Exhibit 99.1

FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

October 2024

Forward Looking Statements

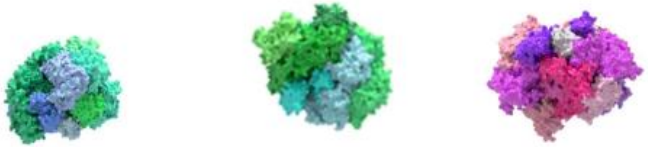
This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on information currently available to management. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “could,” “may,” “might,” “will,” “likely,” “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” 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 potential outcomes from our collaboration agreement with Lilly; the initiation, timing, progress and results of our research and development programs and pre-clinical studies and clinical trials, including with respect to our Phase 1 trial of FHD-286 in combination with decitabine in relapsed and/or refractory AML patients and anticipated timing of release of clinical data, and the planned Phase 1 dose escalation trial of FHD-909 with Lilly; our ability to advance product candidates that we may develop and to 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 exogeneous factors, including macroeconomic and geopolitical circumstances, on our and our collaborators’ business operations, including our research and development programs and pre-clinical 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 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, our future products and our Gene Traffic Control Platform; and our use of proceeds from capital-raising transactions, estimates of our expenses, capital requirements, and needs for additional financing. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation. Additional important factors to be considered in connection with forward-looking statements are described in the Company's filings with the Securities and Exchange Commission, including withing the section entitled "Risk Factors" in the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 2023. Any forward-looking statements represent the Company’s views only as of the date of this presentation 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.

Foghorn is the Pioneer in Chromatin Biology, an Untapped Area for Therapeutics

What if … It were possible to develop a therapeutic approach to treat half of all cancers?

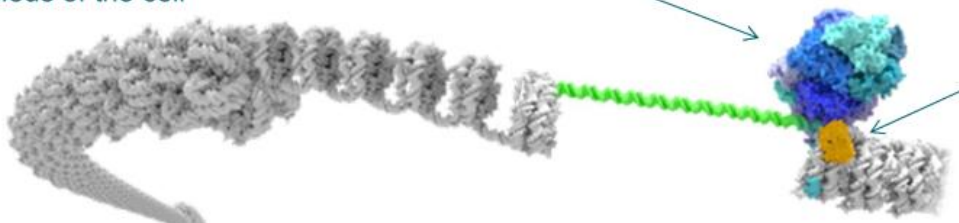
Chromatin biology is implicated in up to 50% of tumors	~2.5 million cancer patients	Potential for therapeutic area expansion (e.g., I&I)

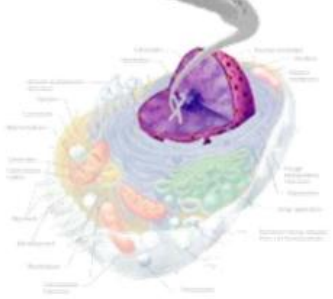
Chromatin Regulatory System Orchestrates Gene Expression; Multiple Opportunities for Targets and Therapeutics



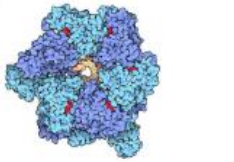
Chromatin Remodeling Complex – specialized multiprotein machineries that allow access to DNA

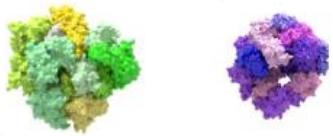
Chromatin – compacted form of DNA inside the nucleus of the cell





Helicases & Other Chromatin Binding Proteins involved in gene expression / function





Gene expression in the right cells at the right time is critical for normal growth, development, and homeostasis

Transcription Factor – proteins that help turn specific genes "on" or "off" by working in concert with the chromatin remodeling complex to bind to DNA

Foghorn has Progressed Multiple Programs Against Challenging Targets

SMARCA2 / SMARCA4: Implicated across solid and hematologic malignancies
Challenge: Can dual inhibition yield clinical benefit?

FHD-286
dual inhibitor in the clinic
Data H2 '24

SMARCA2: Potential in up to 5% of all solid tumors
Challenge: Industry has failed to develop a selective inhibitor

FHD-909
first selective inhibitor in the clinic

CBP: Role in bladder, colorectal, breast, gastric, lung cancers
Challenge: Toxicities with dual inhibition, difficulty engineering selectivity

Selective CBP Degrader
IND enabling studies anticipated by end of year

EP300: Role in both solid and heme malignancies
Challenge: Toxicities with dual inhibition, difficulty engineering selectivity

Selective EP300 Degrader
IND enabling studies anticipated in 2025

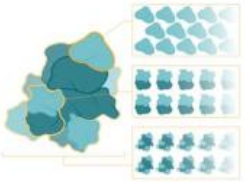
ARID1B: Role in ovarian, endometrial, colorectal cancer
Challenge: Industry has had no success with selective target engagement

Selective ARID1B binder identified. Critical step towards degradation

SMARCA2 = BRM
SMARCA4 = BRG1

… and more.

Foghorn's Gene Traffic Control® Platform Designed to Deliver Precision, First-in-Class Therapeutics: Integrated, Scalable, Efficient, Repeatable

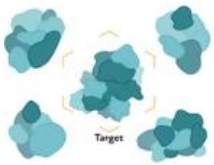


2. Assays & Biochemistry Capabilities

Engineering selectivity via unique assays and protein capabilities

- Protein purification, production & interrogation
- High fidelity, difficult to make proteins
- In silico modeling and computational chemistry

"Where to Drug"



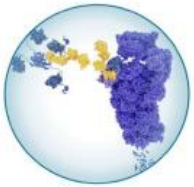
1. Chromatin Biology

Deep mechanistic understanding of chromatin regulatory system

- Bioinformatics
- Genomics
- Epigenomics

Identify Dependencies

"What to Drug"



3. Chemistry & Drugging

Biology first, small molecule modality agnostic

- Selective, small molecules (inhibitors, protein degraders, TF disruptors)
- Protein degradation platform
- Formulation & long-acting delivery

"How to Drug"

Legend: Patents | Know How / Trade Secret

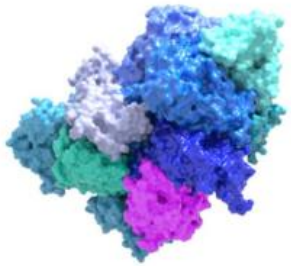
Foghorn's Unique Platform Capabilities Evolved from Drugging a Specific Chromatin Remodeling Complex (BAF)*

Challenge: produce, manipulate, study, and drug a 1.5 megadalton multi-protein complex

Assays and Biochemistry Capabilities

- Purification & recombinant production of large proteins and protein complexes
- Biochemistry & biophysics of intrinsically disordered proteins
- High throughput screening for binders and inhibitors

BAF Chromatin Remodeling Complex



Challenge: drug highly similar proteins that have no enzymatic function

Protein Degrader Platform

- Proprietary linker library
- Suite of assays specific to degradation (i.e., synthesis kinetics, degradation kinetics)
- Optimal E3 ligase pairing
- Ternary complex modeling
- Long-acting formulation technology

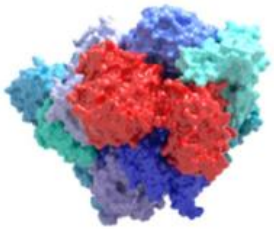
Current and Future Applications

- **Selectively drugging highly similar proteins / hard to drug proteins**
- **Disease area expansion**
- **Going beyond chromatin – novel biology with complex proteins**
- **Payloads for ADCs***

*Brahma-Associated Factor (BAF). Antibody Drug Conjugates (ADCs).

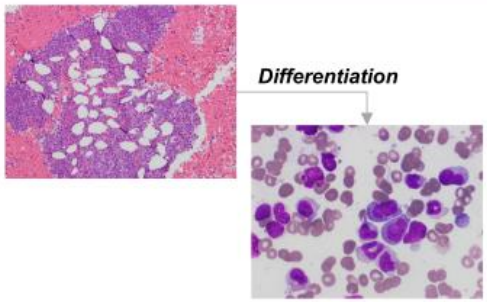
The Next Foghorn Chapter: Delivering Multiple Potential Blockbusters into the Clinic

Pioneering BAF and Chromatin Biology
(2016 – 2020)



- ✓ Built platform and developed deep understanding of biology
- ✓ Producing BAF and transcription factors at scale
- ✓ **Demonstrated druggability of chromatin regulatory system**

POC, Platform & Pipeline Expansion
(2021 – 2023)



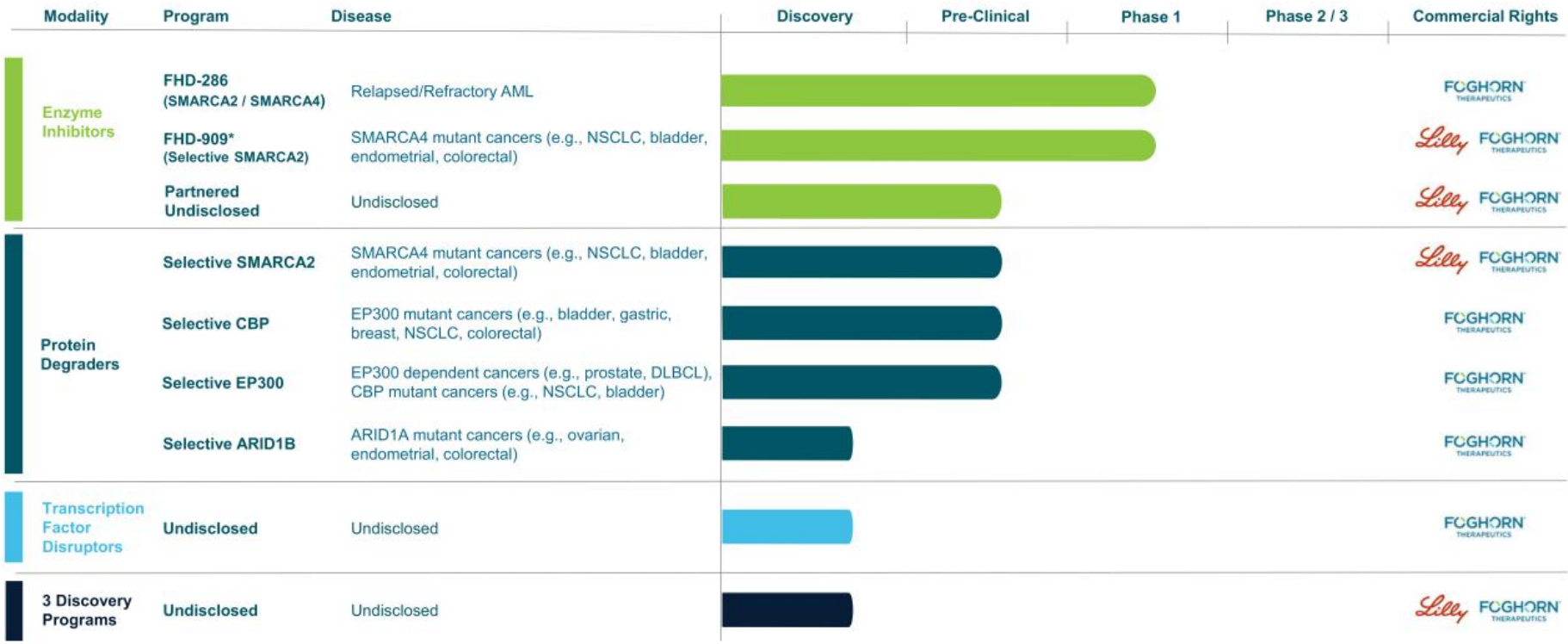
- ✓ **Lilly strategic collaboration**
- ✓ FHD-286 demonstrated mutation-agnostic differentiation effect in acute myeloid leukemia (AML)
- ✓ Initiated efforts on CBP and EP300
- ✓ Expansion of protein degrader platform

Progress Multiple High Value Assets into the Clinic
(2024 – 2027)

- **Proof of concept data for SMARCA2 Selective Inhibition (FHD-909) in NSCLC***
- **Registrational trials for FHD-286 in AML**
- **Potential for 5 additional INDs**
- **Pipeline, platform, disease area expansion**

*Non-small cell lung cancer (NSCLC)

Foghorn Is Advancing a Pipeline of First-in-Class Precision Therapeutics with Potential for Broad Application in Oncology …



*LY4050784
SMARCA2 = BRM
SMARCA4 = BRG1

… with Multiple Near-Term Value Inflection Points through 2026

FHD-286	AML Combination Dose Escalation Data	Q4 2024
FHD-909 (Selective SMARCA2 Inhibitor)	Phase 1: First Patient Dosed	October 2024
	Phase 1 Dose Escalation Data	Confidential
Selective SMARCA2 Degrader	IND Filing / Phase 1 Initiation	Confidential
Selective CBP Degrader	Initiate IND-Enabling Studies	Year End 2024
Lilly Target #2	Target Disclosure and IND Filing	Confidential
Selective EP300 Degrader	Initiate IND-Enabling Studies	2025
Selective ARID1B Degrader	Development Candidate	H1 2026

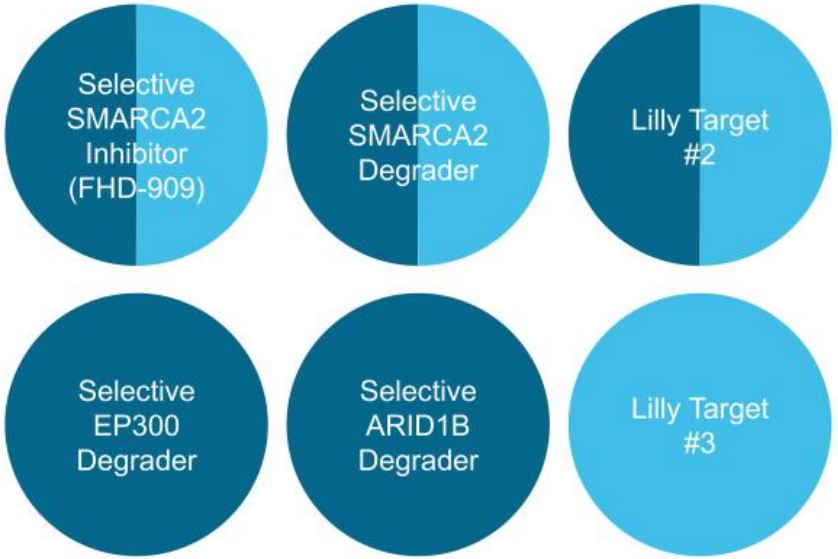
SMARCA2 = BRM
SMARCA4 = BRG1

Potential Multi-Billion Dollar Opportunities in Oncology

$500M to $2B Market Opportunities Each

- FHD-286 (R/R AML, Other)
- Selective CBP Degrader

Greater than $2B Market Opportunities Each



Foghorn Owned

Partnered w Lilly

Potential for therapeutic area expansion (e.g., immunology and inflammation)

FOGHORN THERAPEUTICS

Clinical & Pre-Clinical Programs

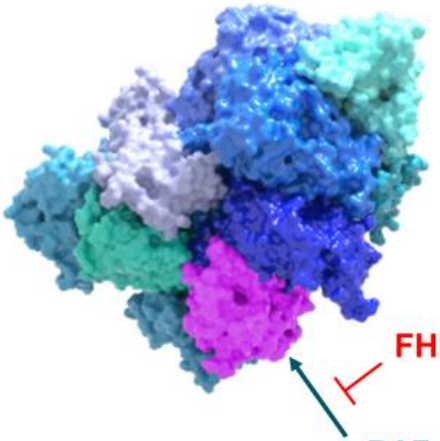
- FHD-286 – Dual SMARCA2 / SMARCA4 Inhibitor
- FHD-909 (LY4050784) – Selective SMARCA2 Inhibitor
- Selective CBP Degrader
- Selective EP300 Degrader
- Selective ARID1B Program

FHD-286: Dual SMARCA2 / SMARCA4 Inhibition

Targeting BAF Dependency in Cancer

SMARCA2 = BRM
SMARCA4 = BRG1

Exploring BAF Dependency in Cancer with FHD-286 – Potent, First-in-Class, Oral Dual Inhibitor of SMARCA2 and SMARCA4



FHD-286:

- **Allosteric modulation inhibiting the activity of both SMARCA2 and SMARCA4**
- **Oral, daily, potent, first-in-class, small molecule inhibitor**

Current and Potential Future Opportunity

Mutations	Pre-clinical data support ability to address BAF mutations
Differentiation	Clinical and pre-clinical data demonstrated broad-based differentiation across AML and multiple solid tumors
Overcoming Drug Resistance	Pre-clinical data support ability to overcome drug resistance (i.e., EGFR NSCLC, enzalutamide-resistant CRPC, PD-1 refractory)
Immune Modulation	Clinical data demonstrated an increase of CD8+ T-cells and a reduction of T-regulatory cells

Significant Opportunity for Novel, Effective Therapy in Patients with R/R AML

Most cases of AML are not curable

- >50% of patients relapse
- Intensive chemo – still standard of care

40% of AML cases have no actionable mutations

- No meaningful developments for broad AML patient population since Venetoclax
- Recent developments focused on actionable mutations (e.g., FLT3, IDH1/2, MLL**)

Initial FHD-286 Opportunity

~17,000 Drug Treatable R/R Patients*

- **Post Ven/Aza:**
  - **No standard of care**
  - **CRc rates 15-17%**
  - **Median OS ~3mo**
- **High unmet need**

FHD-286 Opportunity: R/R Patients and Potentially Newly Diagnosed Patients

*Source: Decision Resources Group 2025 Forecast: **Menin inhibitors not yet approved; R/R: relapsed/refractory; CRc: composite complete response

FHD-286 Demonstrated Promising Mutation-Agnostic Differentiation Effects in Single Agent Phase 1 Trial

Dose Level	Mutations	Cytogenetics Risk	Starting CD11b%	Max CD11b%	CD11b+ Fold Change	Starting CD34%	Min CD34%	CD34+ % Decrease
10mg	N/A	Adverse	7	62	9.2x	94	27	(71%)
7.5mg	CBFB (locus at 16q22)		2	94	59.4x	70	2	(97%)
7.5mg	KMT2A rearrangement	Adverse	3	58	21.4x	85	9	(90%)
7.5mg	RUNX1, KRAS, ASXL1, JAK2, TET2, EZH2, ETNK	Adverse	5	73	15x	95	18	(81%)
7.5mg	N/A	Adverse	8	52	6.3x	94	33	(65%)
7.5mg	ASXL1, TP53, U2AF1	Adverse	19	63	3.3x	92	51	(45%)
5mg	RUNX1, NRAS, KRAS, SF3B1, ASXL2, CSF3R, GATA2	Adverse	3	74	29x	94	19	(80%)
5mg	RUNX1, NRAS, ASLX1	Adverse	4	97	22.8x	98	7	(93%)
5mg	N/A	Adverse	6	79	13x	93	11	(88%)
5mg	TET2, WT1 GATA2 PLCG2 ARHGEF28, BRD4, CDK12, DDX41, KMT20, PARP1, ZRSR2		3	24	8.1x	86	62	(27%)
5mg	N/A	Adverse	4	28	6.5x	93	66	(29%)
5mg	DNMT3a, TET2		21	88	4.1x	30	4	(88%)
2.5mg	NRAS, WT1	Adverse	3	13	4.8x	93	89	(4%)

CD11b (marker of differentiation) increases

CD34 (leukemic stem cell marker) decreases

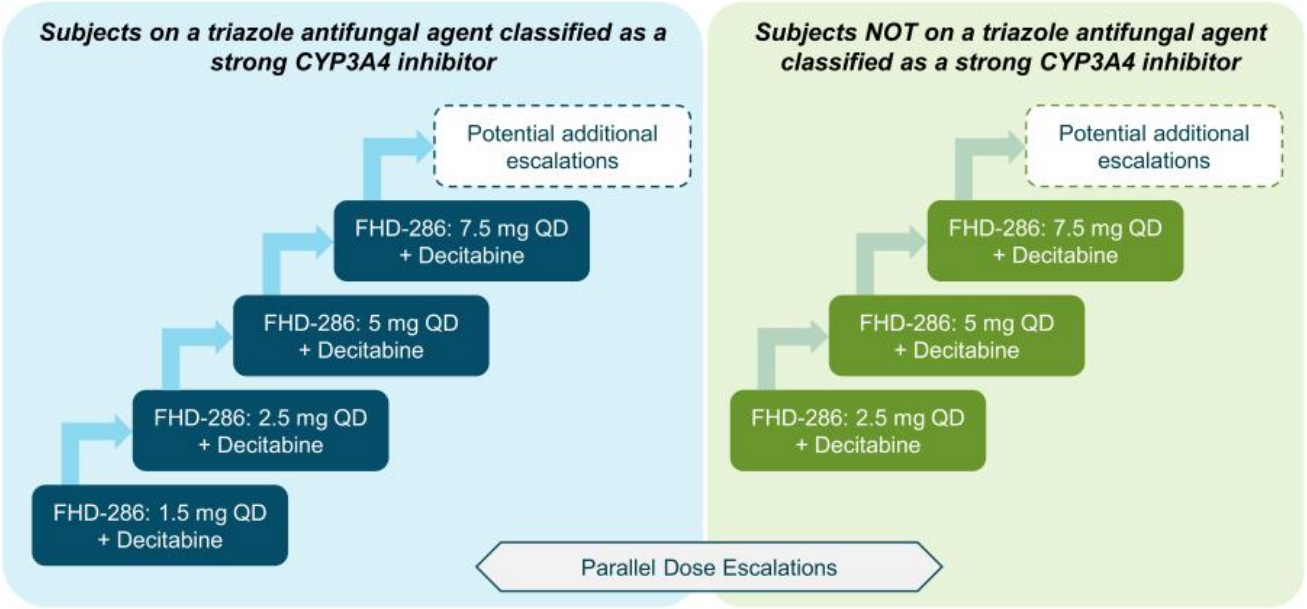
Dose Escalation Trial Design in Combination with Decitabine in AML

Target Indication:

- R/R AML

Treatment Plan & Dose Escalation:

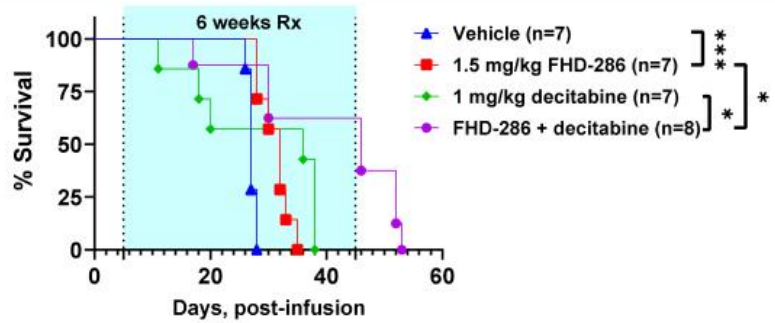
- 3+3 escalation design
- Oral FHD-286, QD, 28-day cycles
- Standard decitabine dose schedule



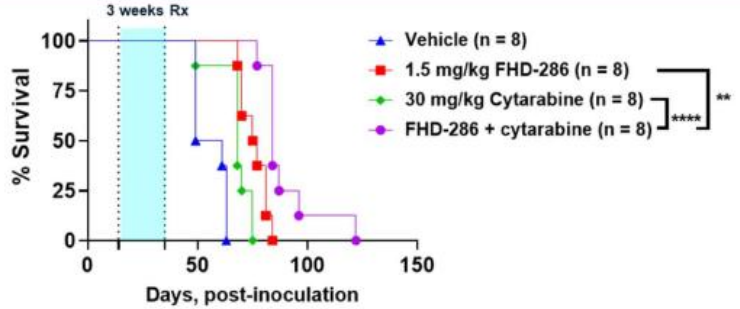
Key Objectives	
Primary	• Safety/Tolerability • Maximum Tolerated Dose (MTD) and/or Recommended Phase 2 Dose (RP2D) determinations
Secondary	• Preliminary clinical activity • PK parameters of FHD-286 in combination with Decitabine in subjects on/off triazole antifungal agents classified as strong CYP3A4 inhibitors
Exploratory	• PD effects of FHD-286 in combination with Decitabine • MRD

Pre-Clinical Data Demonstrated Combination Potential with Multiple Agents in AML

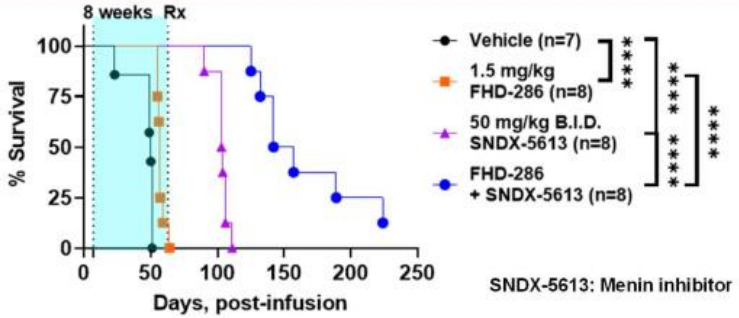
FHD-286 + decitabine
(MLL-AFP + FLT3 TKD Luc/GFP PDX)



FHD-286 + cytarabine
(MV4, 11 FLT3 ITD CDX)

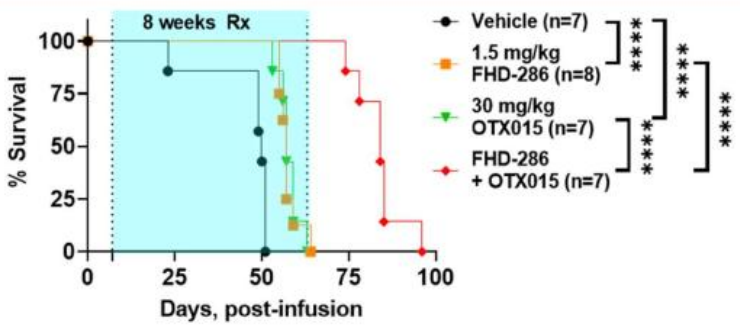


FHD-286 + menin inhibitor
(mtNPM1 + FLT3 ITD Luc/GFP AML PDX)



SNDX-5613: Menin inhibitor

FHD-286 + BET inhibitor
(mtNPM1 + FLT3 ITD Luc/GFP AML PDX)



FHD-286 Has Potential in Multiple High-Value Oncology Indications

R/R AML combinations **(e.g., decitabine, menin inhibitors, others)**	**Front-Line AML Combination**
TKI Combination	**Other Hematologic and Solid Tumors**

Selective SMARCA2 Modulators

For SMARCA4 Mutated Cancers

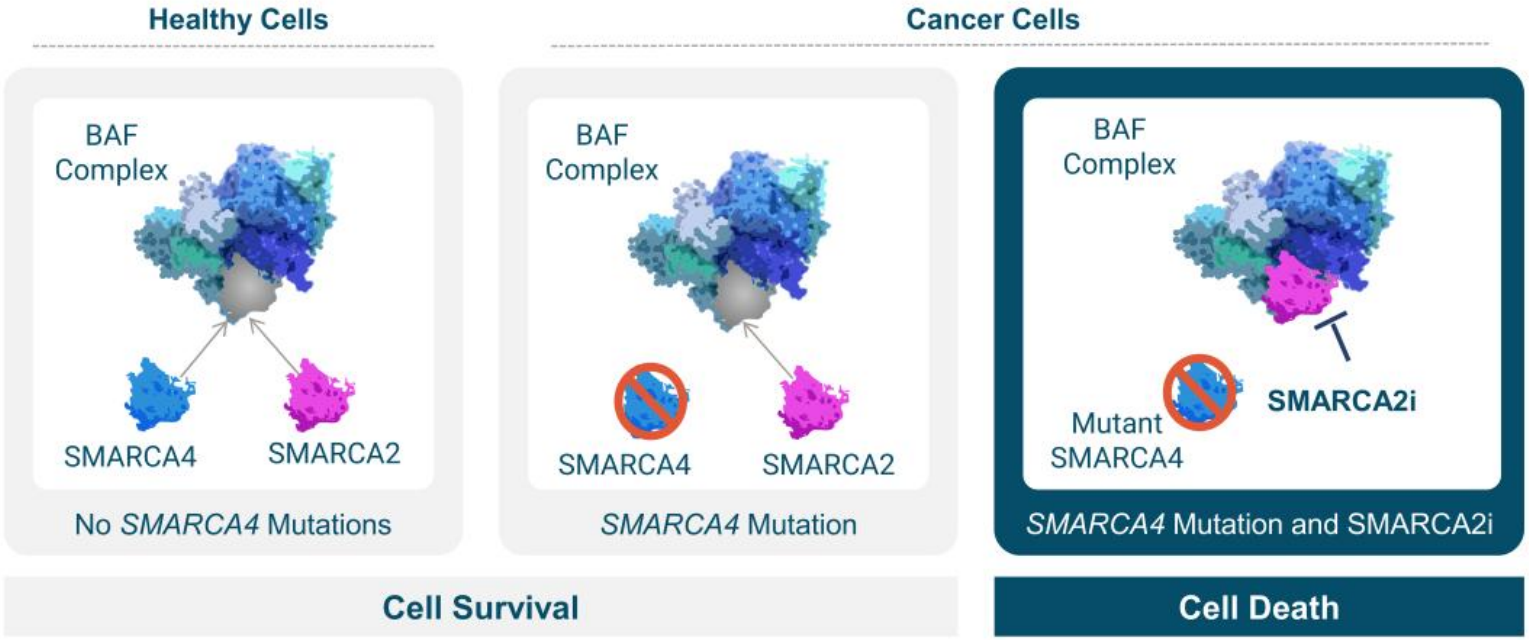
SMARCA2 = BRM
SMARCA4 = BRG1

FHD-909, SMARCA2 Selective Inhibitor in Phase 1 Trial; Selective SMARCA2 Degrader Continues Late-Stage Pre-Clinical Development

	SMARCA2 Selective Inhibitor (FHD-909*)	SMARCA2 Selective Degrader
Biology	Exploit the synthetic lethal relationship between SMARCA2 and mutated SMARCA4	
Stage	Phase 1 dose escalation trial	Advancing in parallel through late pre-clinical development
Opportunity	SMARCA4 mutated cancer including ~10% of NSCLC and up to 5% of all solid tumors	
Lilly Partnership	50/50 global R&D cost share | 50/50 U.S. economics | tiered ex-U.S. royalties starting in the low double-digit range and escalating into the twenties	

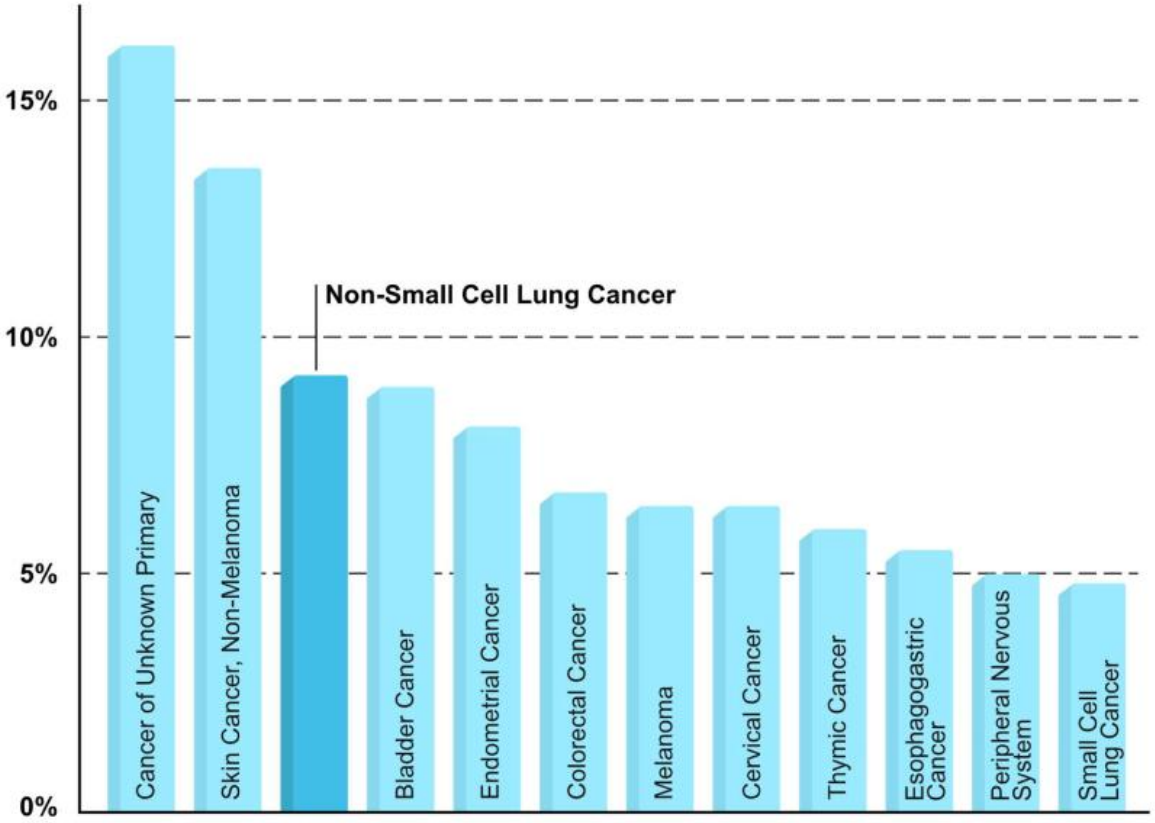
*LY4050784

Selective SMARCA2 Inhibition: Promising Strategy to Exploit Synthetic Lethal Relationship Between SMARCA2 and Mutant SMARCA4



Precision medicine targeting synthetic lethal relationships is a proven clinical approach now used in multiple cancers (e.g., PARP inhibitors)

SMARCA4 is Mutated in Up to 10% of NSCLC; Up to 5% of Solid Tumors



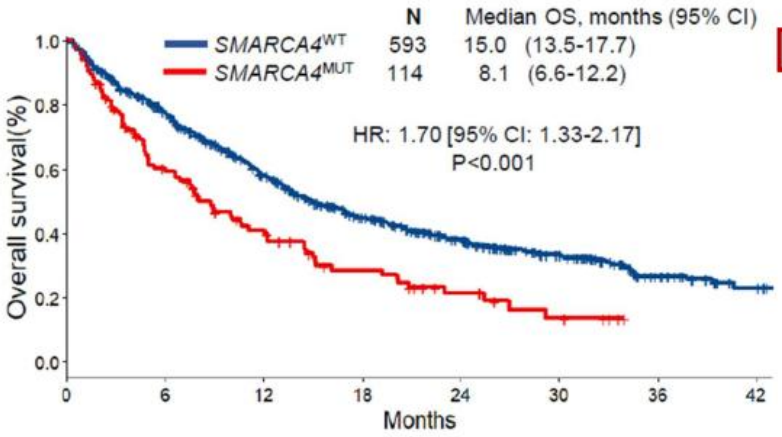
SMARCA4 mutated across a broad range of tumors

Accounts for ~5% of solid tumors

AACR GENIE via cBioPortal

Patients with NSCLC Harboring SMARCA4 Mutations Have Significantly Worse Clinical Outcomes and Define a High Unmet Need Patient Population

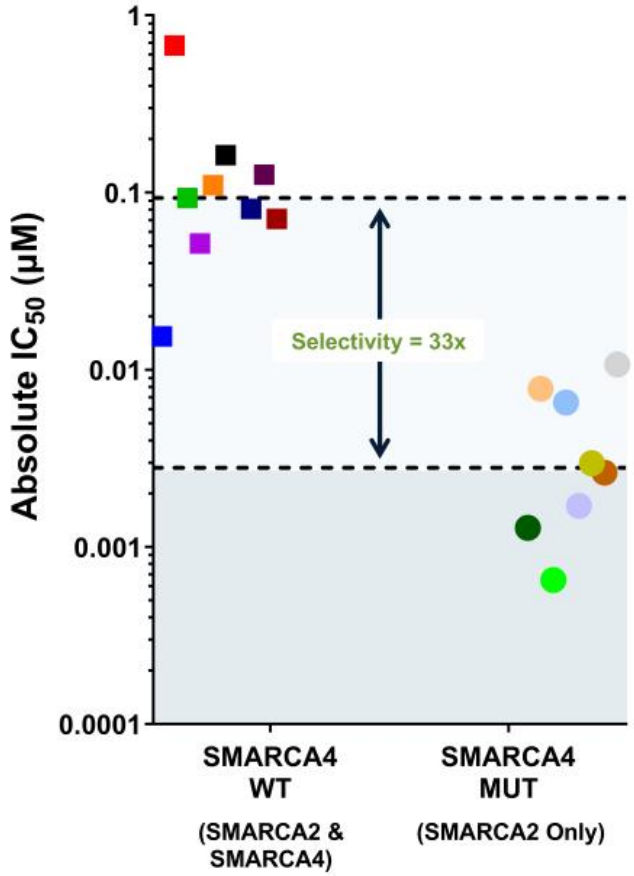
Overall Survival for SMARCA4wt vs SMARCA4mut[1]



SMARCA4 mutated in up to 10% of NSCLC tumors, minimal overlap with other mutations[2]

1. Alessi JV, et al., 2021; 2. TCGA via cBioPortal

FHD-909 Demonstrated Approximately 33-fold Selectivity Across 17 SMARCA4 Mutant and Wild-Type Cell Lines *In Vivo*



SMARCA4 (WT)	SMARCA4 (MUT)
NCI-H2122	NCI-H1944
CALU6	NCI-H1299
NCI-H2172	A549
NCI-H441	RERFLCAI
NCI-H2170	NCI-H1693
NCI-H460	NCI-H1568
NCI-H1703	NCI-H2023
NCI-H358	NCI-H838
NCI-H2009	

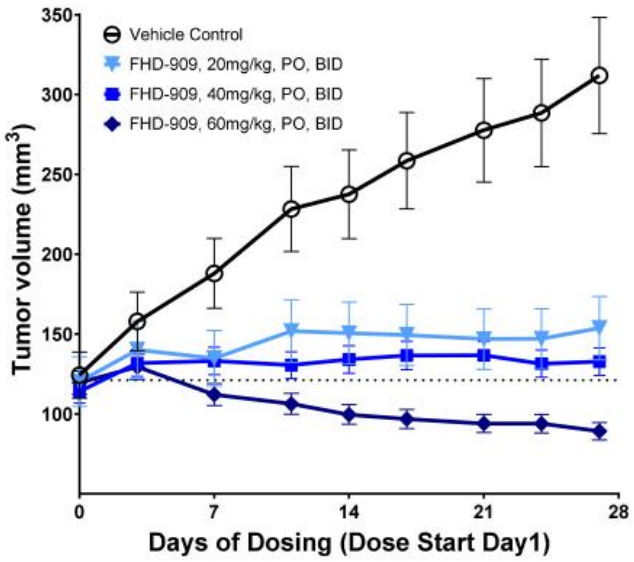
	Median IC_{50} (µM)
SMARCA4 WT	0.0932
SMARCA4 MUT	0.0028

Spread in potency for wild type versus mutant cell lines indicates

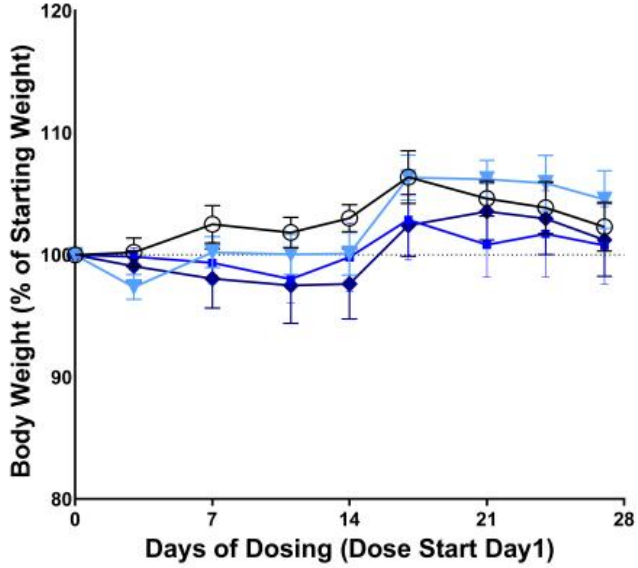
33-fold selectivity observed

FHD-909 Monotherapy Demonstrated Regression *In Vivo* in H2126 SMARCA4 Mutant NSCLC Model and Was Well Tolerated

H2126 Reduction in Tumor Volume



H2126 Body Weight

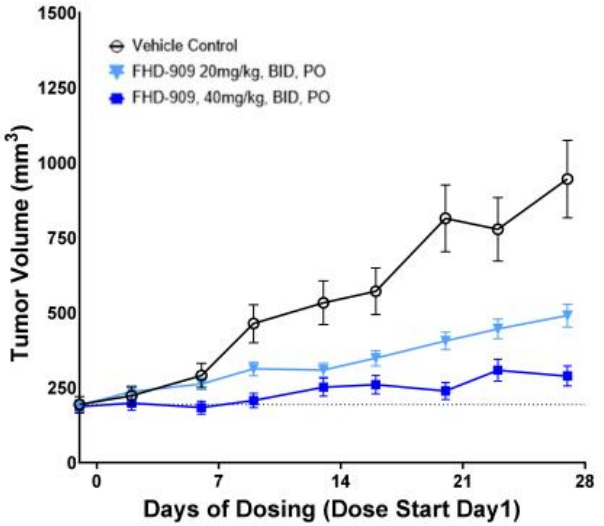


Genetic Background: *SMARCA4 W764R, TP53 E62*, STK11-/-, CDKN2A-/-, KEAP1 R272C*

NOTE: All doses were well tolerated. Dosing holidays were applied at the high dose, as appropriate

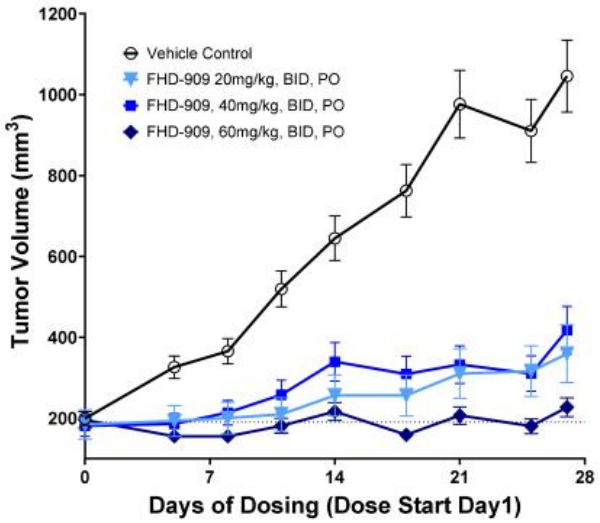
FHD-909 Monotherapy Demonstrated 96% TGI in A549 and Tumor Stasis in RERF-LC-AI Mutant NSCLC Models

A549 Model



Genetic Background	*SMARCA4, Q729fs / H736Y, KRAS G12S, STK11-/-, CDKN2A-/-, KEAP1 G333C*

RERF-LC-AI Model

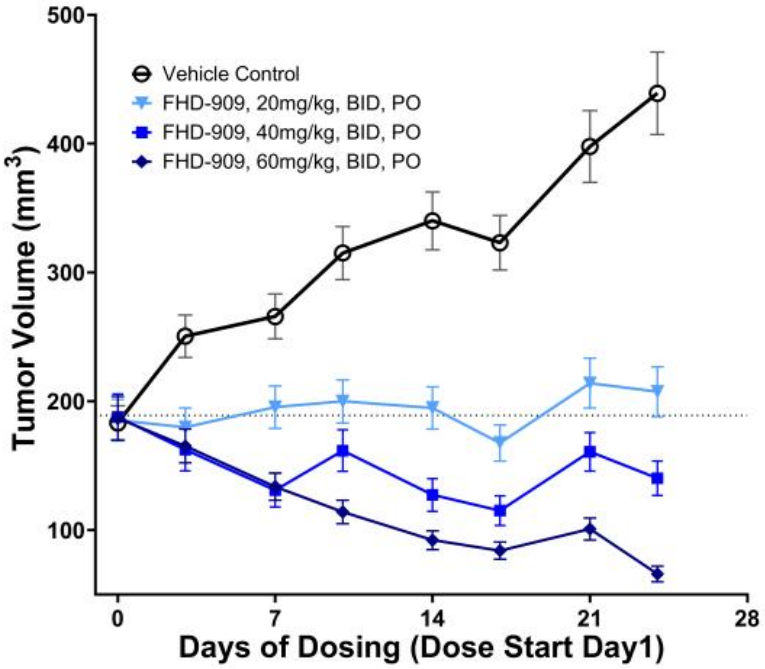


Genetic Background	*SMARCA4 mut p.E1496*, TP53 p.Q104*, NF1 p.E1699**

NOTE: All doses were well tolerated. Dosing holidays were applied at the high dose, as appropriate

FHD-909 Monotherapy Demonstrated Regression in H1793 SMARCA4 Mutant NSCLC Model

H1793 Model



Genetic Background | *SMARCA4, E514*, TP53 R209* R273H, ARID1A C884**

- **FHD-909** delivered across range of SMARCA4 mut xenograft models
- Results ranging from impressive TGI to regression as monotherapy
- All doses across all four models were well tolerated

NOTE: All doses were well tolerated. Dosing holidays were applied at the high dose, as appropriate

FHD-909 Trial Design

Dose Escalation

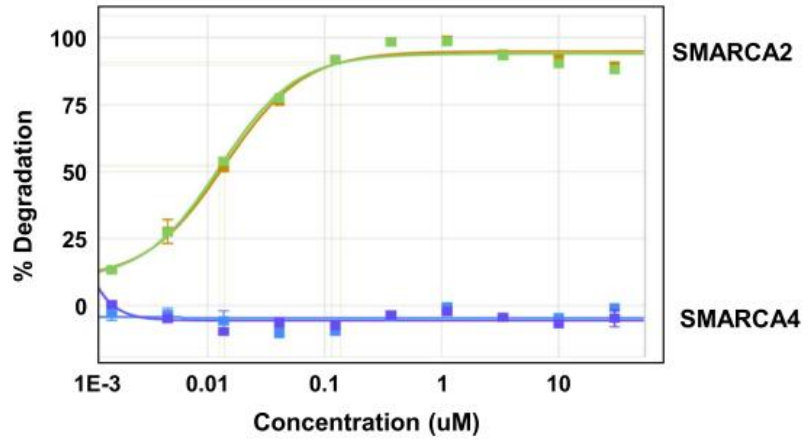
- Restricted to SMARCA4 mutated tumors
- SMARCA4 mutant status confirmed by standard NGS panel
- Further enrichment for NSCLC patients as trial progresses
- Tumor histology agnostic

Dose Expansion

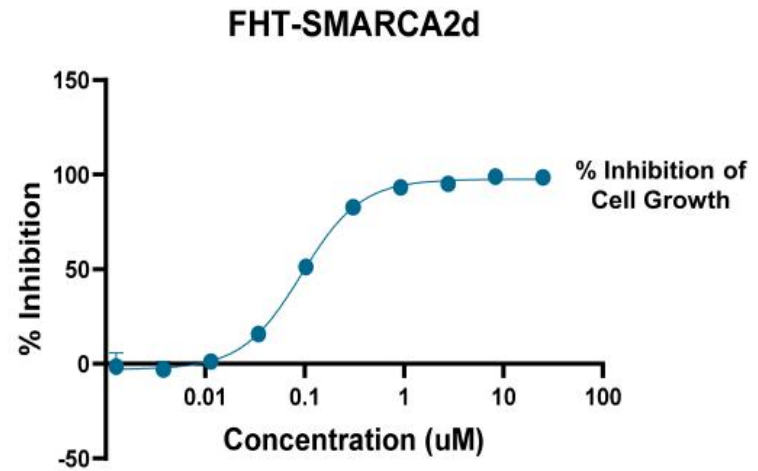
- Arm 1: SMARCA4 mutant NSCLC
- Arm 2: Other SMARCA4 mutant tumors (e.g., bladder, endometrial, colorectal)
- Potential for combination arm(s)

SMARCA2 Selective Degrader Achieved Complete SMARCA2 Degradation and Cell Growth Inhibition *In Vitro*

SMARCA2 / SMARCA4 HIBIT Data



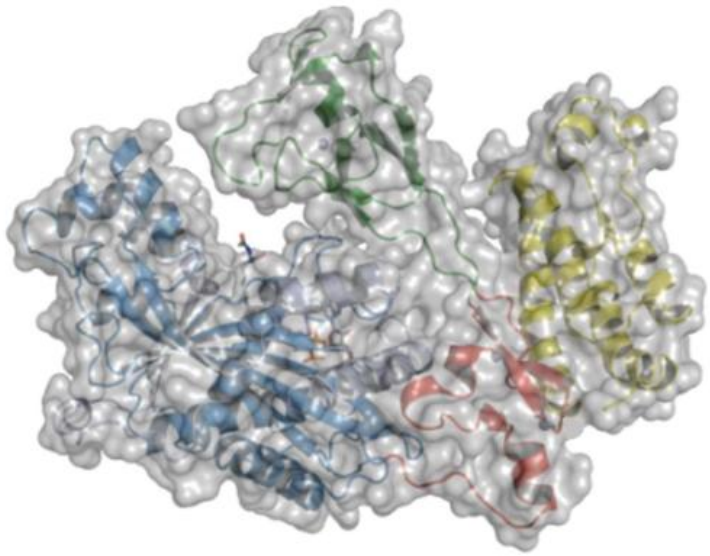
A549 Ten-Day Proliferation Assay



Degraders Caused Time- and Dose-Dependent SMARCA2 Degradation Antiproliferative Effects in A549 Mutant NSCLC Model

Data as of Q4 2021

CBP and EP300 Proteins – A Decades Long Challenge in Selectivity



- **CBP** and **EP300** are chromatin regulators and histone acetyltransferases
- **CBP** and **EP300** are virtually identical, thus achieving selectivity is a significant challenge
    - Dual targeting has revealed tolerability and safety issues

Foghorn is working on two separate programs, each with their own defined dependencies and patient populations

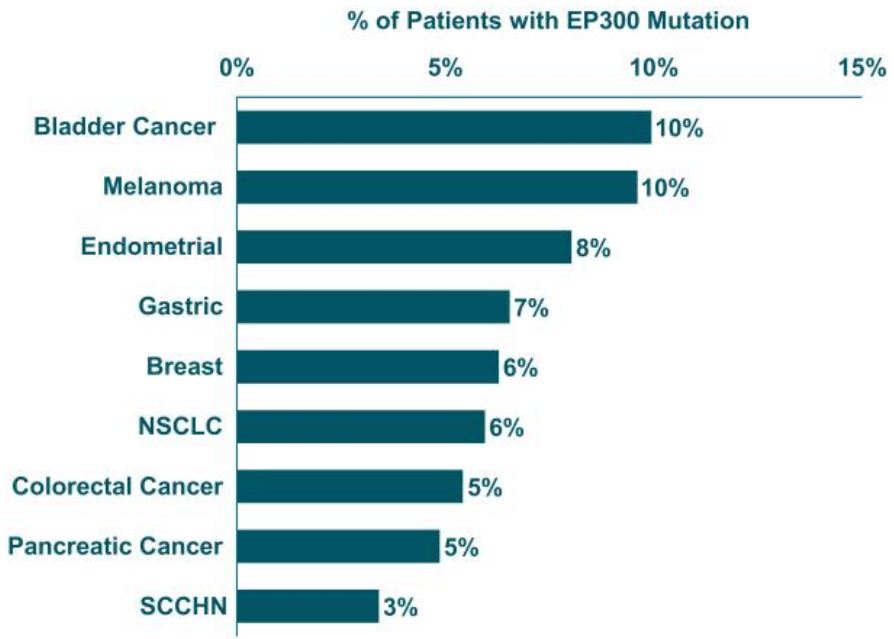
Selective CBP Protein Degrader

For EP300 Mutated Cancers

Summary: Selective CBP Protein Degrader for EP300 Mutated Cancers

Target / Approach	• CREB binding protein (CBP) • Targeted protein degrader
Initial Indication	• EP300 mutated cancers (e.g., subsets of bladder, colorectal, breast, gastric and lung cancers)
Mutation / Aberration	• EP300 mutated cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

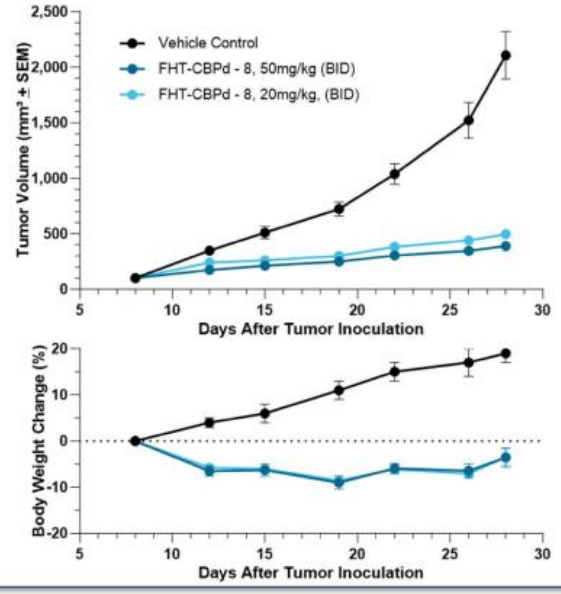
Commercial Opportunity



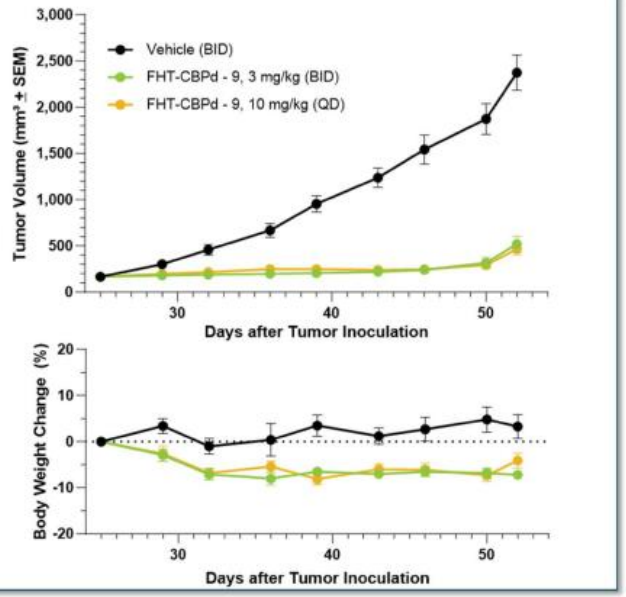
* Per year incidence in the U.S., EU5, Japan . Source: Clarivate DRG Mature Markets Data.

Selective CBP Degradation Resulted in Significant Tumor Growth Inhibition in Colorectal & Bladder and Regression in Gastric EP300 Null Models

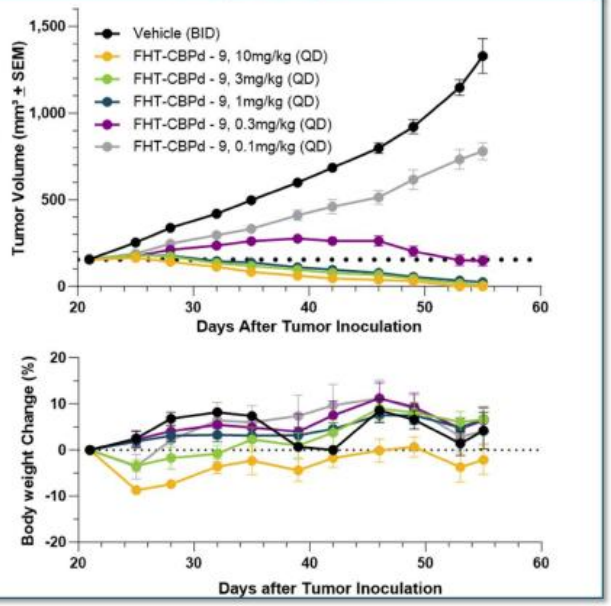
Colorectal
RKO (EP300 Null) Model



Bladder
639V (EP300 Null) Model

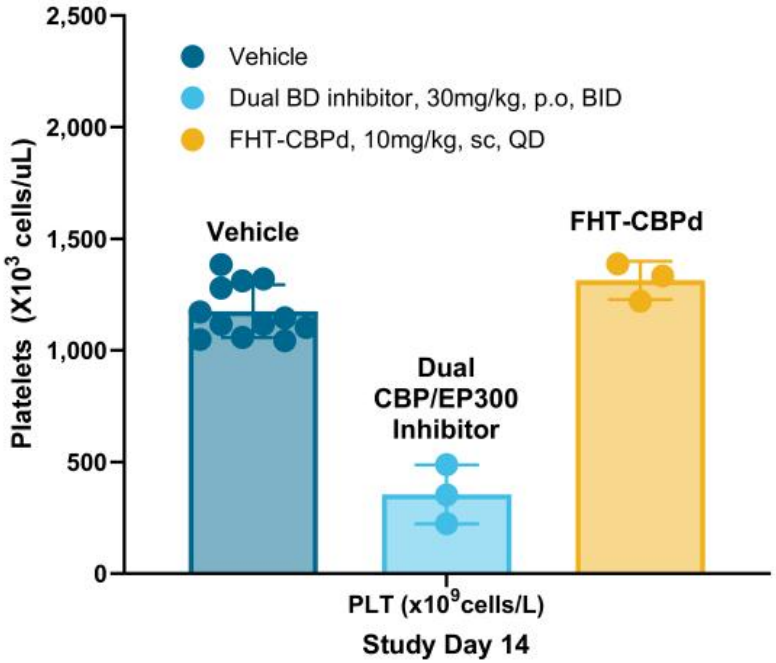


Gastric
AGS (EP300 Null) Model

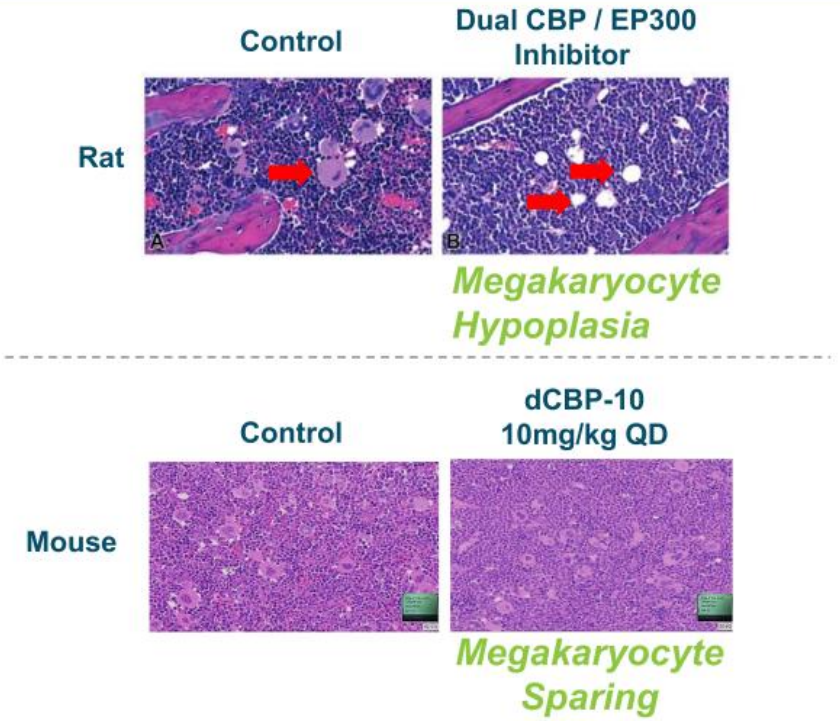


Pre-Clinical Studies Indicate Selective CBP Degradation Did Not Show Thrombocytopenia and Spares Megakaryocytes *In Vivo*

Platelet Counts Post Two Weeks of Dosing (*In Vivo*)

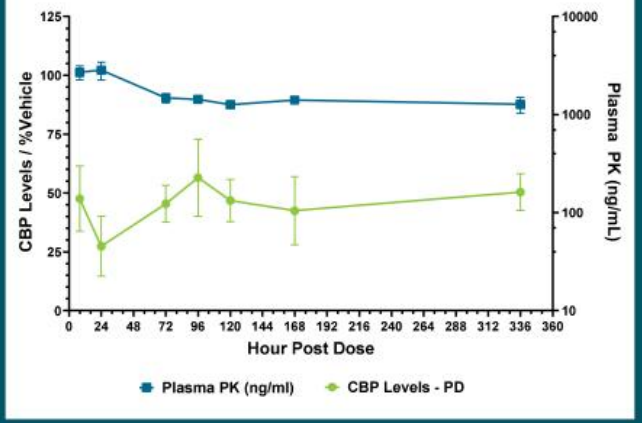


Bone Marrow Staining (*In Vivo*)

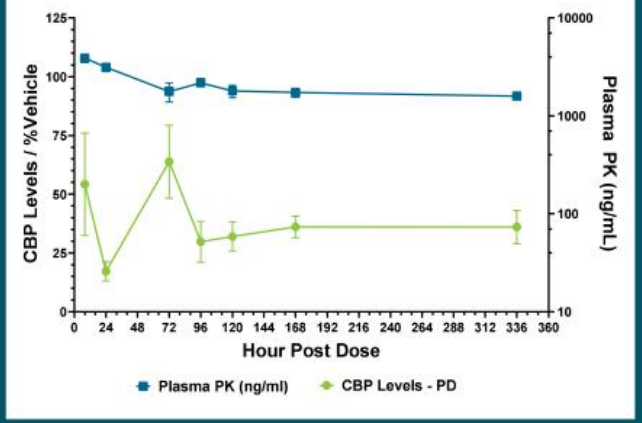


Pre-Clinical Studies Indicate Long-Acting Injectable Formulations of CBP Degrader Could Enable At Least Once Every 2 Weeks Dosing

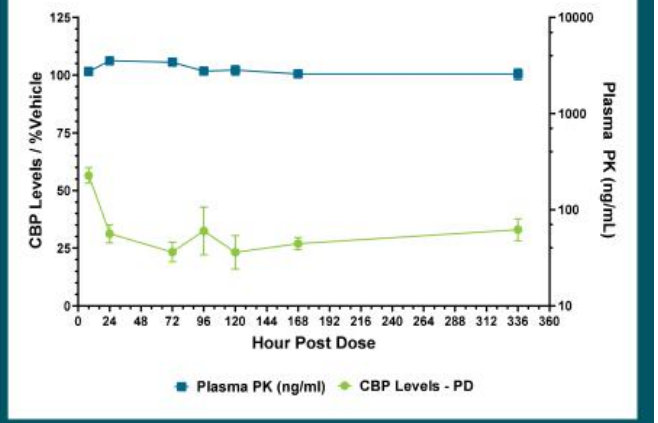
dCBP-9 PK/PD (60mg/kg, sc)



dCBP-9 PK/PD (150mg/kg, sc)



dCBP-9 PK/PD (150mg/kg, im)



Selective EP300 Protein Degrader

For CBP Mutated and EP300 Dependent Cancers

Summary: Selective EP300 Protein Degrader for CBP Mutant & EP300 Dependent Cancers

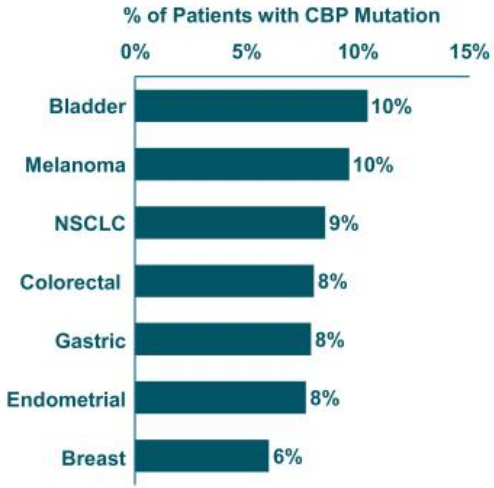
Target / Approach	• E1A binding protein p300 (EP300) • Targeted protein degrader
Initial Indications	• AR+ Prostate • DLBCL • Bladder, melanoma, others
Mutation / Aberration	• EP300 dependent cancers • CBP mutant cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

Commercial Opportunity

EP300 Dependent Cancers

- **Solid Tumors**
  - AR+ mCRPC
  - HR+ breast
- **Hematologic malignancies**
  - DLBCL
  - Multiple Myeloma

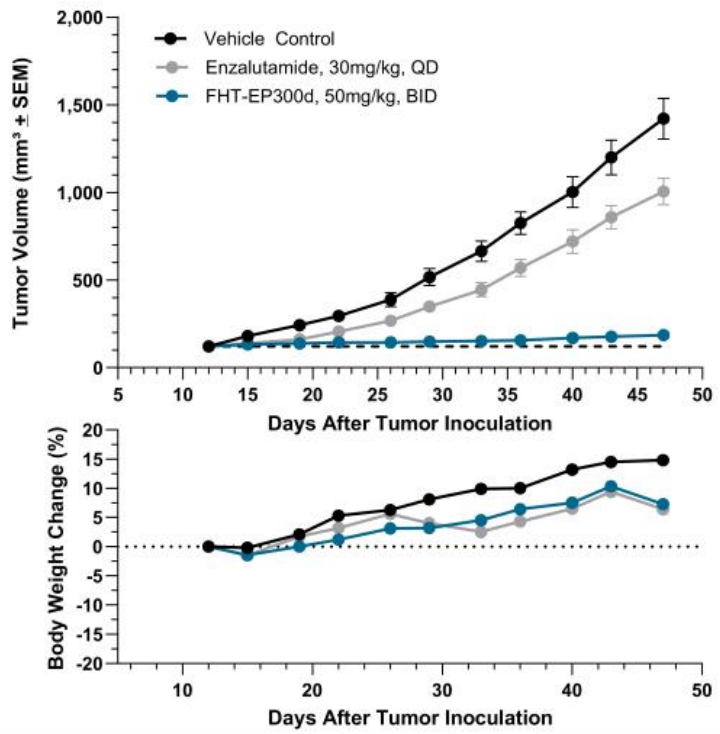
CBP Mutant Cancers



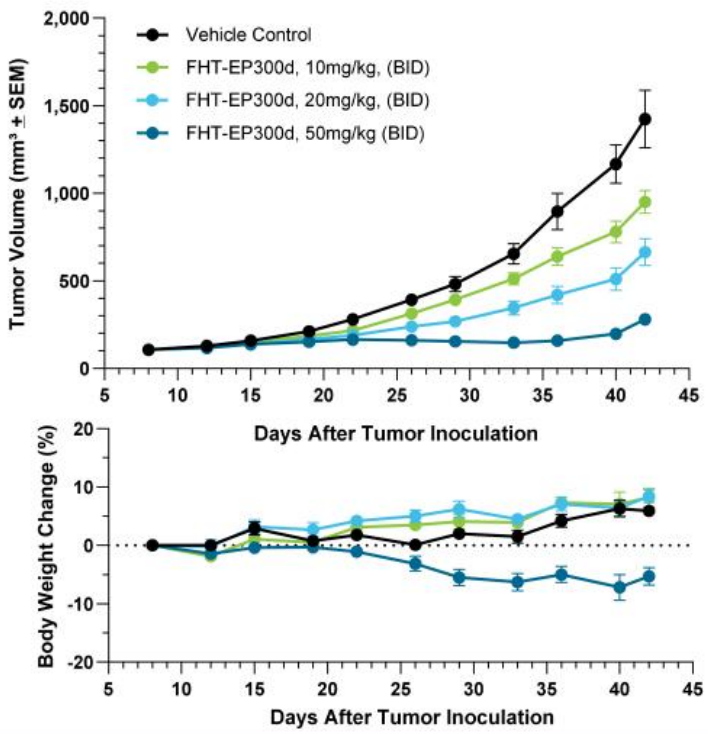
* Per year incidence in the U.S., EU5, Japan. Source: Clarivate DRG Mature Markets Data.

EP300 Degradation Results in Significant Tumor Growth Inhibition in AR+ VCAP Prostate and KARPAS422 DLBCL Models

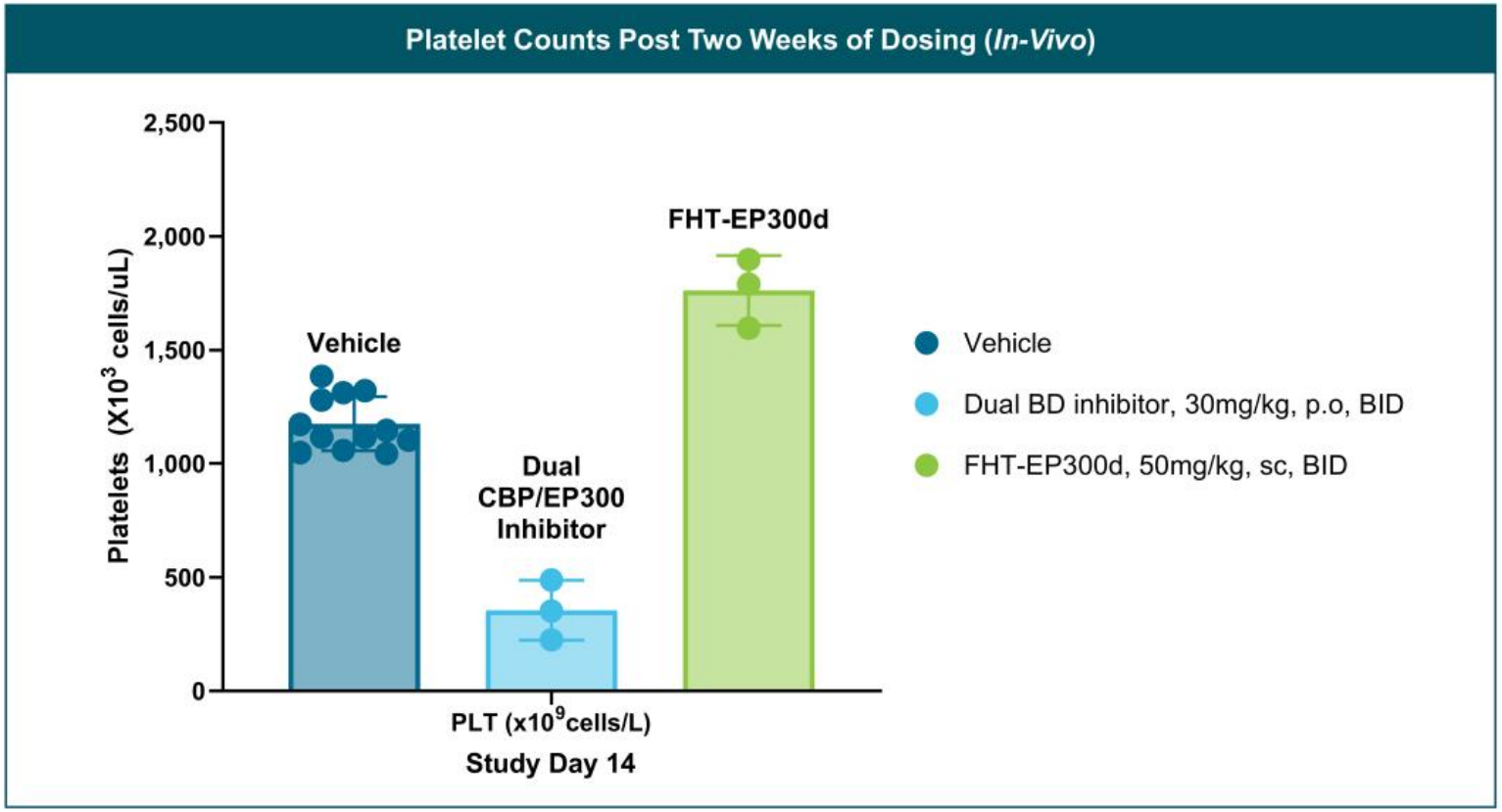
Prostate
AR+ VCAP Model



DLBCL
KARPAS422 (CBP Null) Model



Selective EP300 Degradation Does Not Show Thrombocytopenia *In Vivo*

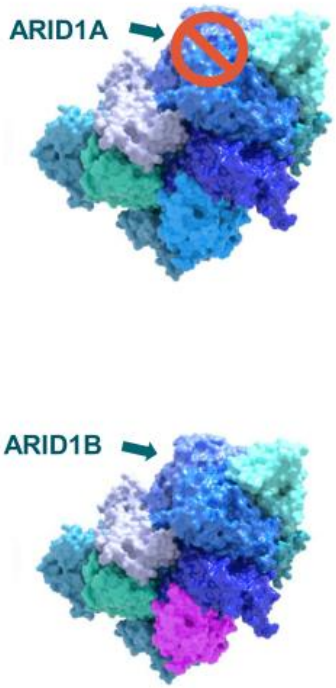


Selective ARID1B Protein Degrader

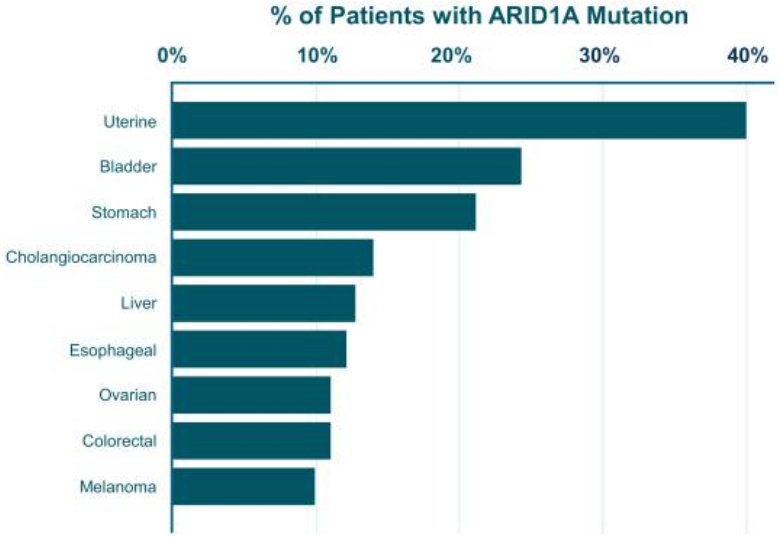
For ARID1A Mutated Cancers

ARID1B is a Major Synthetic Lethal Target Implicated in Up To 5% of All Solid Tumors

Target / Approach	• ARID1B • Targeted protein degrader
Initial Indication	• ARID1A mutated cancers
Mutation / Aberration	• ARID1A mutations (e.g., ovarian, endometrial, colorectal, bladder and other cancers)
Stage	• Pre-clinical
New Patients Impacted / Year*	• > 175,000



Commercial Opportunity

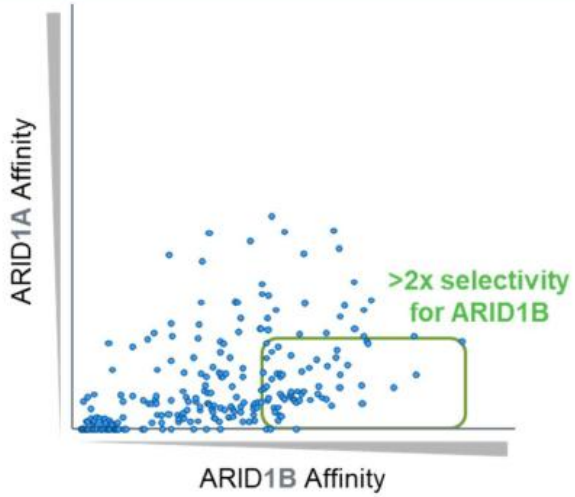


~5% of all solid tumors harbor ARID1A mutations

* Per year incidence in the U.S., EU5, Japan. Source: Clarivate DRG Mature Markets Data.

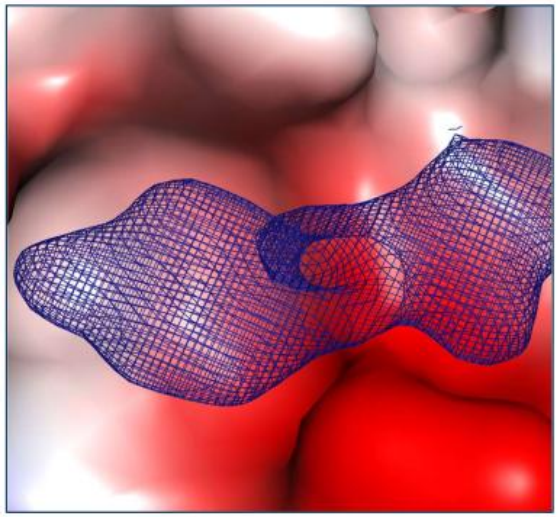
Compound Screening and Structure-Based Optimization Yielded Selective ARID1B Binders

Identification of Selective ARID1B Binders



- Mapped and purified several potential ligandable regions of ARID, which were then screened against various compound libraries
- Characterized binding using multiple biochemical and biophysical techniques: e.g., DSF, ASMS, NMR, and SPR

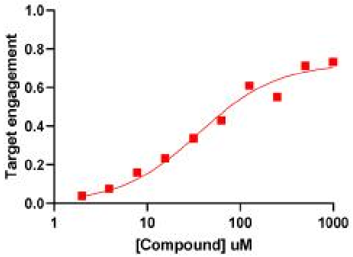
X-Ray Crystal Structures Detail Selective ARID1B Binding

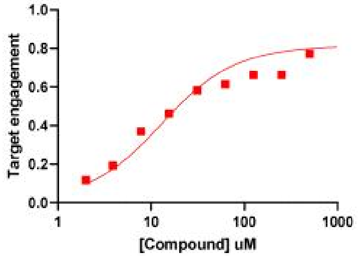


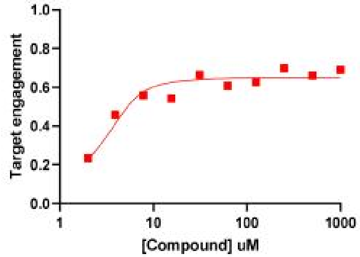
- Determined X-ray crystal structure of ARID ligandable domains with specific binders
- Leveraged these structures to drive binding affinities and expand binding chemotypes

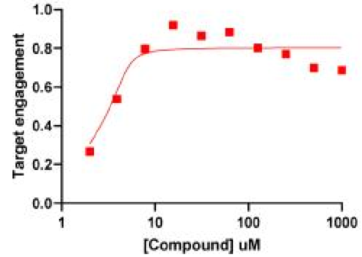
Structure-Based Optimization Drove Improved ARID1B Binding Affinity from 100 uM to less than 200 nM

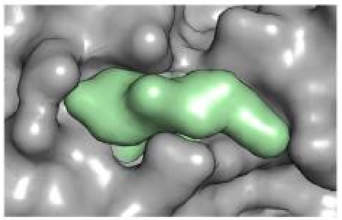
Gen 1: Screening Hit	Gen 2: Early Optimization	Gen 3: Sub-uM Affinity	
ARIDb-1 ARID1B Kd: **100 uM**	**ARIDb-2** ARID1B Kd: **15 uM**	**ARIDb-3** ARID1B Kd: **0.5 uM**	**ARIDb-9** ARID1B Kd: **0.2 uM**



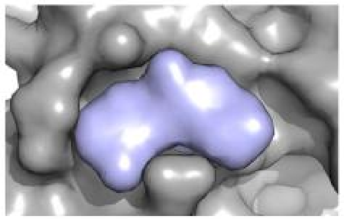




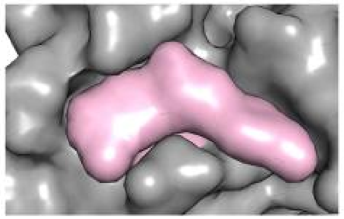




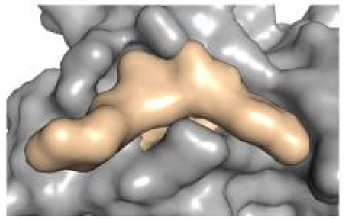
1.4 Å co-xtal structure



2.0 Å soak structure



1.9 Å co-xtal structure



1.7 Å soak structure

… with Multiple Near-Term Value Inflection Points through 2026

FHD-286	AML Combination Dose Escalation Data	Q4 2024
FHD-909 (Selective SMARCA2 Inhibitor)	Phase 1: First Patient Dosed	October 2024
	Phase 1 Dose Escalation Data	Confidential
Selective SMARCA2 Degrader	IND Filing / Phase 1 Initiation	Confidential
Selective CBP Degrader	Initiate IND-Enabling Studies	Year End 2024
Lilly Target #2	Target Disclosure and IND Filing	Confidential
Selective EP300 Degrader	Initiate IND-Enabling Studies	2025
Selective ARID1B Degrader	Development Candidate	H1 2026

SMARCA2 = BRM
SMARCA4 = BRG1

Developing First-In-Class Precision Medicines Targeting Major Unmet Needs in Cancer

Leader in Unique Area of Cancer Biology

Foghorn is a **leader in targeting chromatin biology,** which has the potential to address underlying dependencies of many genetically defined cancers

Platform with initial focus in oncology, **therapeutic area expansion potential**

Large Market Potential

Chromatin biology is implicated in up to **50% of tumors**, potentially impacting **~2.5 million patients**

Foghorn's current pipeline potentially addresses **more than 500,000** of these patients

Broad pipeline across a range of targets and small molecule modalities

Well-Funded

$285.2 million in cash and equivalents

(as of 6/30/2024)

Cash runway into 2027

Shares outstanding: approximately 62.5M*

Value Drivers

Anticipate data from the Phase 1 trial of FHD-286 in combination with decitabine in **Q4'24**

SMARCA2 Selective Inhibitor (FHD-909), partnered with Lilly, in **Phase 1 trial**

Advancement of preclinical assets (SMARCA2 Selective Degrader, CBP, EP300, ARID1B) towards INDs

Major Strategic Collaboration

Strategic collaboration with Lilly; **$380 million upfront;** 50/50 U.S. economic split on two lead programs

**Includes common shares outstanding as of 6/30/2024 as well as common stock and pre-funded warrants issued as part of May 2024 financing*

FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

October 2024

Appendix

Lilly Collaboration Validates Foghorn Approach: Significant Upfront and Deal Economics

$380 Million Up-front

$300 million cash

$80 million in Foghorn common stock at a price of $20 per share

50/50 U.S. Economics on Two Programs

50/50 U.S. economic split on SMARCA2-Selective and another undisclosed program

Tiered ex-U.S. royalties starting in the low double-digit range and escalating into the twenties based on revenue levels

Three Undisclosed Discovery Programs

Option to participate in a percentage of the U.S. economics

Tiered ex-U.S. royalties from the mid-single digit to low-double digit range

$1.3 billion in potential milestones

FHD-286: Dual SMARCA2 / SMARCA4 Inhibition

Targeting BAF Dependency in Cancer

Additional Information

Potential First-in-Class Mutation-Agnostic Differentiation Agent With Significant Combination Potential in AML

Completed Phase I Monotherapy Safety and Efficacy Results

Efficacy

- Differentiation observed in heavily pre-treated patients, regardless of mutational status
- Multiple patients with bone marrow and peripheral blast improvements and associated ANC recovery

Safety

- Adverse data observed to be profile consistent with late-line AML population
  - Most frequent ≥ grade 3 TRAEs: increased blood bilirubin, hypocalcemia, differentiation syndrome (DS), stomatitis, increased ALT
- Adjudicated Differentiation Syndrome rate of 15%

Ongoing Phase I Combination Trial

- Phase I dose escalation trial evaluating oral daily dosing of FHD-286 with fixed dose decitabine or cytarabine
- Standard 3+3 dose escalation design
- Data anticipated in H2'2024

Peripheral Blood and Bone Marrow Blast Count Reduction Led to ANC Recovery in a Subset of Patients

Diagnosis	Dose Level	Mutations	Cytogenetic Risk	Cycles on Study	ANC Recovery	Peripheral Starting Blast %	Peripheral Min Blast %	Peripheral Blast % Change	BMA Starting Blast %	BMA Min Blast %	BMA Blast % Change
AML	10mg	N/A	Adverse	2.2	**YES**	15	0	(100)	40	6	(85)
AML	10mg	DNMT3A, U2AF1, DDX41, CUX1, TP53	Adverse	0.5	N	20	0	(100)	13	2	(85)
AML	10mg	NRAS, SF3B1	Intermediate	7.3	N	2	0	(100)	12	5	(58)
AML	10mg	NRAS, BRCA1, MEN1, CDKN1Ap	Adverse	0.3	N	80	11	(86)	52	-	-
AML	10mg	D17Z1, TP53	Intermediate	0.6	N	9	1	(89)	9	-	-
AML	10mg	GATA2, ETV6, KDR	Intermediate	1.4	N	2	2	0	5	-	-
AML	7.5mg	RUNX1, KRAS, ASXL1, JAK2, TET2, EZH2, ETNK1	Intermediate	2.9	N	83	1	(99)	83	2	(98)
AML	7.5mg	ASXL1, TP53, U2AF1	Adverse	1.3	N	-	5	-	36	14	(61)
AML	7.5mg	KMT2A rearrangement	Adverse	2.8	**YES**	97	5	(95)	89	48	(46)
AML	7.5mg	N/A	Adverse	4.1	**YES**	28	4	(86)	25	15	(40)
* MDS	7.5mg	DNMT3A, TP53	Adverse	1.4	N	-	0	-	8	5	(38)
AML	7.5mg	DNMT3A, KRAS, NRAS	Adverse	1.8	N	32	2	(94)	47	49	4
AML	7.5mg	CBFB (locus at 16q22)	Favorable	1.7	**YES**	32	0	(100)	27	29	7
AML	7.5mg	N/A	Adverse	0.1	N	35	19	(46)	72	-	-
AML	7.5mg	ASXL1, BCOR, FLT3ITD, NF1, CBL, H1-B, NFE2	Adverse	0.7	N	8	7	(13)	25	-	-
AML	7.5mg	N/A	-	0.5	N	0	0	0	8	-	-
AML	7.5mg	NRAS, ASXL2, SRSF2	Adverse	0.1	N	93	-	-	17	-	-
AML	7.5mg	ASXL1, DNMT3A, TET2, TP53	Adverse	0.5	N	-	4	-	-	-	-
AML	7.5mg	FLT3ITD	Favorable	0.8	N	0	39	-	12	-	-

* MDS Patient

Peripheral Blood and Bone Marrow Blast Count Reduction Leading to ANC Recovery in a Subset of Patients

Diagnosis	Dose Level	Mutations	Cytogenetic Risk	Cycles on Study	ANC Recovery	Peripheral Starting Blast %	Peripheral Min Blast %	Peripheral Blast % Change	BMA Starting Blast %	BMA Min Blast %	BMA Blast % Change
AML	5mg	RUNX1, NRAS, ASLX1	Adverse	3.1	**YES**	29	0	(100)	35	12	(66)
AML	5mg	N/A	Adverse	8.0	N	-	2	-	11	7	(36)
AML	5mg	N/A	Adverse	1.8	**YES**	6	0	(100)	24	16	(33)
AML	5mg	ASXL1, DNMT3A, KRAS, PTPN11, WT1, GRIN2AWT1	Adverse	2.0	N	32	38	19	49	52	6
* MDS	5mg	RUNX1, NRAS, KRAS, SF3B1, ASXL2, CSF3R, GATA2	Adverse	1.0	**YES**	5	13	160	11	14	27
* MDS	5mg	DNMT3a, TET2	Intermediate	1.9	**YES**	0	0	0	1	2	100
AML	5mg	TET2, WT1, GATA2, PLCG2, ARHGEF28, BRD4, CDK12, DDX41, KMT20, PARP1, ZRSR2	Intermediate	1.7	**YES**	9	0	(100)	18	46	156
AML	5mg	KRAS, PTNP11, IRF8, MSH6, RUNX1	-	1.3	N	17	7	(59)	-	80	-
AML	5mg	TP53	Adverse	0.7	N	41	20	(51)	18	-	-
AML	5mg	TP53	Adverse	0.5	N	44	35	(20)	55	-	-
AML	5mg	PPM1D, TP53	Adverse	0.5	N	15	12	(20)	18	-	-
AML	5mg	KRAS, TET2	Adverse	0.6	N	37	32	(14)	56	-	-
* MDS	5mg	ASXL1, DNMT3A, IDH1, SRSF2, SF3B1, TET2	-	0.4	N	0	0	0	0	-	-
AML	5mg	N/A	Adverse	0.5	N	10	11	13	-	-	-
AML	5mg	ASXL1, NRAS, EP300, STAG2, RUNX1, TET2	Adverse	0.1	N	25	32	25	11	-	-
AML	5mg	CEBPA, KMT2C, NCOR1, CBL	-	0.3	N	48	75	56	64	-	-
AML	2.5mg	NRAS, WT1	Adverse	1.4	N	36	62	72	45	74	64
AML	2.5mg	BCR/ABL, PMLRARA, RUNX1, TET2	-	2.4	N	68	28	(59)	30	-	-
AML	2.5mg	N/A	Adverse	0.8	N	7	0	(100)	22	-	-
AML	2.5mg	DNMT3A, KRAS, TP53	Adverse	0.8	N	28	40	46	45	-	-
AML	2.5mg	DNMT3A, TP53	Adverse	1.0	N	4	-		25	-	

* MDS Patient

Signs of Differentiation in Heavily Pre-Treated, Secondary AML Patient with Abnormal Karyotype in Phase 1 Dose Escalation Trial

Patient Background:

- 47-year-old male, secondary AML
- Abnormal karyotype: Del (7Q), Inv (3), Der (7;12), -8, ADD(1)

Prior AML Treatment:

- Progressive disease: 4 lines prior treatment and 2 bone marrow transplants

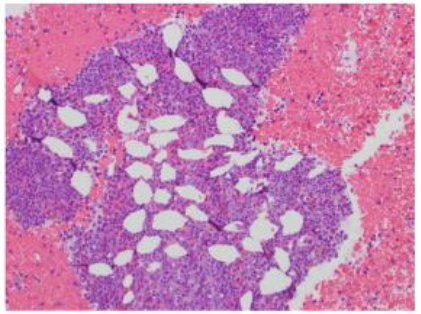
Prior non-AML treatment:

- MDS with inv(3) and der(7;12) and ASXL1 mut. Received AZA x 4.

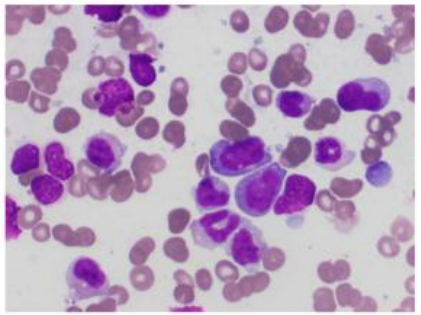
Initiation of FHD-286 at 10 MG Dose:

- Bone marrow blast from 40% to 6% with clear evidence of differentiation with persistence of cytogenetics abnormalities. ANC recovery.

Bone Marrow Blast Reduction from 40% to 6%



Bone Marrow Aspirate: Clear Evidence of Differentiation



Clinical Benefit in Heavily Pre-Treated Patient in Phase 1 Dose Escalation Trial

Patient Background:

- 25-year-old male, treatment-related AML
- KMT2A rearrangement

Prior AML Treatment:

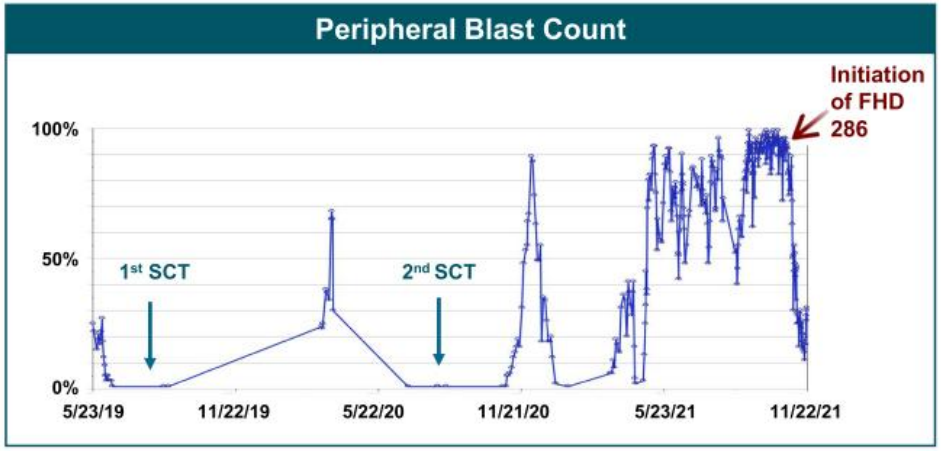
- Progressive disease with CNS Leukemia: 7 lines prior treatment and 2 bone marrow transplants

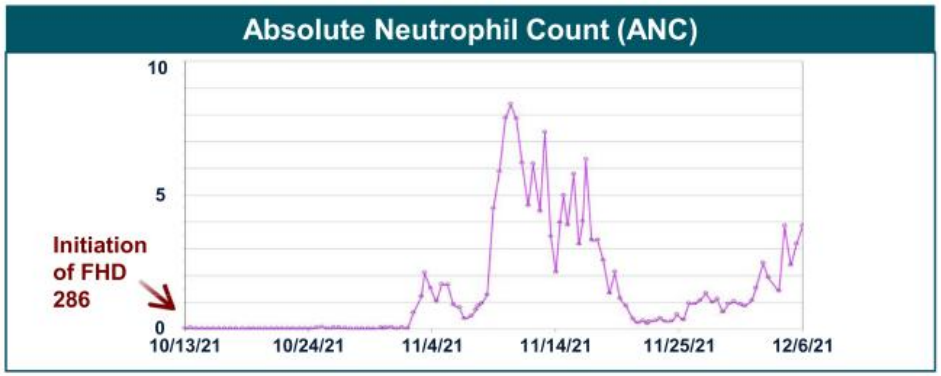
Prior non-AML treatment:

- Ewing's sarcoma: Treated with Chemo/RT/Surgery (VCR, doxo, cyclophos, ifos, etoposide)

Initiation of FHD-286 at 10 MG Dose:

- Drop in peripheral blast, 97% to 5%
- Bone marrow reduction from 89% to 48%, with ANC recovery





Transcription Factors

A Novel Approach

Foghorn's Novel Approach to Drugging Transcription Factors Enabled by Its Protein Production and Discovery Capabilities

Transcription Factors are Compelling Drug Targets…

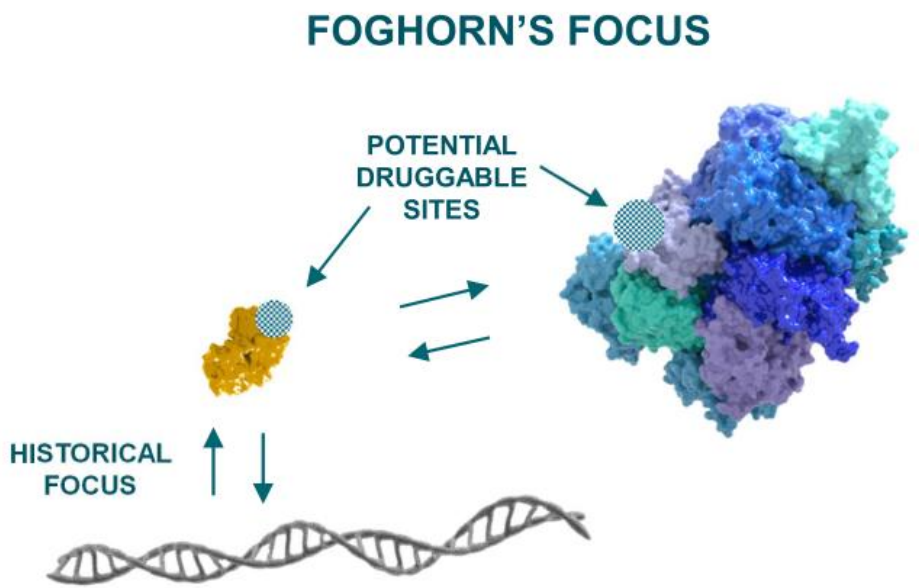
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…But Historically Difficult to Target…

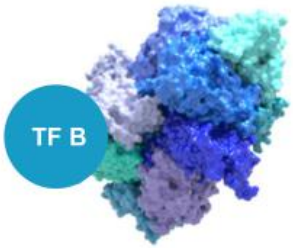
- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities

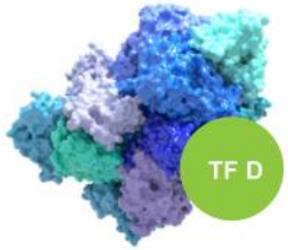
Foghorn Has a New Approach Focusing on Interaction with BAF

- Druggable binding pockets
- Druggable affinities



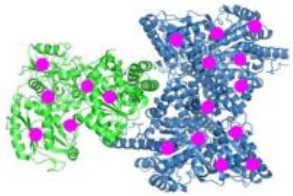
Transcription Factors Bind to BAF Directly with Specificity; Unique Insights into Where and How Transcription Factors Bind



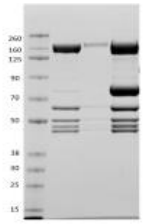


Mapping the TF-BAF Interaction

Mass spec. foot-printing



Pull-down assays

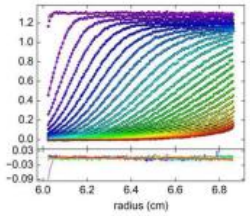


Foghorn's collection of BAF sub-complexes and domains

Validating the TF-BAF Interaction

Biophysical

AUC / SPR / ITC

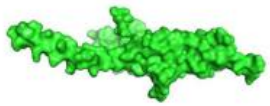


Biochemical

TR-FRET / FP

Structural

Crystal / NMR



FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

October 2024