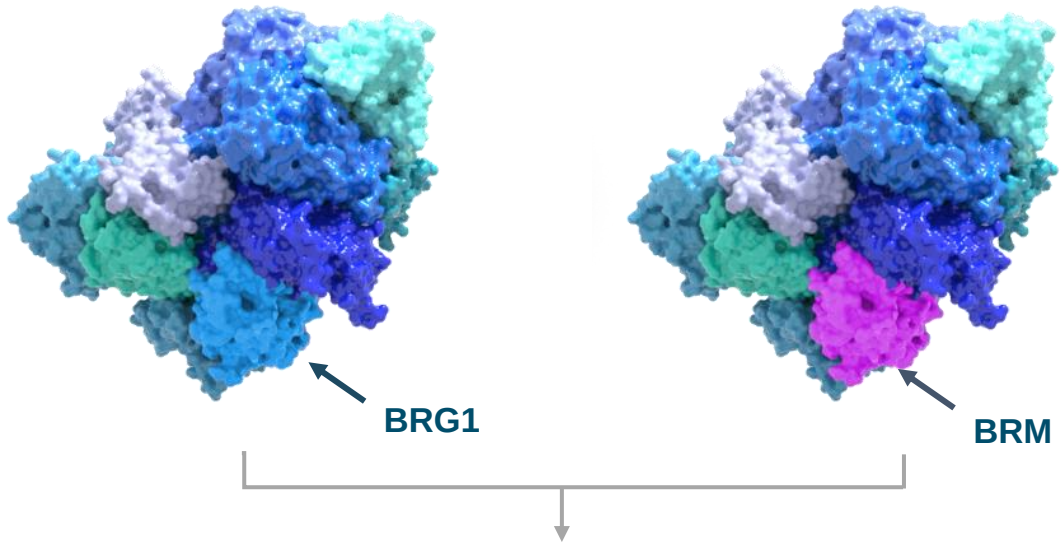
Chief Medical Officer, Foghorn Therapeutics

FHD-286 Targets Abnormal Dependencies on BAF in Cancer

Target / Approach	• BRG1/BRM ATPase • Small molecule, allosteric, oral enzymatic inhibitor
Indications	• Acute myelogenous leukemia (AML) • Uveal melanoma • Indication expansion work ongoing in multiple solid tumors
Mutation / Aberration	• **AML**: Elevated BRG1-BAF / TF activity in AML blast cells • **Uveal Melanoma**: GNAQ/GNA11 mutated UM is driven by dependency on BAF / TF activity
Program Status / Milestones	• Phase I studies enrolling in AML and metastatic uveal melanoma • Phase I data as early as Q4'21
New Patients Impacted / Year*	• **AML**: **Over 20,000 relapsed and/or refractory patients** • **Uveal melanoma: Over 5,000 patients**

* US, EU5, Japan

BAF Chromatin Remodeling Complex



- **BRM/BRG1 is the engine (ATPase) of the BAF chromatin remodeling complex**
- **BRG1 & BRM are highly similar proteins**

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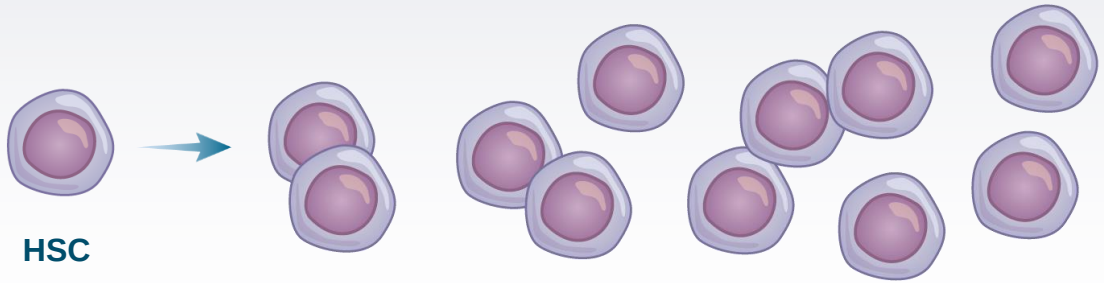
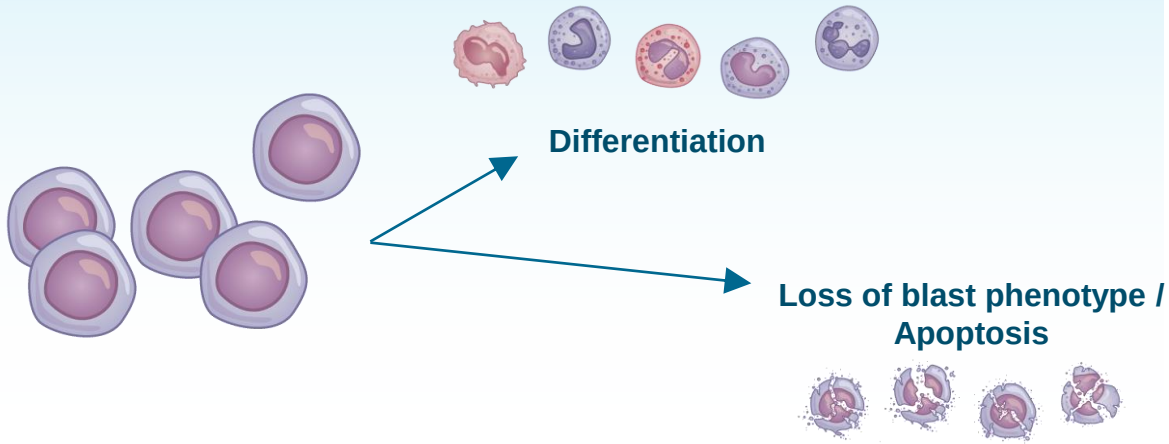
AML & Dependency on BRG1 / Lineage Dependent TF Interactions

Disease State

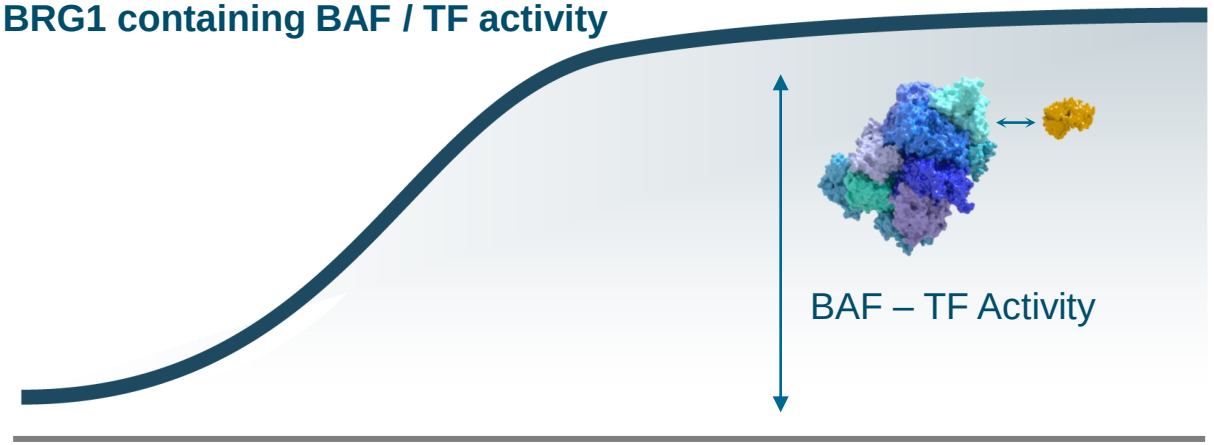


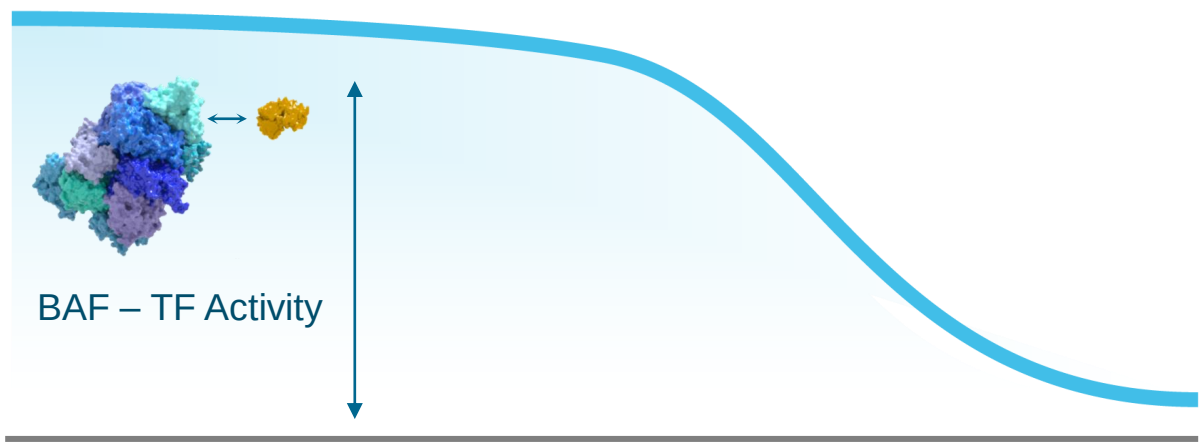
AML blasts stuck in BAF / TF dependent proliferative phase

Treatment with FHD-286



Cancerous blast cells rely on BRG1 containing BAF / TF activity





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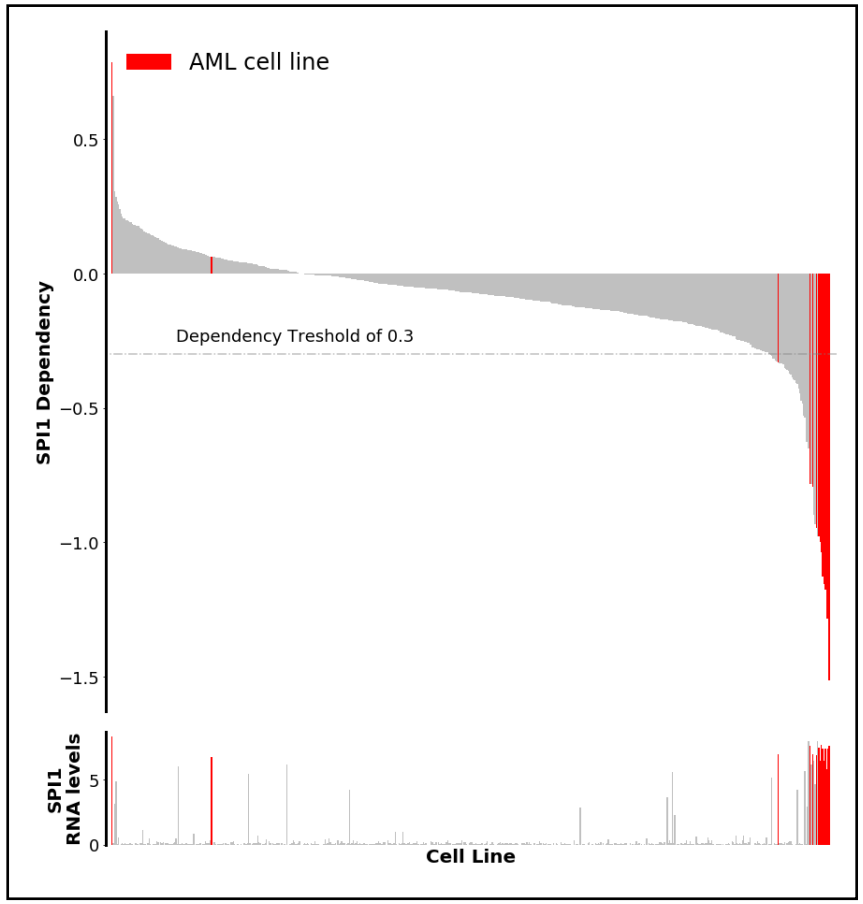
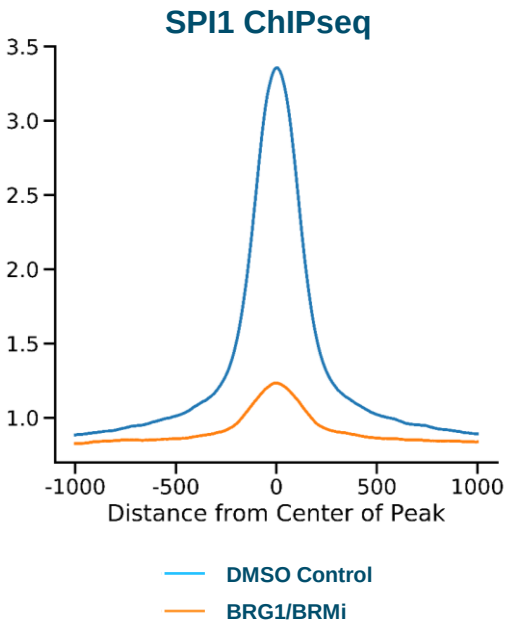
AML Dependent on BRG1 / Lineage TF Interaction

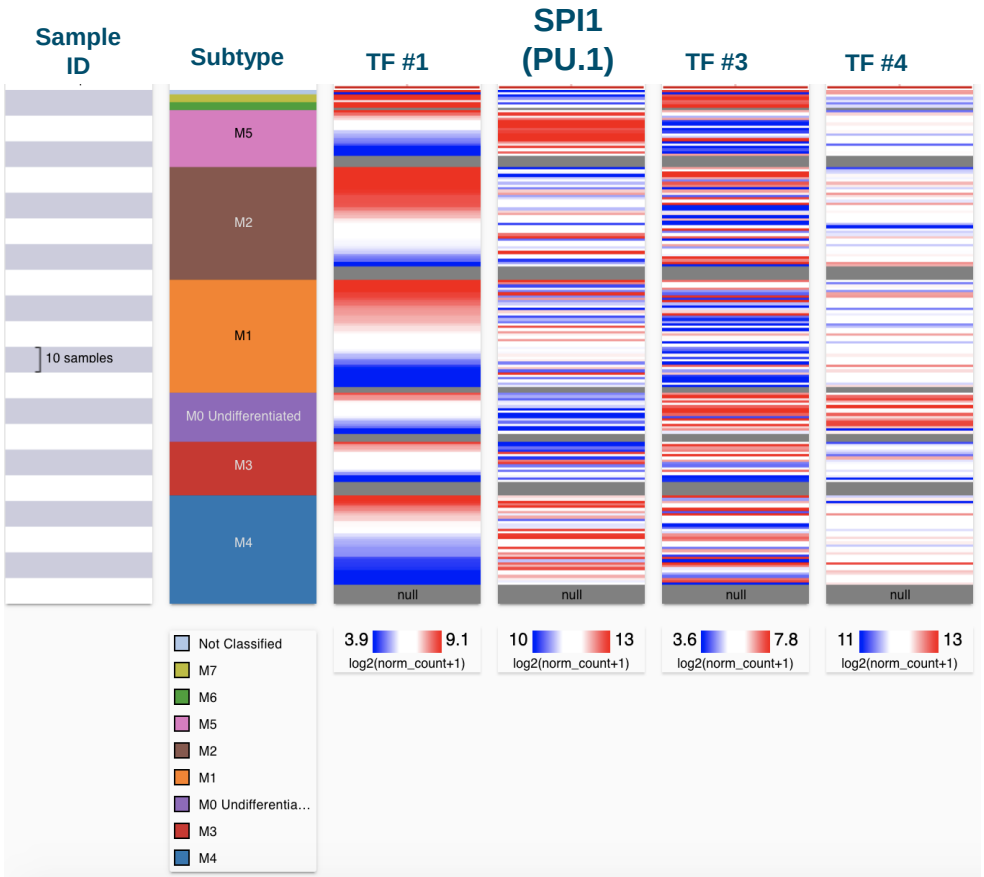
SPI1 (PU.1) / BAF Dependency



BRG1 Inhibition Leads to Loss of SPI1 (PU.1) Occupancy on Chromatin



Four TF's Associated With 70% of AML



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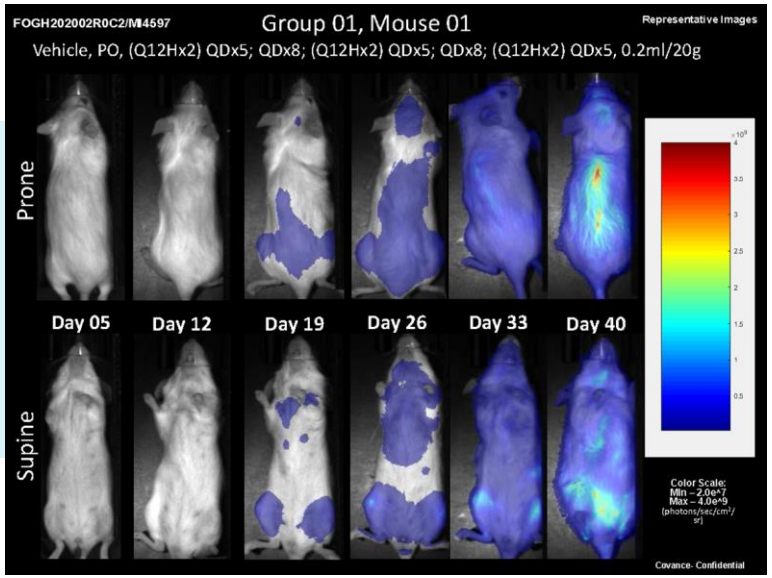
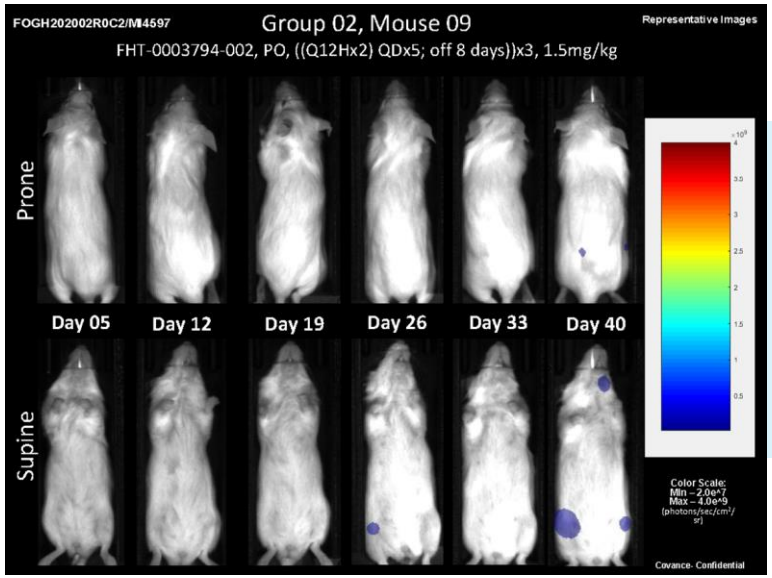
Tumor Growth Inhibition with FHD-286 Treatment Observed by Bioluminescence

Imaging in a Disseminated AML model

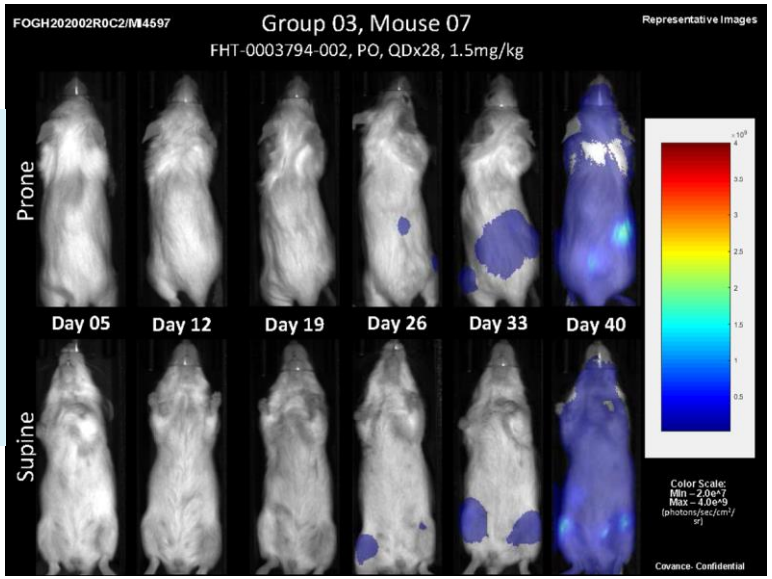
Vehicle

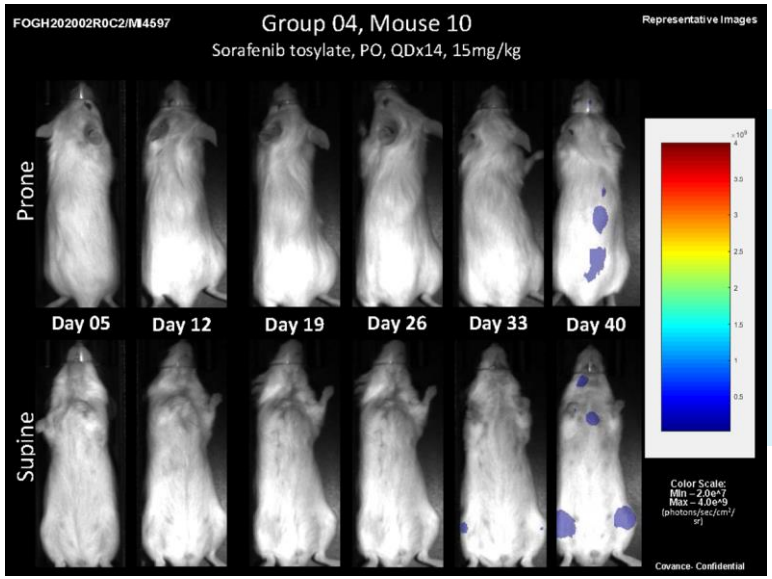




FHD-286
1.5 mg/kg, BID
(5on / 8off) x3

FHD-286
1.5 mg/kg
QDx28





Sorafenib
15 mg/kg,
QDx14

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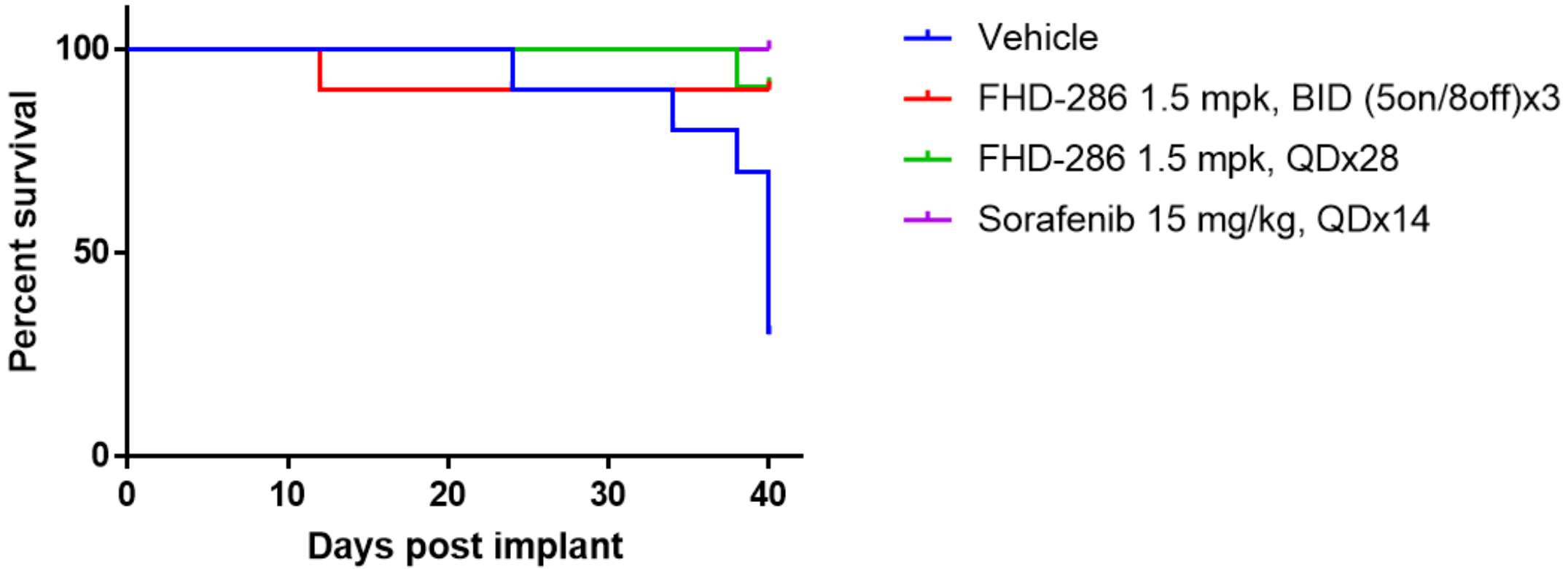
FHD-286 Survival Advantage in Disseminated AML Model

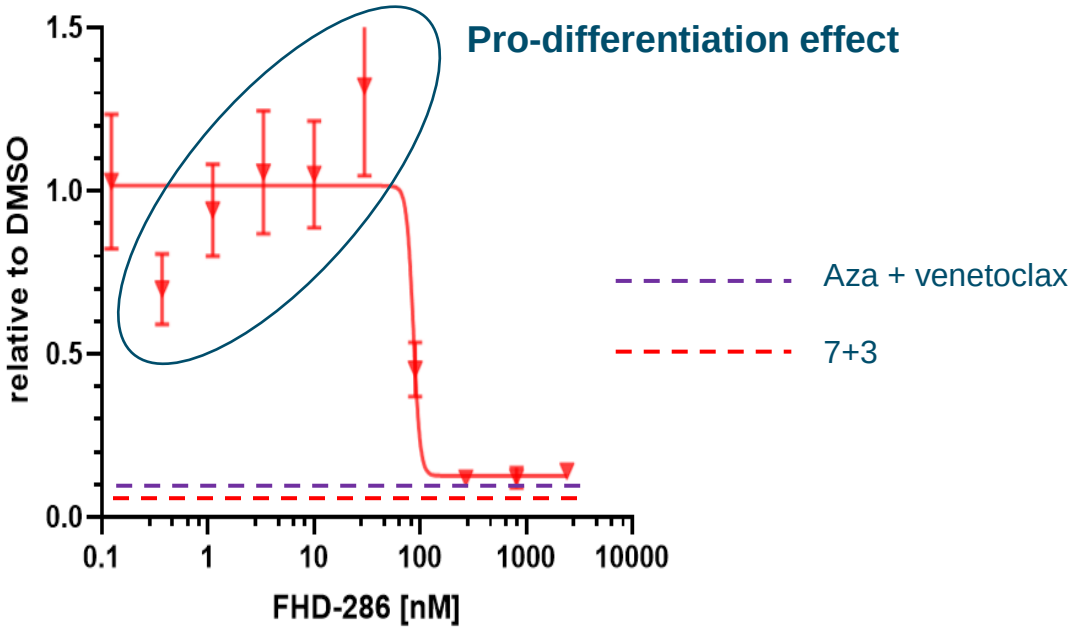


FHD-286 Shows Broad Efficacy Across AML Patient Derived Samples

Notable Patient ID	Deep Response	Pathology Review	Disease Status
1690AML1	Y	AML	Secondary
1695AML1	Y	AML/MDS	Secondary
1696AML1	Y	AML	Secondary
1701AML1	Y	AML	Secondary
1893AML1	Y	AML	R/R
1899AML1	Y	AML	R/R
1990pAML1	Y	AML	R/R
1991pAML1	Y	AML	de novo
2041AML1	Y	N/A	de novo
2043pAML1	Y	AML	R/R
2059AML1	Y	AML	R/R
1682AML1	~	N/A	N/A
1689AML1	~	AML/MDS	de novo
1684AML1	N	CML	R/R
1924AML1	N	AML/MDS	R/R

Y = Deep reduction in blast cells
~ = Partial reduction
N = No response

1695AML1 – BM-secondary AML



- **Response observed in a majority of primary AML samples, irrespective of prior treatment or disease stage**
- **Additional data set from patient derived samples demonstrate mutation agnostic responses**

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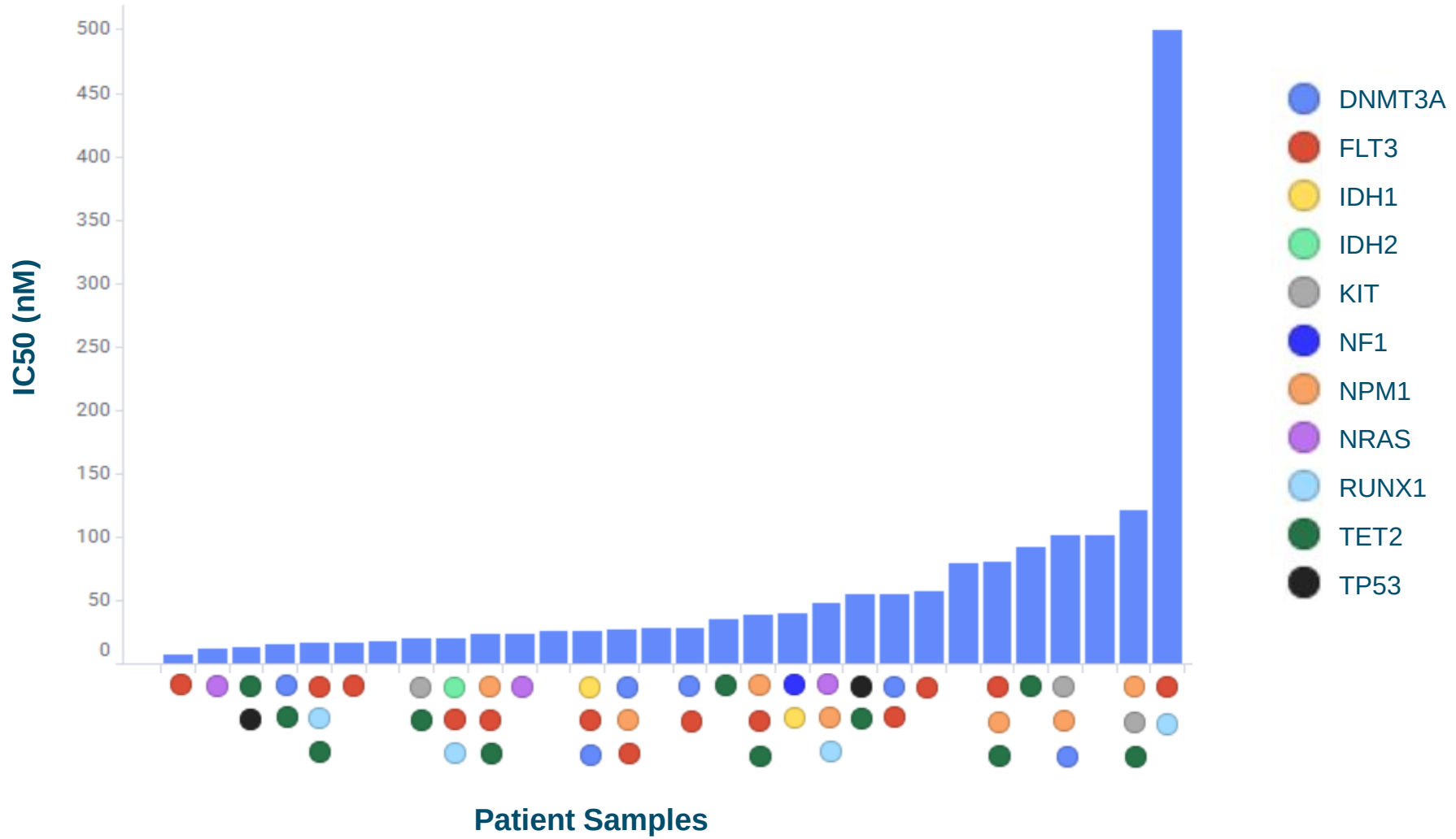
FHD-286 Shows Effect Across a Range of Mutations in AML Patient-Derived Samples



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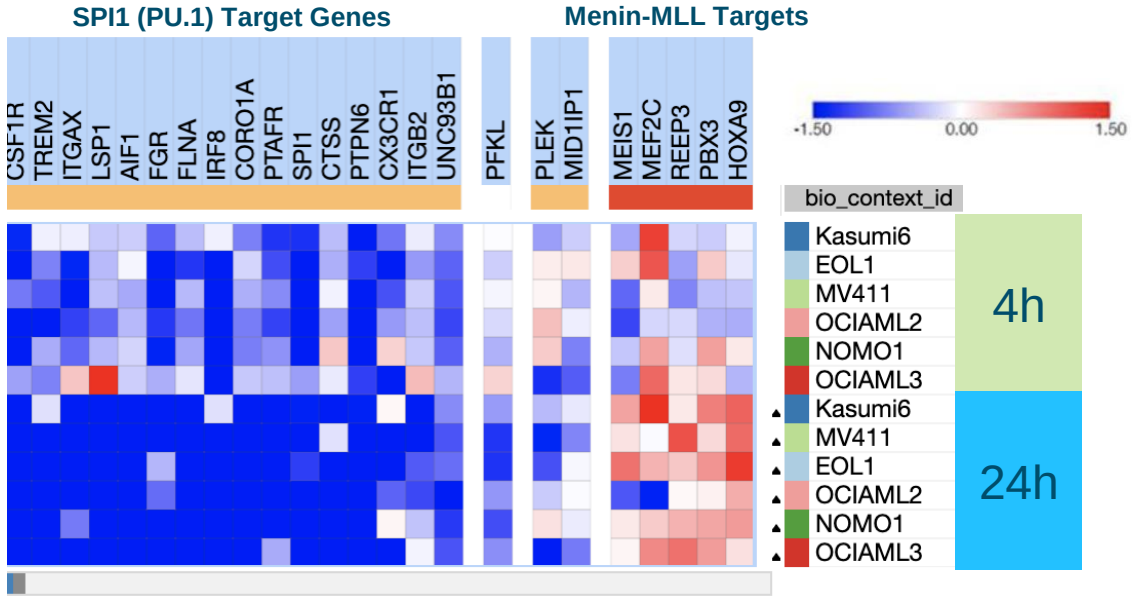
Potential PD Marker: AML TFs and Gene Signatures

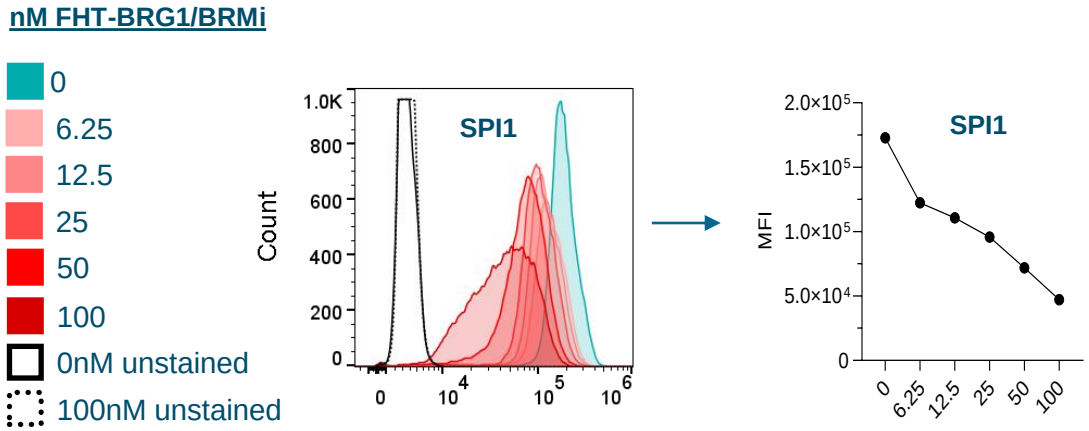
BAF Regulates SPI1, A Key Transcription Factor in AML Subsets

FHD-286 downregulates SPI1 protein and downstream SPI1 target genes

SPI1 target RNA signature is suppressed

SPI1 protein is reduced in a dose-dependent manner





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Uveal Melanoma

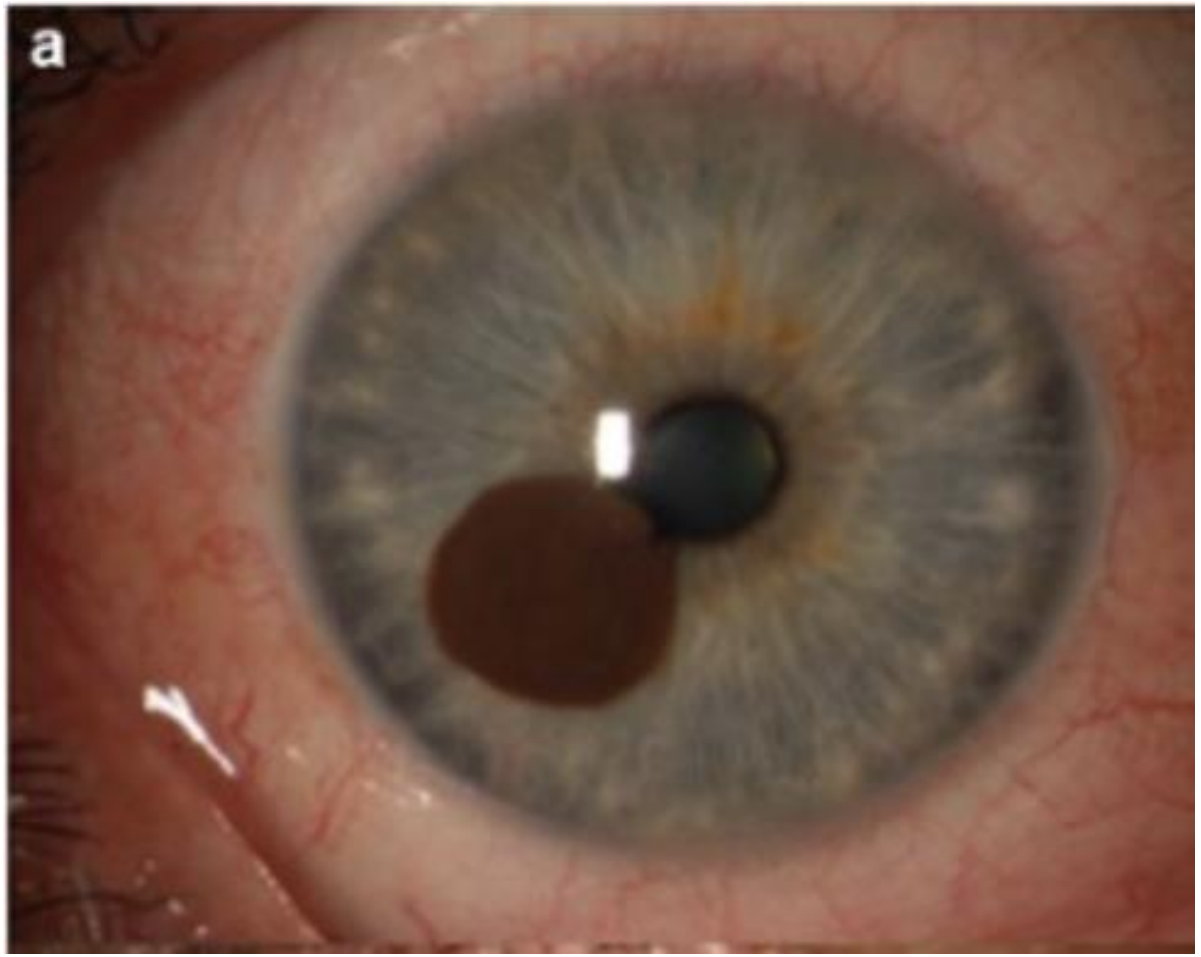
Disease Overview

Primary Disease:

- ~1,500 cases diagnosed each year, US
- Age at diagnosis: early 60s-median, 70s-peak
- ~30% asymptomatic
  - Lesion found on routine eye exam
- Mild symptoms such as blurry vision in one eye
- 95+% are primary disease at diagnosis

Metastatic Disease:

- Over 50% of patients develop metastatic disease, typically in the liver.

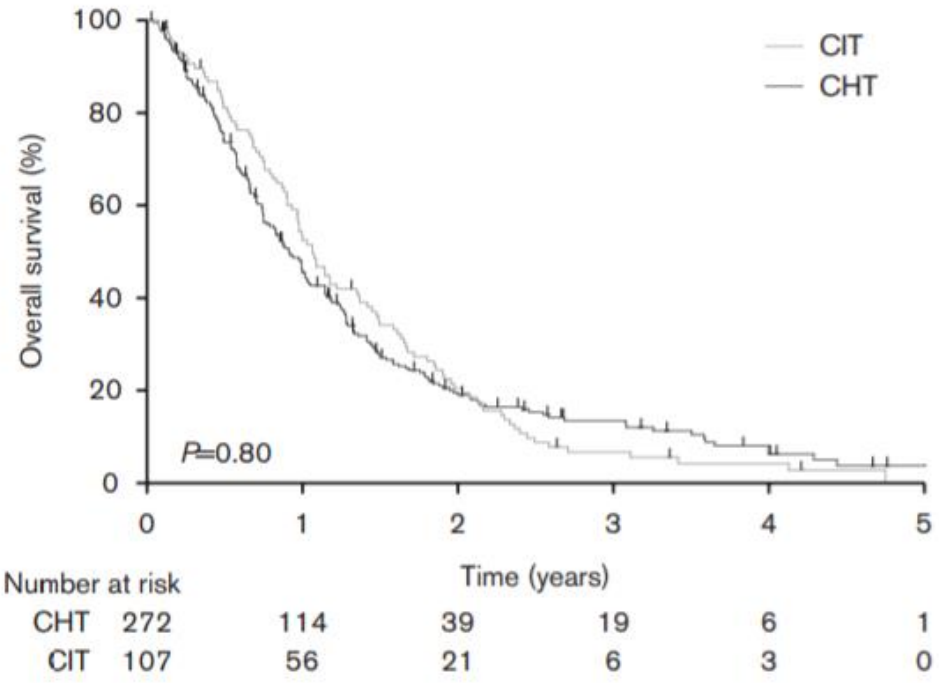


Metastatic Uveal Melanoma

Disease Overview

- Treatment: No approved treatments
  - Liver Directed: Chemoembolization, radio-ablation, immunotherapy
  - Systemic: Ipilimumab + nivolumab, dacarbazine, temozolomide
- Majority (~80%) of patients die within 1 year of detection of metastasis
- Median survival is ~6-9 months

High Unmet Need



Rantala, et. al, Melanoma Research, 2019, Vol 29 No 6

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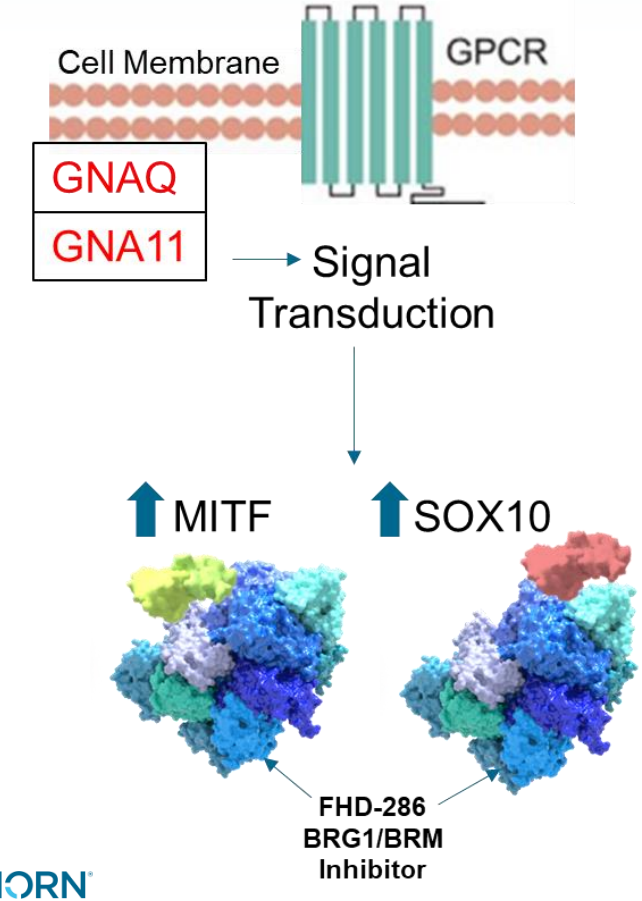
Therapeutic Rationale for Uveal Melanoma: Dependency on MITF / SOX10 TF's and BAF Complex Interaction

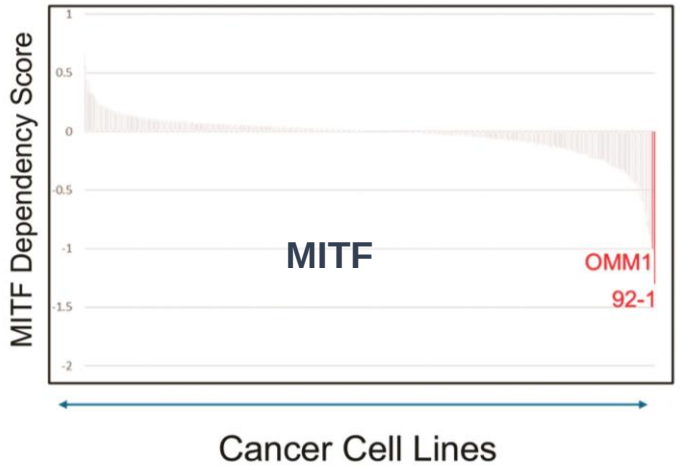
Inhibiting BRG1/BRM to Shut Down the Abnormal TF Interaction with the BAF Complex

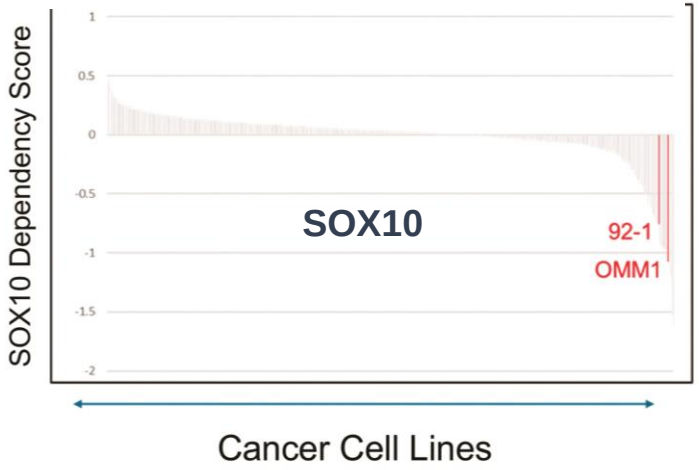
Biology

Over 85% of uveal melanoma cancers have GNAQ or GNA11 mutations

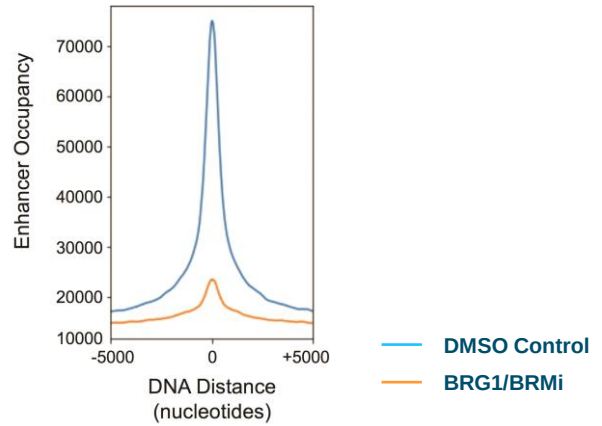


Validation of Dependency and Approach

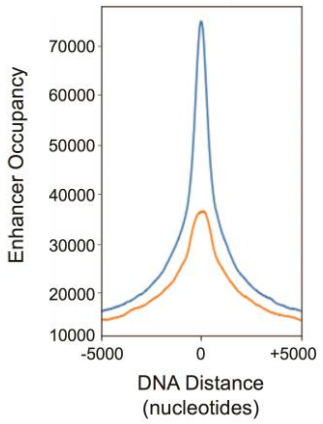




MITF ChIPseq



SOX10 ChIPseq



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Dual BRG1/BRM Inhibition

Disrupts the SOX10-MITF Transcriptional Axis in Uveal Melanoma

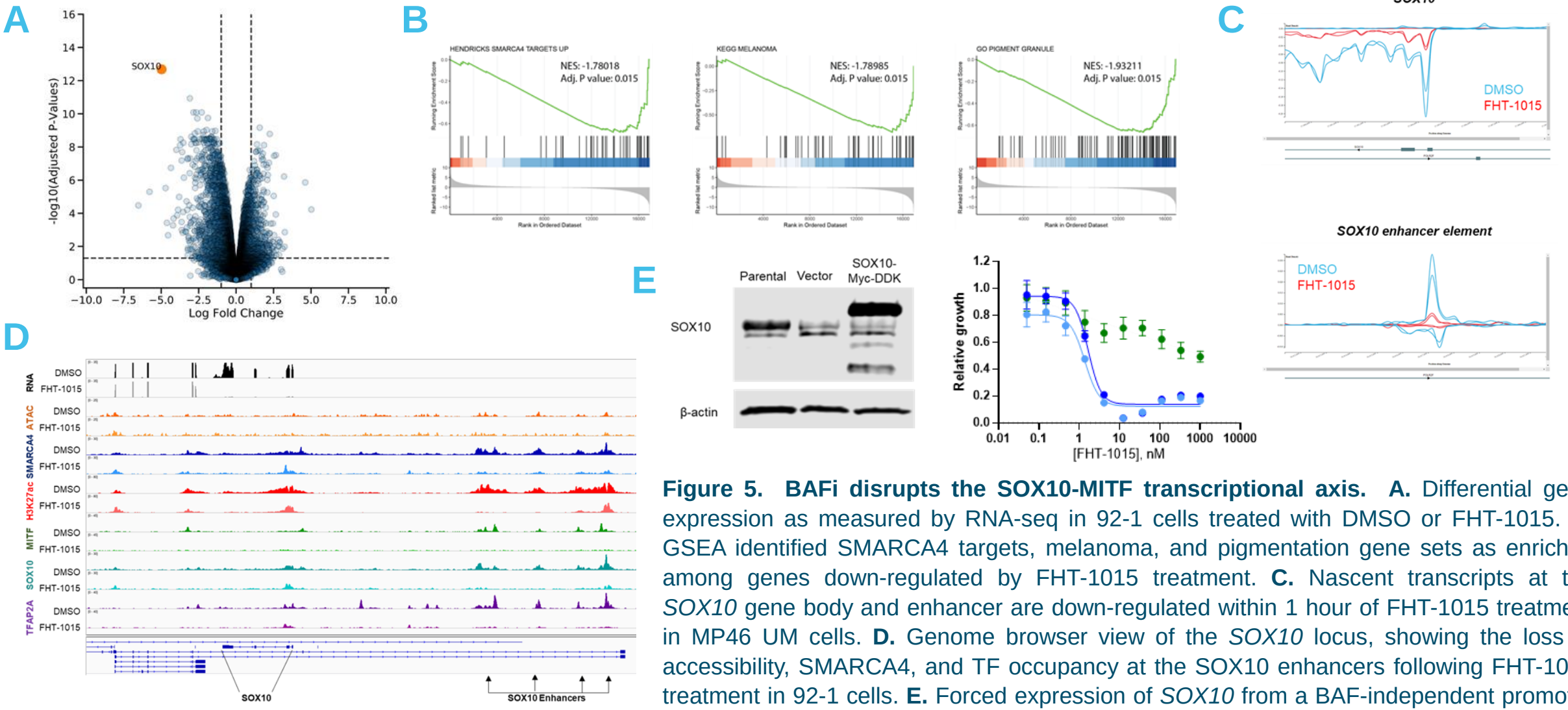

Figure 5. BAFi disrupts the SOX10-MITF transcriptional axis. **A.** Differential gene expression as measured by RNA-seq in 92-1 cells treated with DMSO or FHT-1015. **B.** GSEA identified SMARCA4 targets, melanoma, and pigmentation gene sets as enriched among genes down-regulated by FHT-1015 treatment. **C.** Nascent transcripts at the *SOX10* gene body and enhancer are down-regulated within 1 hour of FHT-1015 treatment in MP46 UM cells. **D.** Genome browser view of the *SOX10* locus, showing the loss of accessibility, SMARCA4, and TF occupancy at the SOX10 enhancers following FHT-1015 treatment in 92-1 cells. **E.** Forced expression of *SOX10* from a BAF-independent promoter can rescue the growth inhibition phenotype elicited by FHT-1015.

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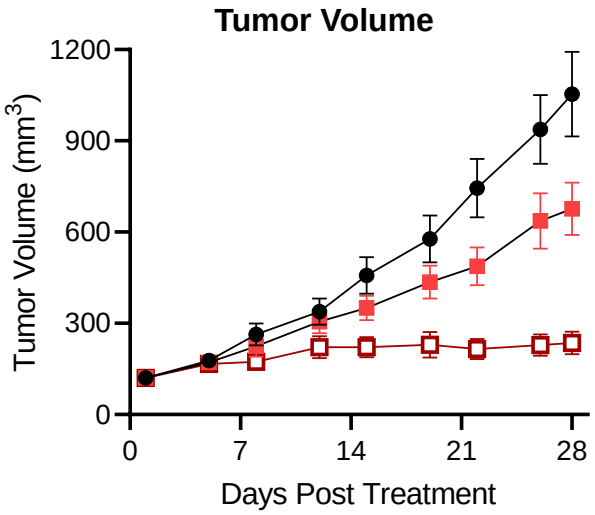
FHD-286 was Associated with Dose-Dependent Tumor Regression in Uveal Melanoma CDX Models at Tolerated Doses

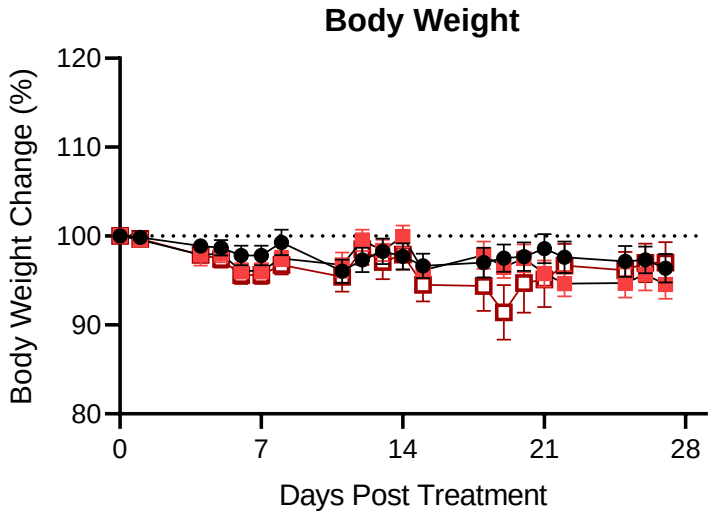
MP-46 uveal melanoma CDX model

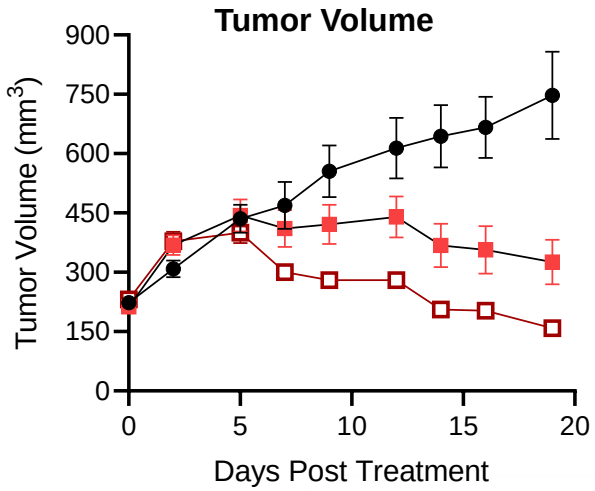
- Dose-dependent tumor growth inhibition
- Well tolerated

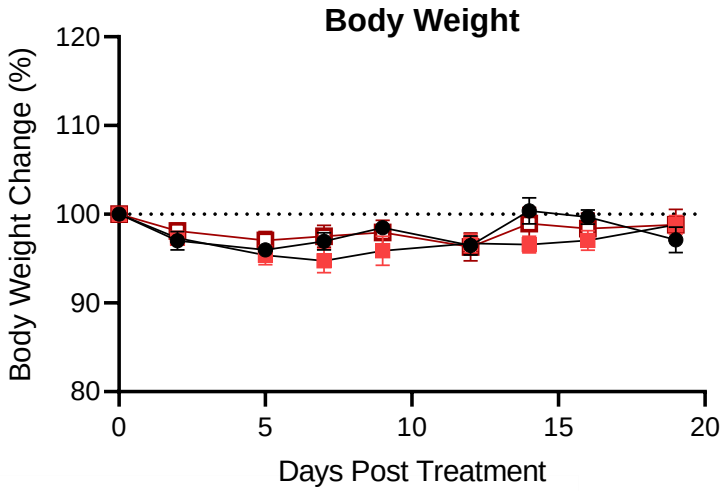
92-1 uveal melanoma CDX model

- Dose-dependent tumor growth inhibition
- Tumor regression at 1.5 mg/kg, PO, QD
- Well tolerated









Vehicle
FHD-286, 0.5 mg/kg, PO, QD
FHD-286, 1.5 mg/kg, PO, QD

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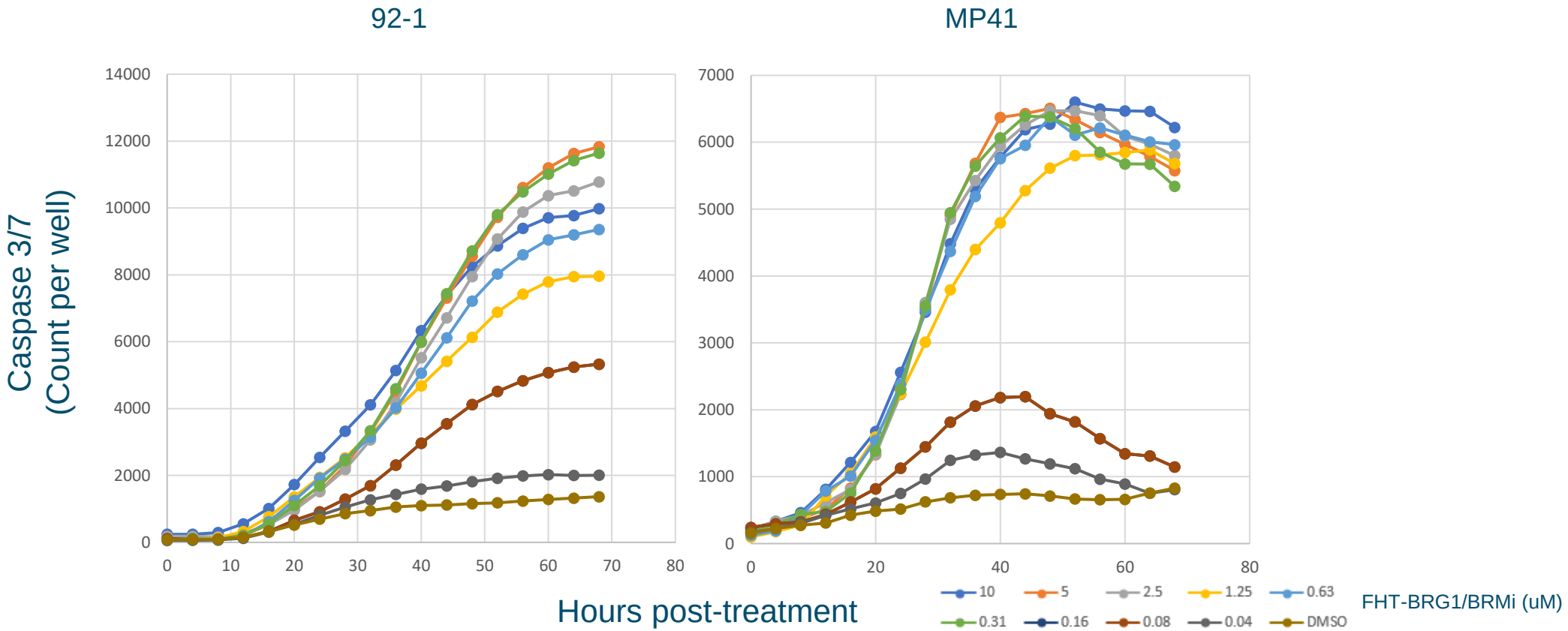
BRG1/BRM Inhibition Induces Apoptosis in UM Cell lines



Significantly faster onset (3 days) and potent growth inhibition with apoptosis in UM cell lines

FOGHORN THERAPEUTICS

FHD-286 Clinical Development Plan

Two Parallel Phase 1 Studies Activated

CLINICAL PLAN

AML & Uveal Melanoma FIH Phase 1 Studies

Relapsed / Refractory AML & MDS

Metastatic Uveal Melanoma

Trial Designs

- Single patient accelerated titration (n=1)
- Convert to 3+3 once relevant PK/PD, safety or clinical activity observed
- Retrospective biomarker analysis to further evaluate safety and efficacy
- Assess safety, PK, biomarkers and efficacy

Expansion cohorts in AML, UM and potentially other indications

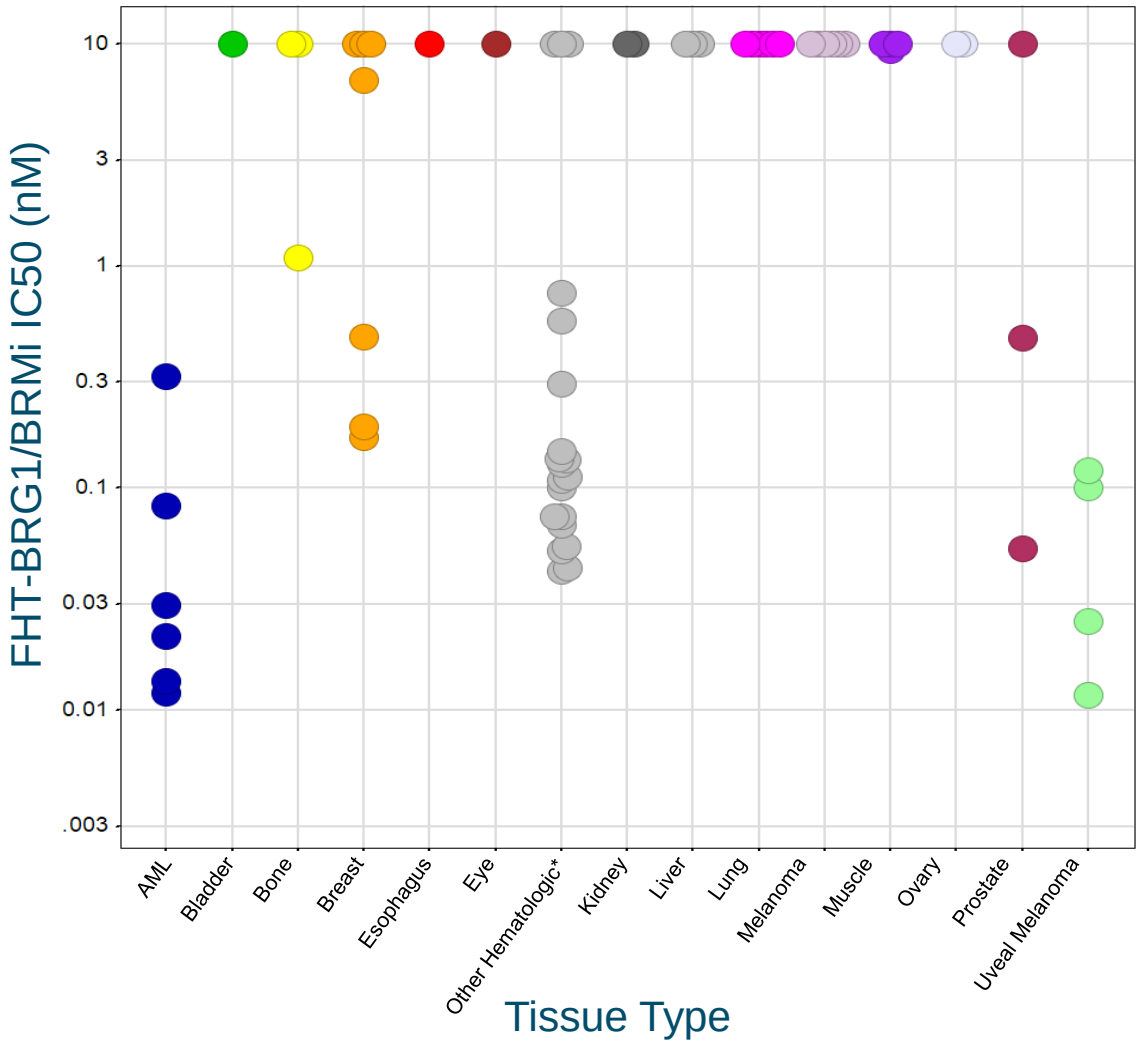
Potential for entry into definitive efficacy trials in AML

Potential for entry into definitive efficacy trials in metastatic uveal melanoma

Potential for Indication Expansion Beyond AML and UM

Clinical data as early as Q4 2021

Indication Expansion Opportunities for FHD-286



*DLBCL, B-ALL, T-ALL, MDS, MM, Burkitt's Lymphoma, Mantle Cell Lymphoma

FOGHORN THERAPEUTICS

nature COMMUNICATIONS

ARTICLE

Check for updates

https://doi.org/10.1038/s41467-020-19328-1 OPEN

Role of specialized composition of SWI/SNF complexes in prostate cancer lineage plasticity

Joanna Cyrta et al.#

SCIENTIFIC REPORTS

nature research

SMARCA2-deficiency confers sensitivity to targeted inhibition of SMARCA4 in esophageal squamous cell carcinoma cell lines

Katharina Ehrenhöfer-Wölfer[1], Teresa Puchner[1], Cornelia Schwarz[1], Janine Rippka[1], Silvia Blaha-Ostermann[1], Ursula Strobl[1], Alexandra Hörmann[1], Gerd Bader[1], Stefan Kornigg[1], Stephan Zahn[1], Wolfgang Sommergruber[1], Norbert Schweifer[1], Thomas Zichner[1], Andreas Schlattl[1], Ralph A. Neumüller[1], Junwei Shi[2], Christopher R. Vakoc[3], Manfred Kögl[1], Mark Petronczki[1], Norbert Kraut[1], Mark A. Pearson[1] & Simon Wöhrle[1]

Memorial Sloan Kettering Cancer Center

Acute Myeloid Leukemia in 2021

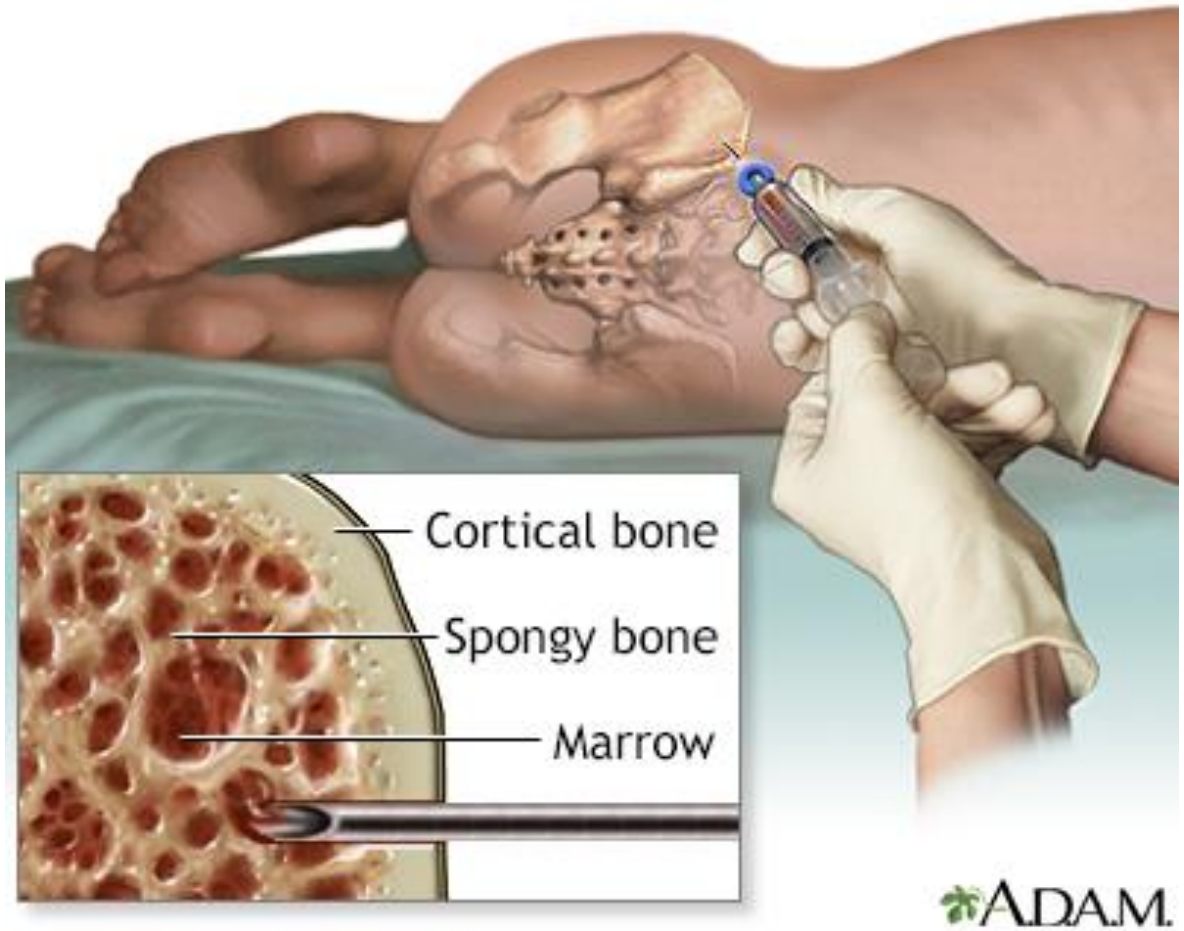
Eytan M. Stein, MD

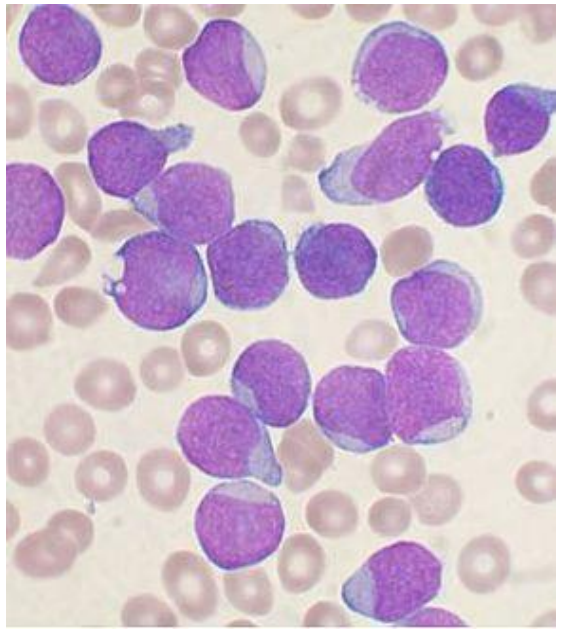
Director, Program for Drug Development in Leukemia

Leukemia Service
Memorial Sloan Kettering Cancer Center

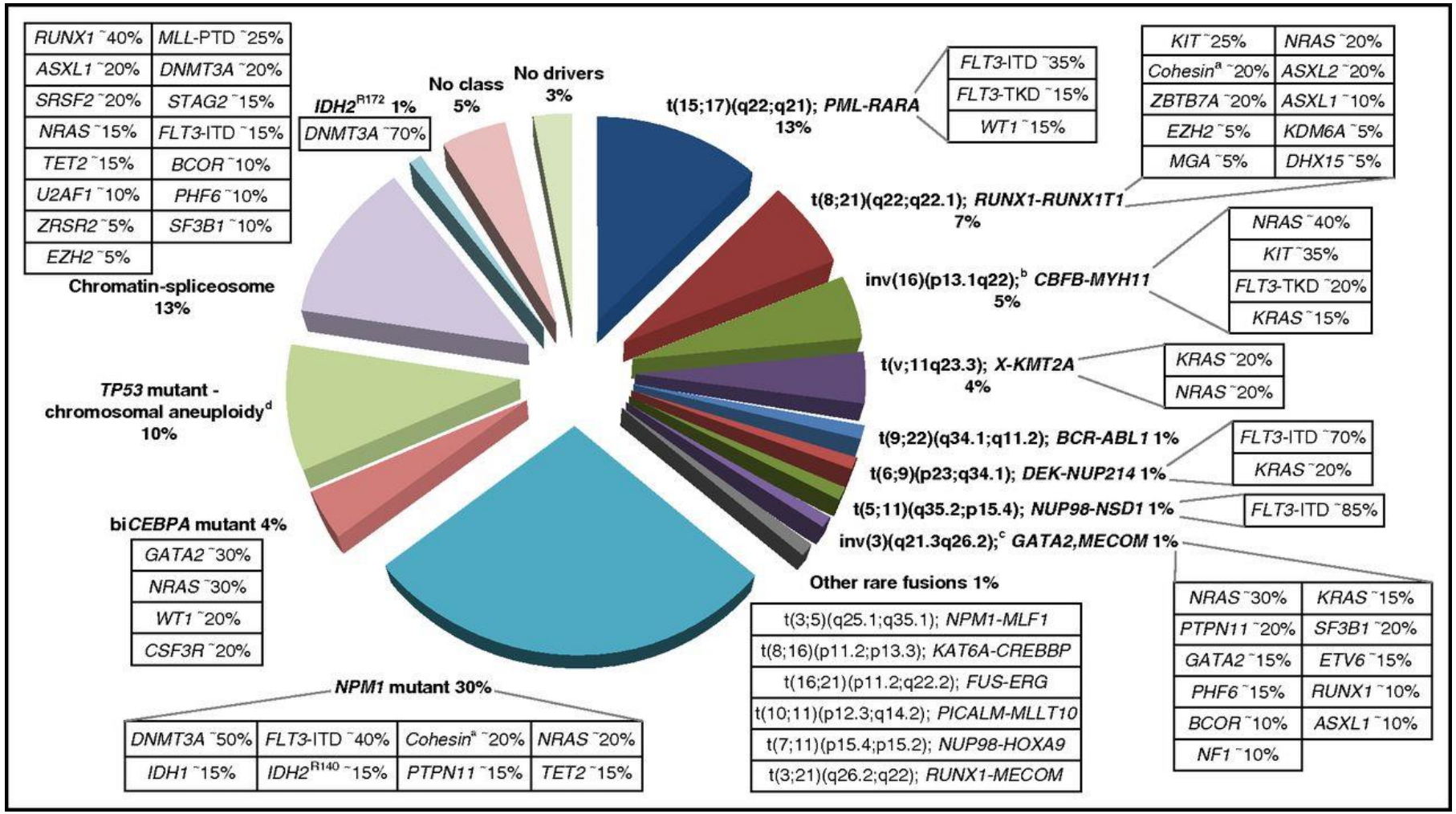
New York, New York

Acute Myeloid Leukemia





AML is Complicated

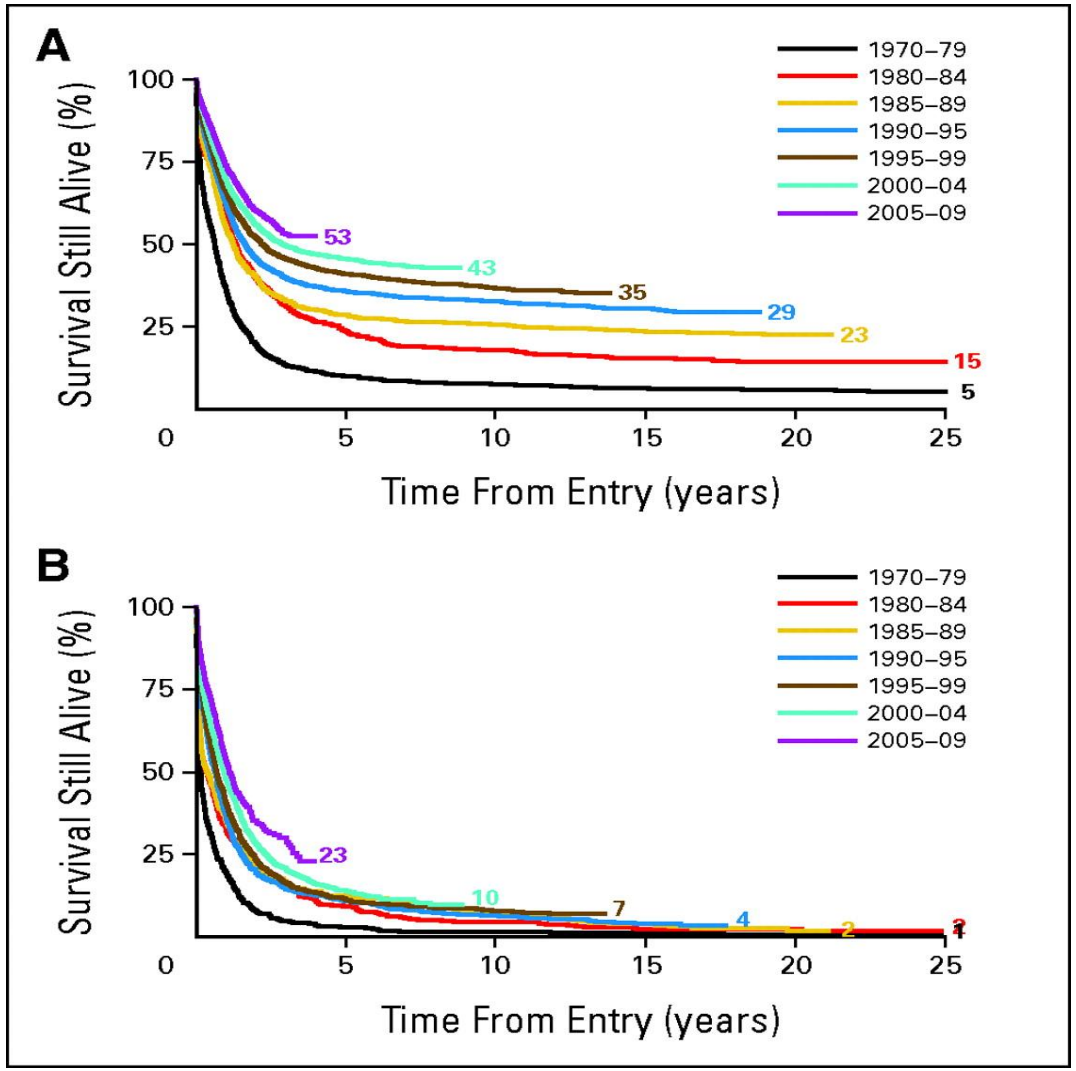


Dohner H, et. al, Blood, 2017

Overall Survival in AML

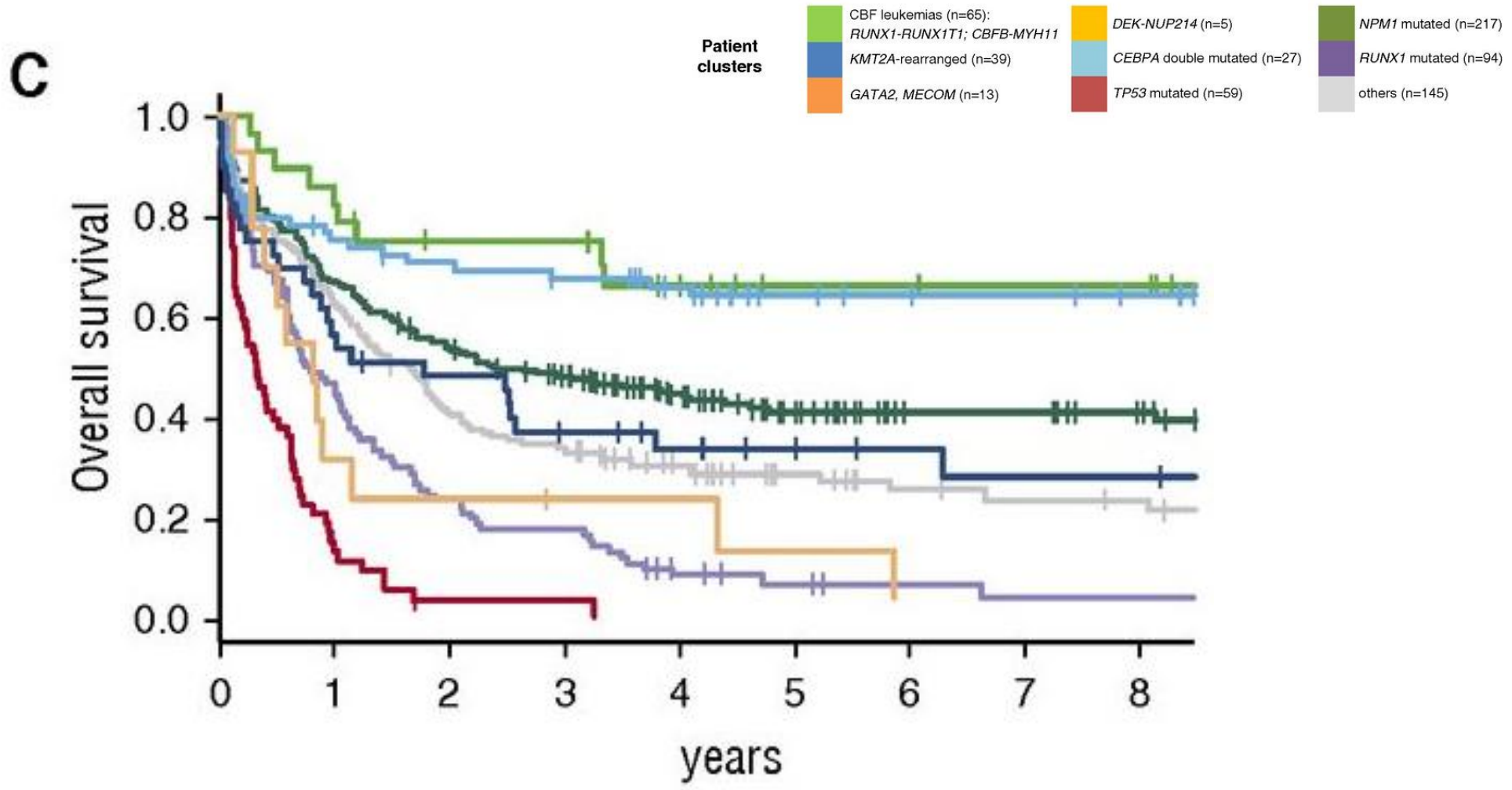
Younger than 60

Older than 60



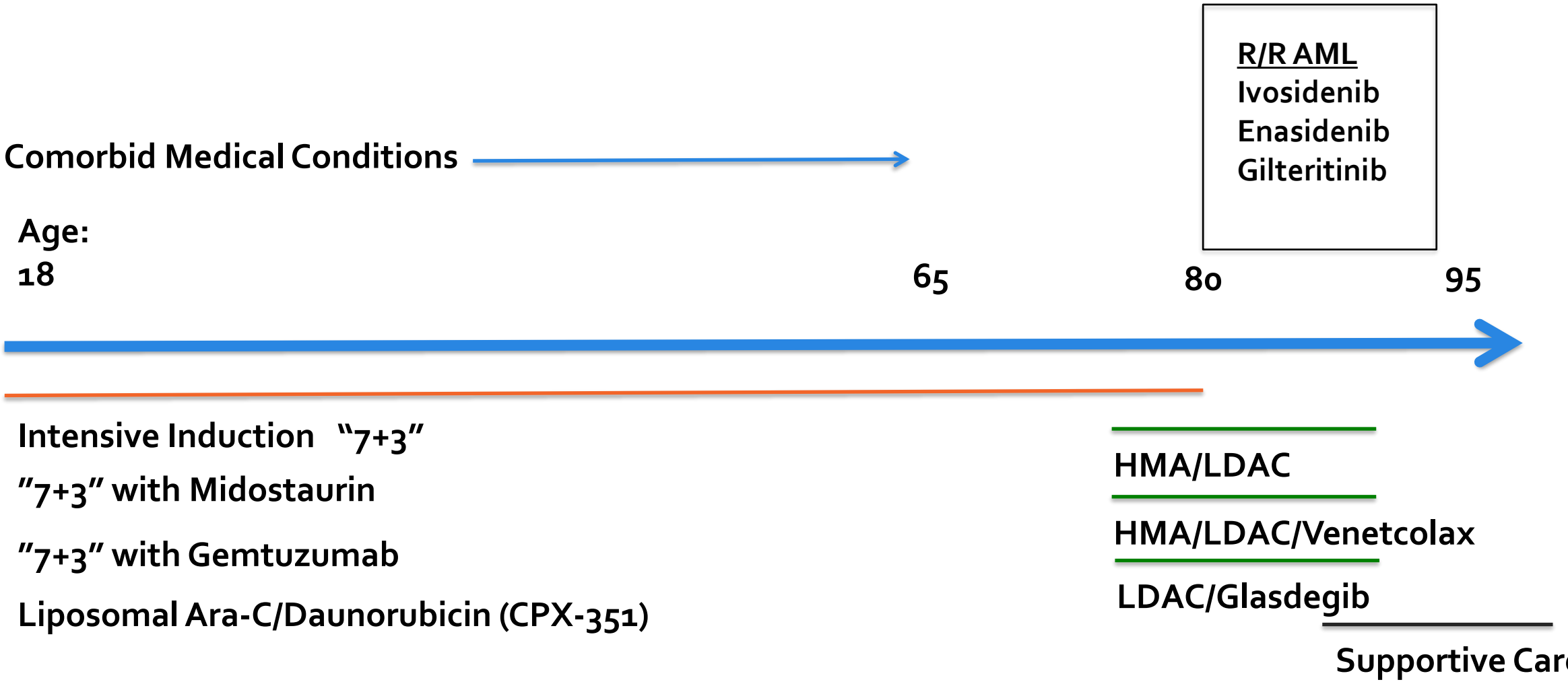
Burnett A et al. JCO 2011;29:487-494

Survival Can be Stratified Based on Genetics

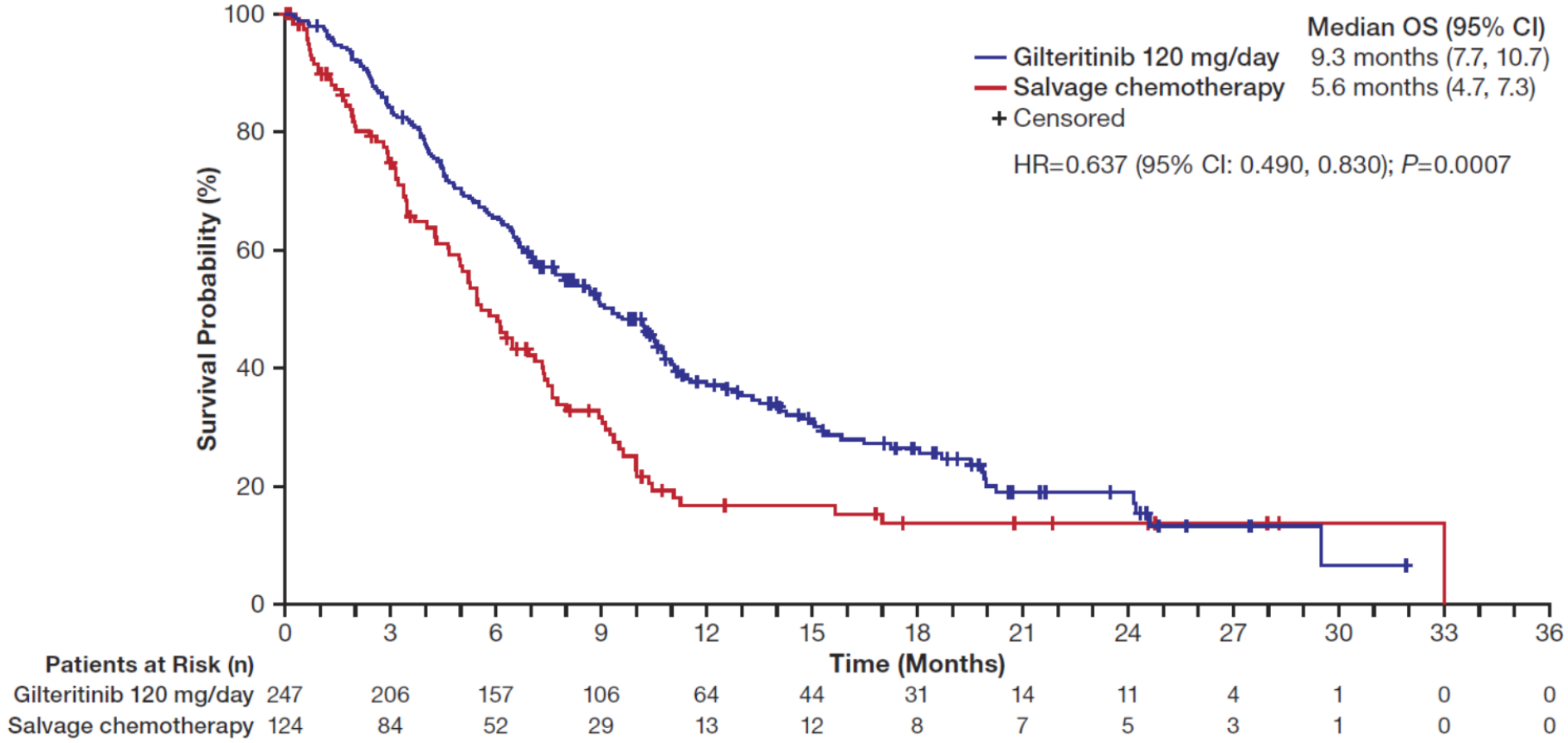


Metzeler, Blood 2016

Paradigms for Treating Newly Diagnosed AML

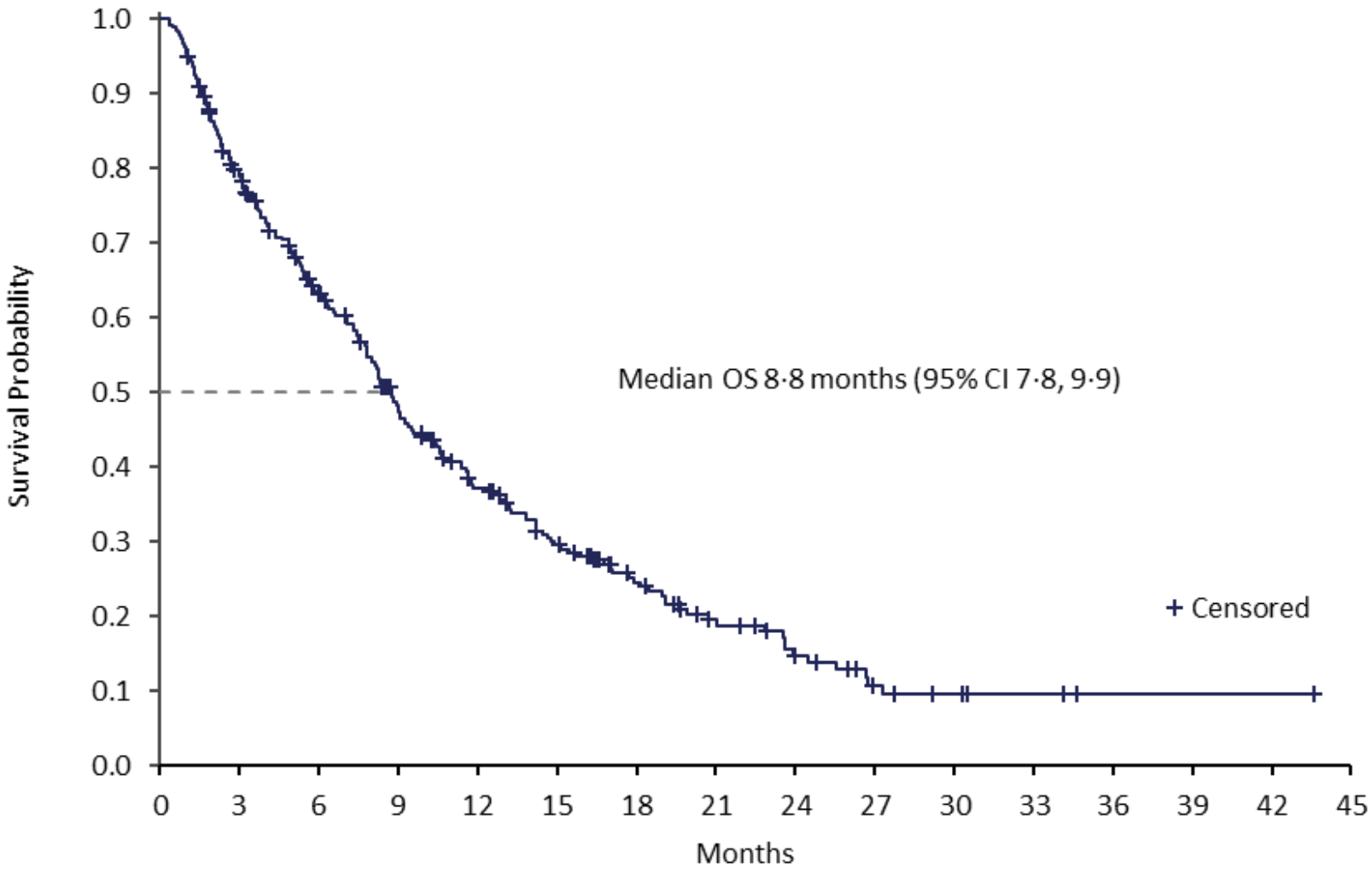


Gilteritinib for Relapsed/Refractory AML with FLT3 Mutation



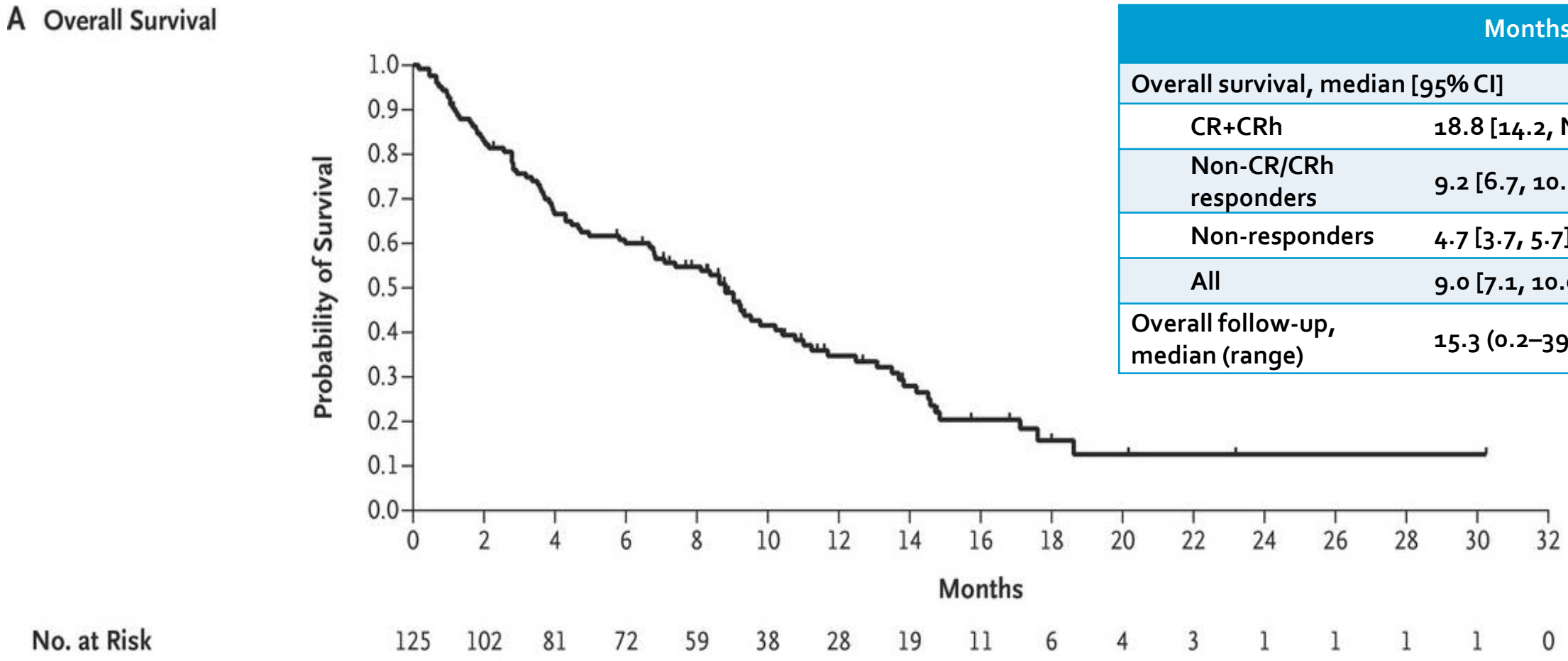
Perl A, et. al, NEJM 2019

Enasidenib (IDH2) Overall Survival – All R/R Patients



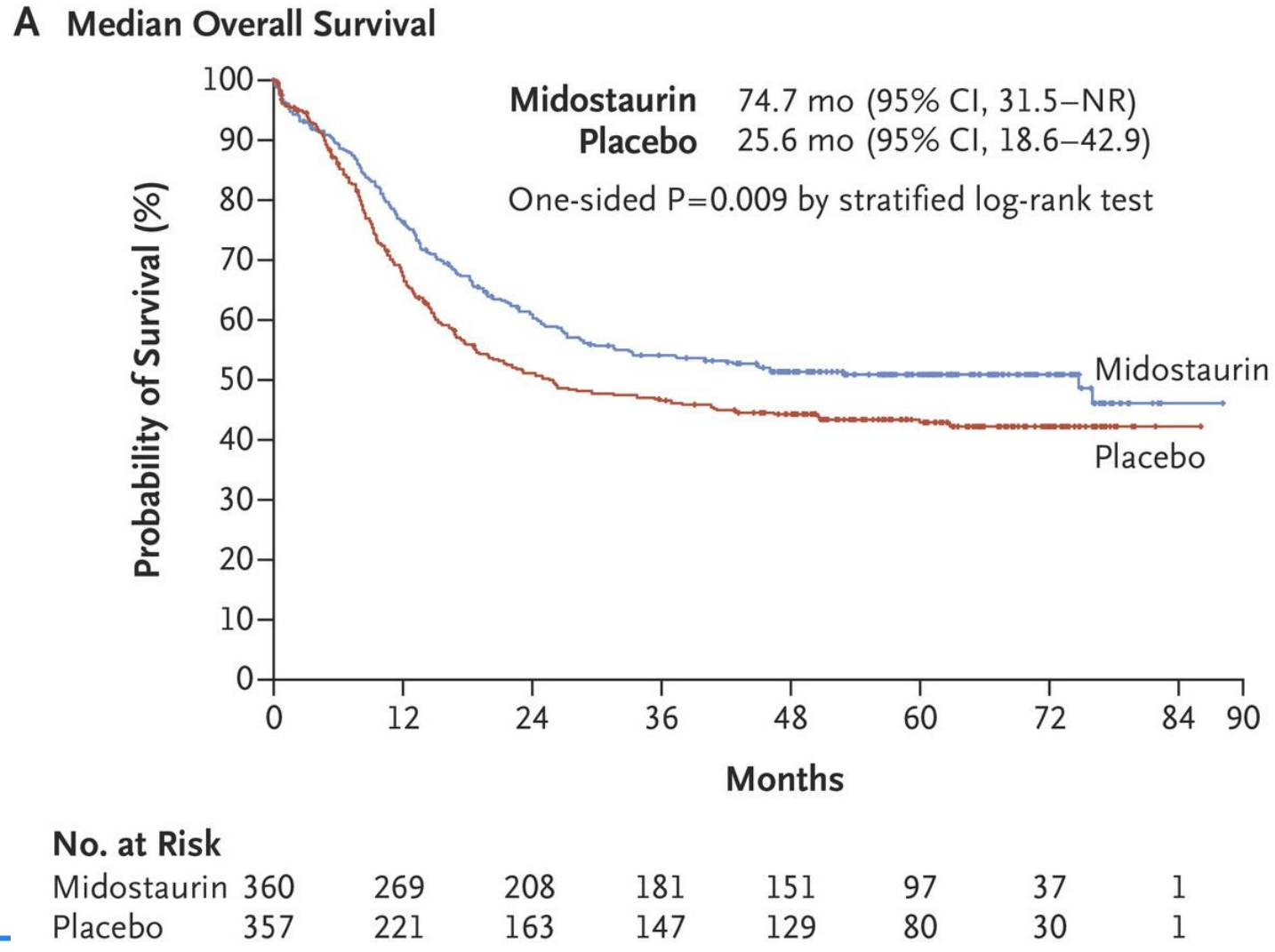
Stein EM, Dinardo CD, et al, Blood 2017

Ivosidenib (IDH1) Overall Survival – All R/R Patients



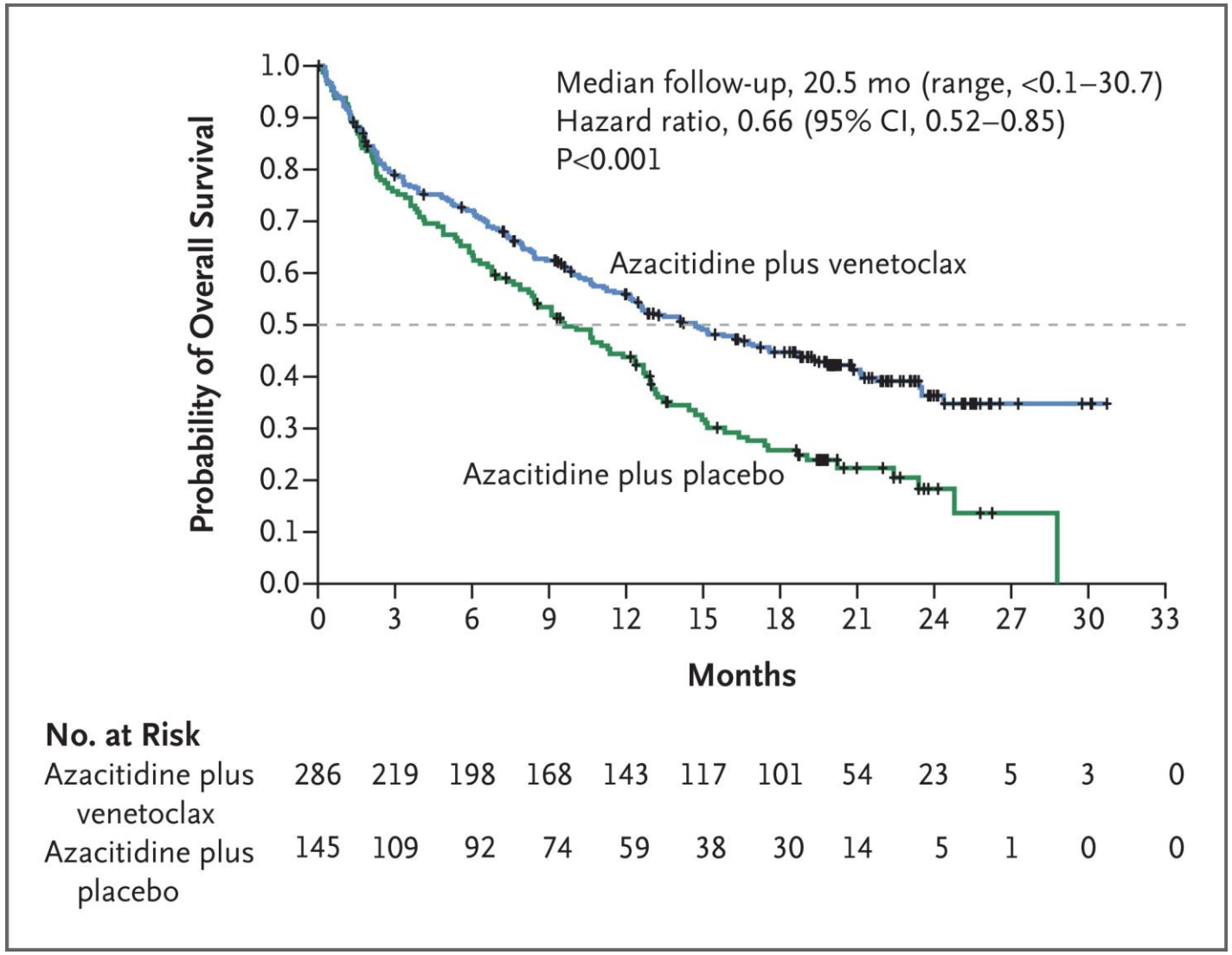
	Months
Overall survival, median [95% CI]	
CR+CRh	18.8 [14.2, NE]
Non-CR/CRh responders	9.2 [6.7, 10.8]
Non-responders	4.7 [3.7, 5.7]
All	9.0 [7.1, 10.0]
Overall follow-up, median (range)	15.3 (0.2–39.5)

Combining Midostaurin with Chemotherapy



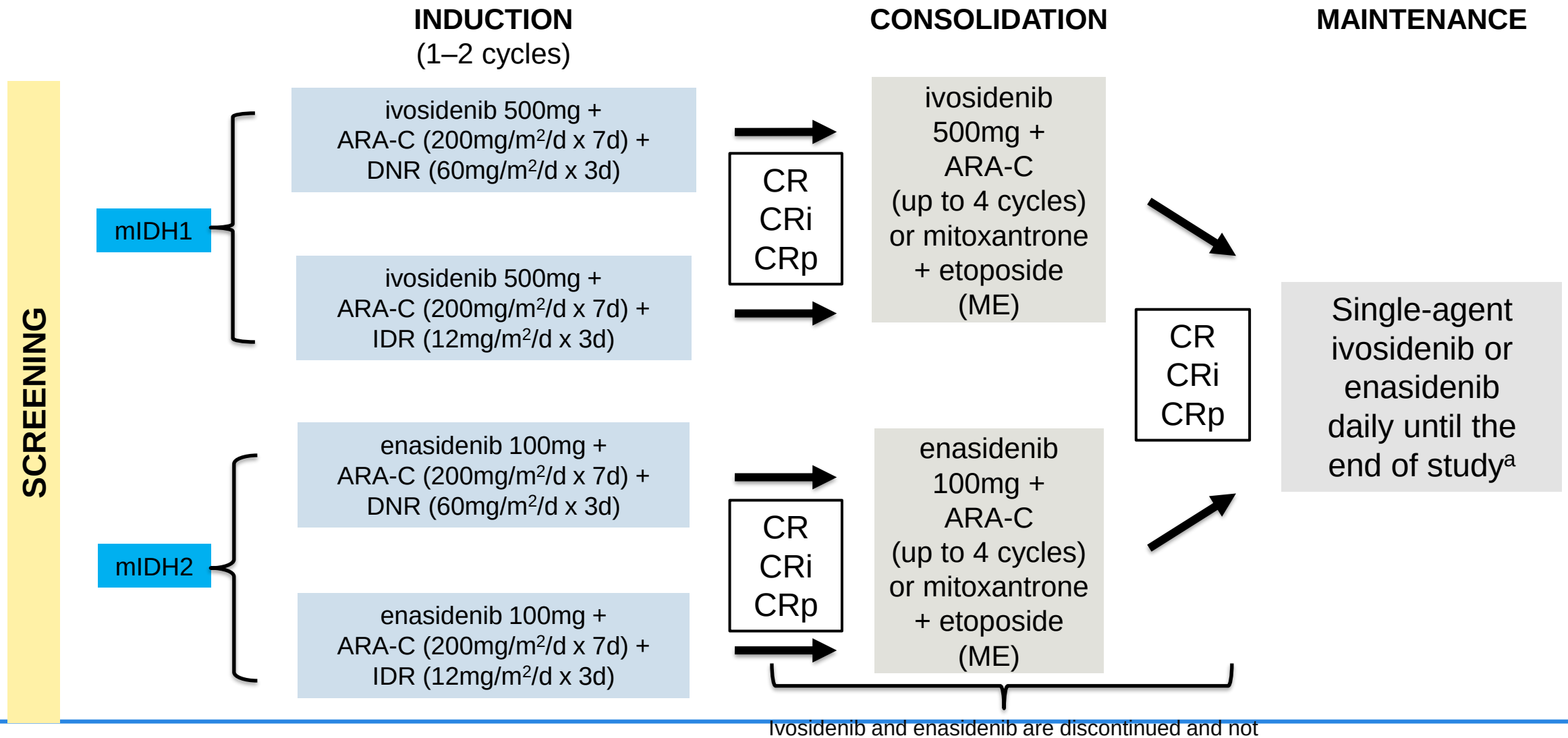
Stone R, et. al, NEJM 2017

Aza/Ven



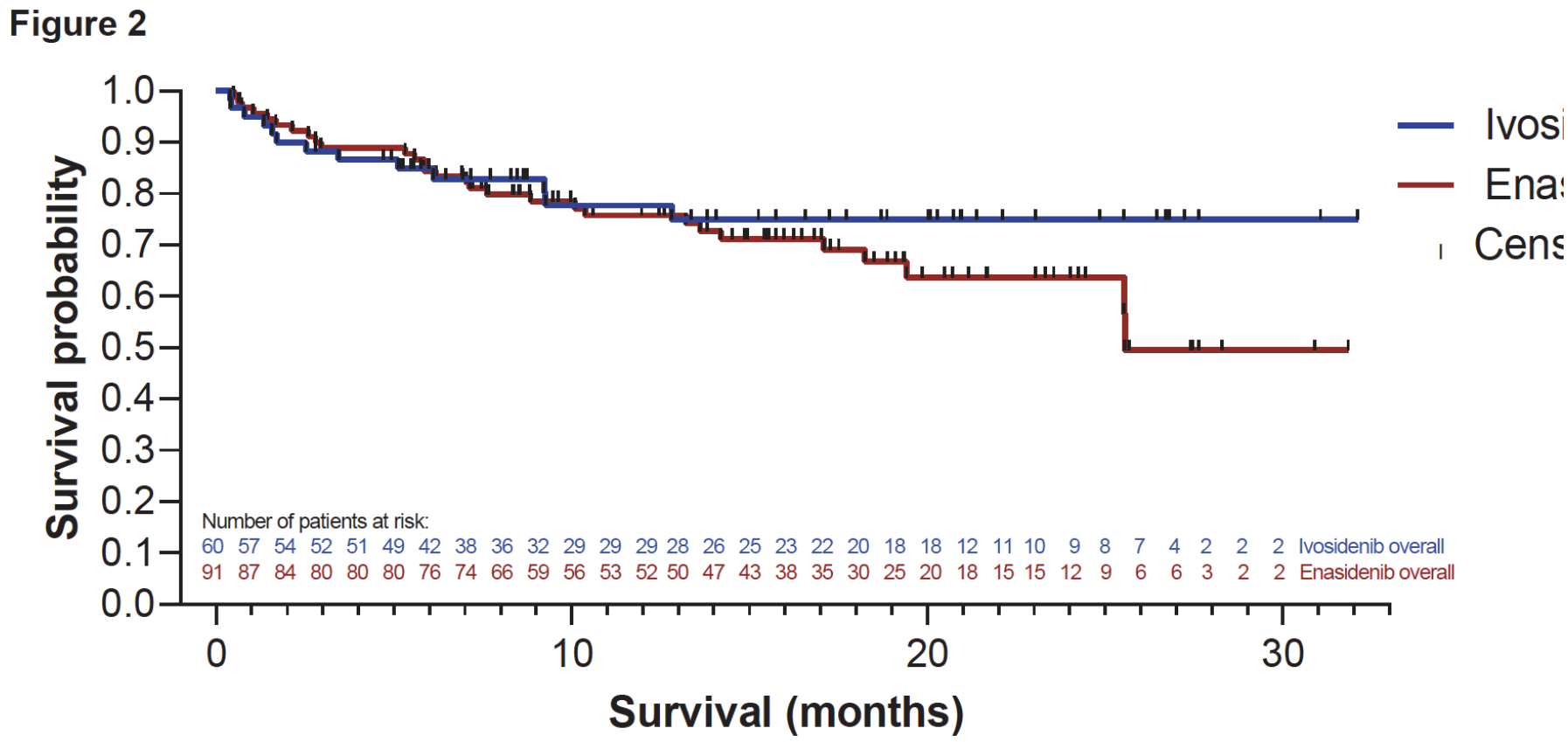
Dinardo C, NEJM 2020

Enasidenib/Ivosidenib with Induction Chemotherapy



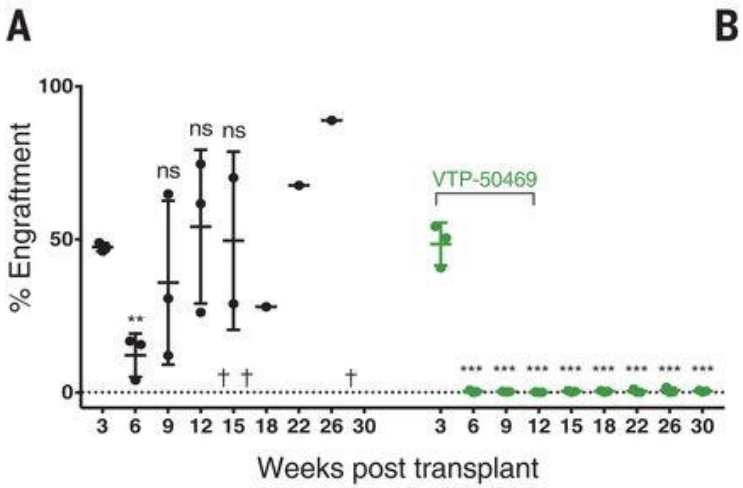
ARA-C = cytarabine; DNR = daunorubicin; IDR = idarubicin

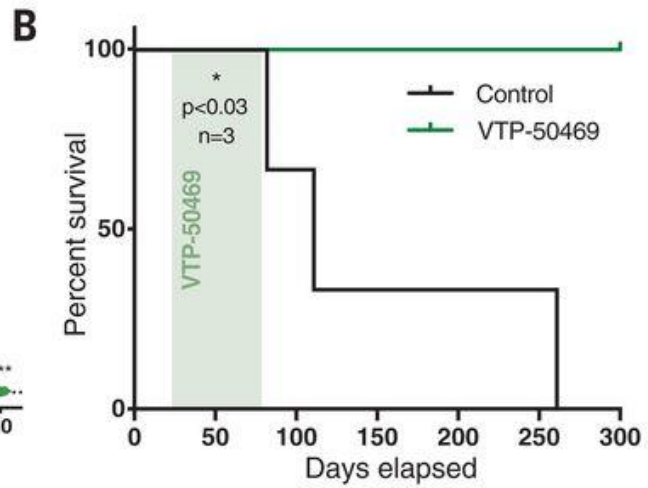
Enasidenib/Ivosidenib with Induction Chemotherapy

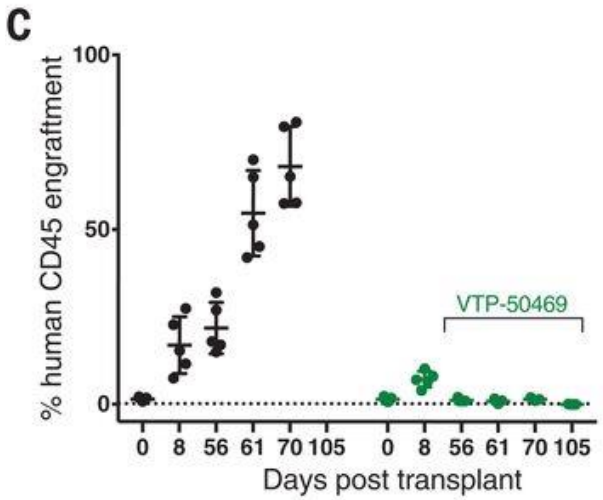


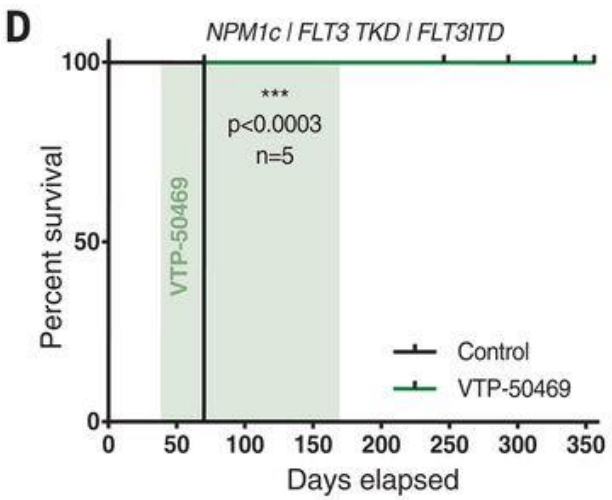
Stein E, et. al Blood 2020

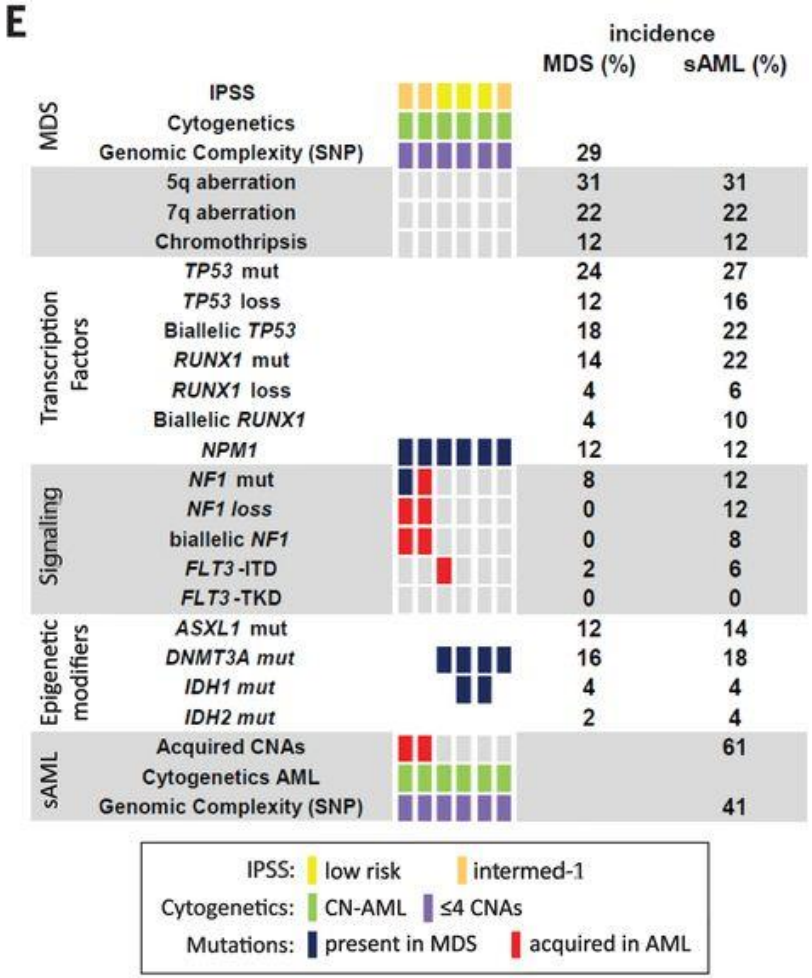
Novel Investigational Therapies are Effective











Hannah J. Uckelmann et al. Science 2020;367:586-590

Transcription Factor Modifiers in Action - Menin Inhibitors

Best Response at data cutoff	Response Evaluable n = 31 (%)
Overall Response Rate*	**15/31 (48%)**
CR/CRh	5
CRp	5
CRi/MLFS	5
MRD negative^ ORR	10/15 (67%)
MLLr overall response rate	13/24 (54%)
mNPM1 overall response rate	2/7 (29%)

4 MRD- patients went on to receive stem cell transplant

Conclusions

- Molecular studies are now part of the routine assessment of patients with newly diagnosed and relapsed acute myeloid leukemia
- Despite the approval of novel agents, the median and two year overall survival with these agents leaves many opportunities for the use of novel agents
  - Overall survival at two years with aza-ven is 40%
- Moving effective agents into combinations earlier in the course of therapy, are crucial for deriving the maximum benefit from novel agents
- Foghorn is exploring a novel approach to altering gene expression through dual inhibition of BRG1/BRM, and subsequent transcription factor modulation, such as SPI1 and others.

Thank You!

steine@mskcc.org

FHD-609: Clinical Entry Point – Synovial Sarcoma

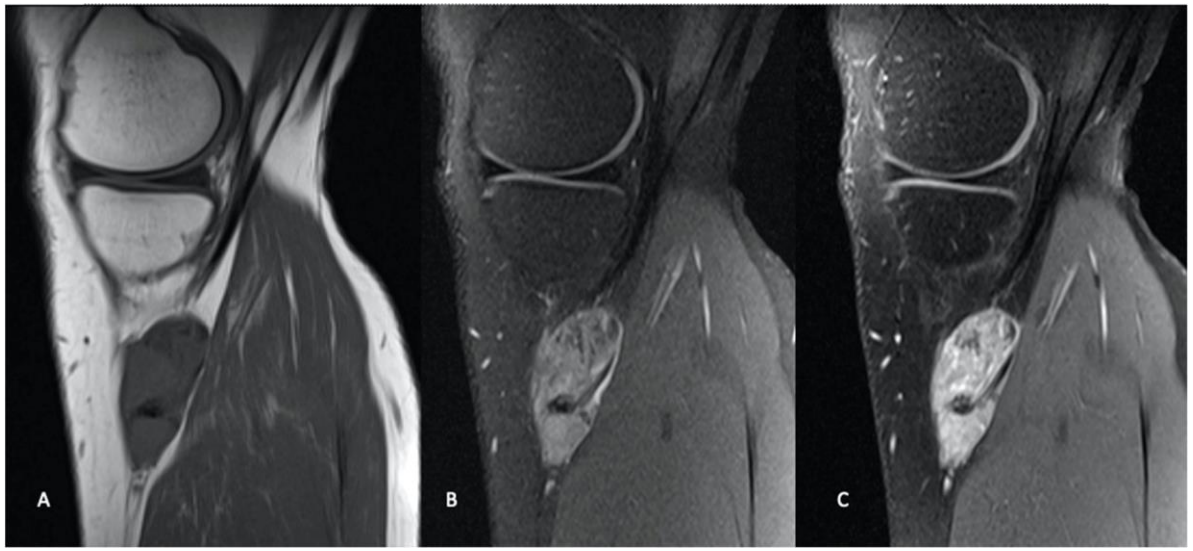
Sam Agresta, MP, MPH & TM

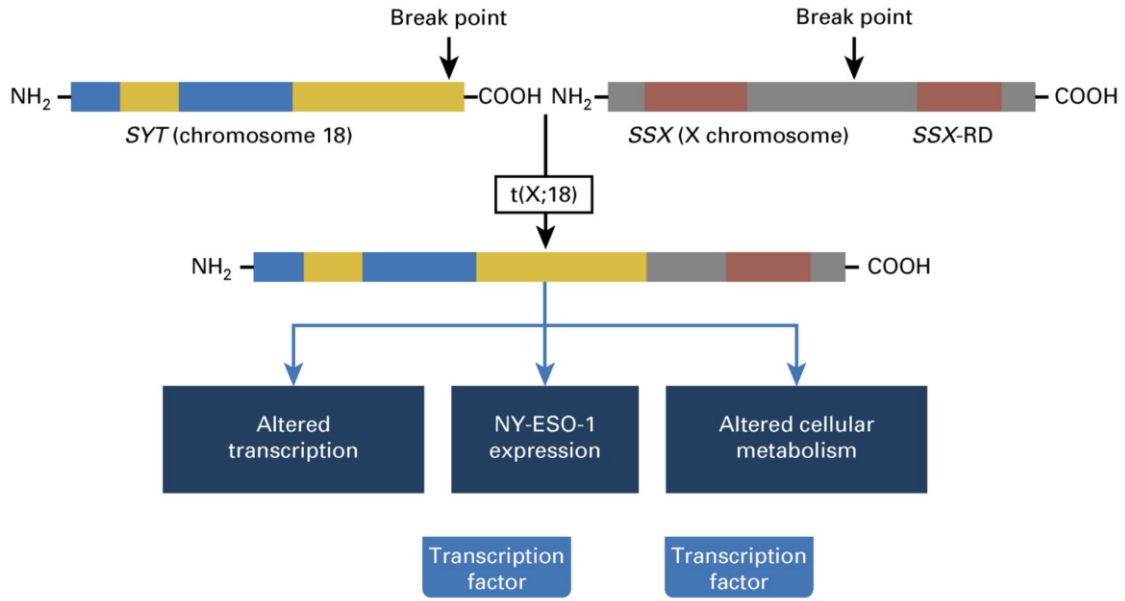
Chief Medical Officer, Foghorn Therapeutics

Synovial Sarcoma

Disease Overview

- 5-10% of all soft tissue sarcomas
- Most common presentation: Soft tissue tumor of the extremities in young adults
- Characterized by the translocation t(X;18)(p11.2;q11.2)
- *SS18* gene product:
  - Encodes for a protein subunit of the mSWI/SNF (BAF) chromatin remodeling complex
  - *SS18-SSX* gene competes with the endogenous SS18 protein, forming an altered complex lacking the tumor suppressor BAF4





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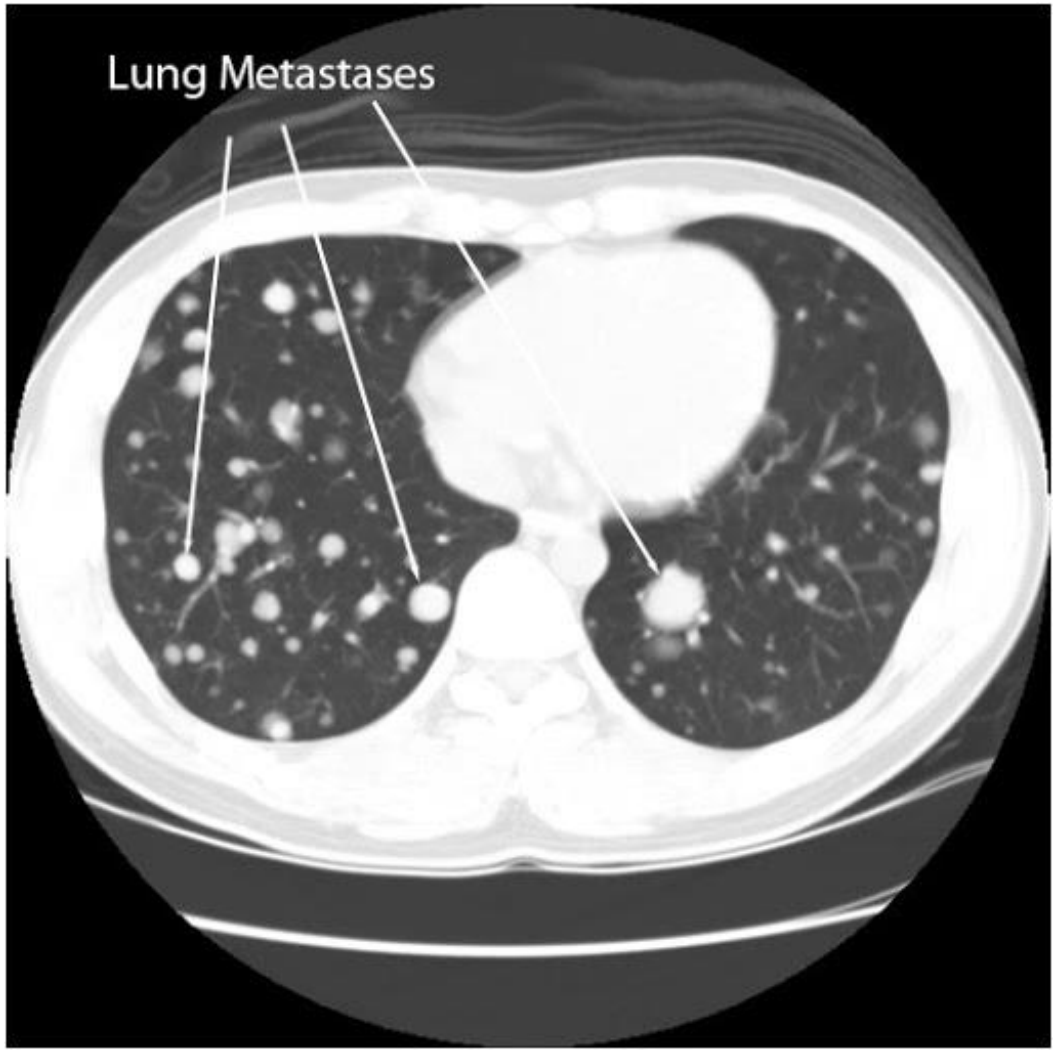
Metastatic Synovial Sarcoma

Disease Overview

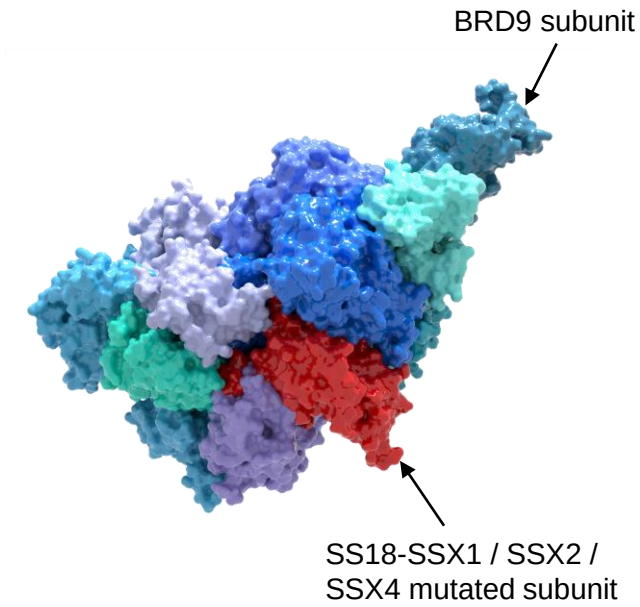
- Surgical resection of metastatic disease
  - Can provide long-term relapse-free survival in selected patients.
- Chemotherapy
  - Administered with palliative intent, with the goals of decreasing tumor bulk, diminishing symptoms, improving quality of life, and prolonging survival.
- Advanced unresectable
  - Candidates for clinical trials
- Prognosis:
  - Localized disease: 5-year PFS range from 26% - 80.7% and 5-year OS from 40% - 90.7%
  - Metastatic disease: 5-year OS is very low, 0-10%

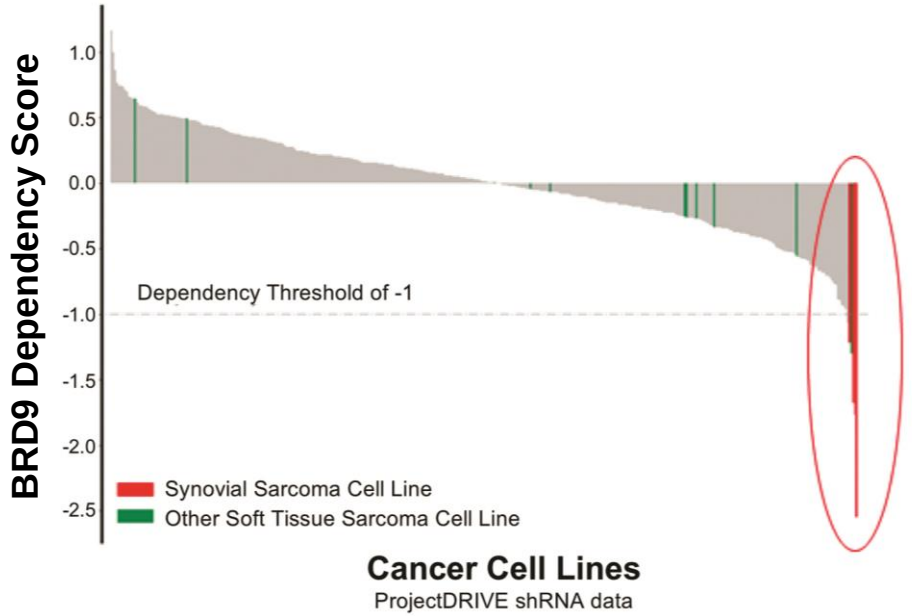


FHD-609 Targets and Degrades the BRD9 subunit of BAF which is Required for Synovial Sarcoma Cells to Survive

Selective, Potent BRD9 Targeted Protein Degrader

Target / Approach	• BRD9 • Intravenous Protein Degrader
Initial Indication	• Synovial Sarcoma
Mutation / Aberration	• SS18-SSX1 / SSX2 / SSX4 protein fusions
Program Status / Milestones	• Phase I data as early as H1’22
New Patients Impacted / Year*	• **Synovial Sarcoma: Over 1,800 patients / year**





BRD9 is required for the survival of synovial sarcoma cells

* US, EU5, Japan

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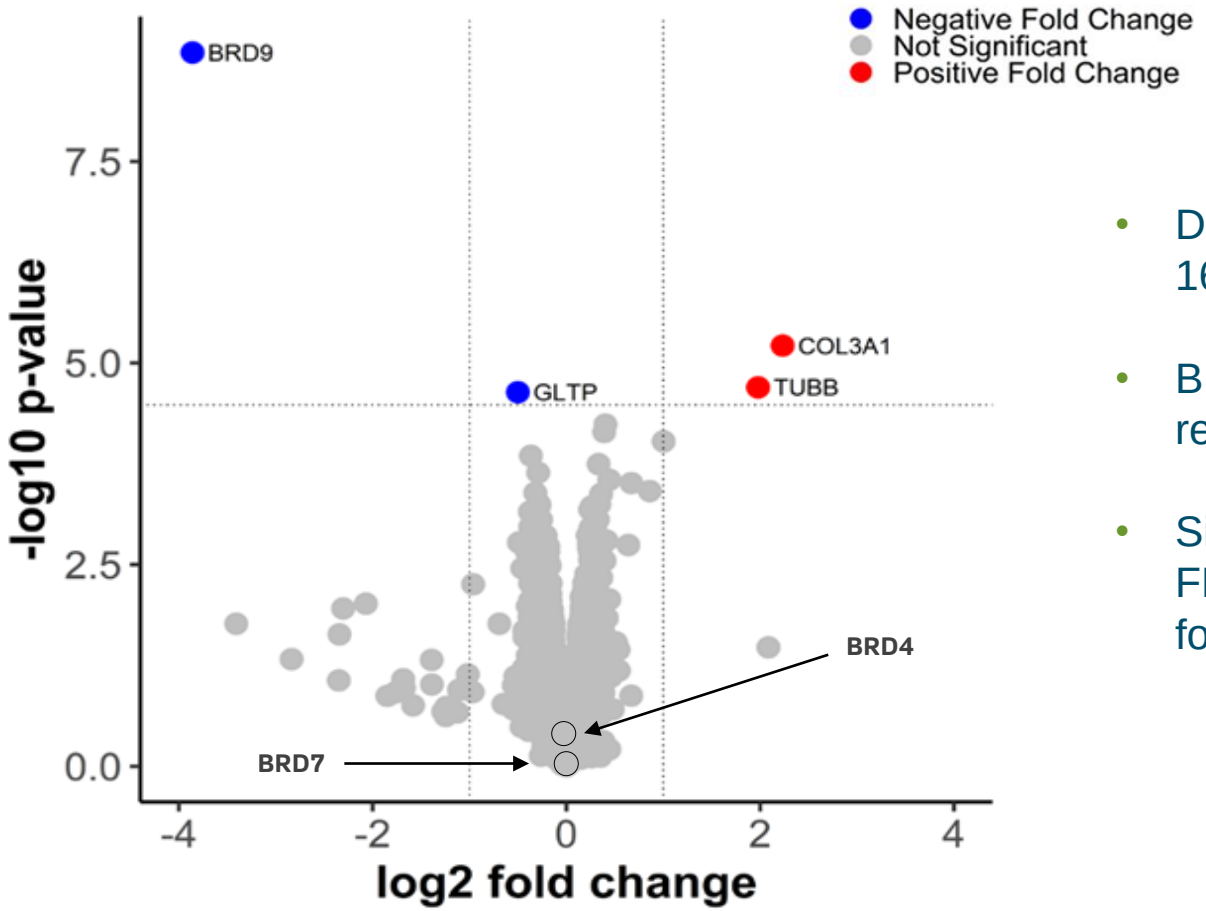
FHD-609 Selectively Degrades BRD9: Global Proteomic Assessment



- Data shown for SYO1 synovial sarcoma cells treated with 16nM of FHD-609 (~200x DC50) for 4h.
- BRD9 is the only protein significantly degraded, with 16-fold reduction, by quantitative MS analysis.
- Similar selectivity observed for 24h treatment of 16nM FHD-609, or higher concentration of 78nM (~1000x DC50) for 4h, data not shown

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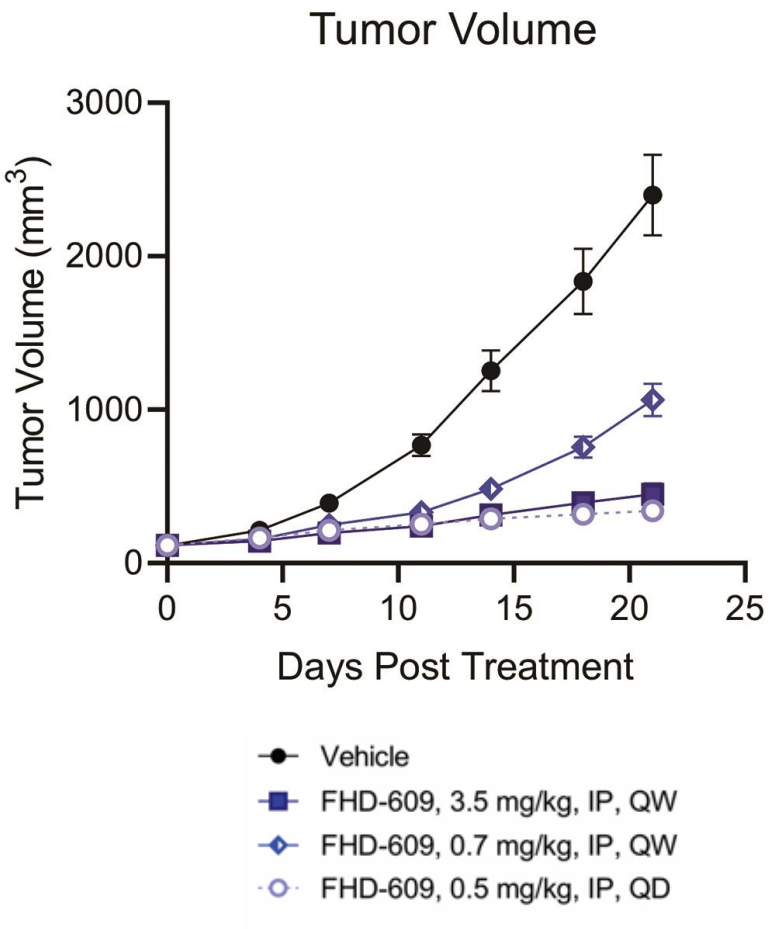
Robust *in vivo* Activity Observed in Synovial Sarcoma Model and BRD9 Degradation Associated with FHD-609 Treatment

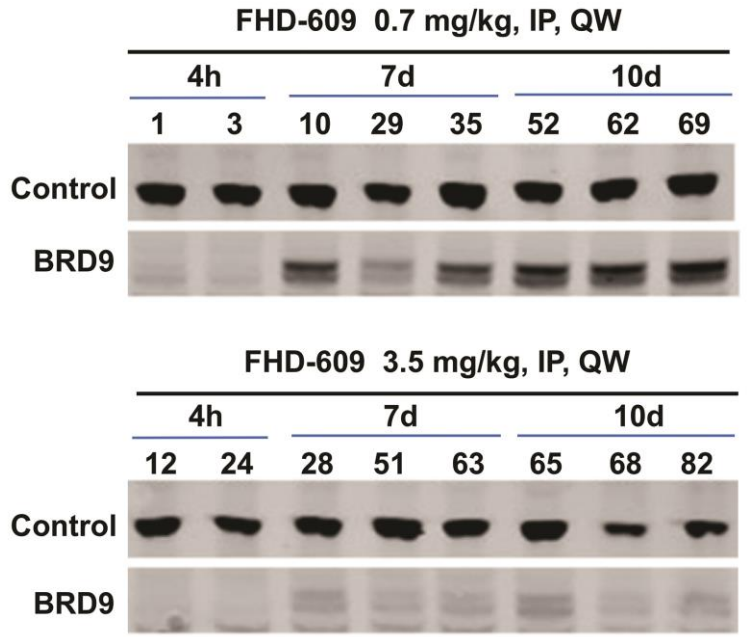
Weekly Dosing of FHD-609 Achieved Sustained BRD9 Degradation

SY01 Synovial Sarcoma CDX Model

- Mutation: **SS18-SSX2**
- Inhibited tumor growth
- Dose dependent BRD9 degradation correlated with anti-tumor activity



Sustained BRD9 Degradation



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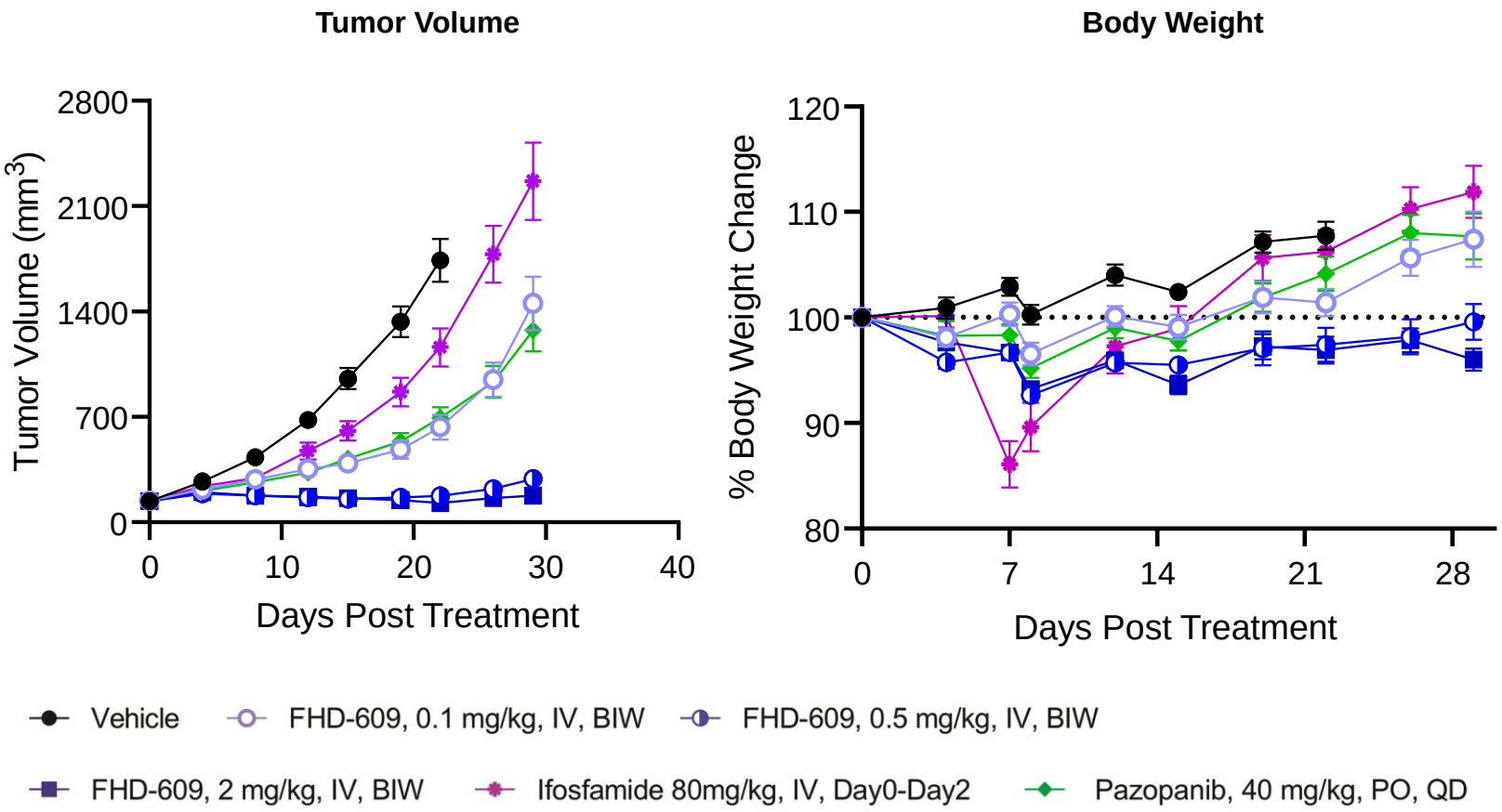
Superior Tumor Growth Inhibition of FHD-609 in a Synovial Sarcoma Model as Compared to Ifosfamide and Pazopanib

ASKA CDX Model

- Mutation: **SS18-SSX1**
- Superior tumor growth inhibition compared to ifosfamide and pazopanib
- Complete suppression observed over 30 days at 2 mg/kg of FHD-609



FOGHORN THERAPEUTICS

FHD-609 Clinical Development Plan

CLINICAL PLAN

Synovial Sarcoma FIH Phase 1

Metastatic Synovial Sarcoma

Trial Designs

- Single patient accelerated titration (n=1)
- Convert to 3+3 once relevant PK/PD, safety or clinical activity observed
- Assess safety, PK, clinical activity and biomarkers

Biomarkers:

- SS18-SSX1, SS18-SSX2 or SS18-SSX4 translocation

Synovial Sarcoma expansion cohorts

SMARCB-1 deleted tumors and potentially other indications

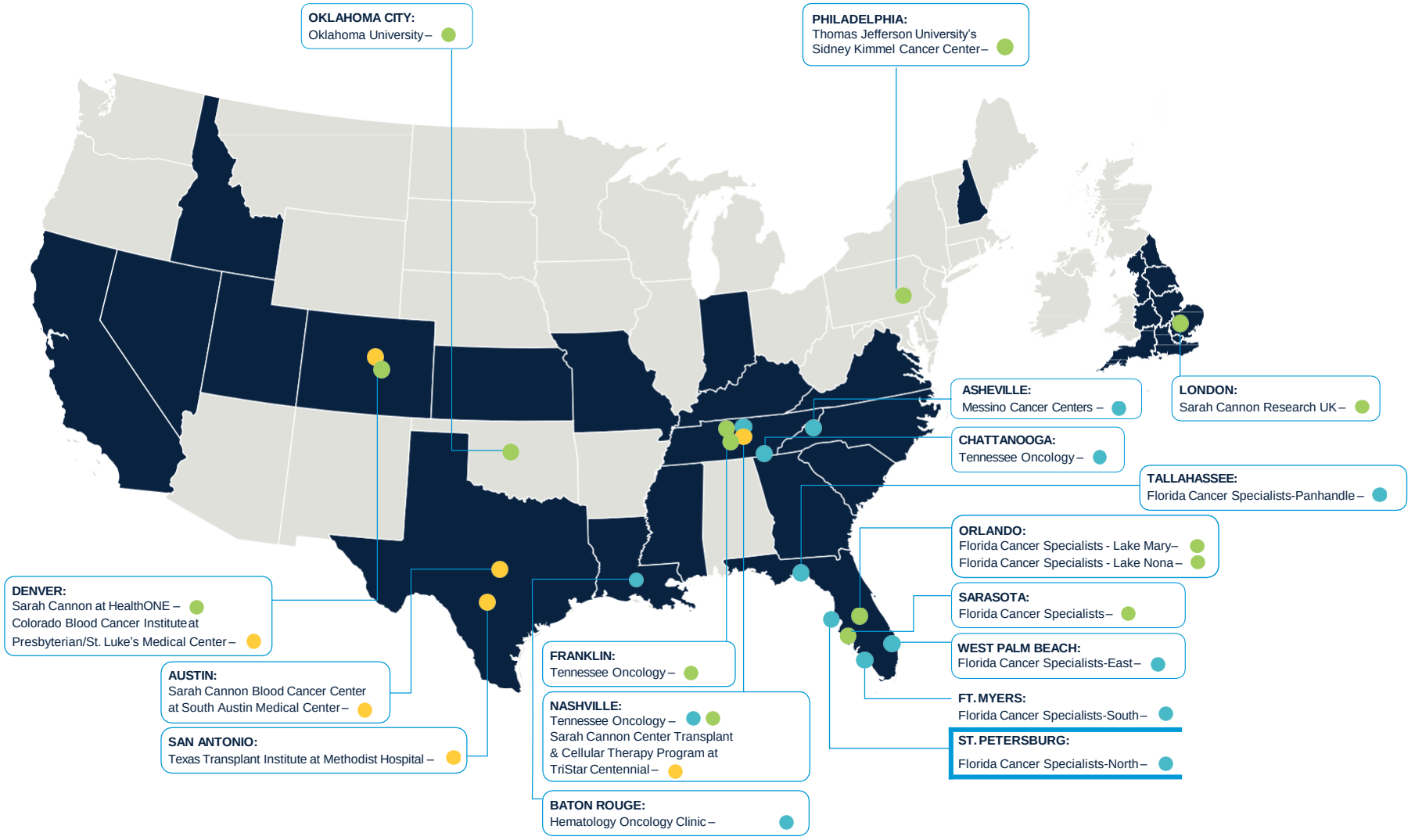
Potential for entry into definitive efficacy trials in synovial sarcoma

Clinical data as early as H1 2022

Howard “Skip” Burris III, MD, FASCO, FACP
Sarah Cannon Research Institute

SARAH CANNON

Sarah Cannon Research Sites



- States with HCA Healthcare Hospitals
- Strategic Sites
- DDU / Phase 1
- Sarah Cannon Transplant & Cellular Therapy Network

SARAH CANNON

Phase I Drug Development Program

50+
Phase 1 Studies in Presentations at ASCO 2020

500+
First-in-Human Trials Conducted

Clinical Trial Leader in the **Majority** of Approved Cancer Therapies over the Last 10 Years

200+
Unique Agents per Year

1,200+
Patients Treated on Phase 1 Trials in 2020

250+
Physicians who Engage in P1 Research

Tennessee Oncology
Nashville
Franklin

Florida Cancer Specialists
Sarasota
Lake Mary
Lake Nona

Oklahoma Univ. Medical Center
Oklahoma City

Sarah Cannon UK
London

Sarah Cannon HealthONE
Denver

SKCC-Jefferson Health
Philadelphia

SARAH CANNON

First Two Programs in the Clinic, Broad Pipeline Advancing

Precision Oncology / Breadth and Depth

Program / Target	Modality	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3	Global Rights
FHD-286 (BRG1 / BRM)	Enzyme inhibitor	AML		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
		Uveal melanoma		Early Clinical Data (Q4 2021)			FOGHORN THERAPEUTICS
FHD-609 (BRD9)	Protein degrader	Synovial sarcoma		Early Clinical Data (H1 2022)			FOGHORN THERAPEUTICS
Selective BRM	I) Enzyme inhibitor	BRG1 mutated cancers	IND 2022				FOGHORN THERAPEUTICS
	II) Protein degrader	BRG1 mutated cancers					
Selective ARID1B	Protein degrader	ARID1A mutated cancers					FOGHORN THERAPEUTICS
Synthetic Lethal Targets (multiple)	I) Enzyme inhibitors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Transcription Factors (multiple)	I) Transcription factor disruptors						FOGHORN THERAPEUTICS
	II) Protein degraders						
Partnered program (undisclosed)	Transcription factor disruptor						MERCK

Gene Traffic Control® Platform

Thank you

SARAH CANNON

FOGHORN® THERAPEUTICS

Concluding Remarks
Q&A

Adrian Gottschalk

Chief Executive Officer, Foghorn Therapeutics