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): August 8, 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 2.02 Results of Operations and Financial Condition.

On August 8, 2024, Foghorn Therapeutics Inc. (the "Company") issued a press release announcing certain of the Company's financial results for the quarter ended June 30, 2024. A copy of the press release is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Item 2.02 (including Exhibit 99.1 attached hereto) is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference into any filing by the Company under the Securities Act of 1933, as amended (the "Securities Act"), or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 7.01 Regulation FD Disclosure.

The Company is furnishing as Exhibit 99.2 to this Current Report on Form 8-K a presentation, dated August 2024, which the Company intends to use in meetings with or presentations to investors.

The information in this Item 7.01 (including Exhibit 99.2 attached hereto) is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference into any filing by the Company under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press Release issued on August 8, 2024
99.2	Investor Presentation dated August 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: August 8, 2024

Foghorn Therapeutics Provides Second Quarter 2024 Financial and Corporate Update

Topline Phase 1 dose escalation data for FHD-286 in combination with decitabine, in relapsed and/or refractory AML patients, anticipated in the fourth quarter of 2024

Dosing of first patient in a Phase 1 trial for FHD-909, a potential first-in-class SMARCA2 selective inhibitor, anticipated in the second half of 2024; primary target population in SMARCA4 mutated NSCLC

IND-enabling studies for Selective CBP degrader program on track to begin by year-end 2024

Successful $110 million financing strengthens balance sheet with cash, cash equivalents and short-term investments of $285.2 million as of June 30, 2024 and extends expected cash runway into 2027

CAMBRIDGE, Mass. -- (GLOBE NEWSWIRE) – August 8, 2024 -- Foghorn® Therapeutics Inc. (Nasdaq: FHTX), a clinical-stage biotechnology company pioneering a new class of medicines that treat serious diseases by correcting abnormal gene expression, today provided a financial and corporate update in conjunction with the Company's 10-Q filing for the quarter ended June 30, 2024. With an initial focus in oncology, Foghorn's Gene Traffic Control® Platform and resulting broad pipeline have the potential to transform the lives of people suffering from a wide spectrum of diseases.

"We anticipate topline data from our Phase 1 combination trial with FHD-286 in patients with relapsed and/or refractory AML in the fourth quarter of 2024. We believe FHD-286 has the potential to be a first-in-class, mutation-agnostic differentiation therapeutic," said Adrian Gottschalk, President and Chief Executive Officer of Foghorn. "Additionally, the IND for FHD-909 cleared in May and we anticipate FHD-909 to be the first SMARCA2 selective inhibitor to enter the clinic. Dosing of the first patient in our Phase 1 trial, with primary target population in SMARCA4 mutated NSCLC, is planned for the second half of the year. We are also on track to initiate IND-enabling studies in the fourth quarter of 2024 for our Selective CBP degrader program targeting tumors harboring EP300 mutations including bladder, gastric and endometrial cancers."

Mr. Gottschalk continued, "The biological foundation for the development of therapeutics targeting the chromatin regulatory system in oncology and other disease areas continues to get stronger. In April, we were pleased to present preclinical data at AACR reinforcing the potential of our platform to deliver innovative medicines across cancers by selectively drugging historically very challenging targets. The conviction in our pipeline was further strengthened by our recent successful financing with new and long-term investors, which extended our expected cash runway into 2027 and through key inflection points, strongly positioning us for continued advancement."

Corporate Updates

Strengthened Balance Sheet to Advance Pipeline. In May, Foghorn successfully closed an approximately $110 million registered direct offering to advance the Company's pipeline. The offering included new investors BVF Partners, Deerfield Management and other leading healthcare specialist investors as well as current investors, including founding investor Flagship Pioneering.

Presented at AACR Annual Meeting. In April, Foghorn presented preclinical data highlighting pipeline progress on the advancement of multiple potential first-in-class medicines, including the first presentation of preclinical data for FHD-909, at the 2024 American Association for Cancer Research (AACR) Annual Meeting.

Hosted Chromatin Regulation Summit. In April, Foghorn hosted the first Future of Disease and Chromatin Regulation Summit at the Foghorn corporate headquarters in Cambridge, Massachusetts. The live event featured presentations and panel discussions with world-renowned industry and academic key opinion leaders on therapeutic opportunities in chromatin regulatory biology.

Strengthened Executive Leadership. In April, Foghorn appointed Kristian Humer as Chief Financial Officer. Mr. Humer joined Foghorn with over 14 years of diversified financial strategy and business development experience in the life science industry and more than 20 years of experience in the financial industry.

Key Recent Program Updates and Upcoming Milestones

FHD-286. FHD-286 is a potent, first-in-class, selective inhibitor of the SMARCA2 (BRM) and SMARCA4 (BRG1) subunits of the BAF chromatin remodeling complex where dependency on SMARCA2/SMARCA4 is well-established preclinically with multiple tumor types, including acute myeloid leukemia (AML)/myelodysplastic syndrome (MDS), non-small cell lung cancer (NSCLC) and prostate cancer.

- **AML Phase 1 trial.** The ongoing Phase 1 dose escalation trial is evaluating the safety, tolerability, pharmacokinetics (PK), pharmacodynamics (PD), and preliminary clinical activity of FHD-286 in combination with decitabine or low-dose cytarabine in patients with relapsed and/or refractory AML who have failed multiple previous courses of therapy. FHD-286 previously demonstrated a promising mutation-agnostic differentiation effect in a single-agent dose escalation trial.
    - Topline clinical data are anticipated in the fourth quarter. We anticipate this will include topline safety, tolerability, initial efficacy and PK/PD data.

- **Overcoming Tyrosine Kinase Inhibitor Resistance**. Data published in *Cancer Cell* in August 2023, together with additional preclinical studies conducted by Foghorn, suggest that FHD-286 may play an important role in overcoming resistance in EGFR/KRAS tumors. Preclinical data profiling the ability of FHD-286 to amplify and/or restore tumor sensitivity will be presented with FHD-286 Phase 1 dose escalation data.

FHD-909. FHD-909 is a first-in-class oral SMARCA2 (BRM) selective inhibitor that has demonstrated in preclinical studies to have high selectivity over its closely related paralog SMARCA4 (BRG1), two proteins that are the catalytic engines across all forms of the BAF complex. SMARCA4 mutations are common across tumor types, including approximately 10% of NSCLC, and result in tumors being dependent on SMARCA2 activity for their survival. Selectively blocking SMARCA2 activity is a promising synthetic lethal strategy intended to induce tumor death while sparing healthy cells.

- In December 2021, Foghorn announced a strategic collaboration with Lilly to create novel oncology medicines. The collaboration includes a U.S. 50/50 co-development and co-commercialization agreement for Foghorn’s Selective SMARCA2 oncology program, which includes a selective inhibitor and a selective degrader, and an additional undisclosed oncology

target. In addition, the collaboration includes three discovery programs using Foghorn's proprietary Gene Traffic Control platform.

- In April 2024, Foghorn and Lilly presented new preclinical data as a poster presentation at the AACR Annual Meeting and during a pipeline update call demonstrating at tolerable doses high SMARCA2 selectivity and dose-dependent single agent activity in SMARCA4 mutated cancers.
- In May 2024, the investigational new drug (IND) application was approved by the Food and Drug Administration. The primary target patient population is SMARCA4 mutated NSCLC.
- Dosing of the first patient in the Phase 1 trial for FHD-909 is planned to begin in the second half of 2024.

Selective CBP degrader program and Selective EP300 degrader program.
Foghorn is advancing two separate programs targeting either CBP or EP300, paralog histone acetyltransferases with a synthetic lethal relationship in tumor cells. Attempts to selectively drug CBP or EP300 have been challenging due to the high level of similarity between the two proteins. Additionally, dual inhibition of CBP/EP300 has been historically limited by hematopoietic toxicity.

Selective CBP degrader program. In April, Foghorn presented new pharmacodynamic and pharmacokinetic preclinical data at the 2024 AACR Annual Meeting and during a pipeline update call highlighting:

- Deep and sustained CBP degradation significantly inhibited tumor growth in mouse xenograft solid tumor models.
- Robust monotherapy preclinical anti-tumor activity that was not associated with significant body weight loss, thrombocytopenia or anemia.
- Identification of potent and selective CBP protein degraders with first-in-class potential to address tumors harboring EP300 mutations in many types of cancer, including bladder, gastric and endometrial cancers.
- IND-enabling studies on track to initiate by the fourth quarter of 2024.

Selective EP300 degrader program. In April, Foghorn presented new pharmacodynamic and pharmacokinetic preclinical data at the 2024 AACR Annual Meeting and during a pipeline update call highlighting:

- Well-tolerated *in vivo* with no observed decrease in platelet levels, with no effects on megakaryocyte viability at pharmacologically relevant concentrations in *ex vivo* studies.
- Identification of potent and selective EP300 degraders with anti-tumor activity in prostate and hematological malignancies, including prostate cancer, multiple myeloma and diffuse large B cell lymphoma (DLBCL).

Selective ARID1B degrader program.
ARID1A is the most mutated subunit in the BAF Complex and amongst the most mutated proteins in oncology. These mutations lead to a dependency on ARID1B, in several types of cancer, including ovarian, endometrial, colorectal and bladder. Attempts to selectively drug ARID1B have been challenging because of the high degree of similarity between ARID1A and ARID1B and the fact that ARID1B has no enzymatic activity to target.

- In April, Foghorn presented data at the AACR Annual Meeting demonstrating potent and selective small molecule binders to ARID1B. The Company is in the process of converting these selective binders into heterobifunctional degraders.

Second Quarter 2024 Financial Highlights

- **Collaboration Revenues.** Collaboration revenue was $6.9 million for the three months ended June 30, 2024, compared to $5.6 million for the three months ended June 30, 2023. The increase year-over-year was primarily driven by the continued advancement of programs under the Lilly Collaboration Agreement.

- **Research and Development Expenses.** Research and development expenses were $23.8 million for the three months ended June 30, 2024, compared to $29.2 million for the three months ended June 30, 2023. This decrease was primarily due to lower personnel-related costs and lower development program spend following the shutdown of two clinical studies (FHD-286 in metastatic uveal melanoma and FHD-609 (BRD9 degrader) in synovial sarcoma).

- **General and Administrative Expenses.** General and administrative expenses were $7.3 million for the three months ended June 30, 2024, compared to $8.4 million for the three months ended June 30, 2023. This decrease was primarily due to lower personnel-related costs.

- **Net Loss.** Net loss was $23.0 million for the three months ended June 30, 2024, compared to a net loss of $29.5 million for the three months ended June 30, 2023.

- **Cash, Cash Equivalents and Marketable Securities.** As of June 30, 2024, the Company had $285.2 million in cash, cash equivalents and marketable securities, providing expected cash runway into 2027.

About FHD-286
FHD-286 is a highly potent, first-in-class, selective, allosteric, and orally available small-molecule, enzymatic inhibitor of SMARCA2 (BRM) and SMARCA4 (BRG1), two highly similar proteins that are the ATPases, or the catalytic engines, of the BAF complex, one of the key regulators within the chromatin regulatory system. In preclinical studies, FHD-286 has shown anti-tumor activity across a broad range of malignancies including both hematologic and solid tumors.

About AML
Adult acute myeloid leukemia (AML) is a cancer of the blood and bone marrow and the most common type of acute leukemia in adults. AML is a diverse disease associated with multiple genetic mutations. It is diagnosed in about 20,000 people every year in the United States.

About FHD-909
FHD-909 (LY4050784) is a potent, first-in-class, allosteric and orally available small molecule that selectively inhibits the ATPase activity of SMARCA2 (BRM) over its closely related paralog SMARCA4 (BRG1), two proteins that are the catalytic engines across all forms of the BAF complex, one of the key regulators of the chromatin regulatory system. In preclinical studies, tumors with mutations in SMARCA4 rely on SMARCA2 for BAF function. FHD-909 has shown significant anti-tumor activity across multiple SMARCA4-mutant lung tumor models.

About Foghorn Therapeutics
Foghorn® Therapeutics is discovering and developing a novel class of medicines targeting genetically determined dependencies within the chromatin regulatory system. Through its proprietary scalable Gene Traffic Control® platform, Foghorn is systematically studying, identifying and validating potential drug

targets within the chromatin regulatory system. The Company is developing multiple product candidates in oncology. Visit our website at www.foghorntx.com for more information on the Company, and follow us on X (formerly Twitter) and LinkedIn.

Forward-Looking Statements

This press release contains "forward-looking statements." Forward-looking statements include statements regarding the Company's clinical trials, including its ongoing Phase 1 study of FHD-286 in combination with decitabine in relapsed and/or refractory AML patients and the planned Phase 1 trial of FHD-909, pre-clinical product candidates, expected timing of clinical data, expected cash runway, expected timing of regulatory filings, and research efforts and other statements identified by words such as "could," "may," "might," "will," "likely," "anticipates," "intends," "plans," "seeks," "believes," "estimates," "expects," "continues," "projects" and similar references to future periods. Forward-looking statements are based on our current expectations and assumptions regarding capital market conditions, our business, the economy and other future conditions. Because forward-looking statements relate to the future, by their nature, they are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict. As a result, actual results may differ materially from those contemplated by the forward-looking statements. Important factors that could cause actual results to differ materially from those in the forward-looking statements include regional, national or global political, economic, business, competitive, market and regulatory conditions, including risks relating to our clinical trials and other factors set forth under the heading "Risk Factors" in the Company's Annual Report on Form 10-K for the year ended December 31, 2023, as filed with the Securities and Exchange Commission. Any forward-looking statement made in this press release speaks only as of the date on which it is made.

Condensed Consolidated Balance Sheets
(In thousands)

	June 30, 2024	December 31, 2023
Cash, cash equivalents and marketable securities	$ 285,177	$ 234,057
All other assets	43,408	51,859
Total assets	**$ 328,585**	**$ 285,916**
Deferred revenue, total	$ 290,726	$ 302,665
All other liabilities	52,181	60,441
Total liabilities	**$ 342,907**	**$ 363,106**
Total stockholders' deficit	**$ (14,322)**	**$ (77,190)**
Total liabilities and stockholders' deficit	**$ 328,585**	**$ 285,916**

Condensed Consolidated Statements of Operations
(In thousands, except share and per share amounts)

	Three Months Ended June 30,	
	2024	2023
Collaboration revenue	$ 6,888	$ 5,599
Operating expenses:		
Research and development	23,797	29,248
General and administrative	7,325	8,401
Impairment of long-lived assets	2,398	—
Total operating expenses	**$ 33,520**	**$ 37,649**
Loss from operations	**$ (26,632)**	**$ (32,050)**
Total other income, net	**$ 3,653**	**$ 3,505**
Provision for income taxes	**$ —**	**$ (942)**
Net loss	**$ (22,979)**	**$ (29,487)**
Net loss per share attributable to common stockholders—basic and diluted	(0.45)	(0.70)
Weighted average common shares outstanding—basic and diluted	**51,580,310**	**41,825,555**

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Exhi

FOGHORN®
THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

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Forward Looking Statements

This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on infor currently available to management. All statements other than statements of historical facts contained in this presentation are fo looking statements. In some cases, you can identify forward-looking statements by terms such as “could,” “may,” “might,” “will,” “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” or the negative of these te other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements inclu are not limited to, statements concerning: the potential outcomes from our collaboration agreement with Lilly; the initiation, progress and results of our research and development programs and pre-clinical studies and clinical trials, including with respec Phase 1 trial of FHD-286 in combination with decitabine in relapsed and/or refractory AML patients and anticipated timing of rel clinical data, and the planned Phase 1 dose escalation trial of FHD-909 with Lilly; our ability to advance product candidates t may develop and to successfully complete preclinical and clinical studies; our ability to leverage our initial programs to d additional product candidates using our Gene Traffic Control Platform®; the impact of exogeneous factors, including macroec and geopolitical circumstances, on our and our collaborators' business operations, including our research and development pr and pre-clinical studies; developments related to our competitors and our industry; our ability to expand the target populations programs and the availability of patients for clinical testing; our ability to obtain regulatory approval for FHD-286 and any future p candidates from the FDA and other regulatory authorities; our ability to identify and enter into future license agreemen collaborations; our ability to continue to rely on our CDMOs and CROs for our manufacturing and research needs; reg developments in the United States and foreign countries; our ability to attract and retain key scientific and management personn scope of protection we are able to establish, maintain and enforce for intellectual property rights covering FHD-286, our future p and our Gene Traffic Control Platform; and our use of proceeds from capital-raising transactions, estimates of our expenses, requirements, and needs for additional financing. You should, therefore, not rely on these forward-looking statements as repre our views as of any date subsequent to the date of this presentation. Additional important factors to be considered in connecti forward-looking statements are described in the Company's filings with the Securities and Exchange Commission, including with section entitled "Risk Factors" in the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 202 forward-looking statements represent the Company’s views only as of the date of this presentation and should not be relie as representing its views as of any subsequent date. The Company explicitly disclaims any obligation to update any fo 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

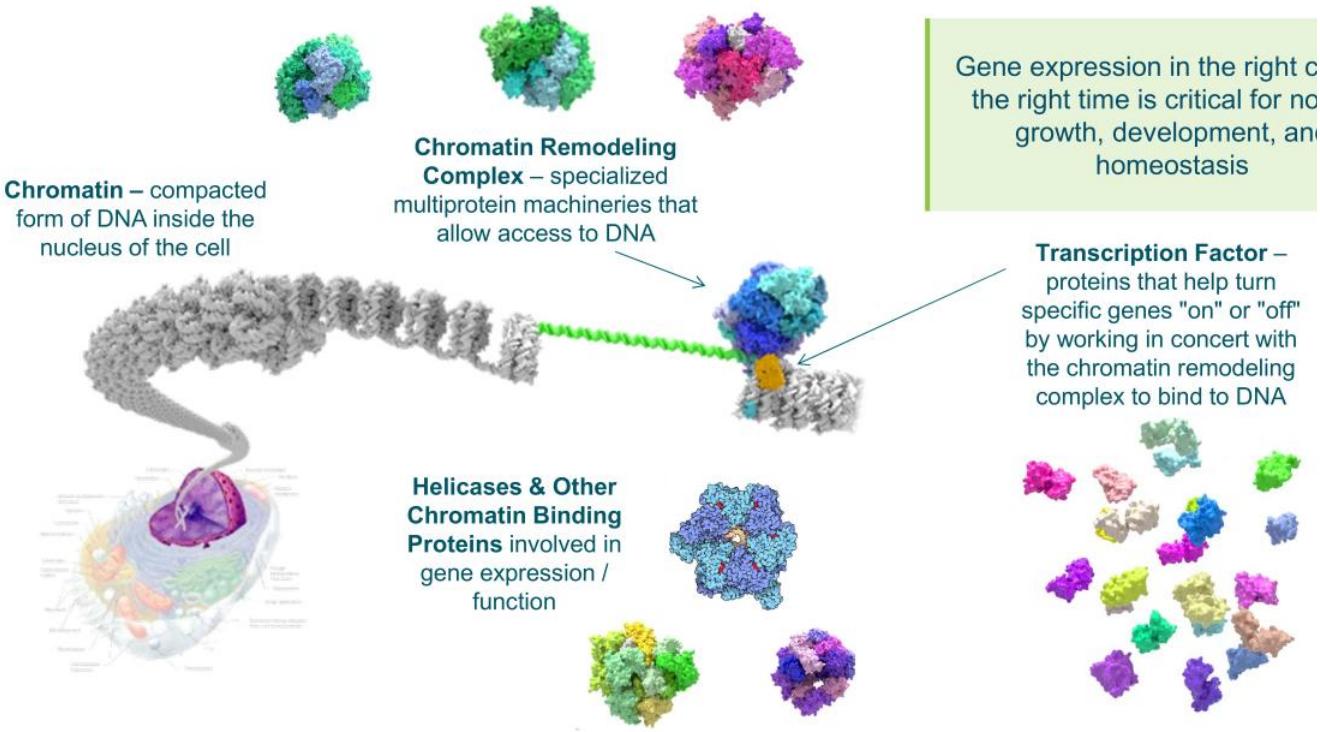
What if … It were possible to develop a therapeutic approach to treat half of all cancers?

Chromatin biology is implicated in up to 50% of tumors

~2.5 million cancer patients

Potential for therapeut area expansion (e.g., I&

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



Gene expression in the right c
the right time is critical for no
growth, development, an
homeostasis

Foghorn has Progressed Multiple Programs Against Challenging Targets

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

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

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

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

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

SMARCA2 = BRM
SMARCA4 = BRG1

… and more.

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

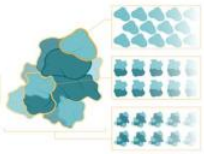
FHD-909
selective inhibitor expec
enter the clinic second h

Selective CBP Degra
IND enabling studies anti
by end of year

Selective EP300 Degra
IND enabling studie
anticipated in 2025

Selective ARID1B bin
identified. Critical step to
degradation

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

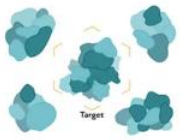


2. Assays & Biochemistry Capabilitie

Engineering selectivity via unique assays and protein capabilities

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

"Where to Drug"



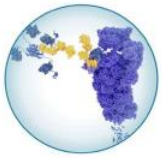
1. Chromatin Biology

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

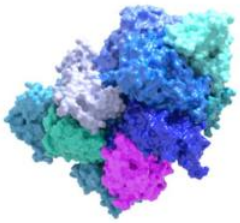
Foghorn's Unique Platform Capabilities Evolved from Drugging a Specific Chromatin Remodeling Complex (BAF)*

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 sim proteins that have no enzyr function

Protein Degrader Platform

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

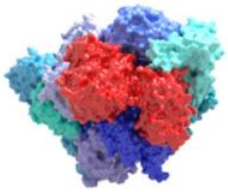
Current and Future Applications

- **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).

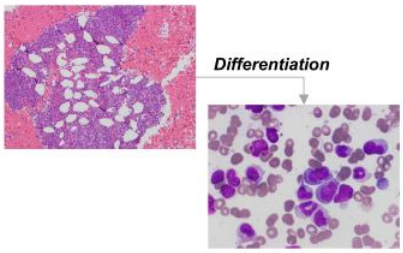
The Next Foghorn Chapter: Delivering Multiple Potential Blockbusters into the Clinic

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)



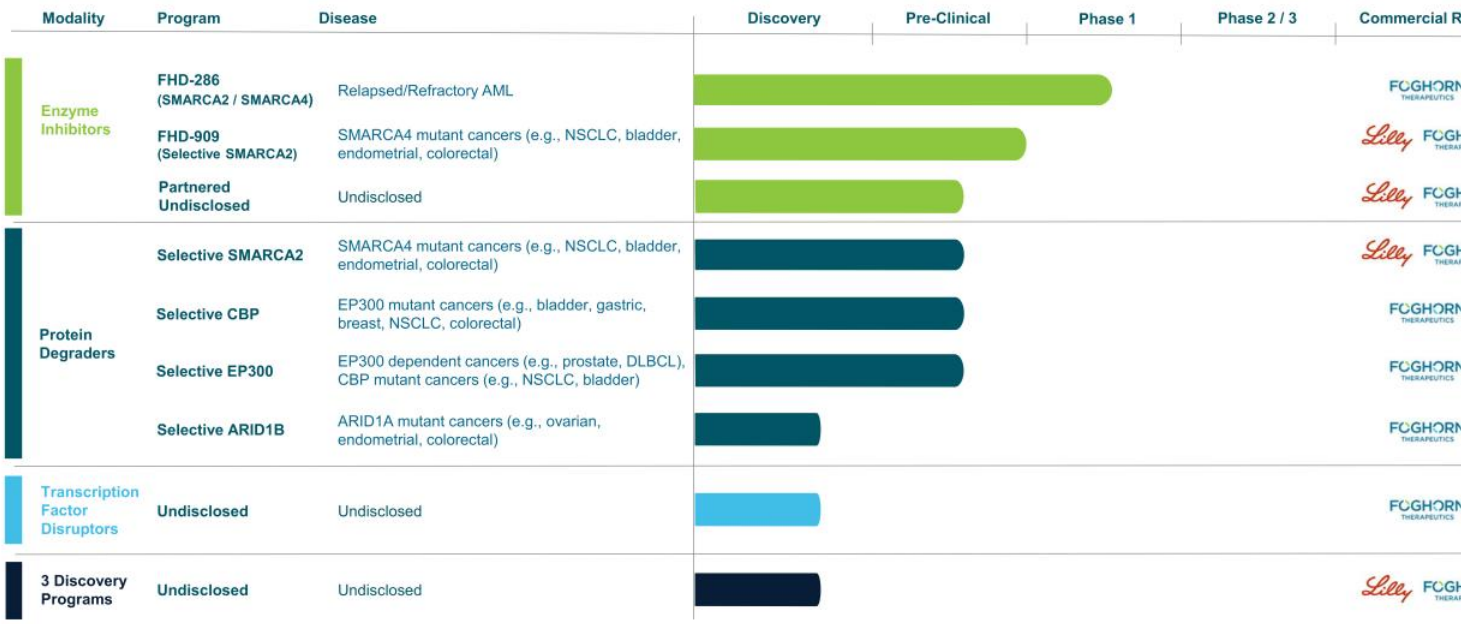
- ✓ **Lilly strategic collaboration**
- ✓ FHD-286 demonstrated mutation-agnostic differentiation effect in acute myeloid leukemia (AML)
- ✓ Initiated efforts on CBP and EP300
- ✓ Expansion of protein degrader platform

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

- **Proof of concept data for SMARC Selective Inhibition (FHD-909) in N**
- **Registrational trials for FHD-286 i**
- **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 …



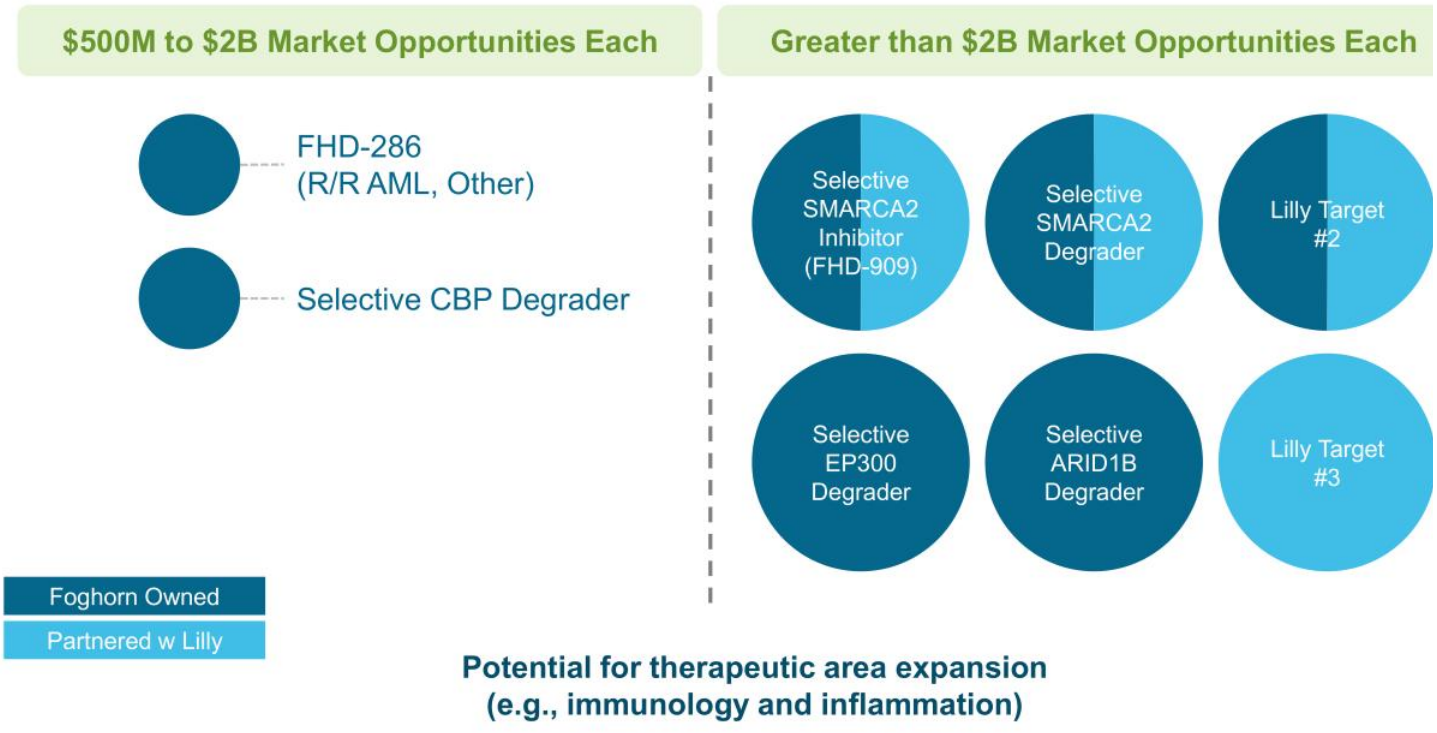
SMARCA2 = BRM
SMARCA4 = BRG1

… 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 Initiation	H2 2024
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



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

FOGHORN THERAPEUTICS

Clinical & Pre-Clinical Programs

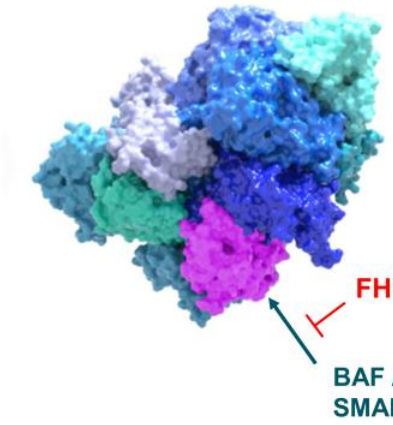
- FHD-286 – Dual SMARCA2 / SMARCA4 Inhibitor
- FHD-909 (LY4050784) – Selective SMARCA2 Inhibitor
- Selective CBP Degrader
- Selective EP300 Degrader
- Selective ARID1B Program

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-Cl
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 B/ mutations
Differentiation	Clinical and pre-clinical data demonstrated bro based differentiation across AML and multip solid tumors
Overcoming Drug Resistance	Pre-clinical data support ability to overcome d resistance (i.e., EGFR NSCLC, enzalutamid resistant CRPC, PD-1 refractory)
Immune Modulation	Clinical data demonstrated an increase of CD T-cells and a reduction of T-regulatory cells

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

Initial FHD-286 Opportunit

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**)

~17,000 Drug Treatable R/R Patie

- **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 (le
cell mark
decrease

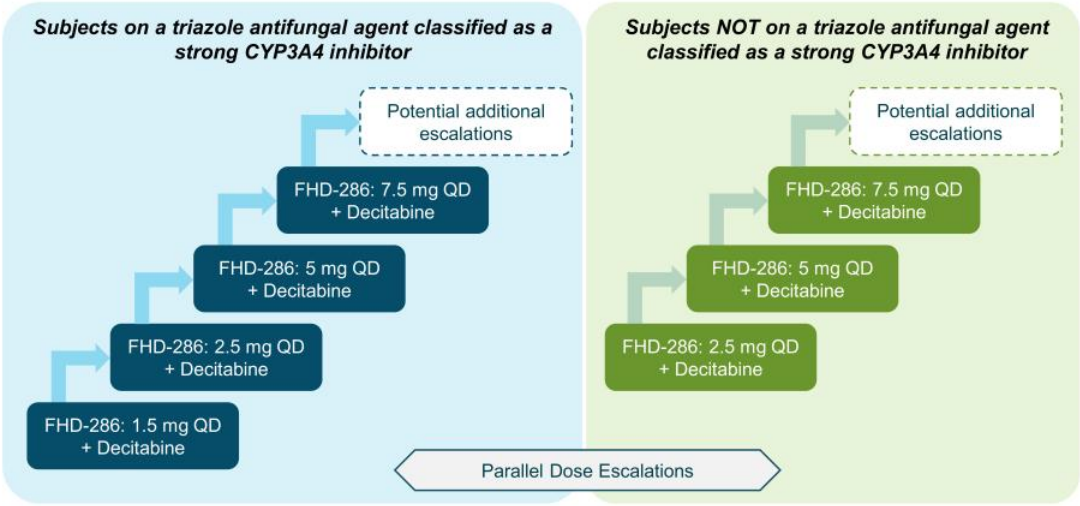
Dose Escalation Trial Design in Combination with Decitabine in AML

Target Indication:

- R/R AML

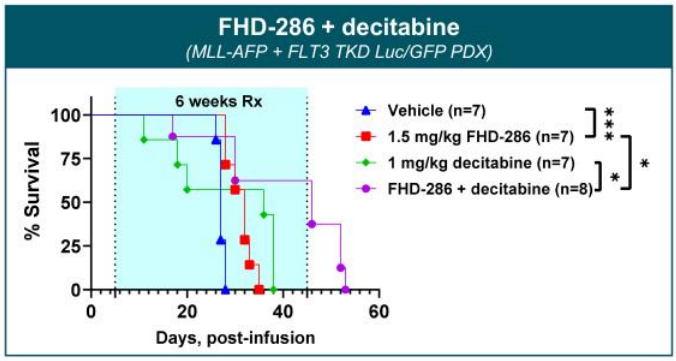
Treatment Plan & Dose Escalation:

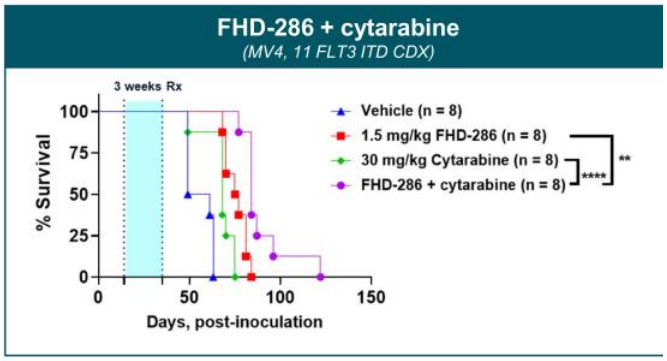
- 3+3 escalation design
- Oral FHD-286, QD, 28-day cycles
- Standard decitabine dose schedule

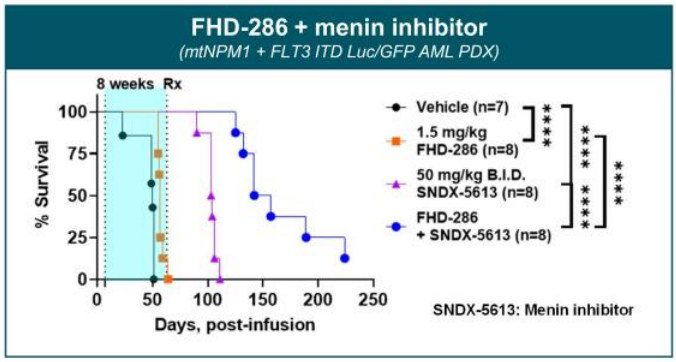


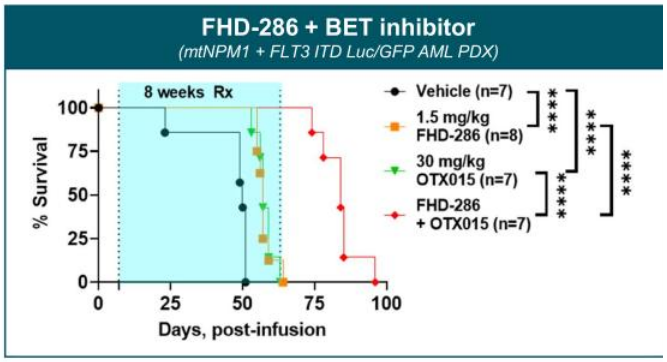
Key Objectives	
Primary	• Safety/Tolerability • Maximum Tolerated Dose (MTD) and/or Recommended Phase 2 Dose (RP2D) determinations
Secondary	• Preliminary clinical activity • PK parameters of FHD-286 in combination with Decitabine in subjects on/off triazole antifungal agents classified as strong CYP3A4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

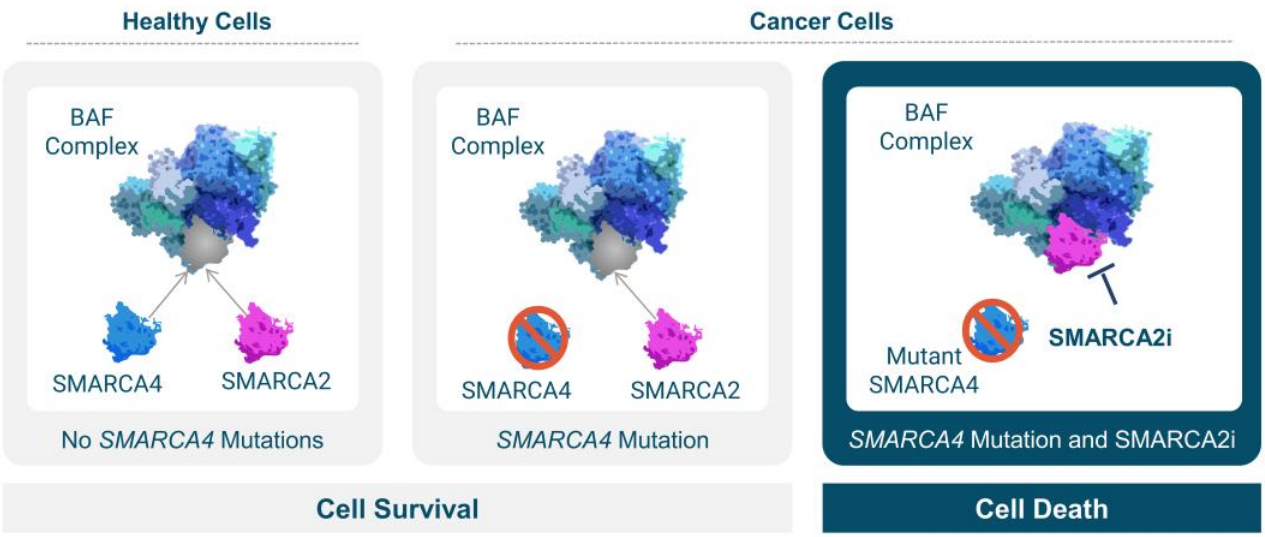
For SMARCA4 Mutated Cancers

SMARCA2 = BRM
SMARCA4 = BRG1

SMARCA2 Selective Inhibitor FHD-909 on Track for Phase 1 Initiation, Selective SMARCA2 Degrader Continues Late-Stage Pre-Clinical Development

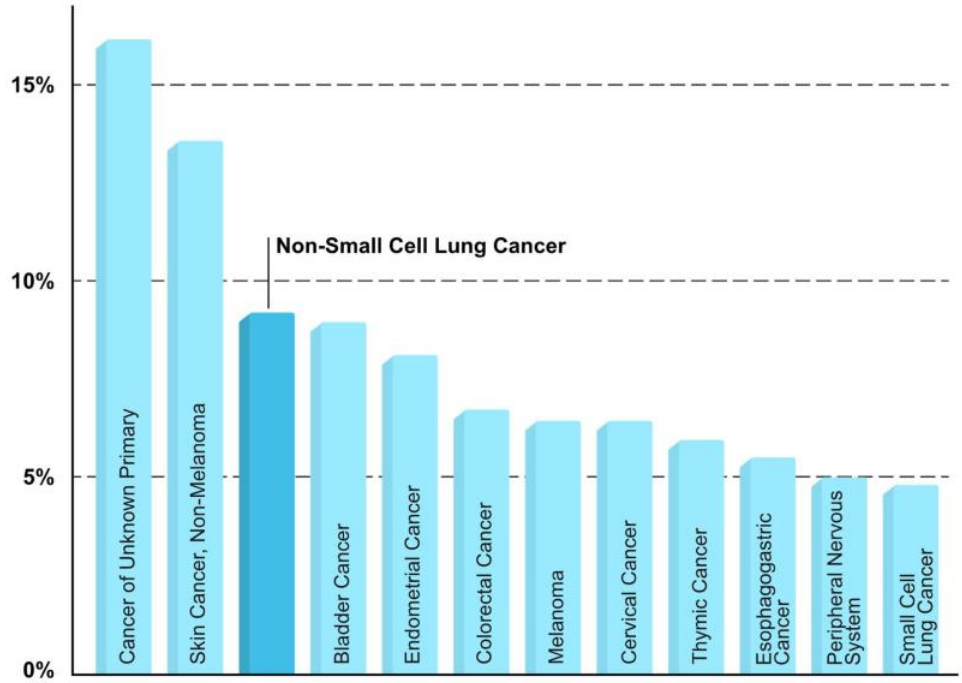
	SMARCA2 Selective Inhibitor (FHD-909)	SMARCA2 Selective Degrader
Biology	Exploit the synthetic lethal relationship between SMARCA2 and mutated SMARCA4	
Stage	IND cleared, Initiating Phase 1 clinical trial in H2'24	Advancing in parallel through late p clinical development
Opportunity	SMARCA4 mutated cancer including ~10% of NSCLC and up to 5% of all solid tum	
Lilly Partnership	50/50 global R&D cost share | 50/50 U.S. economics | tiered ex-U.S. royalties sta in the low double-digit range and escalating into the twenties	

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



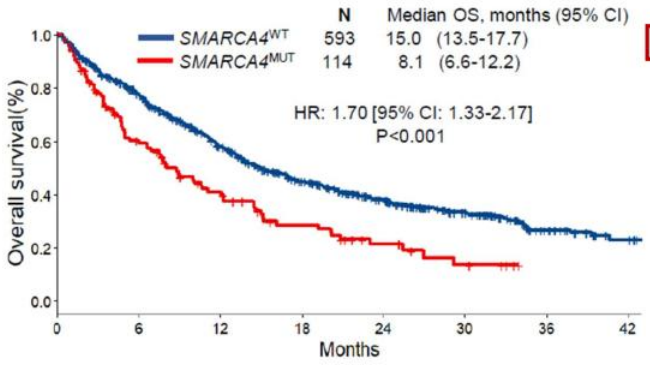
SMARCA4 mutated acro a broad range of tumors

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

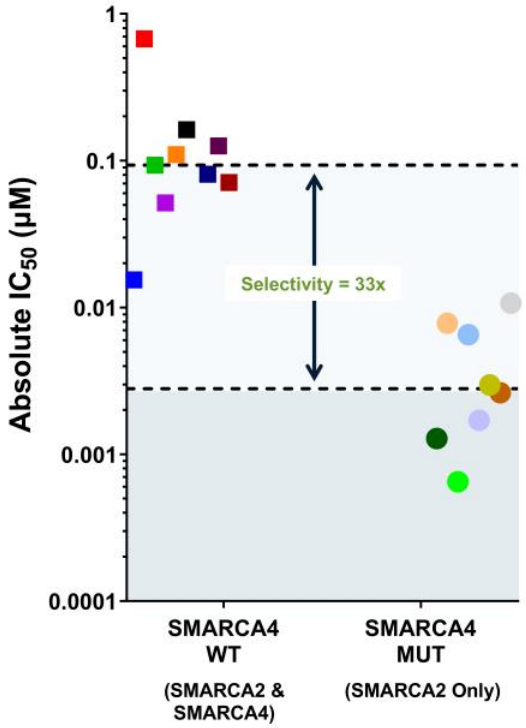
Overall Survival for SMARCA4wt vs SMARCA4mut[1]

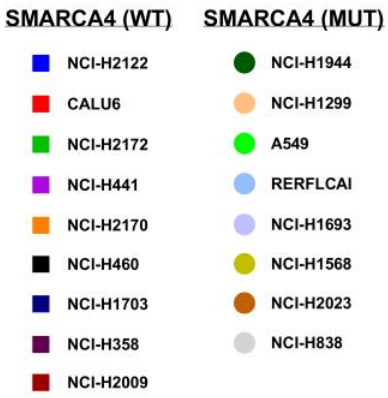


SMARCA4 mutated in up to 10% of NSCLC tumors, minimal overlap with other mutations[2]

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*





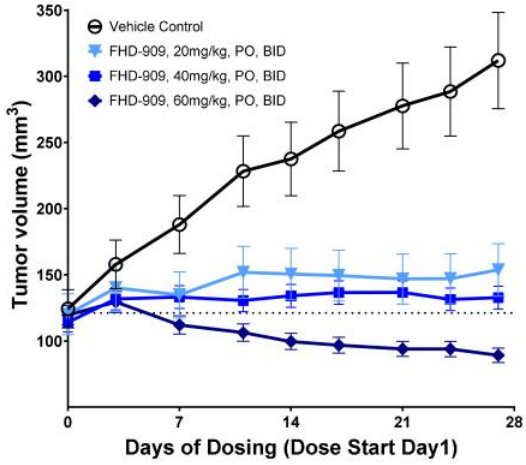
	Median IC_{50} (µM)
SMARCA4 WT	0.0932
SMARCA4 MUT	0.0028

Spread in potency for wil
versus mutant cell lines
indicates

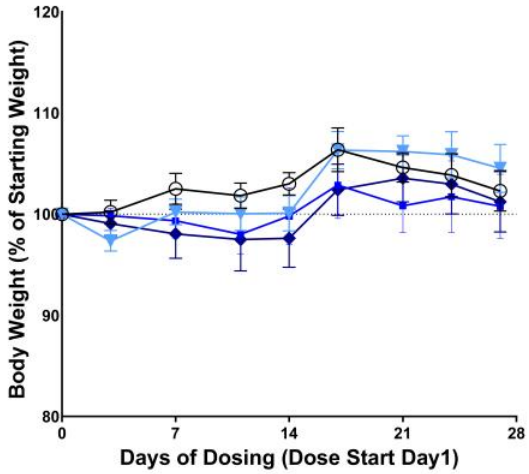
33-fold selectivity observed

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

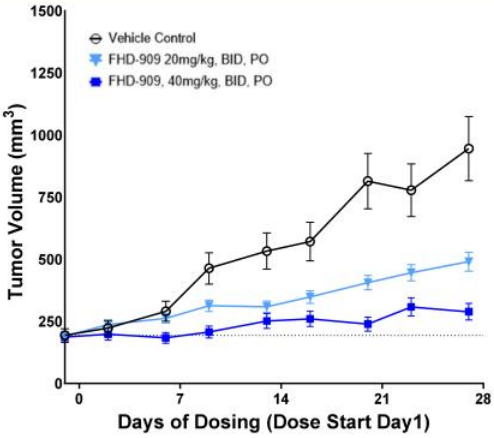


Genetic Background: *SMARCA4 W764R, TP53 E62*, STK11-/-, CDKN2A-/-, KEAP1 R272C*

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

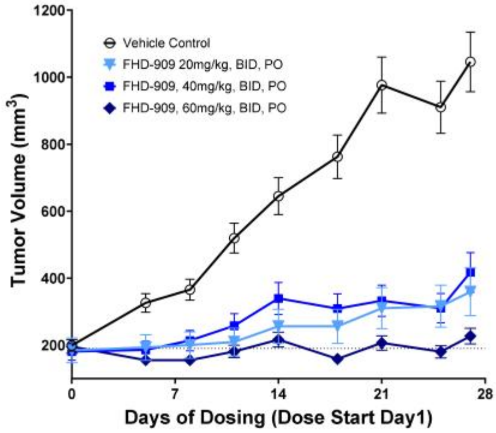
FHD-909 Monotherapy Demonstrated 96% TGI in A549 and Tumor Stasis in RERF-LC-AI Mutant NSCLC Models

A549 Model



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

RERF-LC-AI Model

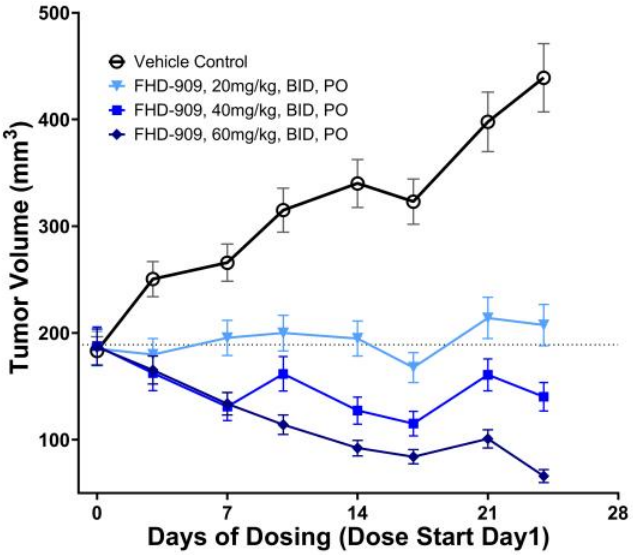


Genetic Background | *SMARCA4 mut p.E1496*, TP53 p.Q104*, NF1 p.E1699**

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 – Phase 1 Initiation On Track

Dose Escalation

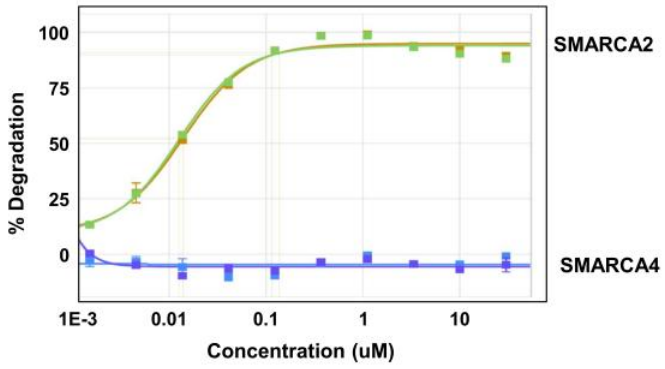
- Restricted to SMARCA4 mutated tumors
- SMARCA4 mutant status confirmed by standard NGS panel
- Further enrichment for NSCLC patients as trial progresses
- Tumor histology agnostic

Dose Expansion

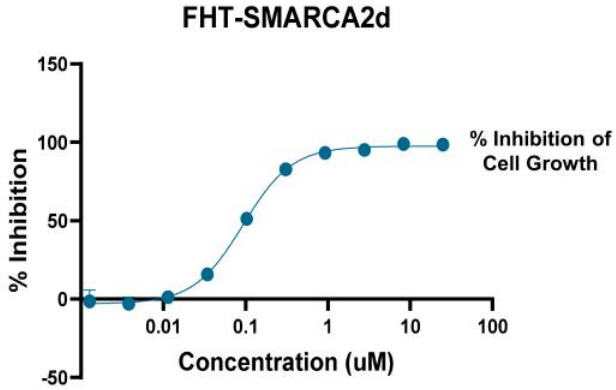
- Arm 1: SMARCA4 mutant NSCLC
- Arm 2: Other SMARCA4 mutant tumors (e.g., bladder, endometrial, colorectal)
- Potential for combination arm(s)

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

SMARCA2 / SMARCA4 HIBIT Data



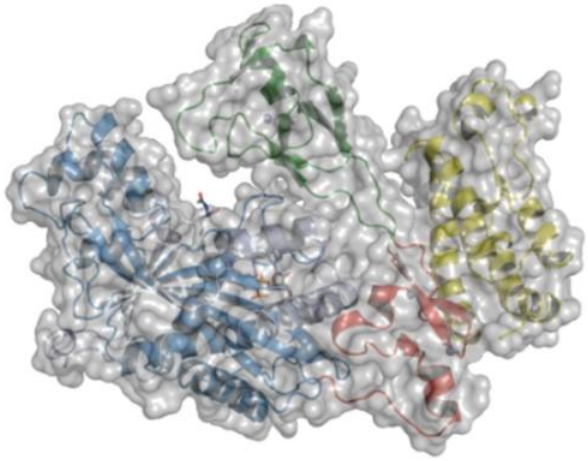
A549 Ten-Day Proliferation Assay



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, thu 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

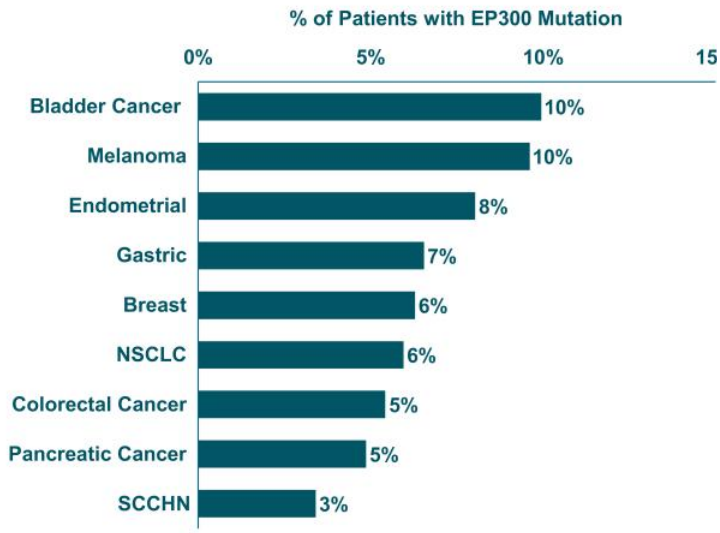
Selective CBP Protein Degrader

For EP300 Mutated Cancers

Summary: Selective CBP Protein Degrader for EP300 Mutated Cancers

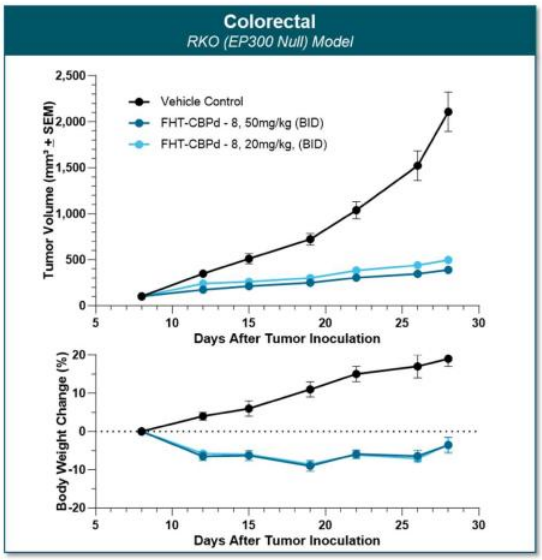
Target / Approach	• CREB binding protein (CBP) • Targeted protein degrader
Initial Indication	• EP300 mutated cancers (e.g., subsets of bladder, colorectal, breast, gastric and lung cancers)
Mutation / Aberration	• EP300 mutated cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

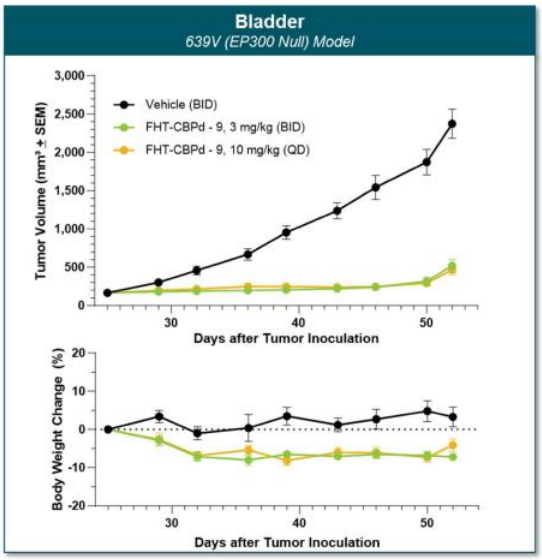
Commercial Opportunity

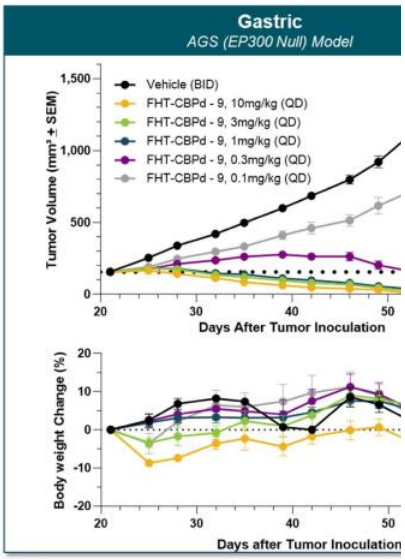


* Per year incidence in the U.S., EU5, Japan . Source: Clarivate DRG Mature Markets Data.

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

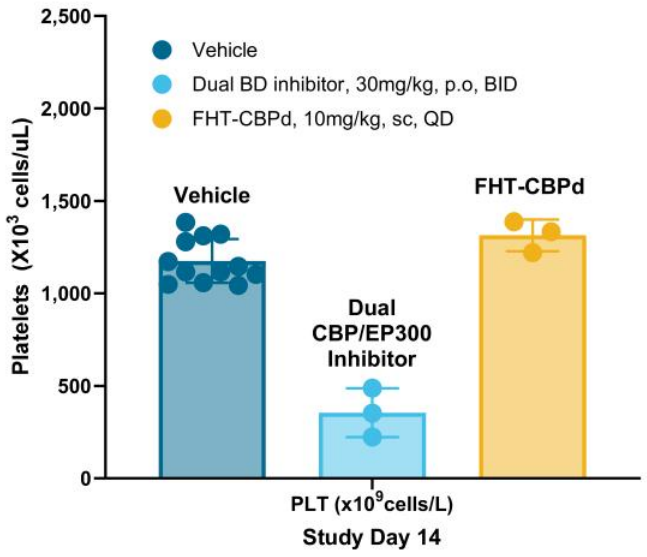




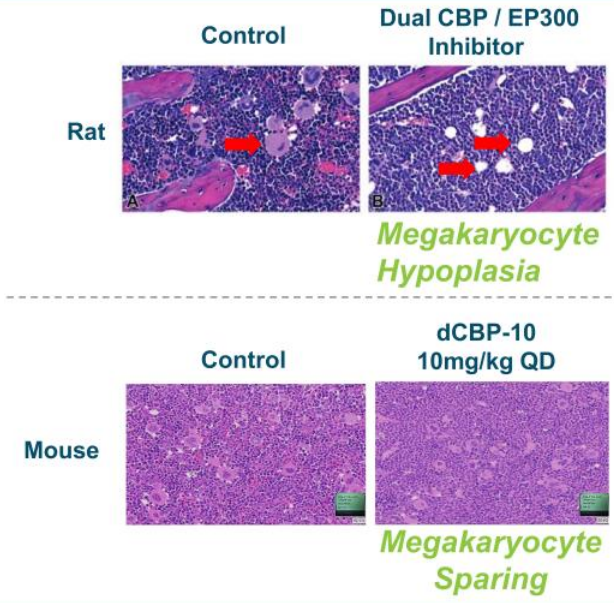


Pre-Clinical Studies Indicate Selective CBP Degradation Did Not Show Thrombocytopenia and Spares Megakaryocytes *In Vivo*

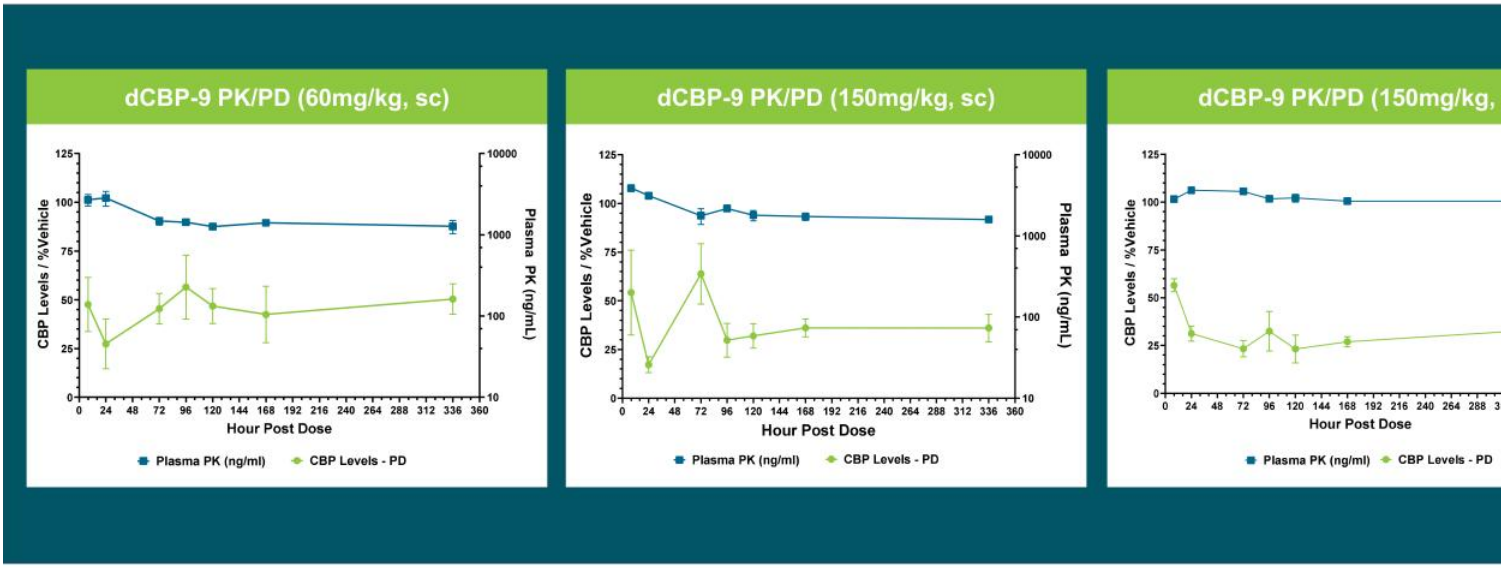
Platelet Counts Post Two Weeks of Dosing (*In Vivo*)



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



Selective EP300 Protein Degrader

For CBP Mutated and EP300 Dependent Cancers

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

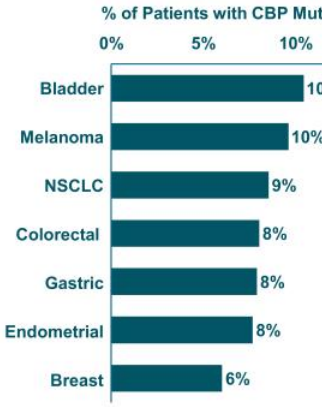
Target / Approach	• E1A binding protein p300 (EP300) • Targeted protein degrader
Initial Indications	• AR+ Prostate • DLBCL • Bladder, melanoma, others
Mutation / Aberration	• EP300 dependent cancers • CBP mutant cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

Commercial Opportunity

EP300 Dependent Cancers

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

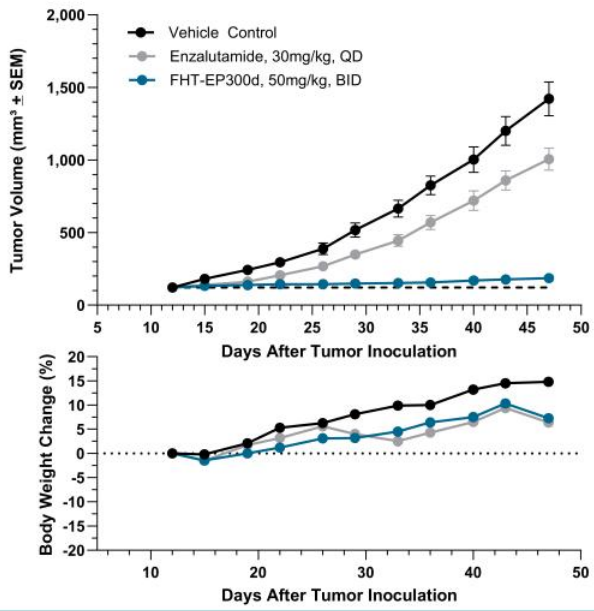
CBP Mutant Cancers



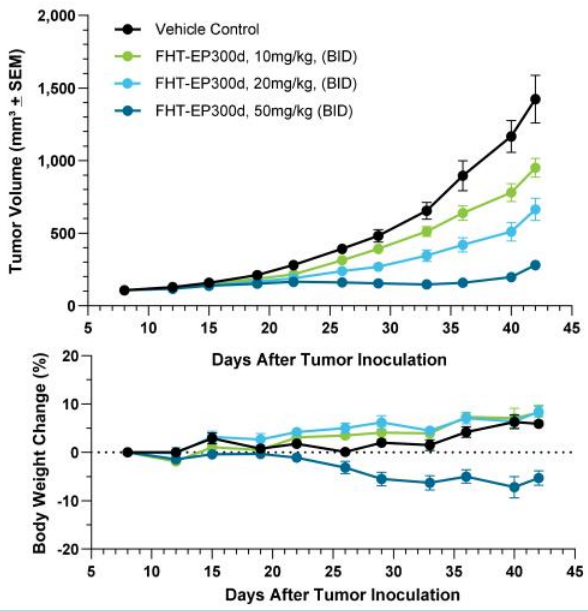
* Per year incidence in the U.S., EU5, Japan. Source: Clarivate DRG Mature Markets Data.

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

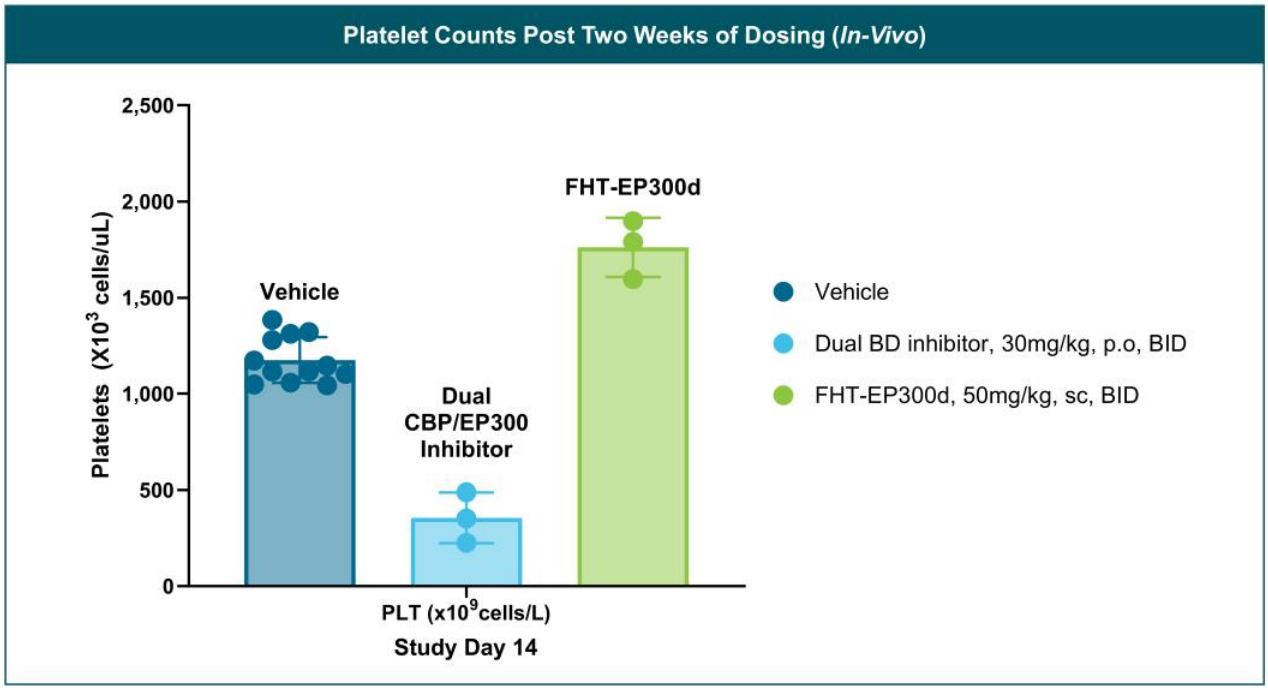
Prostate
AR+ VCAP Model



DLBCL
KARPAS422 (CBP Null) Model



Selective EP300 Degradation Does Not Show Thrombocytopenia *In Vivo*

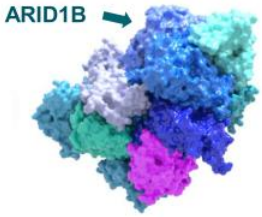


Selective ARID1B Protein Degrader

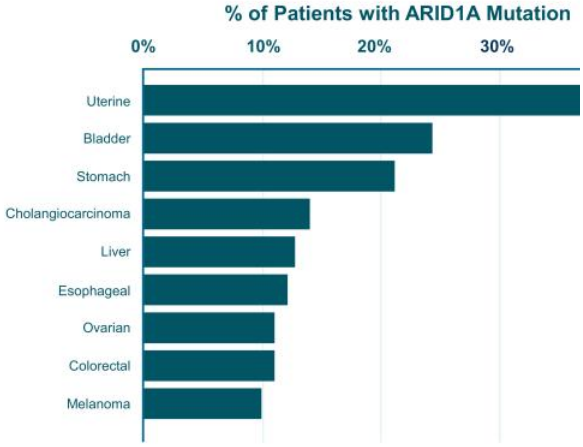
For ARID1A Mutated Cancers

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

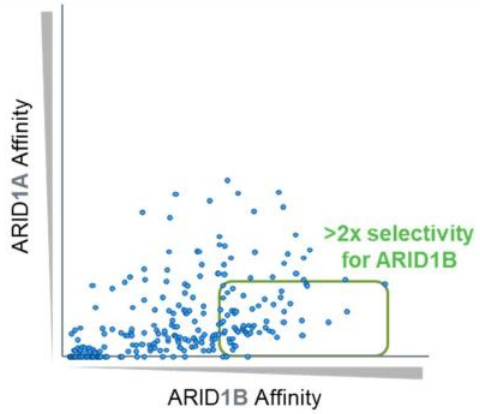


~5% of all solid tumors harbor ARID1A mutati

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

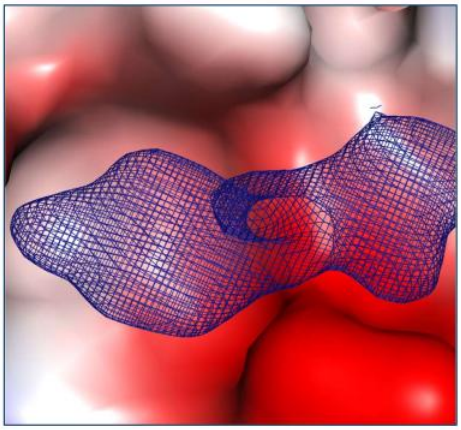
Compound Screening and Structure-Based Optimization Yielded Selective ARID1B Binders

Identification of Selective ARID1B Binders



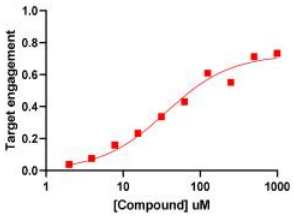
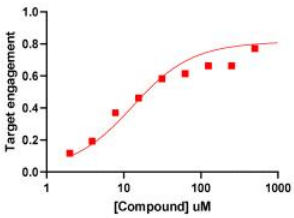
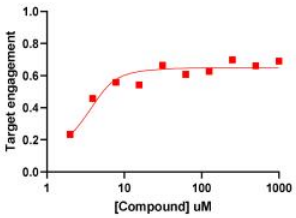
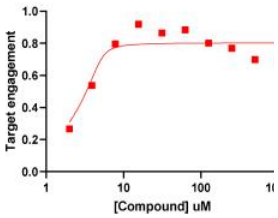
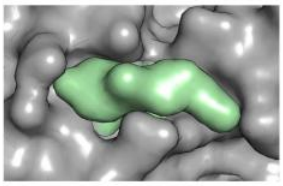
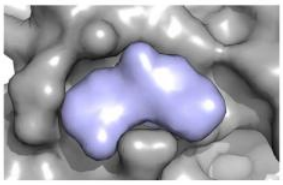
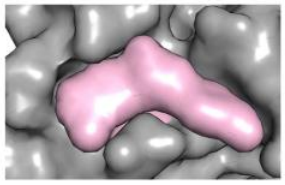
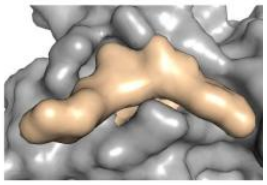
- Mapped and purified several potential ligandable regions of ARID, which were then screened against various compound libraries
- Characterized binding using multiple biochemical and biophysical techniques: e.g., DSF, ASMS, NMR, and SPR

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 Initiation	H2 2024
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

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, **IND cleared, Phase 1 initiation on track in H2'24**

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

Major Strateg Collaboratio

Strategic collaboratio Lilly; **$380 million up** 50/50 U.S. econor split on two lead prog

**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

Aug

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 % Chan
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 AI 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

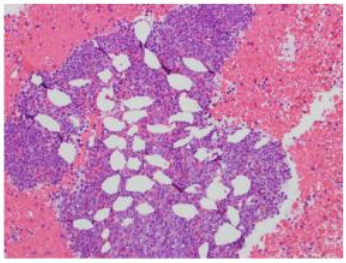
Prior non-AML treatment:

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

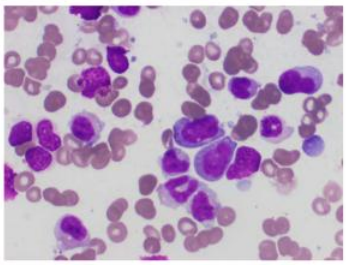
Initiation of FHD-286 at 10 MG Dose:

- 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 Differentiatio



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:

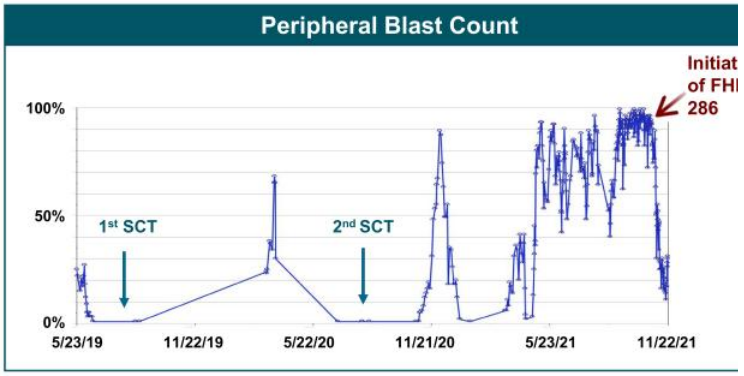
- Progressive disease with CNS Leukemia: 7 lines prior treatment and 2 bone marrow transplants

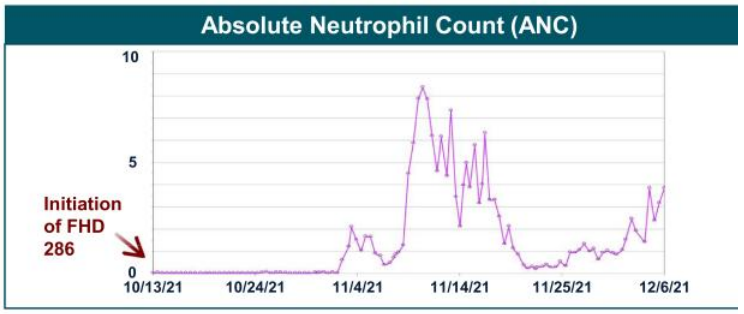
Prior non-AML treatment:

- 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…

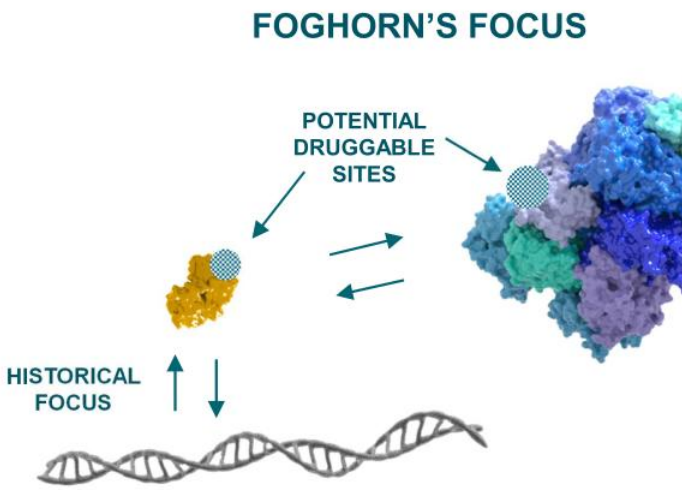
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…But Historically Difficult to Target…

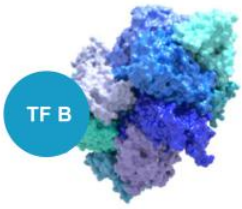
- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities

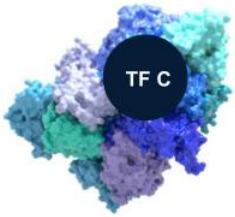
Foghorn Has a New Approach Focusing on Interaction with BAF

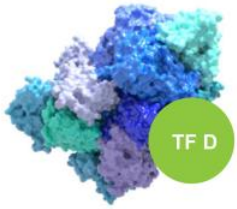
- Druggable binding pockets
- Druggable affinities



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

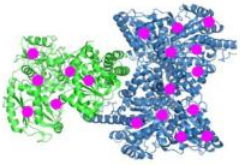




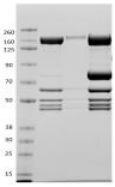


Mapping the TF-BAF Interaction

Mass spec. foot-printing



Pull-down assays

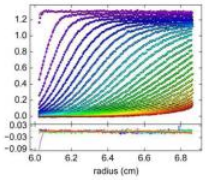


Foghorn's collection of BAF sub-complexes and domains

Validating the TF-BAF Interaction

Biophysical

AUC / SPR / ITC

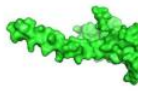


Biochemical

TR-FRET / FP

Structu

Crystal /



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Unique biology

Precision therapeutics

Broad impact

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