FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

January 2025

FHTX
NASDAQ LISTED

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 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-909 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-909, 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 a Leader in Chromatin Biology, Successfully Drugging Challenging Targets

Pioneers in Targeting Chromatin Biology

Chromatin Regulation is Implicated in up to 50% of Tumors

Foghorn has Unlocked Previously Undruggable Targets

Leaders in Targeted Protein Degradation

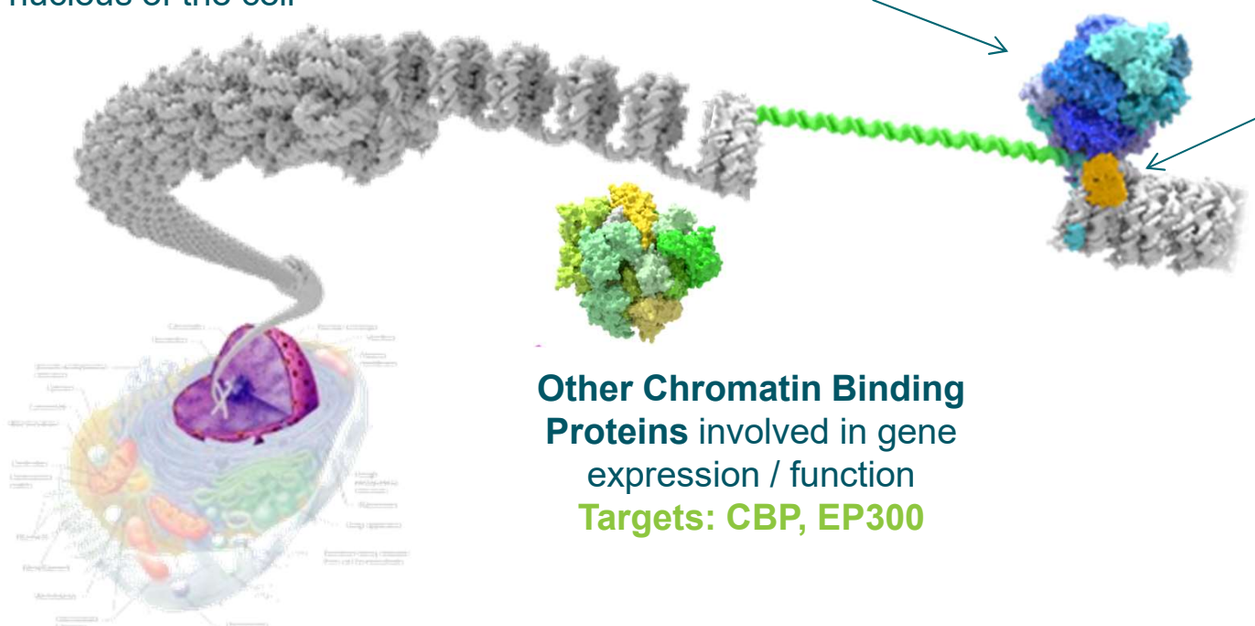
Opening New Biology with Induced Proximity

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

Chromatin Regulatory System genes are **implicated** across a **wide range of cancers**

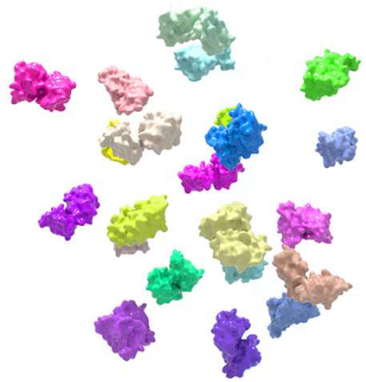
Chromatin – compacted form of DNA inside the nucleus of the cell

Chromatin Remodeling Complexes – specialized multiprotein machines that allow access to DNA
Targets: SMARCA2, ARID1B



Other Chromatin Binding Proteins involved in gene expression / function
Targets: CBP, EP300

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



Leveraging **synthetic lethality** and **lineage dependencies**

Foghorn Has Progressed Multiple Programs Against Challenging Targets

SMARCA2: Potential in up to 10% of NSCLC and 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 expected H1 2026

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

Selective EP300 Degrader
IND expected in H2 2026

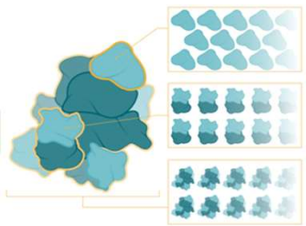
ARID1B: Role in ovarian, endometrial, colorectal cancer. Potential in up to 5% of all solid tumors
Challenge: Industry has had no success with selective target engagement

Selective ARID1B Degrader
Degradation achieved; Program update expected in 2025

… and more.

SMARCA2 = BRM

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

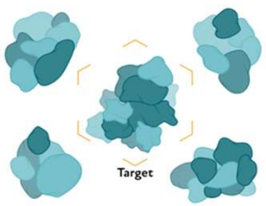


2. Assays & Biochemistry Capabilities

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"



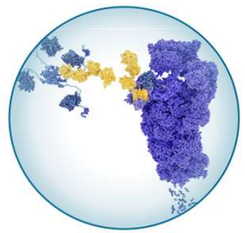
1. Chromatin Biology

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

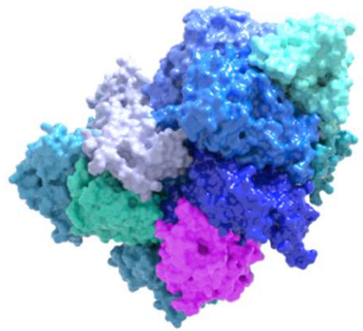
Foghorn's Unique Platform Capabilities Evolved From Drugging a Specific Chromatin Remodeling Complex (BAF)*

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)*

Platform of Proprietary Tools and Unique Know-How Enables Development of Best-in-Class Degraders

Proprietary E3 Ligase Libraries

- Proprietary E3 ligase binders
  - ~ 15 -17 in internal library
  - UBR5

Computational Chemistry *(ML, AI, Virtual Screens)*

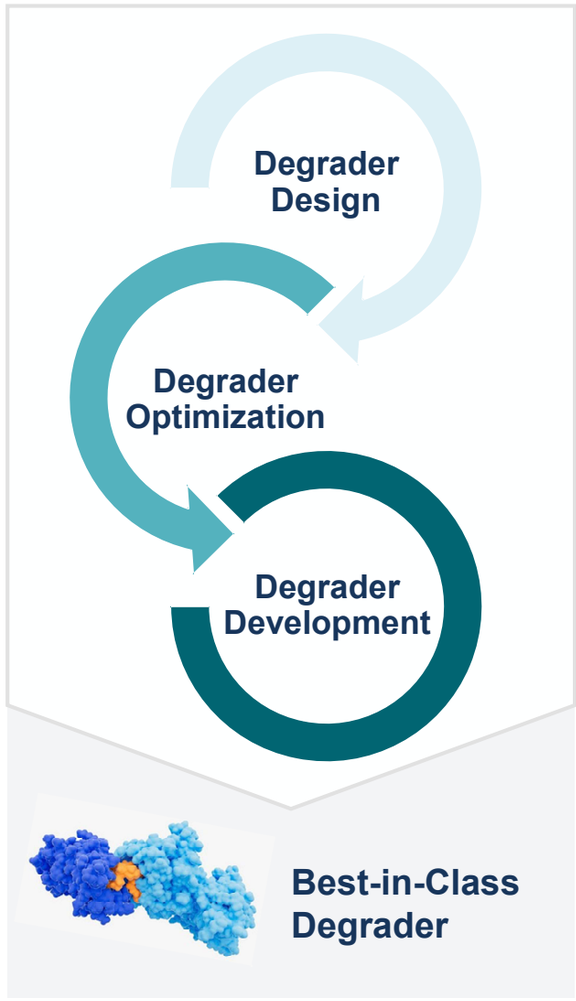
- Structural prediction/ docking

Linker Toolkit

- Well-developed toolkit
- Flexibility-rigidity
- Charge
- Hydrophobicity

Binder Discovery

- Commercial and proprietary covalent and fragment screening libraries
- Target binders



Degrader Mechanics

- Specificity / Selectivity (global proteomics)
- Kinetics (Dmax/DC50)
- Cooperativity (e.g. ternary complex)
- Permeability
- MoA characterization
- Cellular and permeability assays

Exploratory Biology

- *In Vitro* efficacy
- Indication selection
- Combinations
- Resistance mechanisms

In Vivo Validation *(Efficacy and Safety)*

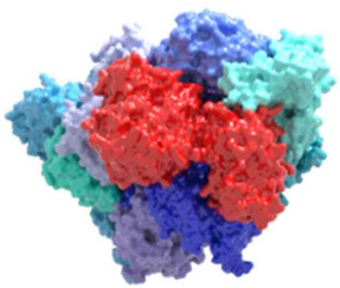
- *In Vivo* models
- PK/PD determination, modeling and dose prediction

Proprietary Formulation & Delivery Platform

- Oral, IV and LAI formulation for drug delivery

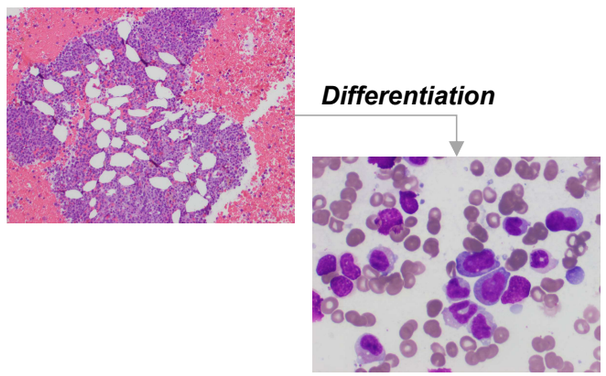
The Next Foghorn Chapter: Delivering Multiple Potential Blockbusters Into the Clinic

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 – 2024)

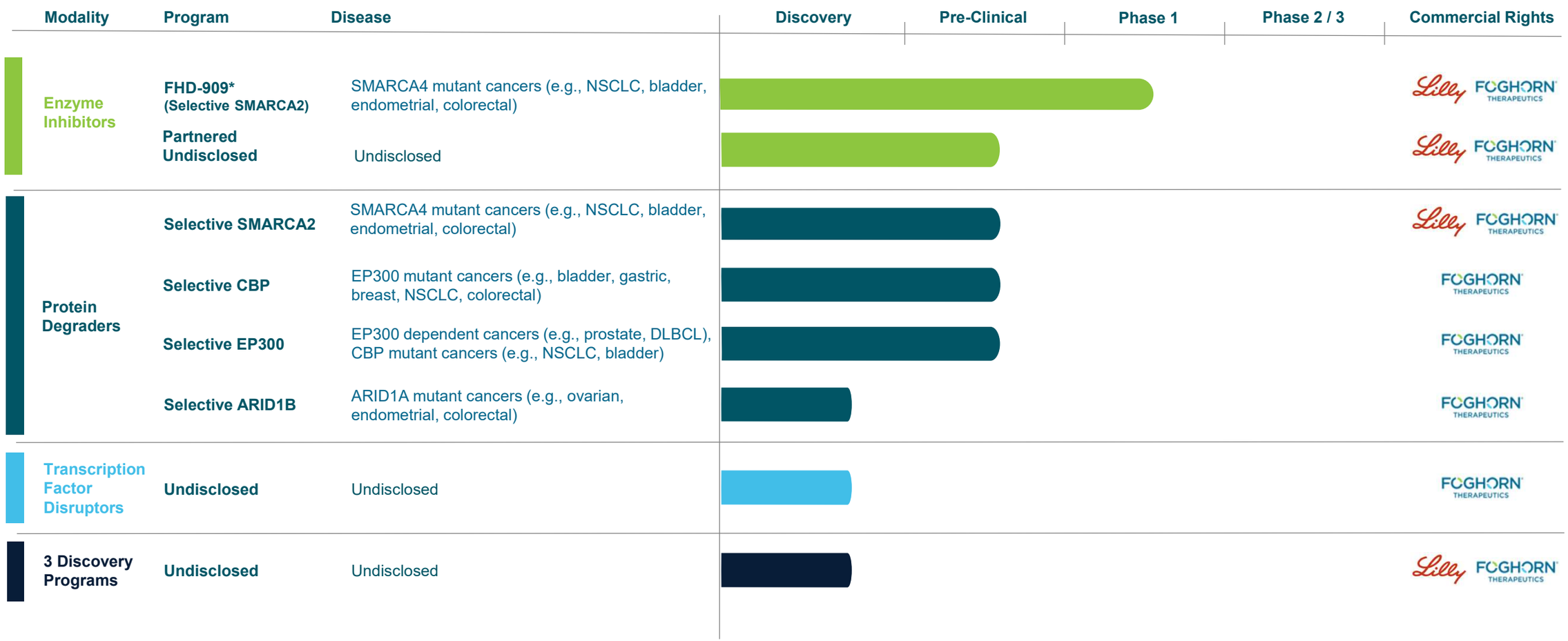


- ✓ **Lilly strategic collaboration**
- ✓ **Developed first-in-class Selective SMARCA2 inhibitor, FHD-909**, and SMARCA2 degrader
- ✓ Advanced Selective CBP degrader, Selective EP300 degrader and Selective ARID1B degrader
- ✓ Expanded protein degrader platform, new applications for Induced Proximity

Progress Multiple High Value Assets into the Clinic
(2025 – 2027)

- **FHD-909 advancing in Phase 1 trial**
- **ARID1B degradation achieved – program update expected in 2025**
- **Potential for 5 additional INDs; Pipeline, platform, disease area expansion**

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



**LY4050784*
SMARCA2 = BRM

…With Multiple Near-Term Value Inflection Points Through 2026

FHD-909 (LY4050784) (Selective SMARCA2 Inhibitor)	Preclinical Combination Data	April 2025 (AACR)
	Phase 1 Dose Escalation Data	Confidential
Selective SMARCA2 Degrader	IND / Phase 1 Initiation	Confidential
Selective CBP Degrader	IND / Phase 1 Initiation	2026
Lilly Target #2	Target Disclosure and IND	Confidential
Selective EP300 Degrader	IND / Phase 1 Initiation	2026
Selective ARID1B Degrader	Selective Degradation Achieved	Program Update 2025

SMARCA2 = BRM
SMARCA4 = BRG1

Potential Multi-Billion Dollar Opportunities in Oncology

Foghorn Owned

Partnered w Lilly

Platform has potential for therapeutic area expansion (e.g., immunology and inflammation)

*LY4050784

FOGHORN THERAPEUTICS

Clinical & Preclinical Programs

- FHD-909 (LY4050784) – Selective SMARCA2 Inhibitor
- Selective CBP Degrader
- Selective EP300 Degrader
- Selective ARID1B Degrader

Selective SMARCA2 Inhibitor and Degrader

For SMARCA4 Mutated Cancers

SMARCA2 = BRM
SMARCA4 = BRG1

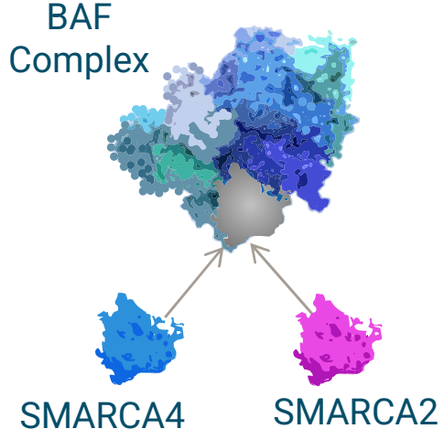
FHD-909 Selective SMARCA2 Inhibitor in Phase 1 Trial; Preclinical Combination Data at AACR 2025

	Selective SMARCA2 Inhibitor FHD-909*	Selective SMARCA2 Degrader
Biology	Exploit the synthetic lethal relationship between SMARCA2 and mutated SMARCA4	
Stage / Next Milestone	Phase 1 dose escalation trial ongoing; Preclinical combination data with pembrolizumab, KRAS and chemo at AACR	Advancing through late preclinical 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

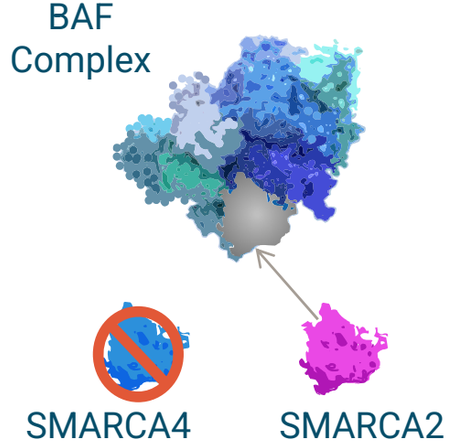
Selective SMARCA2 Inhibition: Promising Strategy to Exploit Synthetic Lethal Relationship Between SMARCA2 and Mutant SMARCA4

Healthy Cells

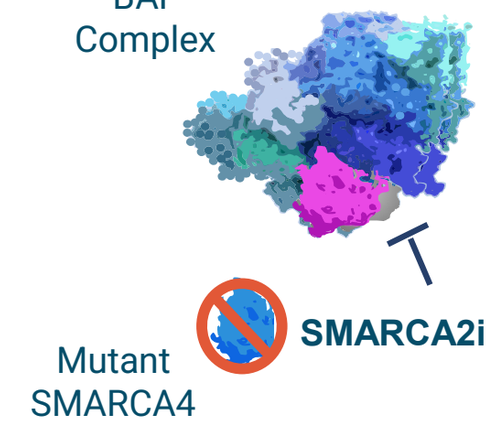


No SMARCA4 Mutations

Cancer Cells



SMARCA4 Mutation



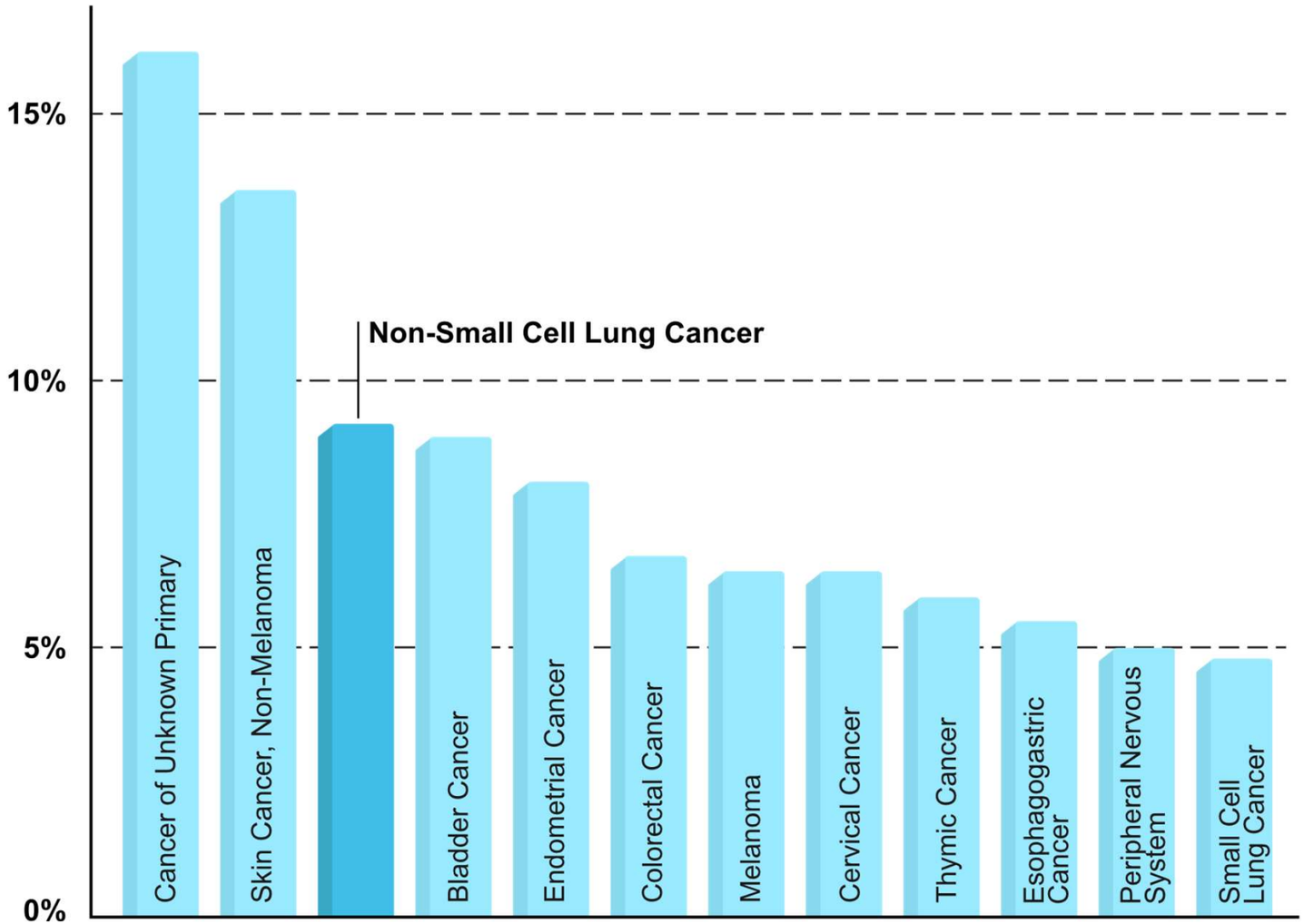
SMARCA4 Mutation and SMARCA2i

Cell Survival

Cell Death

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



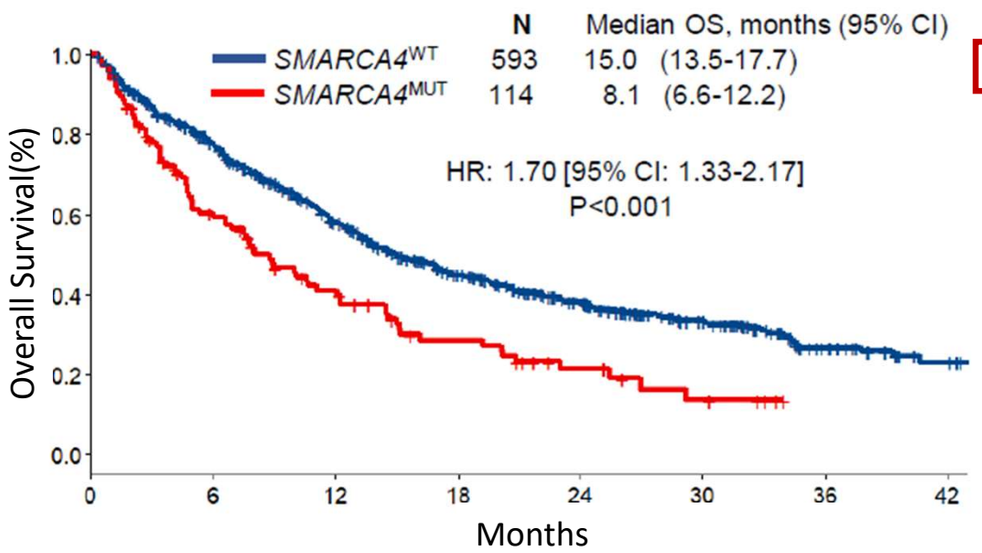
SMARCA4 mutated across a broad range of tumors

Accounts for ~5% of solid tumors

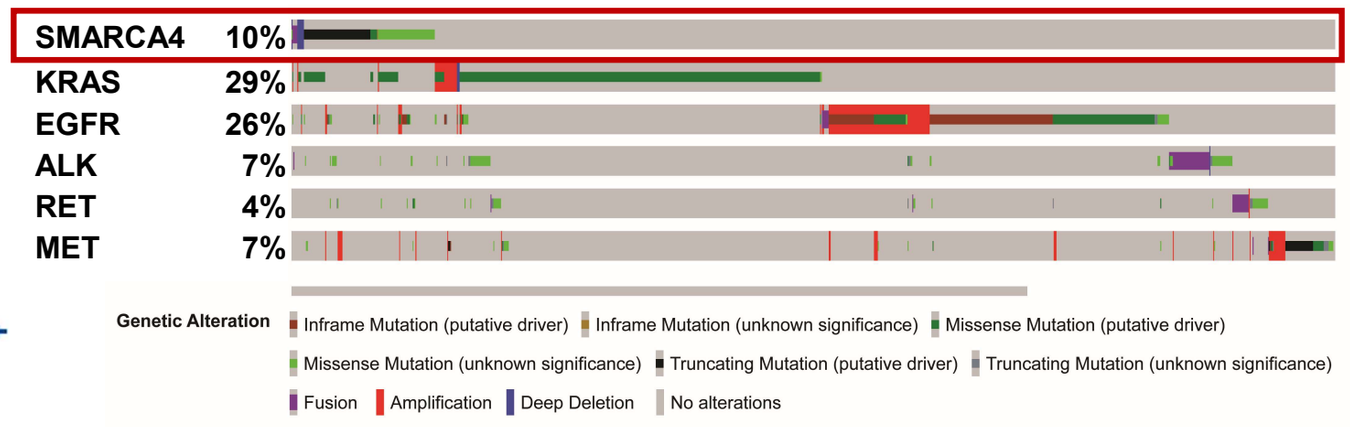
AACR GENIE via cBioPortal

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]

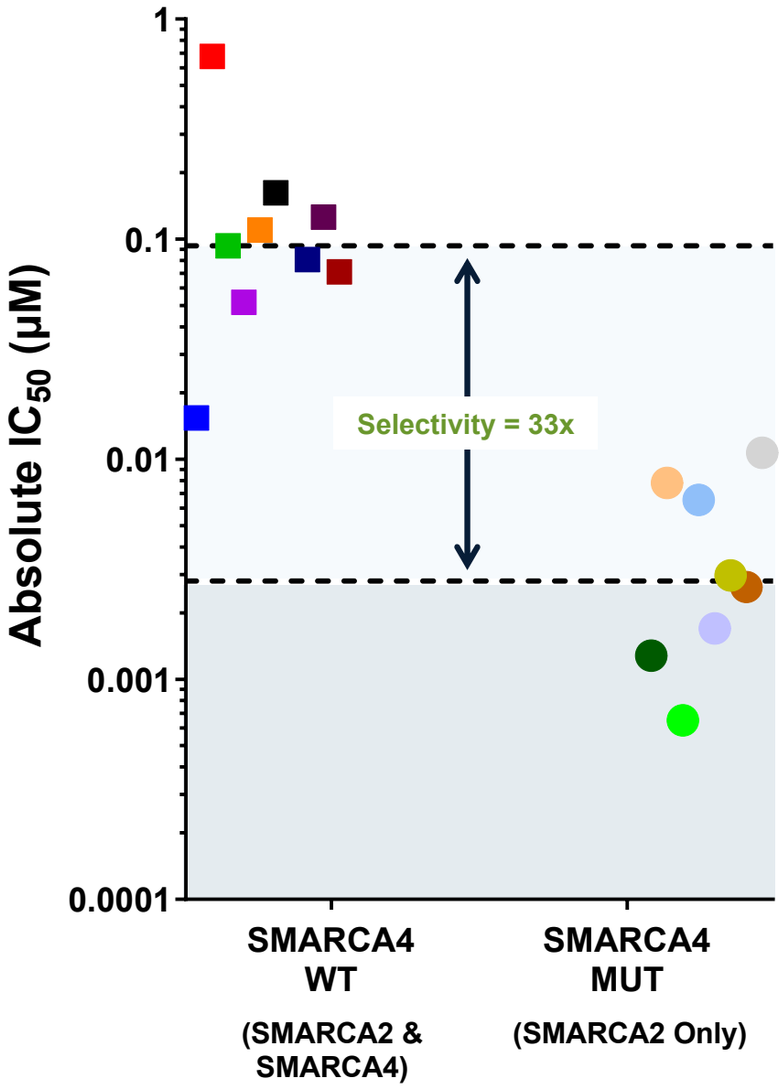


SMARCA4 Mutated in Up to 10% of NSCLC Tumors, Minimal Overlap With Other Mutations[2]



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	

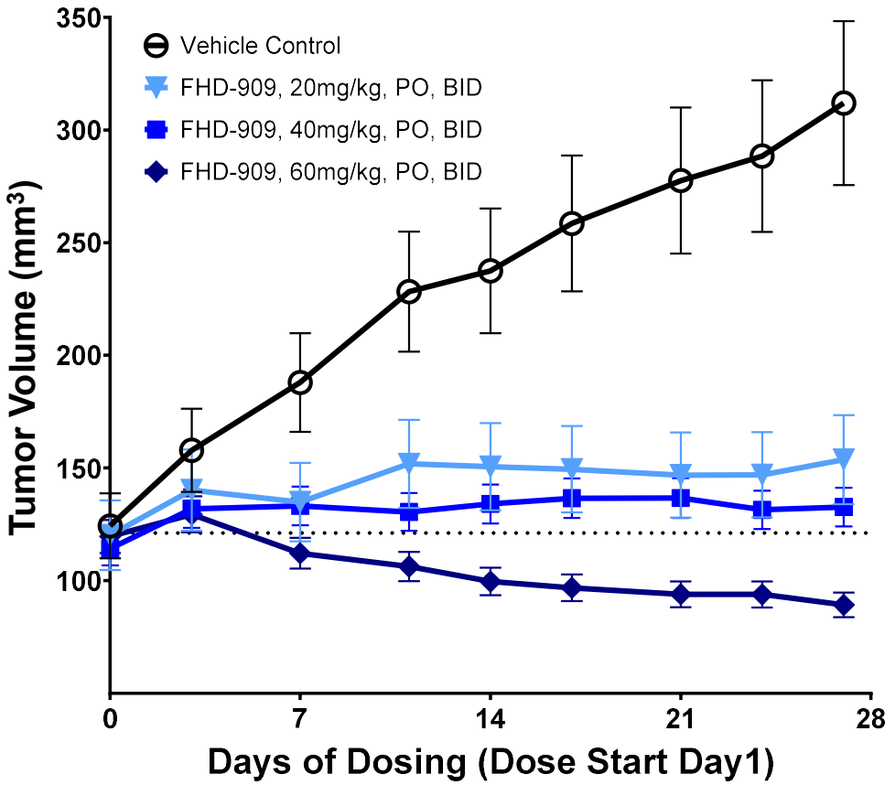
	Median IC_{50} (µM)
SMARCA4 WT	0.0932
SMARCA4 MUT	0.0028

Spread in potency for wild type versus mutant cell lines indicates

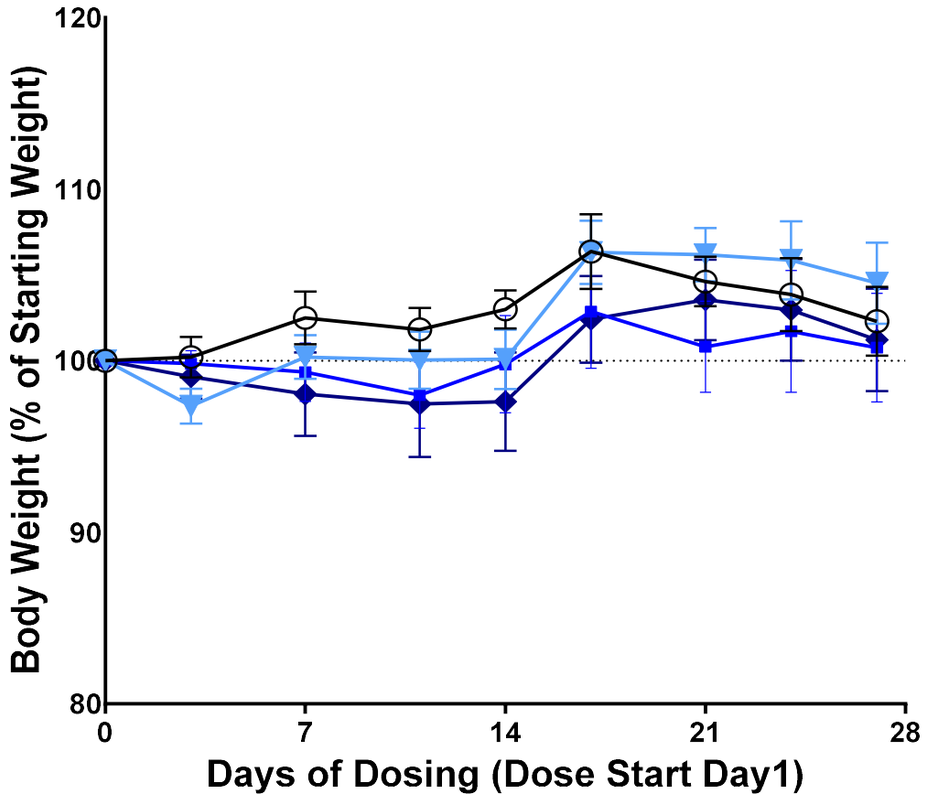
33-fold selectivity observed

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

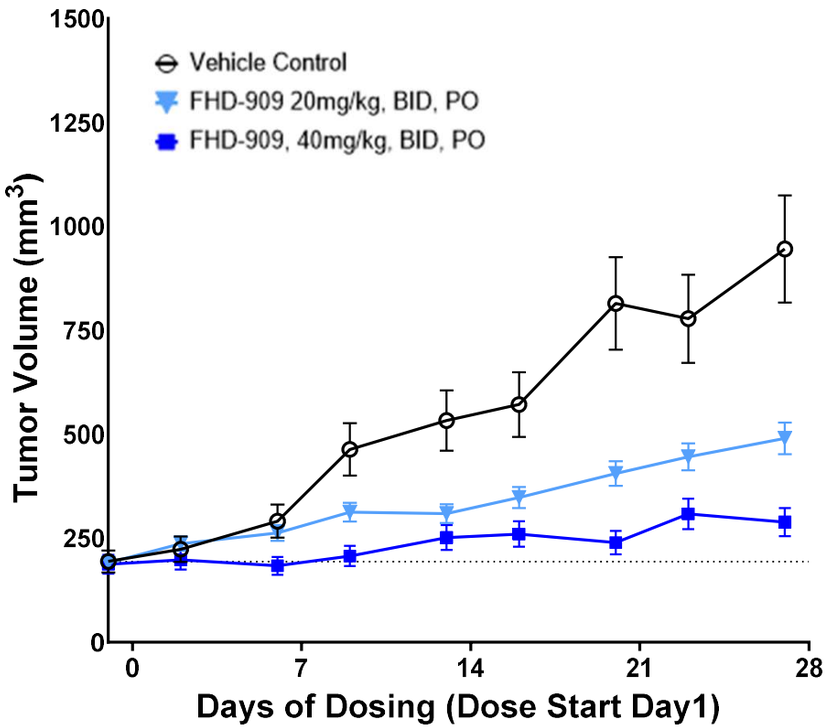


Genetic Background: SMARCA4 W764R, TP53 E62*, STK11-/-, CDKN2A-/-, KEAP1 R272C

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

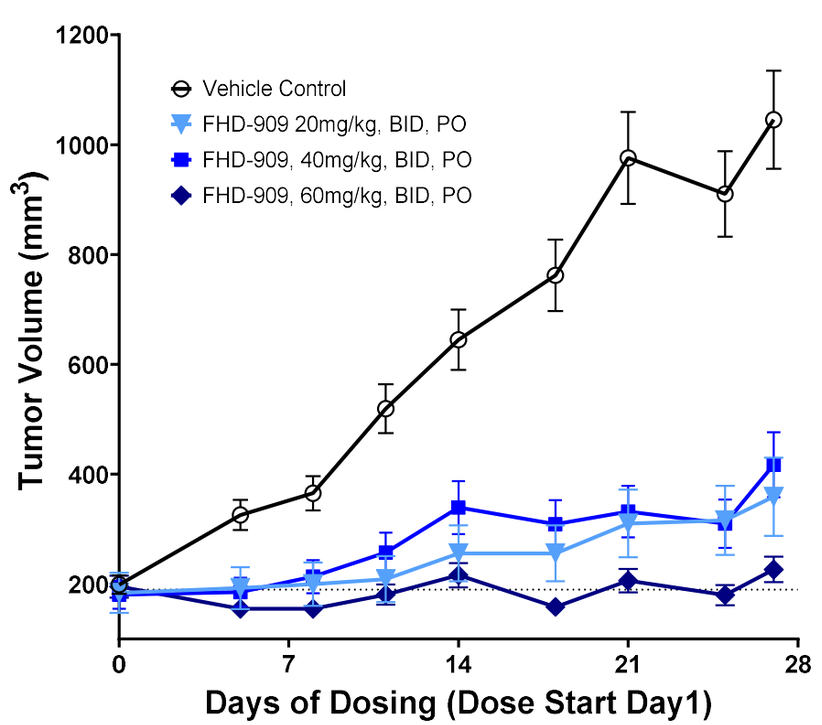
FHD-909 Monotherapy Demonstrated 96% TGI in A549 and Tumor Stasis in RERF-LC-AI Mutant NSCLC Models

A549 Model



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

RERF-LC-AI Model

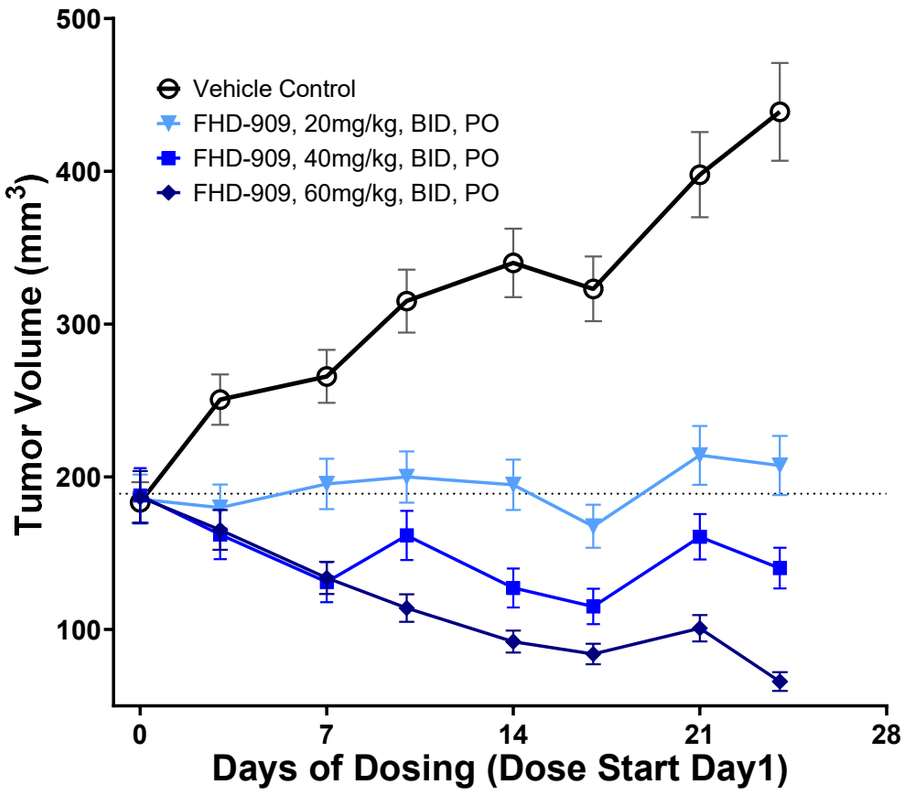


Genetic Background: SMARCA4 mut p.E1496*, TP53 p.Q104*, NF1 p.E1699*

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



- **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

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

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

FHD-909 (LY4050784) 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
- Enrolling in US and Japan

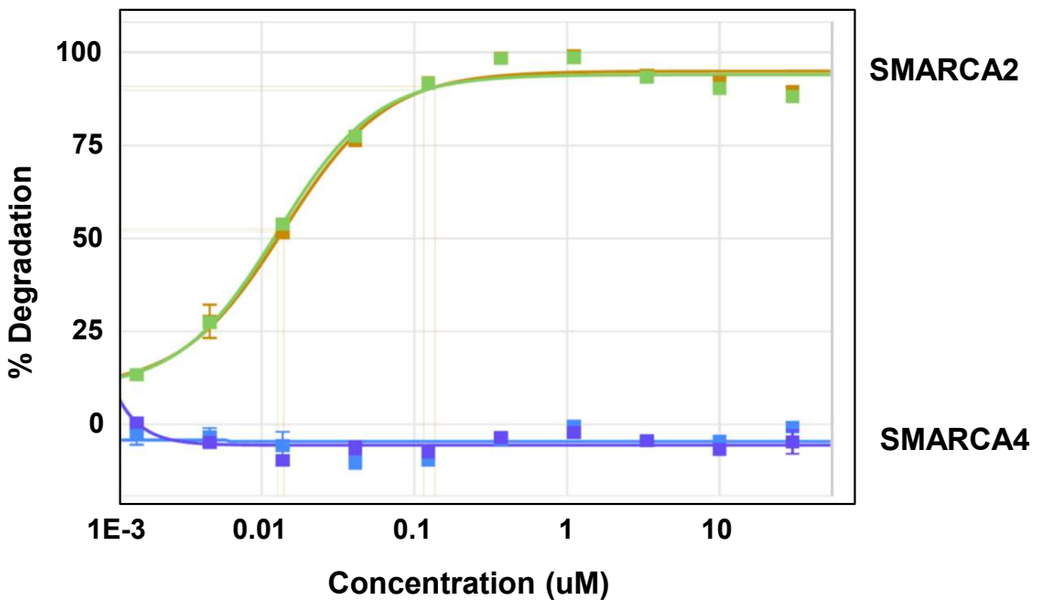
Dose Expansion

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

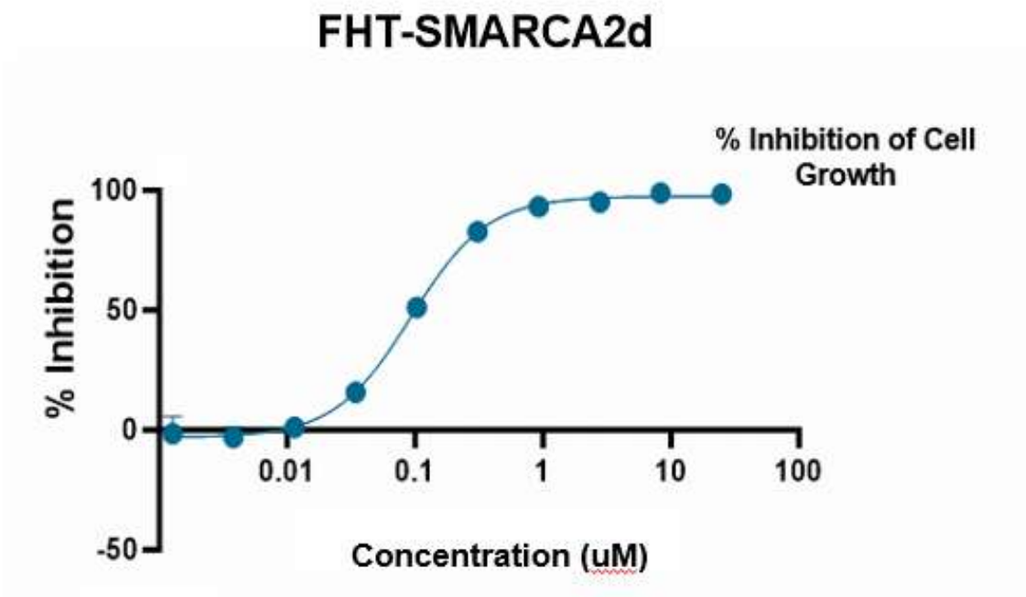
FHD-909 preclinical combination data with pembrolizumab, KRAS inhibitors and chemo to be presented at the AACR Annual Meeting, April 2025

Selective SMARCA2 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

NOTE: Data as of Q4 2021.

Selective CBP Protein Degrader

For EP300 Mutated Cancers

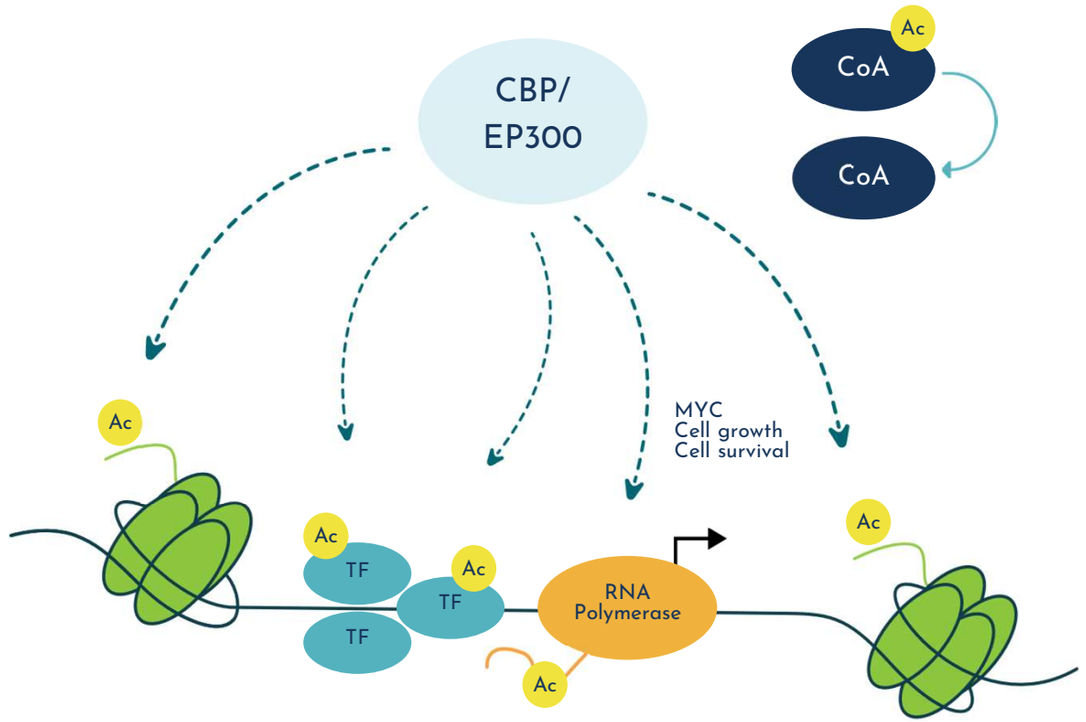
CBP and EP300 Proteins – A Decades Long Challenge in Selectivity

CBP and EP300 Biology

- CBP and EP300 are highly homologous, paralog histone acetyltransferases regulating enhancer-mediated transcription and protein stability
- Dysregulation of CBP and EP300 has been implicated in multiple cancers
- Dual targeting has revealed tolerability and safety issues

Foghorn's Solution... Highly Selective Degradation

- Achieved selective targeting which results in improved tolerability and efficacy
- Advancing two separate programs with defined dependencies and patient populations



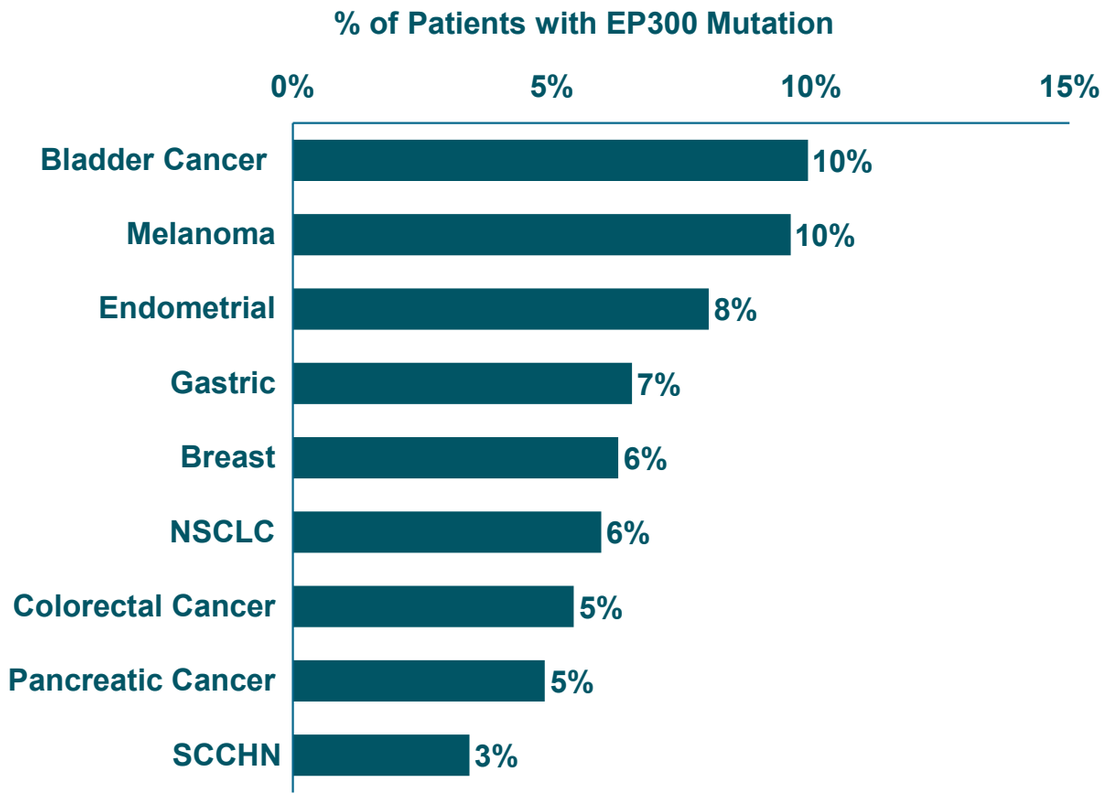
EP300 Degrader Approach
Focus on EP300 Lineage Dependent Cancers

CBP Degrader Approach
Focus on EP300 Mutant Cancers via Synthetic Lethality

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 / Next Milestone	• Preclinical • IND planned for 2026
New Patients Impacted / Year*	• Up to 10% of patients have an EP300 mutation across solid tumors representing ~ 100K addressable population
Key Differentiation	• Highly selective and potent • Increased tolerability relative to non-selective compounds • Long-acting formulation targets Q2-4W dosing • Compelling combination potential

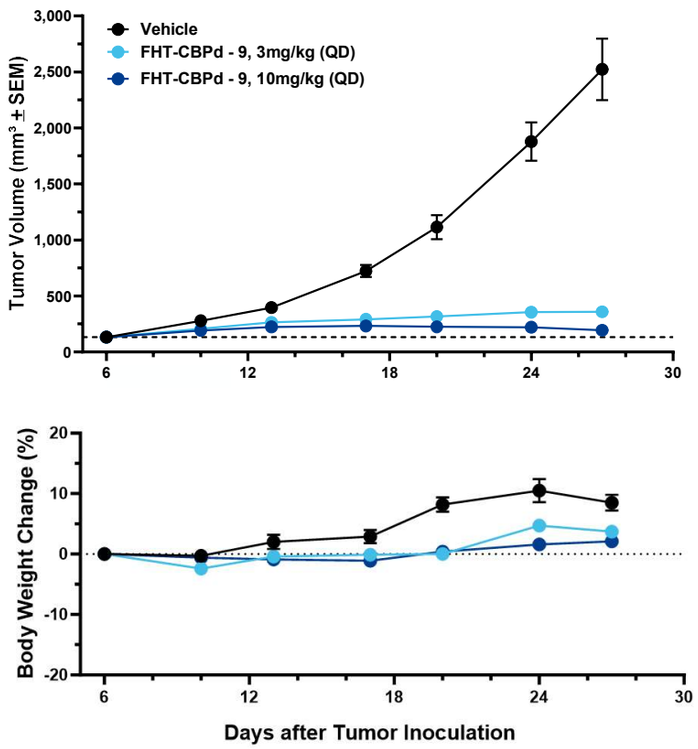
Commercial Opportunity



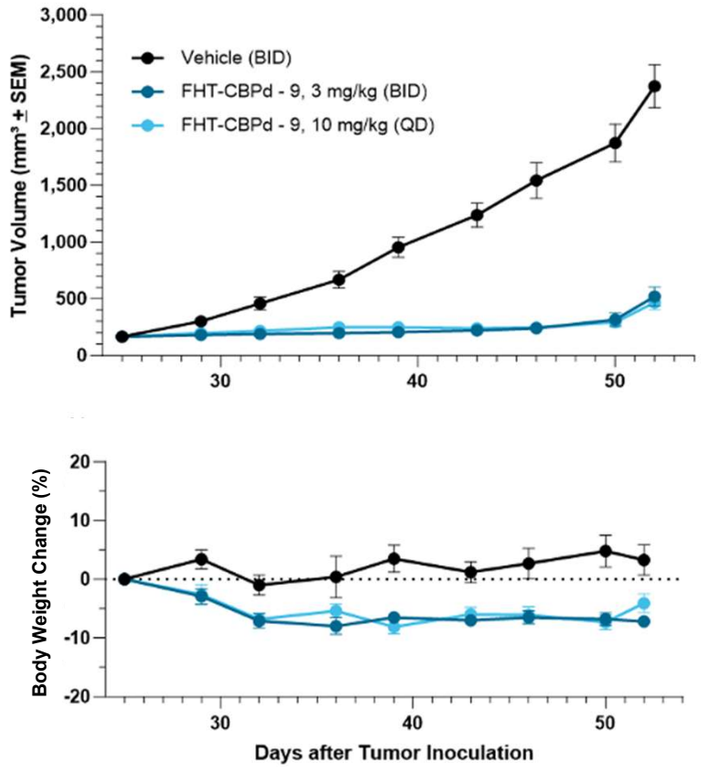
* *Per year incidence in the U.S., EU5, Japan . Source: Clarivate DRG Mature Markets Data.*

Selective CBP Degradation Results in Significant Anti-Tumor Activity in EP300mut Solid Tumor Models

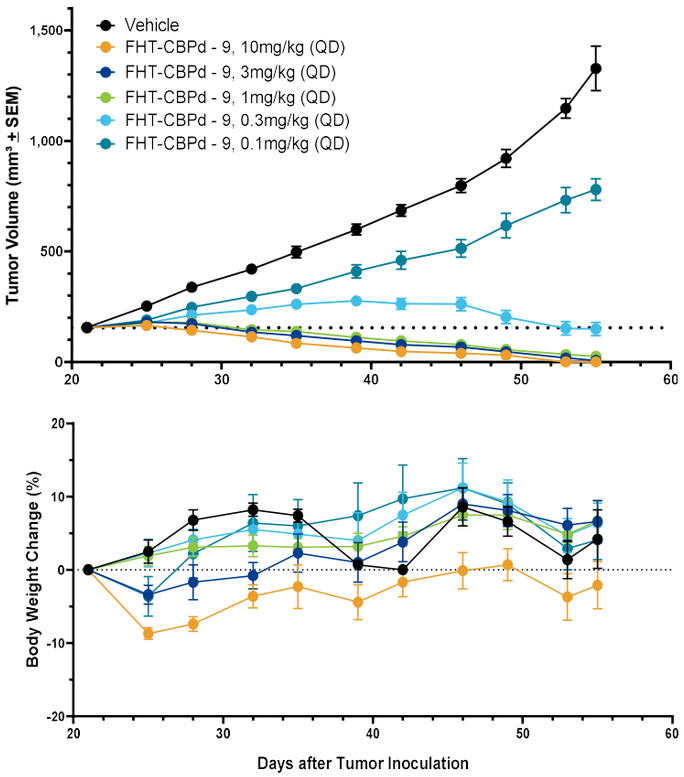
EP300mut Lung Cancer CDX (LU99)



EP300mut Bladder Cancer CDX (639V)



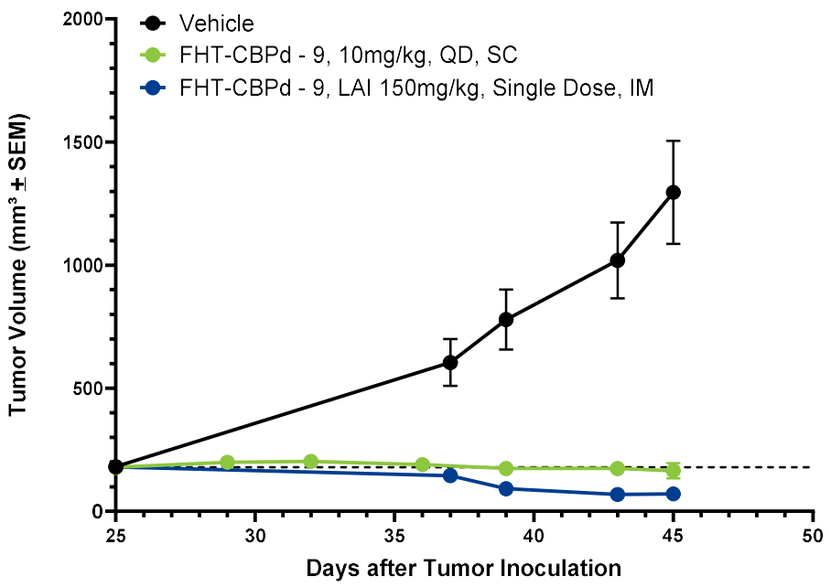
EP300mut Gastric Cancer CDX (AGS)



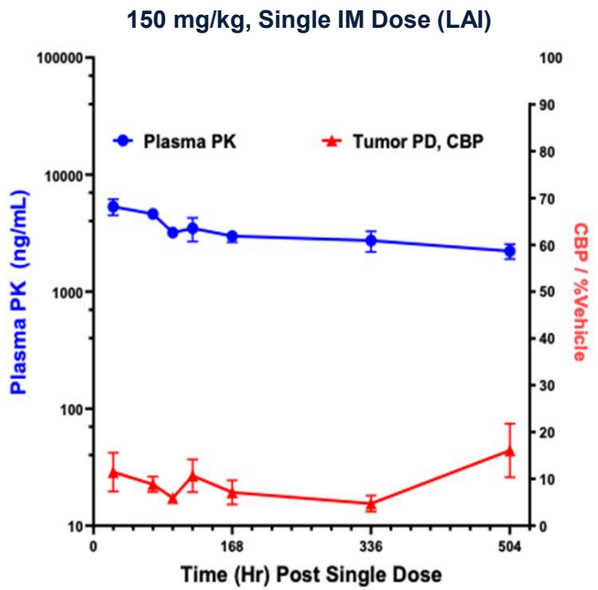
Degrader Selectivity		CBP	EP300	Fold Selectivity
FHT-CBPd-9	*DC_{50} @24h*	0.05 nM	64 nM	>1000x

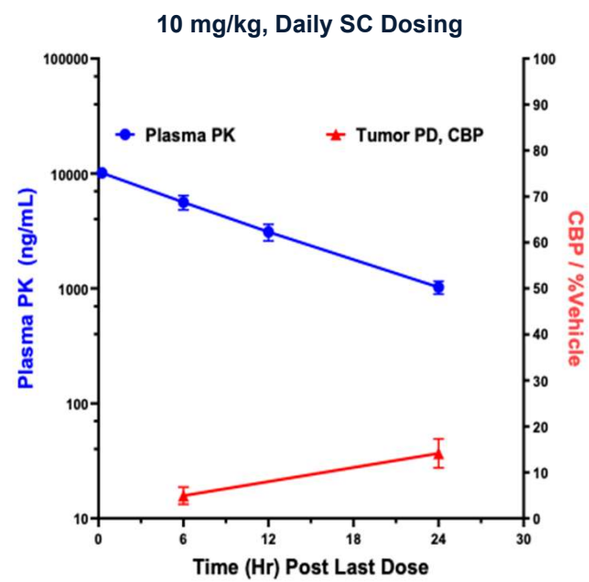
Long-Acting Injectable (LAI) Formulation Provides Sustained Target Coverage, Anti-Tumor Activity and Tolerability With a Single Injection

EP300mut (639V) Bladder CDX
(Daily Dosing and Single Injection of LAI Formulation)



Plasma PK and Tumor PD (CBP) Measured From Bladder CDX Efficacy

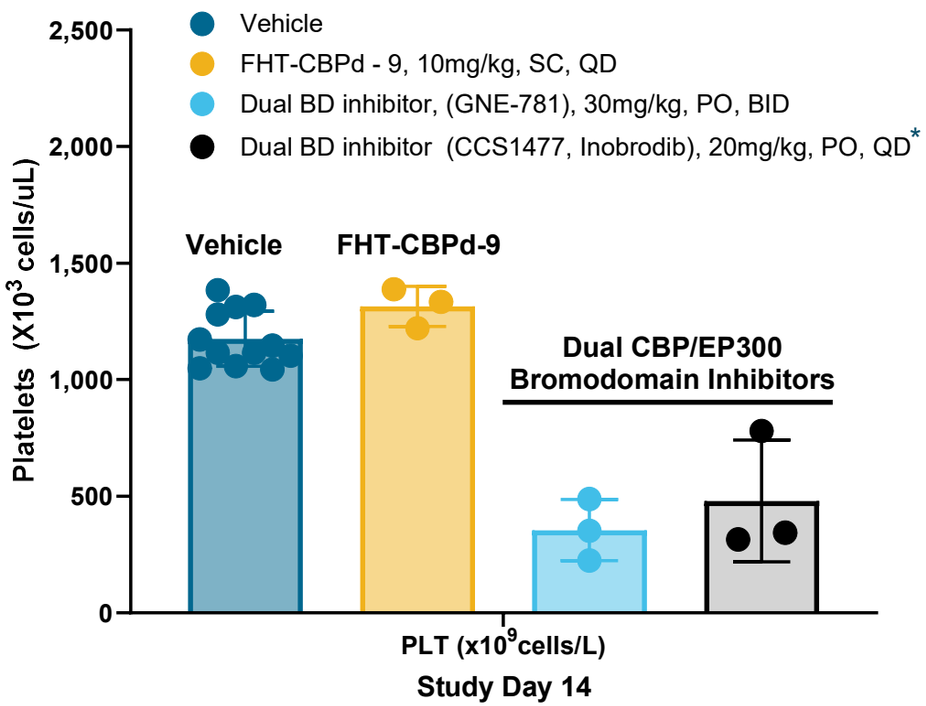




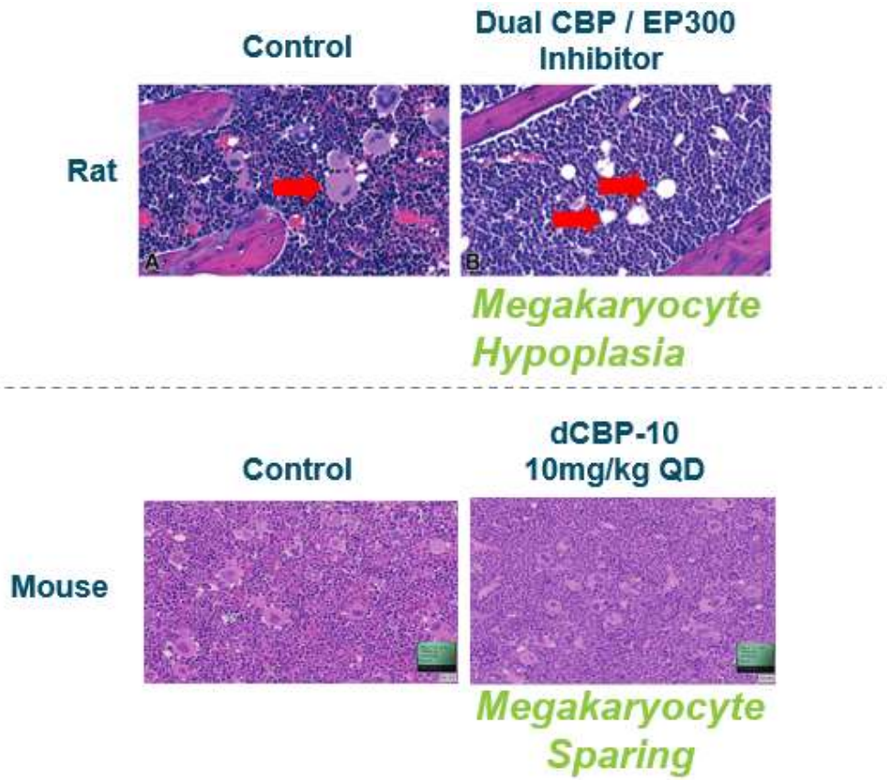
- Single injection of FHT-CBPd-9 in a LAI resulted in sustained plasma exposure (PK) and CBP degradation (PD) throughout the 3-week experiment, ultimately leading to tumor regressions
- No thrombocytopenia was observed through end of study

Preclinical Studies Indicate Selective CBP Degradation Did Not Show Thrombocytopenia and Spared Megakaryocytes *In Vivo*

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



Bone Marrow Staining
(In Vivo)



**Dual CBP/EP300 inhibition study used 3 weeks of dosing.*

Selective EP300 Protein Degrader

For CBP Mutated and EP300 Dependent Cancers

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

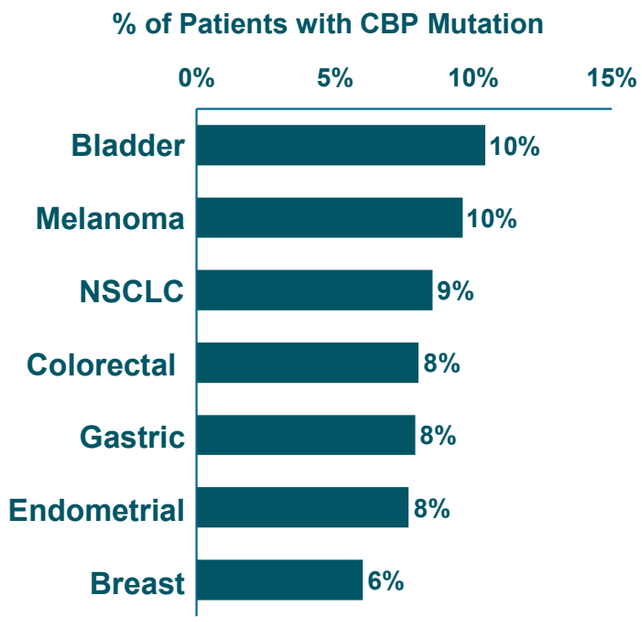
Target / Approach	• E1A binding protein p300 (EP300) • Targeted protein degrader
Indications	• AR+ Prostate • Broad range of heme malignancies • Bladder, melanoma, others
Mutation / Aberration	• EP300 dependent cancers • CBP mutant cancers
Stage / Next Milestone	• Preclinical • IND planned for 2026
New Patients Impacted / Year*	• Over 100,000
Key Differentiation	• Deeper efficacy response with selective degrader vs non-selective molecules • Improved tolerability profile vs non-selective molecules • Patient selection biomarker for Diffuse Large B-Cell Lymphoma (DLBCL)

Commercial Opportunity

EP300 Dependent Cancers

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

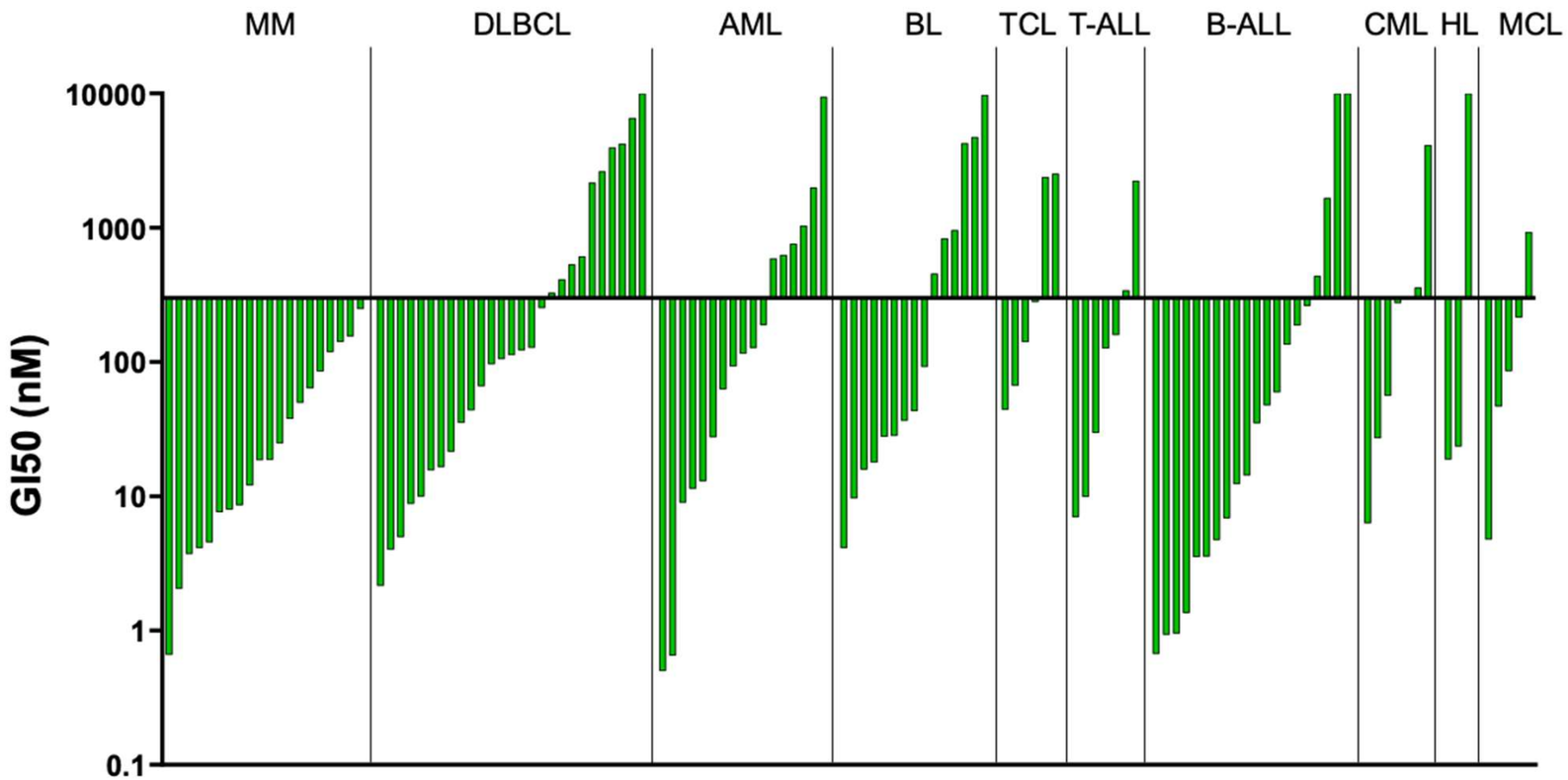
CBP Mutant Cancers



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

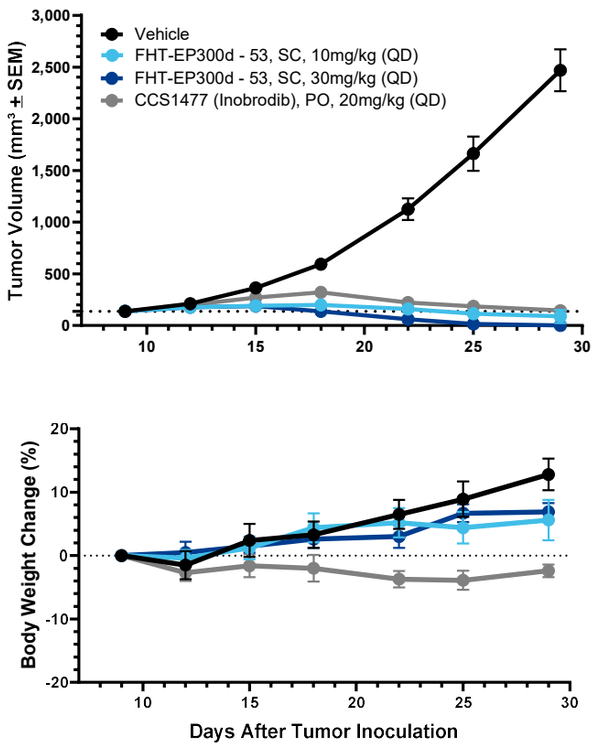
EP300 Degradation Shows Anti-Proliferative Activity in Broad Range of Hematological Malignancies

Anti-Tumor Activity Across Full Range of Heme Sub-Lineages
(~ 70% of All Tested Cell Lines are Sensitive)

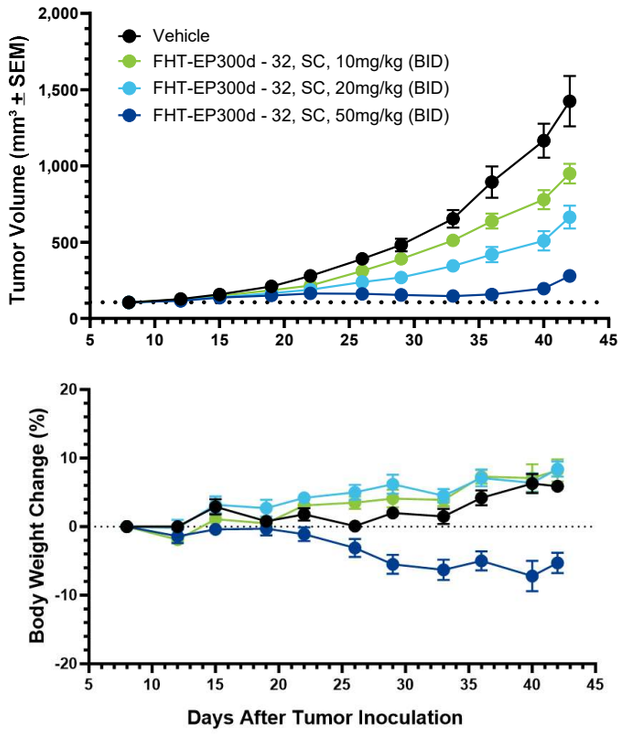


EP300 Degradation Results in Significant Tumor Growth Inhibition in Multiple Myeloma, DLBCL and Prostate Models

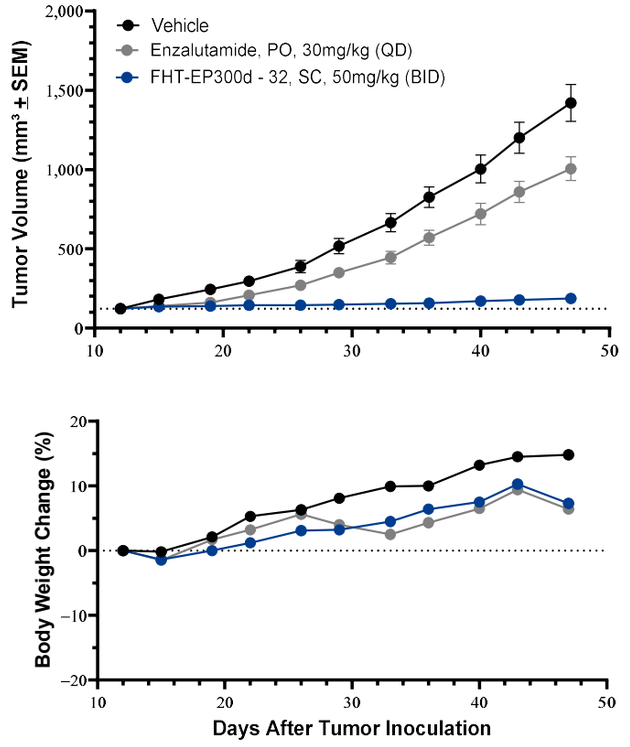
Multiple Myeloma (MM1S) CDX



DLBCL (KARPAS422) CDX



AR+ Prostate (VCaP) CDX

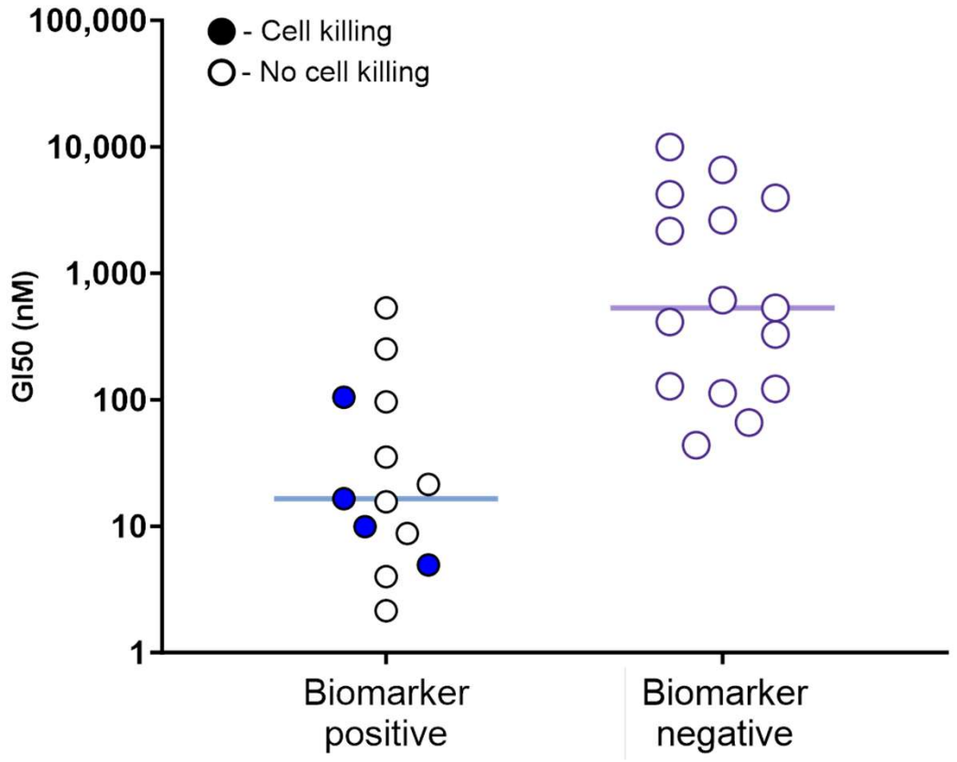


Degrader Selectivity		CBP	EP300	Fold Selectivity
FHT-EP300d-53	*DC_{50} @24h*	>1 uM	0.7 nM	>1000x

Degrader Selectivity		CBP	EP300	Fold Selectivity
FHT-EP300d-32	*DC_{50} @24h*	>1 uM	23 nM	>40x

Identification of a Patient Selection Biomarker in DLBCL

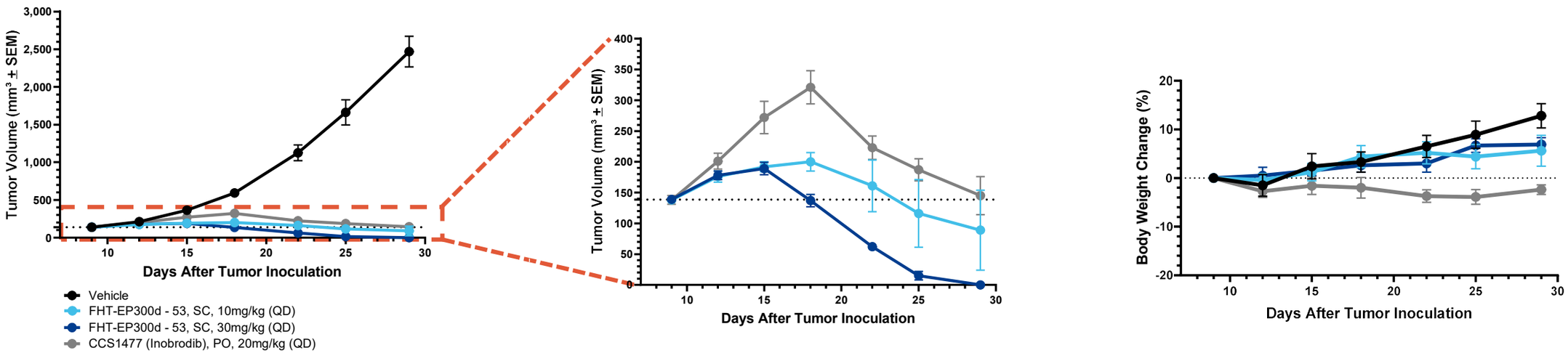
***In Vitro* Anti-Proliferation Screen Using Selective EP300 Degrader**



- Screened 27 DLBCL cell lines; ~60% are sensitive
- Two-step biomarker of sensitivity:
  - EP300 present (no high-impact mutations in EP300) and
  - One of two other mutations
- Mechanistic hypothesis being further validated

Selective EP300 Degrader Demonstrated Complete Response (Tumor Regression) in Multiple Myeloma Model

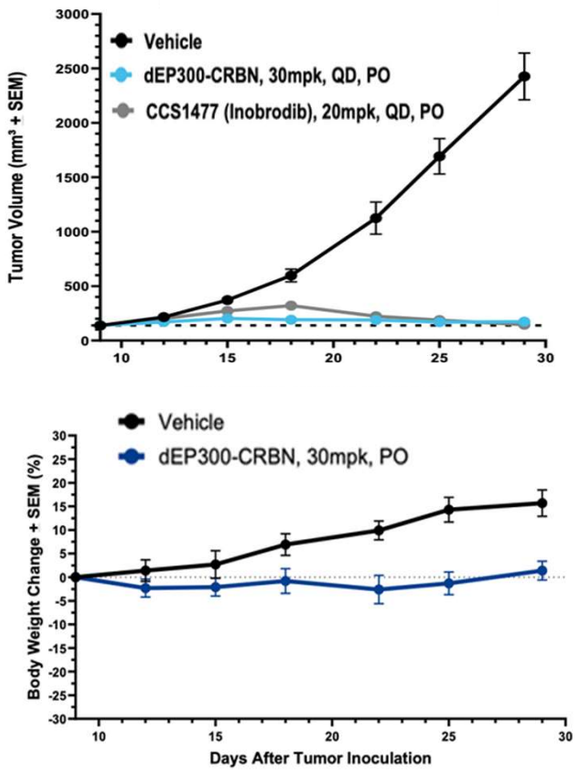
Multiple Myeloma CDX Treated With dEP300 – VHL

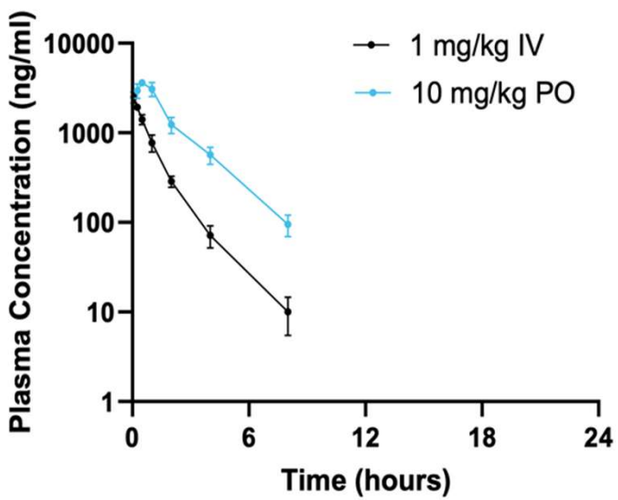


- **Non-selective dual CBP/EP300 inhibitor** shows tumor stasis, but clinical safety (i.e., **thrombocytopenia**) resulted in dosing holidays
- **Selective EP300 degrader** can achieve **deeper responses** (complete tumor regression) with **no thrombocytopenia**
- Selective EP300 degrader with improved therapeutic window enables **sustained target coverage and improved efficacy**

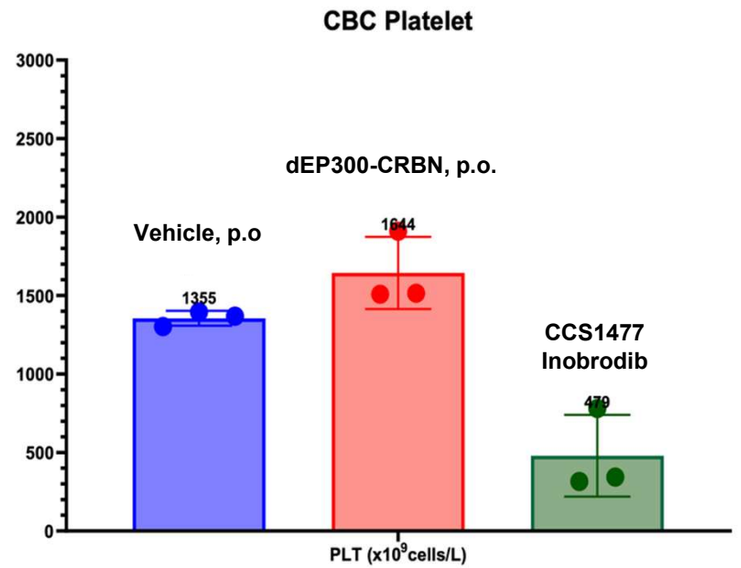
Oral EP300 Selective Degrader Shows Promising Efficacy; Well Tolerated With No Thrombocytopenia

Multiple Myeloma CDX Treated With dEP300 – CRBN





dEP300-CRBN demonstrates 32% oral bioavailability



Degrader Selectivity		CBP	EP300	Fold Selectivity
FHT-EP300d-CRBN	*DC50 @24h*	50 nM	0.2 nM	250x

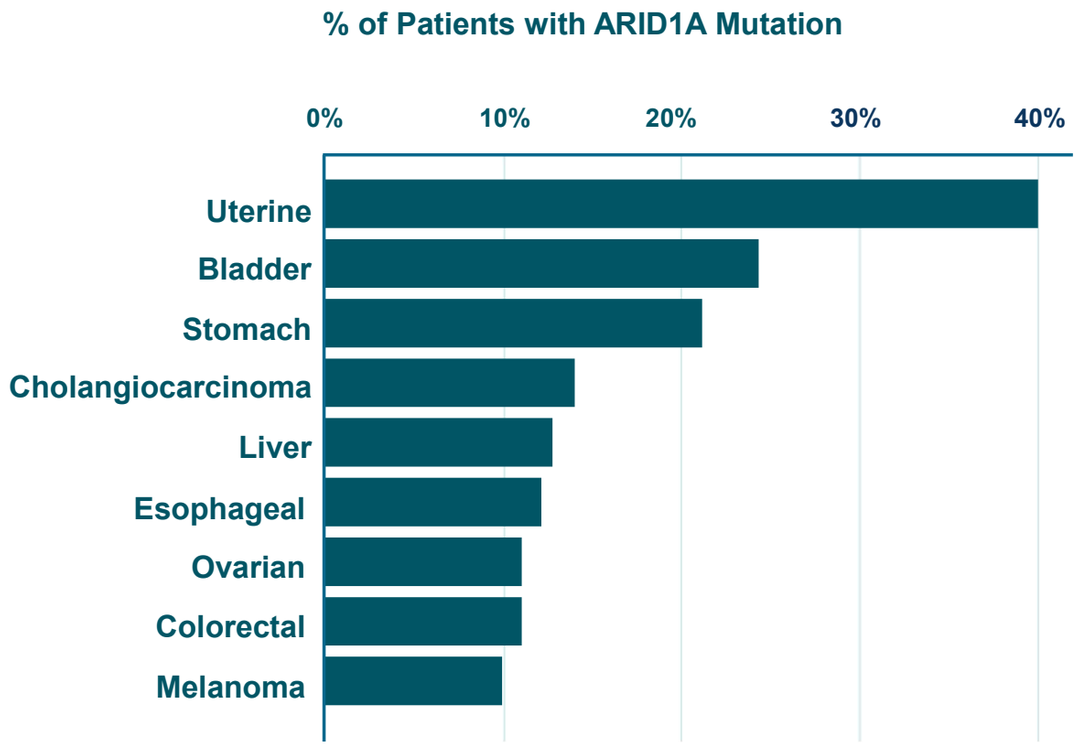
Selective ARID1B Protein Degrader

For ARID1A Mutated Cancers

ARID1B is a Major Synthetic Lethal Target With Potential in Up To 5% of All Solid Tumors; Degradation Achieved, Program Update in 2025

Target / Approach	• ARID1B • Targeted protein degrader
Initial Indication	• ARID1A mutated cancers (e.g. ovarian, endometrial, colorectal, bladder and other cancers)
Mutation / Aberration	• ARID1A mutations
Stage / Next Milestone	• Preclinical • Program update in 2025
New Patients Impacted / Year*	• ARID1A is one of the most mutated protein in cancers (~ 5% of all solid tumors) representing > 175K addressable patients across solid tumors
Key Differentiation	• Multiple ARID1B binders with nM affinity and selectivity • ARID1B degradation achieved

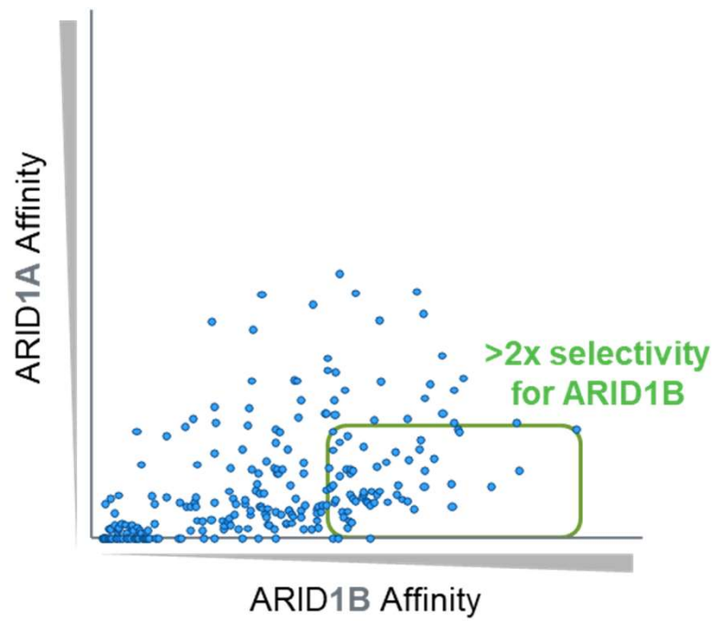
Commercial Opportunity



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

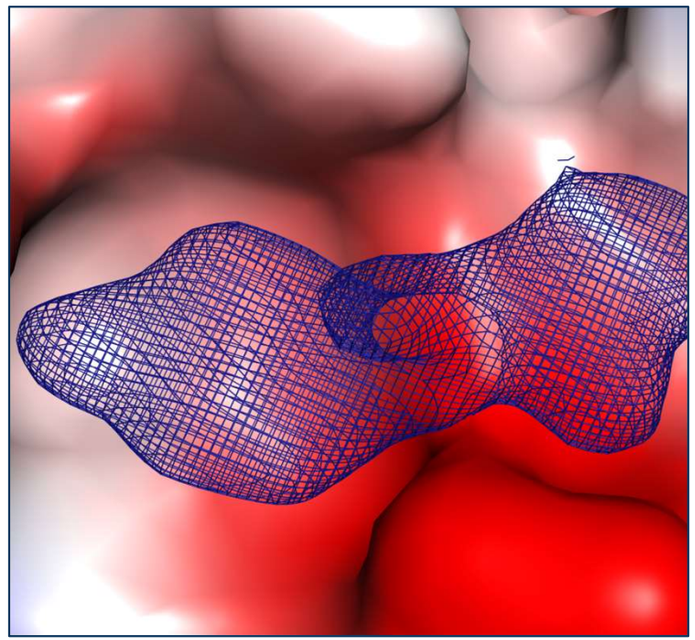
Compound Screening and Structure-Based Optimization Yielded Selective ARID1B Binders

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

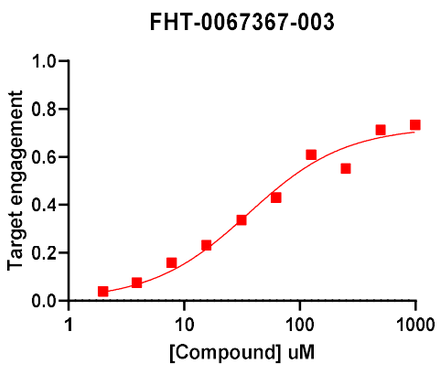


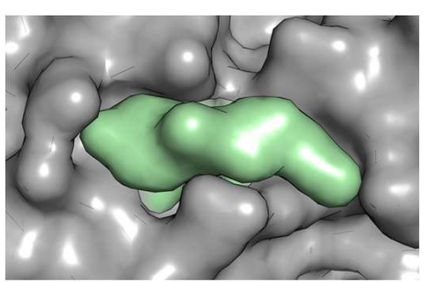
- Determined X-ray crystal structure of ARID ligandable domains with specific binders
- Leveraged these structures to drive binding affinities and expand binding chemotypes

Structure-Based Optimization Drove Improved ARID1B Binding Affinity From 100 uM to Less Than 200 nM

Gen 1: Screening Hit

ARIDb-1
ARID1B Kd: **100 uM**

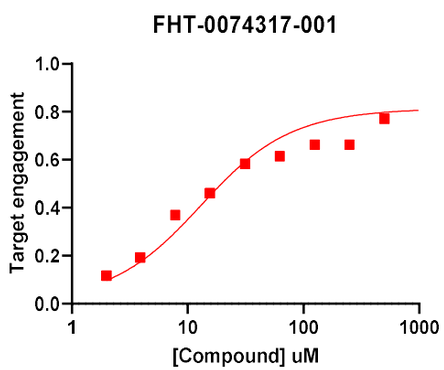


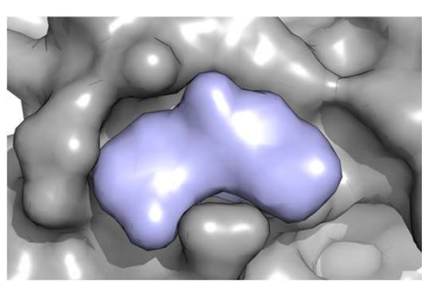


1.4 Å co-xtal structure

Gen 2: Early Optimization

ARIDb-2
ARID1B Kd: **15 uM**

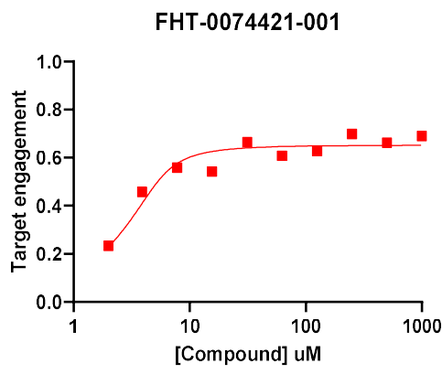


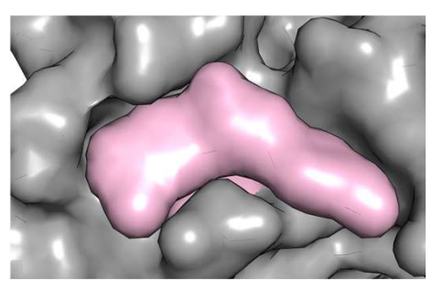


2.0 Å soak structure

Gen 3: Sub-uM Affinity

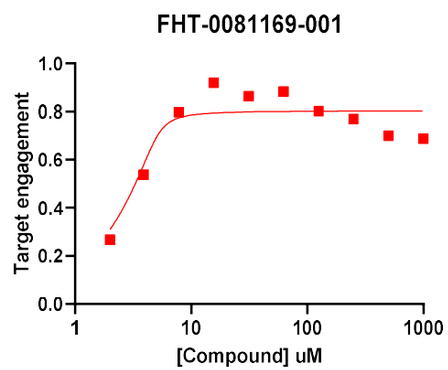
ARIDb-3
ARID1B Kd: **0.5 uM**

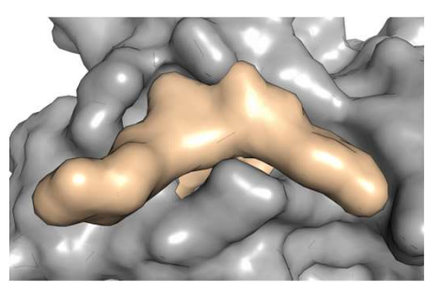




1.9 Å co-xtal structure

ARIDb-9
ARID1B Kd: **0.2 uM**



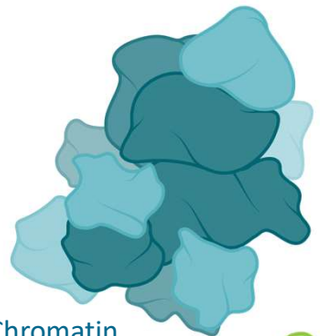


1.7 Å soak structure

Drugging Transcription Factors

Multiple Approaches

Foghorn's Transcription Factor Platform: 4 Approaches



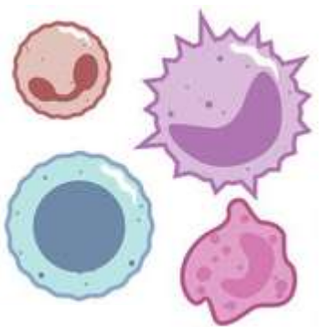
Chromatin Remodeler

Transcription Factor

Modulating TF activity via Protein-Protein Interaction Disruptors

Degrading TFs via recruitment of the native E3 ligase using molecular glue

= MG (Molecular Glue)

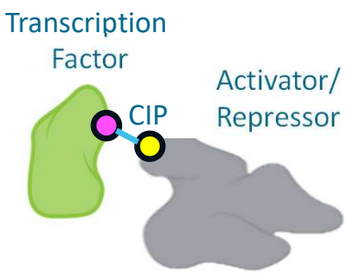


Disease relevant cell lines (change image – copyright?)

Transcription Factor

Leveraging Phenotypic Screening to reduce TF levels in disease relevant settings

Re-wiring transcription through Induced Proximity with TFs



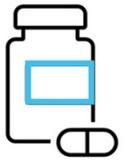
= CIP (Chemical Inducer of Proximity)

Multiple Near-Term Value Inflection Points Through 2026

FHD-909 (LY4050784) (Selective SMARCA2 Inhibitor)	Preclinical Combination Data	April 2025 (AACR)
	Phase 1 Dose Escalation Data	Confidential
Selective SMARCA2 Degrader	IND / Phase 1 Initiation	Confidential
Selective CBP Degrader	IND / Phase 1 Initiation	2026
Lilly Target #2	Target Disclosure and IND	Confidential
Selective EP300 Degrader	IND / Phase 1 Initiation	2026
Selective ARID1B Degrader	Selective Degradation Achieved	Program Update 2025

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

$243.8 million in cash and equivalents (unaudited)

(as of 12/31/2024)

Cash runway into 2027

Shares outstanding: approximately 62.5M*

Value Drivers

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

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

Protein degrader platform with expansion into induced proximity

Major Strategic Collaboration

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

*Includes common shares outstanding and pre-funded warrants as of 12/31/2024.

FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

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