UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): May 6, 2024

Foghorn Therapeutics Inc.

(Exact name of registrant as specified in its charter)

Delaware	**001-39634**	**47-5271393**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

500 Technology Square, Ste 700
Cambridge, MA **02139**
(Address of principal executive offices) (Zip Code)

(Registrant's telephone number, including area code): (617) 586-3100

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.0001 par value per share	**FHTX**	**The Nasdaq Global Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01 Other Events.

On May 6, 2024, the Company posted a presentation to its website which it intends to use in meetings with investors.

A copy of the presentation is attached hereto as Exhibit 99.1 and is incorporated by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Investor Presentation dated May 6, 2024

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

FOGHORN THERAPEUTICS INC.

By: /s/ Kristian Humer
Kristian Humer
Chief Financial Officer

Date: May 6, 2024

Exhibit 99.1

FOGHORN®
THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

May

Forward Looking Statements

This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on informa currently available to management. All statements other than statements of historical facts contained in this presentation are forwa looking statements. In some cases, you can identify forward-looking statements by terms such as “could,” “may,” “might,” “will,” “like “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” or the negative of these terms or o similar expressions, although not all forward-looking statements contain these words. Forward-looking statements include, but are limited to, statements concerning: the potential outcomes from our collaboration agreement with Lilly; the initiation, timing, progress results of our research and development programs and pre-clinical studies and clinical trials, including with respect to our Phase 1 st of FHD-286 in combination with decitabine or cytarabine in relapsed and/or refractory AML patients and anticipated timing of releas clinical data, and the planned Phase 1 dose escalation study of FHD-909 with Loxo@Lilly; our ability to advance product candidates we may develop and to successfully complete preclinical and clinical studies; our ability to leverage our initial programs to deve additional product candidates using our Gene Traffic Control Platform®; the impact of exogeneous factors, including macroeconomic geopolitical circumstances, on our and our collaborators’ business operations, including our research and development programs pre-clinical studies; developments related to our competitors and our industry; our ability to expand the target populations of our progra and the availability of patients for clinical testing; our ability to obtain regulatory approval for FHD- 286 and any future product candida from the FDA and other regulatory authorities; our ability to identify and enter into future license agreements and collaborations; our ab to continue to rely on our CDMOs and CROs for our manufacturing and research needs; regulatory developments in the United Sta and foreign countries; our ability to attract and retain key scientific and management personnel; the scope of protection we are abl establish, maintain and enforce for intellectual property rights covering FHD-286, our future products and our Gene Traffic Cor Platform; and our use of proceeds from capital-raising transactions, estimates of our expenses, capital requirements, and needs additional financing. You should, therefore, not rely on these forward-looking statements as representing our views as of any d subsequent to the date of this presentation. Additional important factors to be considered in connection with forward-looking stateme are described in the Company's filings with the Securities and Exchange Commission, including withing the section entitled "Risk Fact in the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 2023. Any forward-looking statements repres the Company’s views only as of the date of this presentation and should not be relied upon as representing its views as of subsequent date. The Company explicitly disclaims any obligation to update any forward-looking statements. The Company’s busines subject to substantial risks and uncertainties.

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

$206.7 million in cash and equivalents

(as of 3/31/2024)

Provides **runway into H1'26**

Value Drivers

Anticipate data from the Phase 1 study of FHD-286 in combination with decitabine in **H2'24**

BRM Selective Inhibitor (FHD-909), partnered with Loxo@Lilly, **IND submitted to FDA, Phase 1 initiation anticipated in H2'24**

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

Major Strategic Collaboration

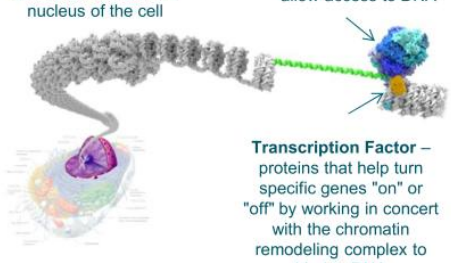
Strategic collaboration wi Loxo@Lilly; **$380 millio upfront;** 50/50 U.S. econo split on two lead program

Unique Insights into Chromatin Biology to Prosecute Untapped Area for Novel Targets and Therapeutics

Chromatin Regulatory System Critical for Gene Expression

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

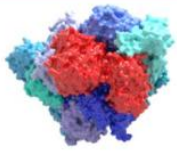
Chromatin Remodeling Complex – specialized multiprotein machineries that allow access to DNA



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

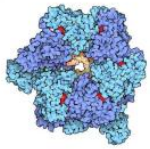
Novel Targets Guided by Genetic Dependencies

Chromatin Remodeling Complex Mutations / Overexpression



Transcription Factor Mutations / Overexpression

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

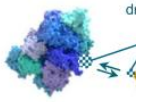


Tailored Drugging Approaches

Enzymatic Inhibito
Highly selective and allo
small molecule inhibit

Targeted Protein Degradation
Molecular glue and bi-functional protein degraders

Transcription Factor Disruptors
Disrupt interactions between chromatin remodeling complexes and transcription factors



Foghorn's Validated Gene Traffic Control© Platform Enables an Integrated, Scalable, Efficient and Repeatable Paradigm

Targeting Disease

Deep mechanistic understanding of the chromatin regulatory system

What to Drug:

Identify disease dependencies with novel targets

Specialized Approach

Biochemistry, biophysics and assays of large complexes and proteins

Where to Drug:

Engineer selectivity via unique assays and protein capabilities

Selective Therapeutics

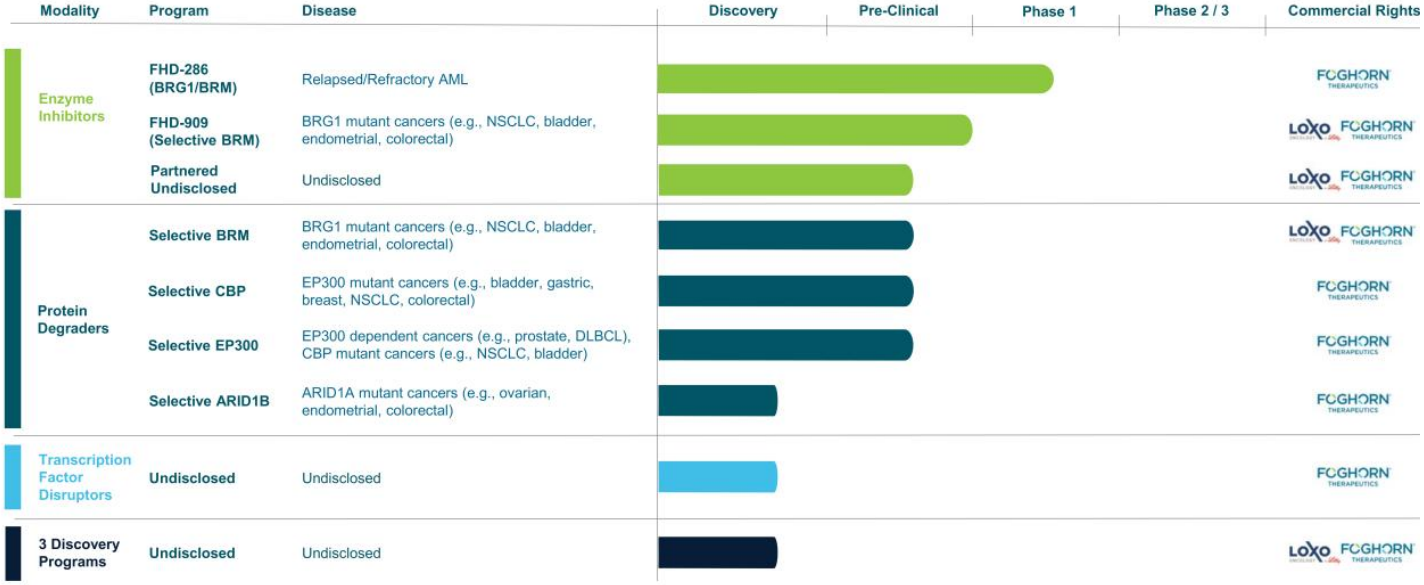
Biology first, small molecule modality agnostic

How to Drug:

Small molecules, degrader and delivery platform

Enzymatic Inhibitors	Targeted Protein Degraders	Transcriptic Factor Disruptors

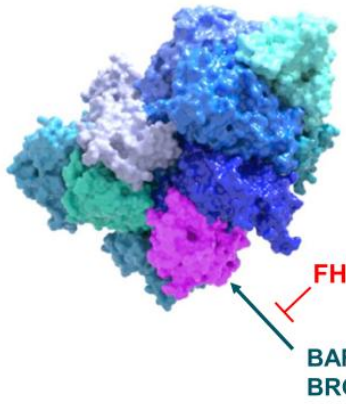
Foghorn Is Advancing a Pipeline of First-in-Class Precision Therapeutics with Potential for Broad Application in Oncology



FHD-286: Dual BRM/BRG1 Inhibition

Targeting BAF Dependency in Cancer

Exploring BAF Dependency in Cancer with FHD-286 – Potent, Small Molecule Inhibitor Targeting BRM and BRG1



FHD-286:

- **Allosteric modulation inhibiting the activity of both BRM and BRG1**
- **Oral, daily, potent small molecule inhibitor**

Current and Potential Future Opportunity

Mutations	Pre-clinical data support ability to address BAF mutated cancers (e.g., BRG1 mutant)
Differentiation	Clinical and pre-clinical data demonstrate 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 demonstrate an increase of CD8+ T-cells and a reduction of T-regulatory cells

First-in-Class Broad-Based 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 event 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 Study

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

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

Initial FHD-286 Opportunity

Most cases of AML are not curable

- More than half of patients will relapse post frontline treatment
- Intensive chemotherapy has been standard of care for four decades with no meaningful improvement

40% of AML cases have no actionable mutations

- No meaningful developments for the broad AML patient population since the approval of Venetoclax
- Recent development has focused predominantly on AML subsets harboring actionable mutations – FLT3, IDH1/2, and MLL**

~17,000 Drug Treatable R/R Patient

- Post Ven/Aza, treatment options are limit – CRc rates 15-17%
- Mortality remains high for this population, mOS ~3mo
- Patients with actionable mutations who relapse post targeted therapy have high unmet need

FHD-286 could provide a meaningful opportunity to improve outcomes in the R/R setting. We believe there is an additional opportunity in the newly diagnosed setting.

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

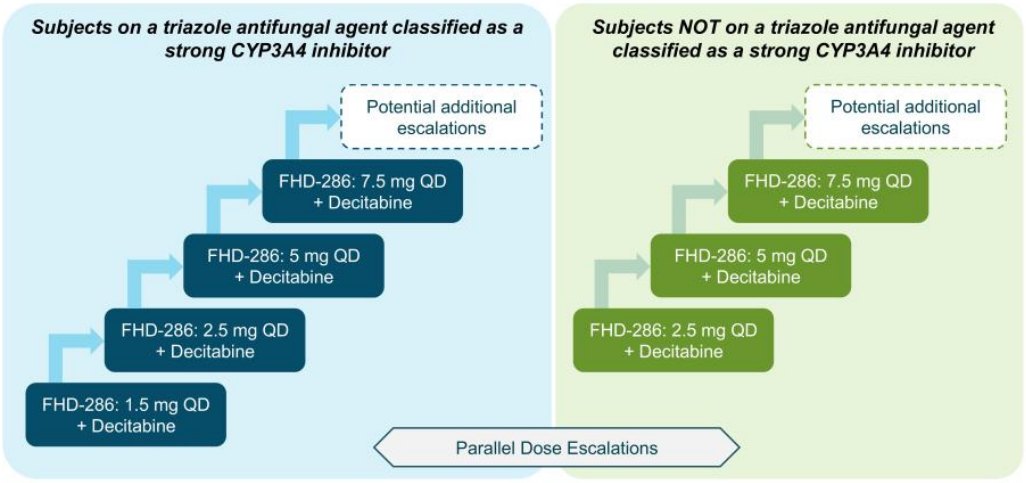
Study Design for FHD-286 Phase 1 Multicenter Dose-Escalation in Combination with Decitabine in AML

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

FHD-286 Demonstrated Differentiation Across a Broad Range of Genetic Backgrounds in 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%)

CD34 (leuken cell marker) decreases

CD11b (marker of differentiation) increases

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

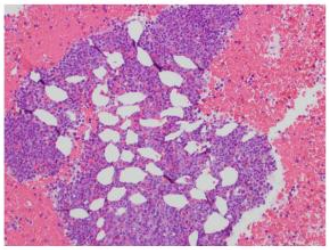
Prior non-AML treatment:

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

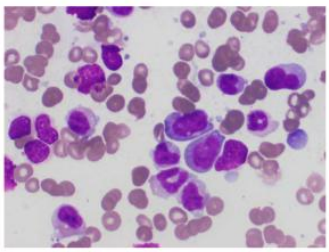
Initiation of FHD-286 at 10 MG Dose:

- 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 Trial

Patient Background:

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

Prior AML Treatment:

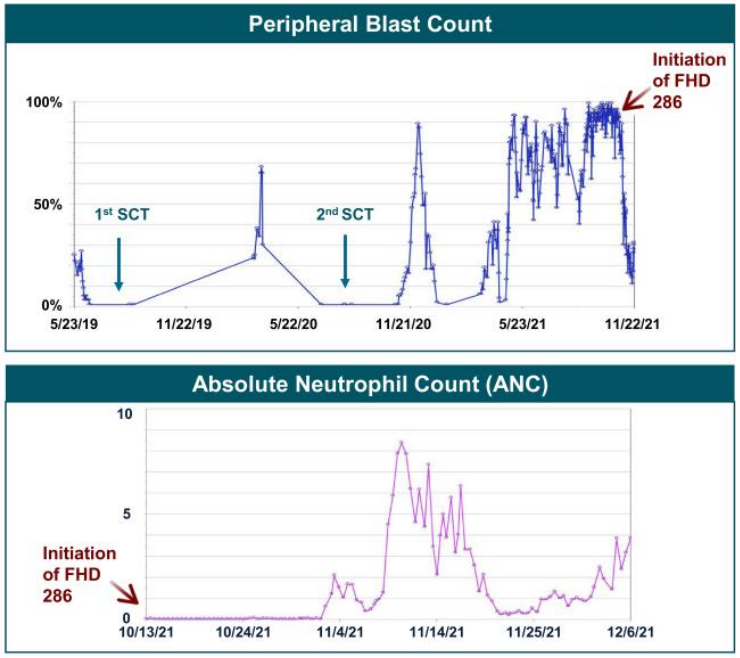
- Progressive disease with CNS Leukemia: 7 lines prior treatment and 2 bone marrow transplants

Prior non-AML treatment:

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

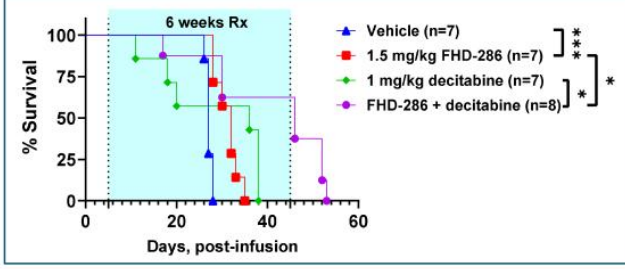
Initiation of FHD-286 at 10 MG Dose:

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

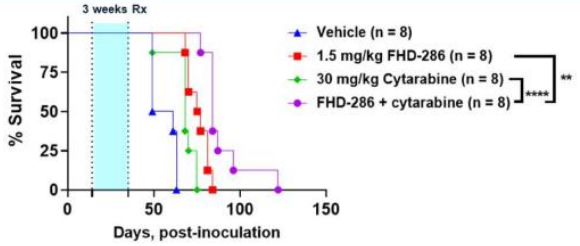


Pre-Clinical Data Demonstrated Combination Potential with Multiple Agents in AML

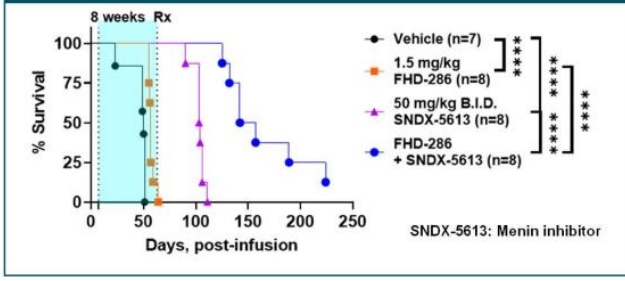
FHD-286 + decitabine
(MLL-AFP + FLT3 TKD Luc/GFP PDX)



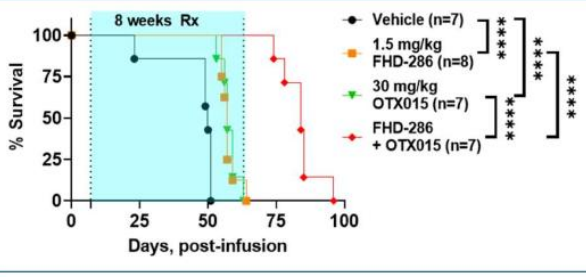
FHD-286 + cytarabine
(MV4, 11 FLT3 ITD CDX)



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



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



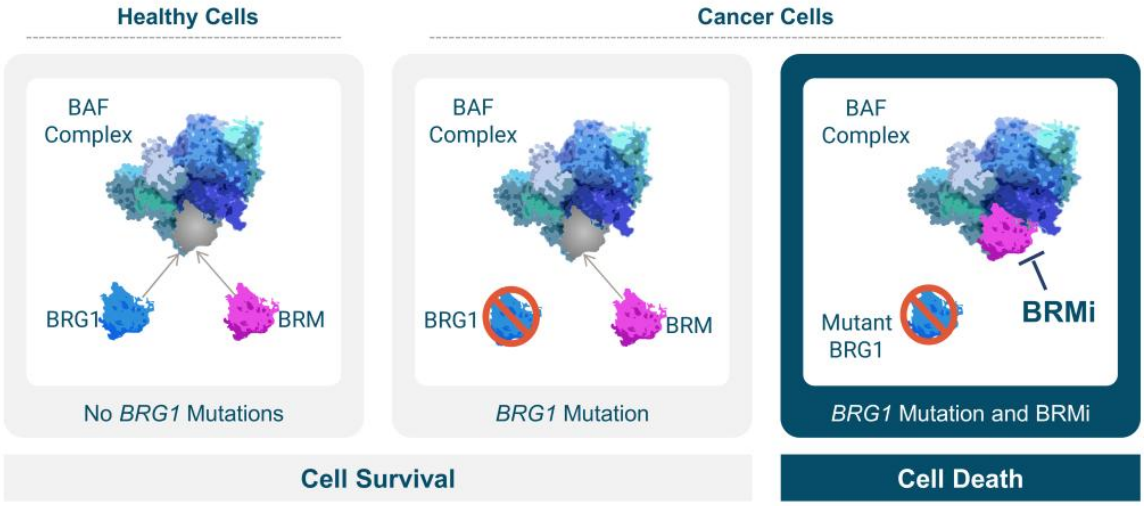
Selective BRM Modulators

For BRG1 Mutated Cancers

BRM Selective Inhibitor FHD-909 IND Submitted in Q2'24, BRM Selective Degrader Continues Late-Stage Pre-Clinical Development

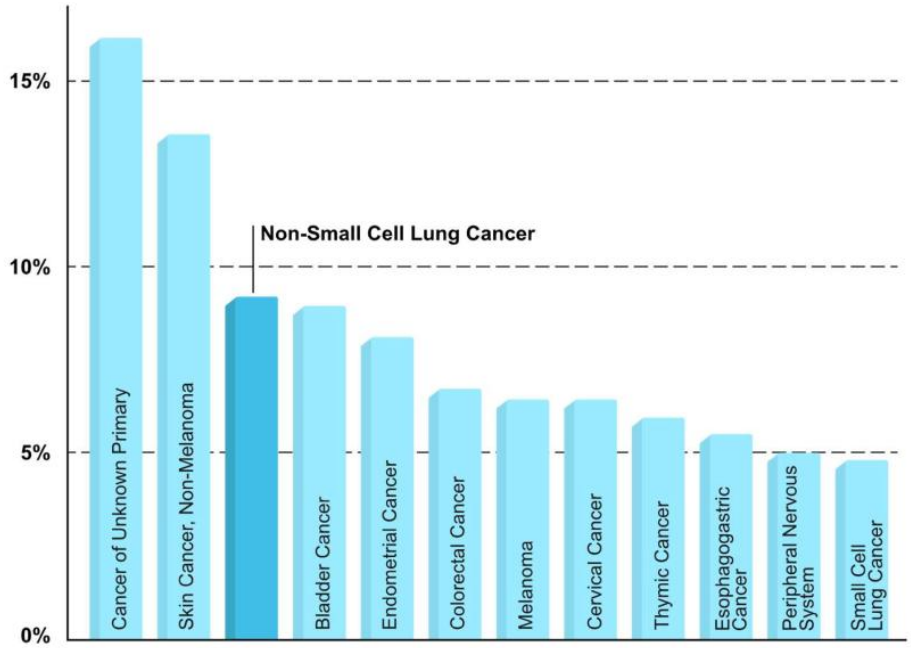
	BRM Selective Inhibitor (FHD-909)	BRM Selective Degrader
Biology	Exploit the synthetic lethal relationship between BRM (SMARCA2) and mutated BRG1 (SMARCA4)	
Stage	IND submitted in Q2'24	Advancing in parallel through late pre-clinical development
Opportunity	BRG1 mutated cancer including ~10% of NSCLC and up to 5% of all solid tumors	
Loxo@Lilly Partnership	50/50 global R&D cost share | 50/50 U.S. economics | tiered ex-U.S. royalties startin in the low double-digit range and escalating into the twenties	

BRM Selective Inhibition is a Promising Strategy to Exploit the Synthetic Lethal Relationship Between BRM and Mutated BRG1



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

BRG1 is Mutated in Up to 10% of NSCLC; Up to 5% of Solid Tumors



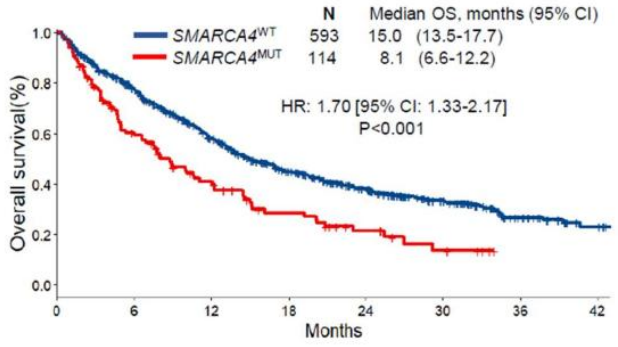
BRG1 mutated across a broad range of tumors

Accounts for ~5% of solid tumors

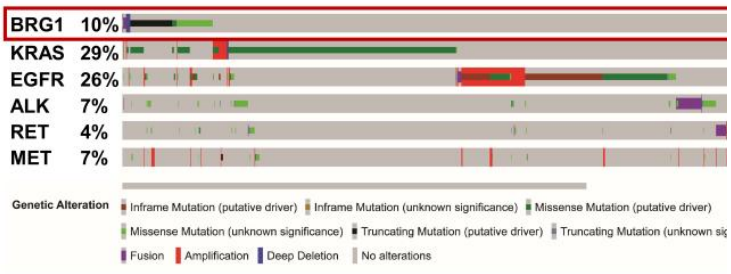
AACR GENIE via cBioPortal

Patients with NSCLC Harboring BRG1 Mutations Have Significantly Worse Clinical Outcomes and Define a High Unmet Need Patient Population

Overall Survival for SMARCA4wt vs SMARCA4mut[1]

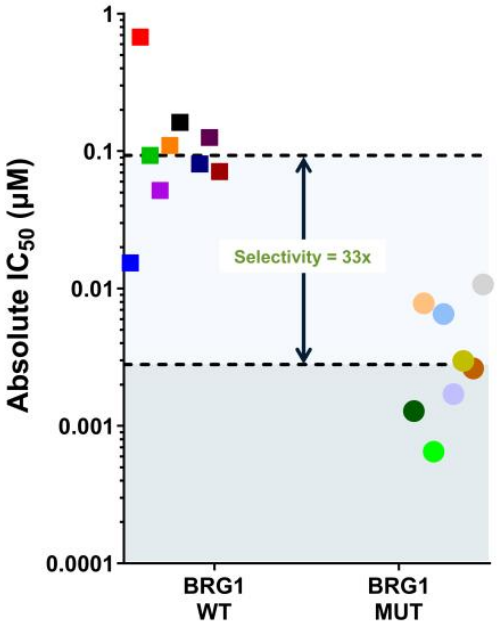


BRG1 mutated in up to 10% of NSCLC tumors, minimal overlap with other mutations[2]



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

FHD-909 Demonstrated Approximately 30-fold Selectivity Across 17 BRG1 (SMARCA4) Mutant and Wild-Type Cell Lines



BRG1 (WT)	BRG1 (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	

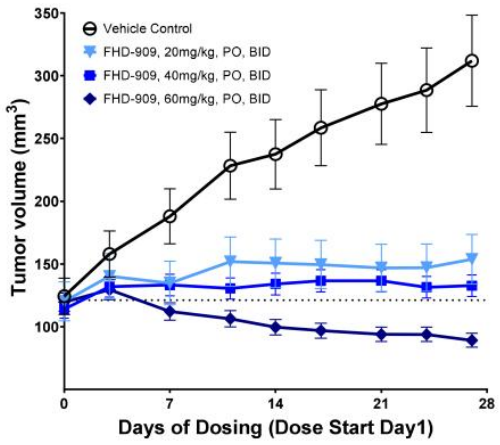
	Median IC_{50} (µM)
BRG1 WT	0.0932
BRG1 MUT	0.0028

Spread in potency for wild type versus mutant cell lines indicates

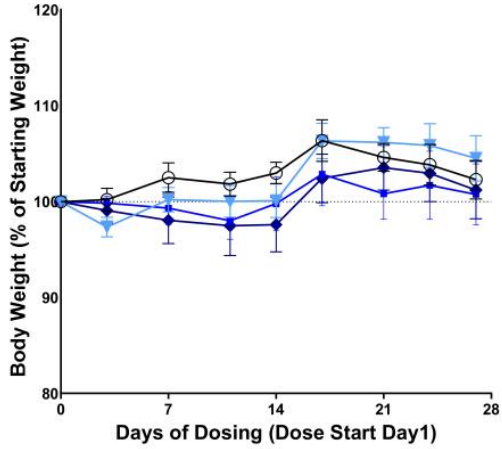
33-fold selectivity observed

FHD-909 Monotherapy Demonstrated *In Vivo* Activity in H2126 BRG1 Mutant NSCLC Model; Well Tolerated

H2126 Reduction in Tumor Volume



H2126 Body Weight

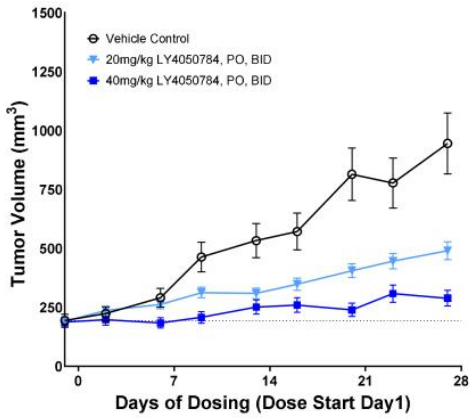


Genetic Background: *BRG1 W764R, TP53 E62*, STK11-/-, CDKN2A-/-, KEAP1 R272C*

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

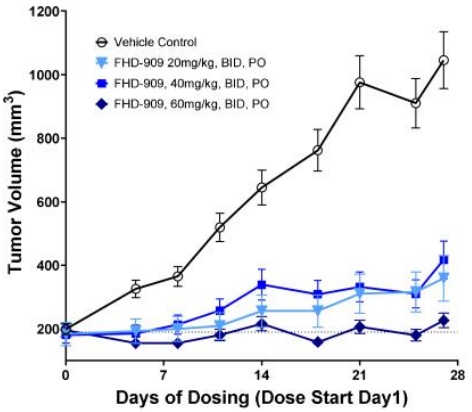
FHD-909 Monotherapy Demonstrated 96% TGI in A549 and Tumor Stasis in RERF-LC-AI Mutant NSCLC Models

A549 Model



Genetic Background	*BRG1 Q729fs / H736Y, KRAS G12S, STK11-/-, CDKN2A-/-, KEAP1 G333C*

RERF-LC-AI Model

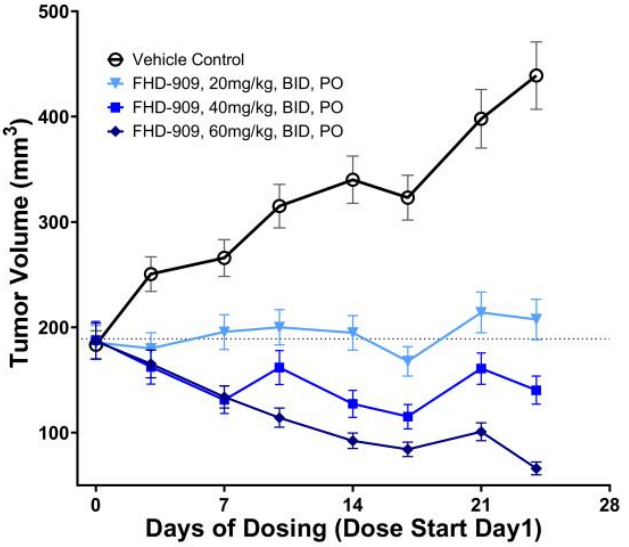


Genetic Background	*BRG1 mut p.E1496*, TP53 p.Q104*, NF1 p.E1699**

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

FHD-909 Monotherapy Demonstrated Regression in H1793 BRG1 Mutant NSCLC Models

H1793 Model



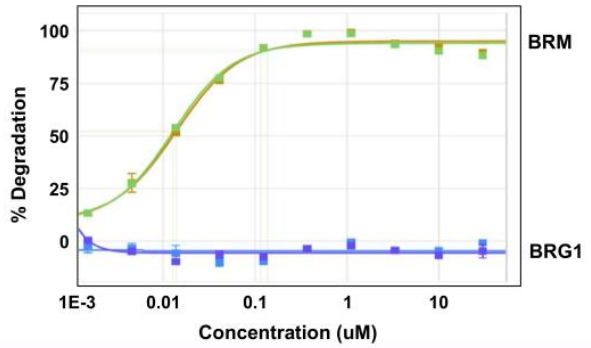
Genetic Background: *BRG1 E514*, TP53 R209* R273H, ARID1A C884**

- **FHD 909** delivered across range of BRG1 mut xenograft models
- Results ranging from impressive TGI to regression as monotherapy
- All doses across all four models were well tolerated

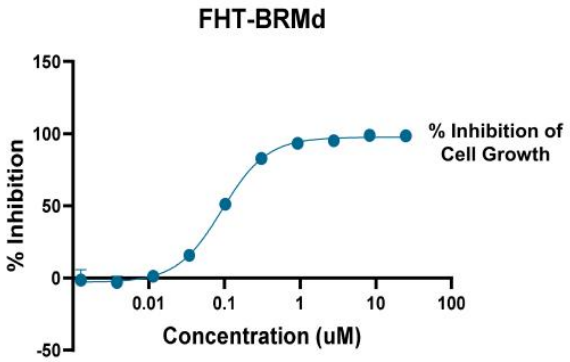
NOTE: All doses were well tolerated. Dosing holidays were applied at the high dose, as appropriate

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

BRM/BRG1 HIBIT Data



A549 Ten-Day Proliferation Assay



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

Data as of Q4 2021

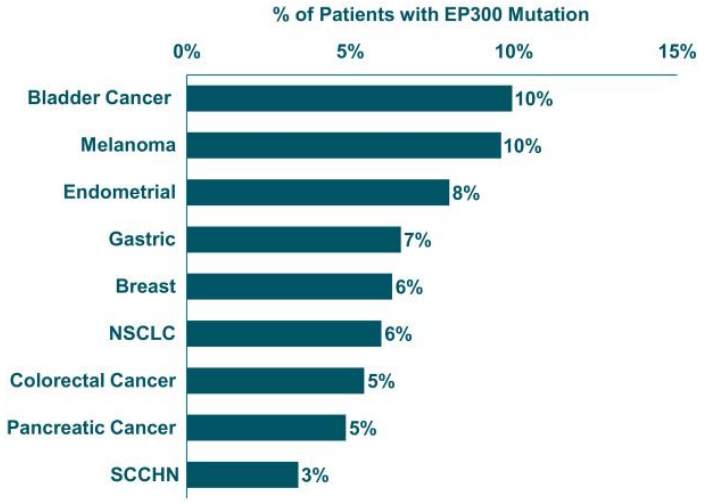
Selective CBP Protein Degrader

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

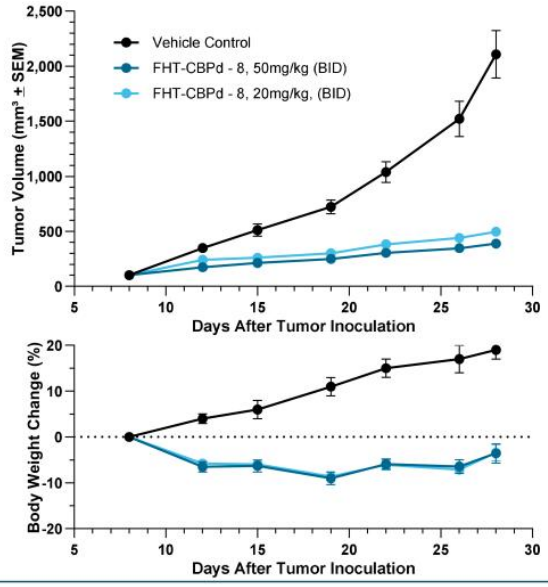
Commercial Opportunity



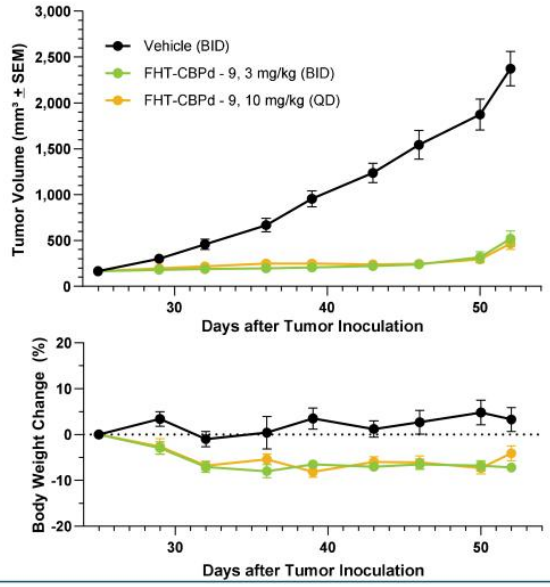
* Per year incidence in the U.S., EU5, Japan Source: Clarivate RDG Mature Markets Data

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

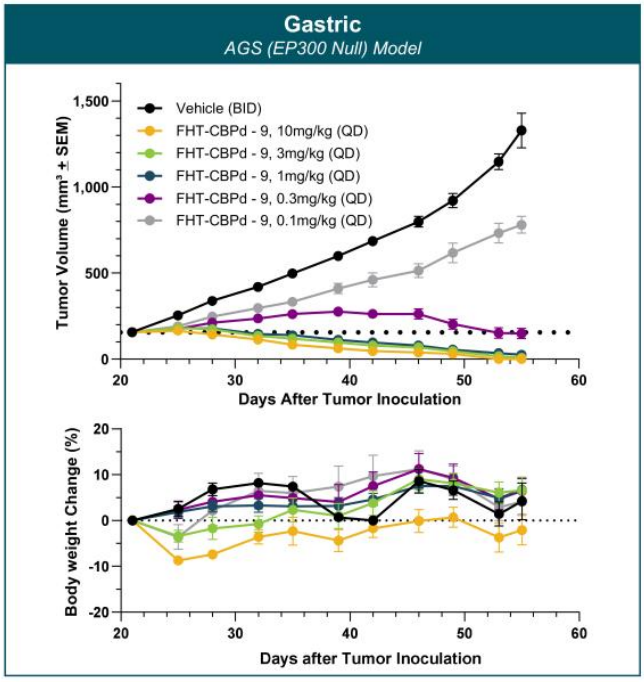
Colorectal
RKO (EP300 Null) Model



Bladder
639V (EP300 Null) Model

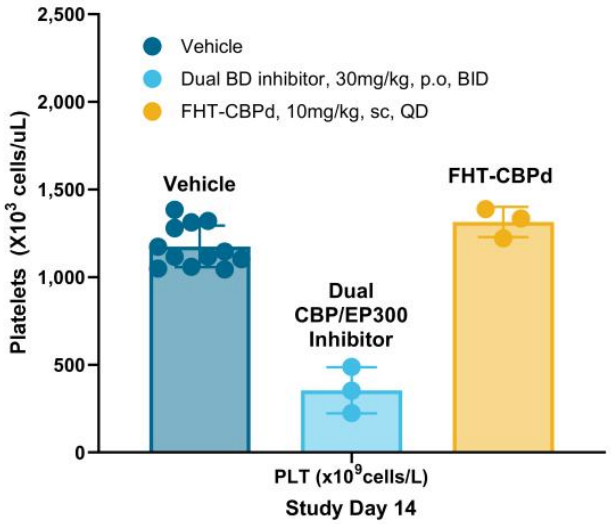


Selective CBP Degradation Resulted in Tumor Regression in Gastric EP300 Null Models

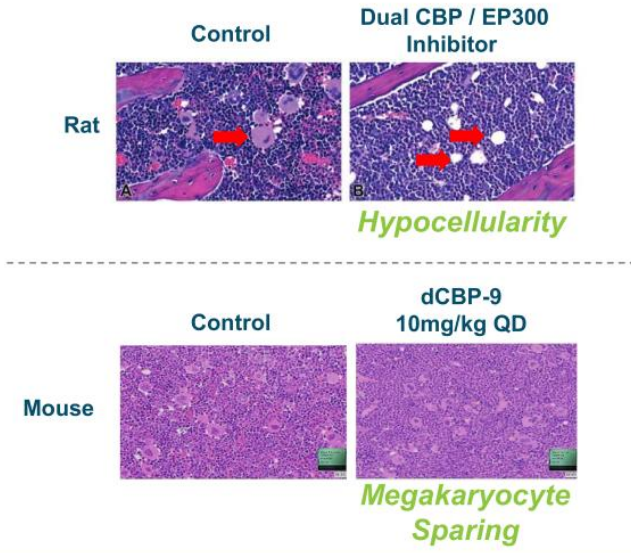


Preclinical Studies Indicated Selective CBP Degradation Did Not Show Thrombocytopenia and Spared Megakaryocytes *In Vivo*

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

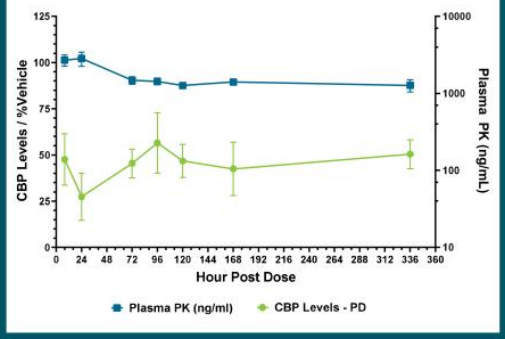


Bone Marrow Staining (*In Vivo*)

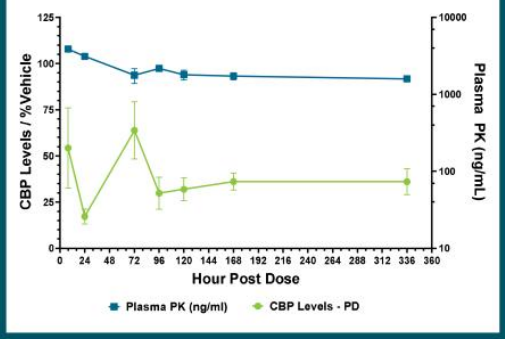


Preclinical Studied Indicated Long-Acting Injectable Formulations of CBP Degrader Could Enable Once Every 2 Weeks, or Less Frequent, Dosing

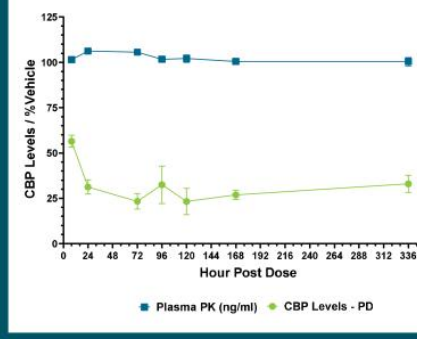
dCBP-9 PK/PD (60mg/kg, sc)



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

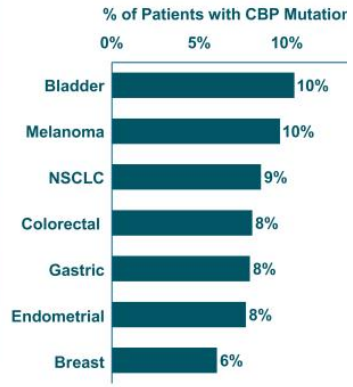
Target / Approach	• E1A binding protein p300 (EP300) • Targeted protein degrader
Initial Indications	• AR+ Prostate • DLBCL • Bladder, melanoma, others
Mutation / Aberration	• EP300 dependent cancers • CBP mutant cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

Commercial Opportunity

EP300 Dependent Cancers

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

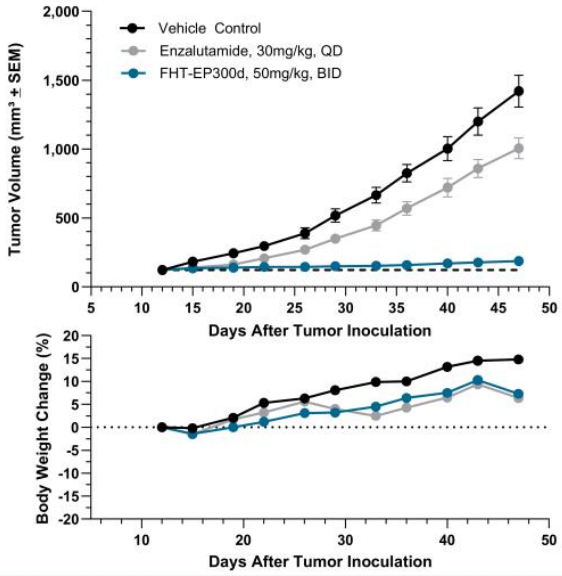
CBP Mutant Cancers



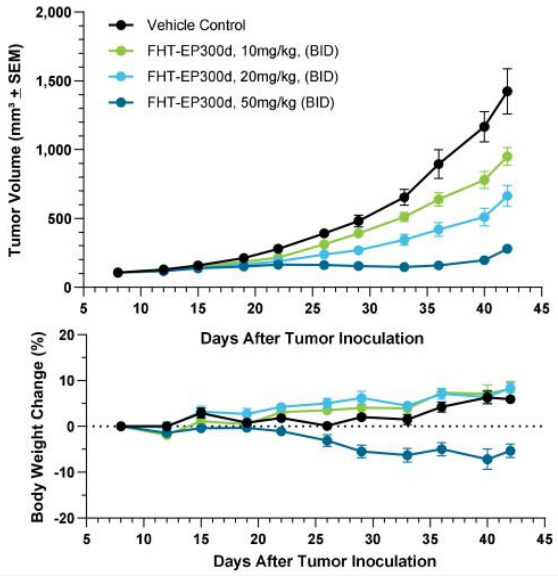
* Per year incidence in the U.S., EU5, Japan Source: Clarivate RDG Mature Markets Data

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

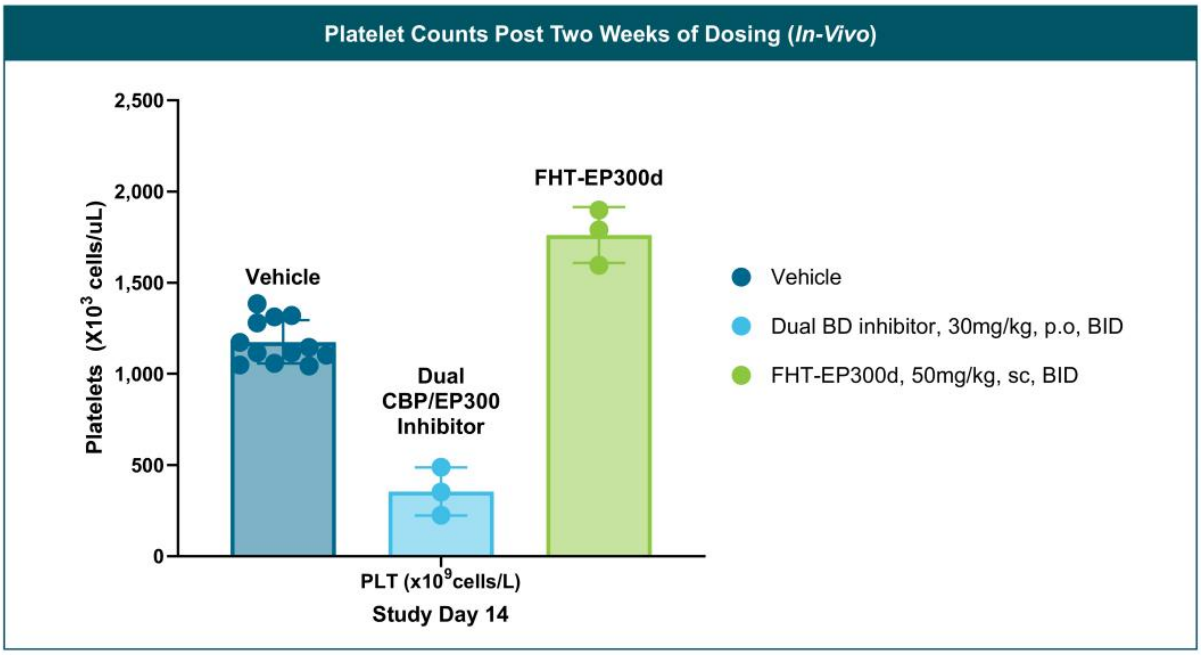
Prostate
AR+ VCAP Model



DLBCL
KARPAS422 (CBP Null) Model



Selective EP300 Degradation Does Not Show Thrombocytopenia *In Vivo*

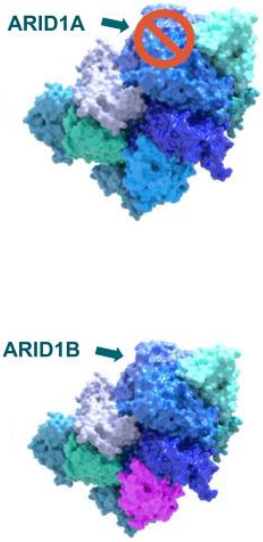


Selective ARID1B Protein Degrader

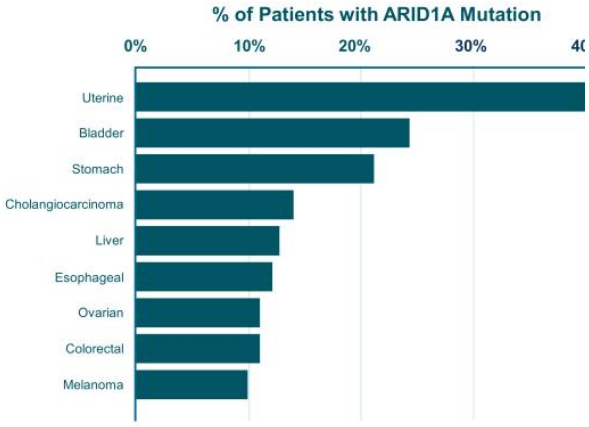
For ARID1A Mutated Cancers

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

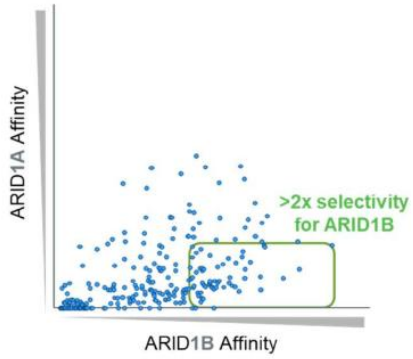


~5% of all solid tumors harbor ARID1A mutations

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

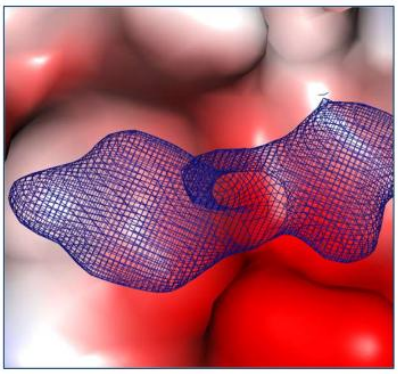
Compound Screening and Structure-Based Optimization Yields Selective ARID1B Binders

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

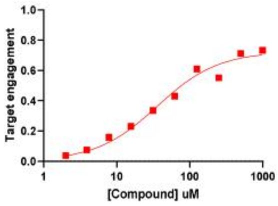
X-Ray Crystal Structures Detail Selective ARID1B Binding

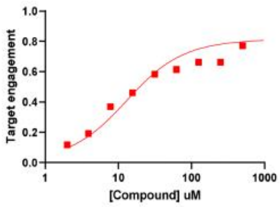


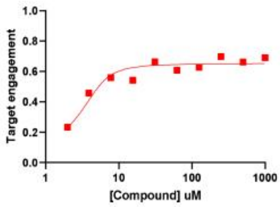
- Determined X-ray crystal structure of ARID ligandable domains with specific binders
- Leveraged these structures to drive binding affinities and expand binding chemotypes

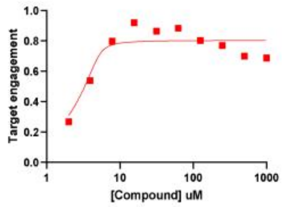
Structure-Based Optimization Drives Improved ARID1B Binding Affinity from 100 uM to less than 200 nM

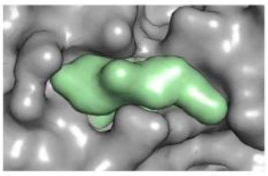
Gen 1: Screening Hit	Gen 2: Early Optimization	Gen 3: Sub-uM Affinity	
ARIDb-1 ARID1B Kd: **100 uM**	**ARIDb-2** ARID1B Kd: **15 uM**	**ARIDb-3** ARID1B Kd: **0.5 uM**	**ARIDb-9** ARID1B Kd: **0.2 uM**



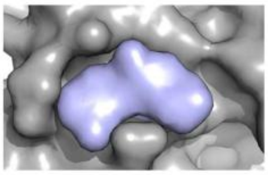




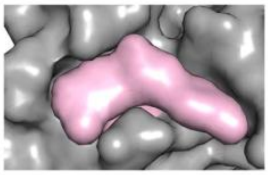




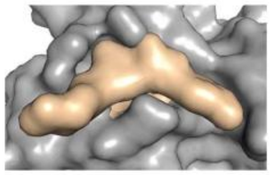
1.4 Å co-xtal structure



2.0 Å soak structure



1.9 Å co-xtal structure



1.7 Å soak structure

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…

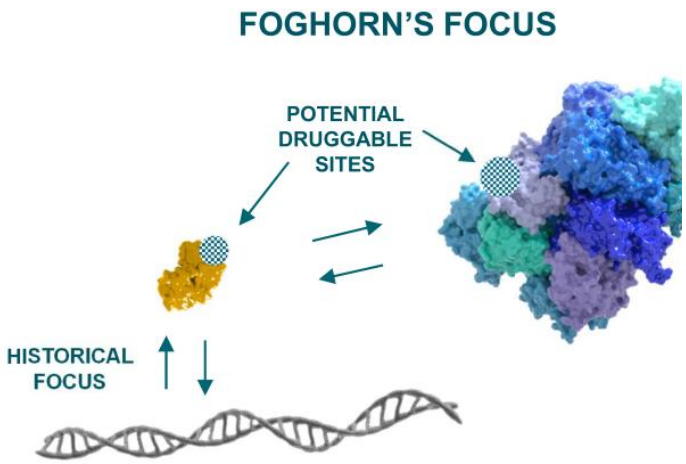
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…But Historically Difficult to Target…

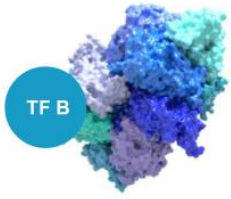
- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities

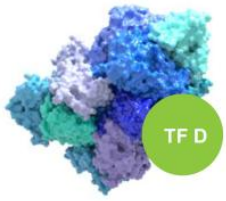
Foghorn Has a New Approach Focusing on Interaction with BAF

- Druggable binding pockets
- Druggable affinities



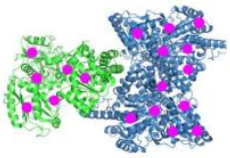
Transcription Factors Bind to BAF Directly with High Degree of Specificity; Unique Insights into Where and How Transcription Factors Bind



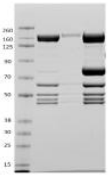


Mapping the TF-BAF Interaction

Mass spec. foot-printing



Pull-down assays

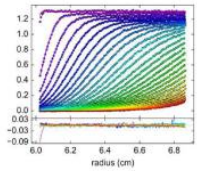


Foghorn's collection of BAF sub-complexes and domains

Validating the TF-BAF Interaction

Biophysical

AUC / SPR / ITC

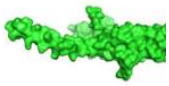


Biochemical

TR-FRET / FP

Structural

Crystal / NMI



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

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

$206.7 million in cash and equivalents

(as of 3/31/2024)

Provides **runway into H1'26**

Value Drivers

Anticipate data from the Phase 1 study of FHD-286 in combination with decitabine in **H2'24**

BRM Selective Inhibitor (FHD-909), partnered with Loxo@Lilly, **IND submitted to FDA, Phase 1 initiation anticipated in H2'24**

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

Major Strategic Collaboration

Strategic collaboration wi Loxo@Lilly; **$380 millio upfront;** 50/50 U.S. econo split on two lead program