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): January 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 7.01 Regulation FD Disclosure.

On January 8, 2024, Foghorn Therapeutics Inc. (the "Company") issued a press release, a copy of which is furnished as Exhibit 99.1 to this Current Report on Form 8-K. Additionally, the Company is furnishing as Exhibit 99.2 to this Current Report on Form 8-K a presentation, dated January 2024, which the Company intends to use in meetings with or presentations to investors.

The information in this Item 7.01 (including Exhibits 99.1 and 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 January 8, 2024
99.2	Investor Presentation dated January 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/ Michael LaCascia
Michael J. LaCascia
Chief Legal Officer

Date: January 8, 2024

Foghorn Therapeutics Highlights Clinical Program Updates and Research Progress and Provides Strategic Objectives for 2024

- FHD-286 combination study in AML continues to progress in the clinic with data anticipated in the second half of 2024; preclinical combination data with FHD-286 and tyrosine kinase inhibitors (TKIs) in EGFR/KRAS resistance anticipated by the second quarter of 2024
- BRM selective inhibitor and degrader programs advancing in partnership with Loxo@Lilly
- Preclinical efficacy and safety demonstrated with selective EP300 and CBP programs; additional preclinical data anticipated in the second quarter of 2024; targeting IND enabling studies for CBP before end of 2024
- Foghorn anticipates at least six new INDs targeting significant oncology patient populations over the next four years, reflecting the continued productivity of its precision medicine platform
- Cash, cash equivalents, and marketable securities of $259.9 million, as of September 30, 2023, provides cash runway into the first half of 2026

CAMBRIDGE, Mass. -- (GLOBE NEWSWIRE) – January 08, 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 announced its strategic objectives for 2024.

“We enter 2024 with several important milestones ahead of us. We anticipate data from our Phase 1 combination study of FHD-286 in AML in the second half of the year and look forward to continued progress with our unique pipeline, including our BRM selective inhibitor and degrader programs in collaboration with Loxo@Lilly,” said Adrian Gottschalk, President and Chief Executive Officer. “We have made significant progress with our preclinical programs, including our selective EP300, CBP and ARID1B degrader programs and are getting ready to share more preclinical data in the first half of the year. This will also include preclinical combination data of FHD-286 with tyrosine kinase inhibitors of EGFR and KRAS. Over the next four years, we anticipate the filing of at least six new INDs, reflecting the productivity of our precision medicine platform. This is all supported by our cash and equivalents position of approximately $259.9 million as of September 30, 2023.”

- **FHD-286**. FHD-286 is a potent, selective inhibitor of the BRG1 and BRM subunits of the BAF chromatin remodeling complex where dependency on BRG1/BRM is well-established preclinically with multiple tumor types, including acute myelogenous leukemia (AML)/myelodysplastic syndrome (MDS), non–small cell lung cancer (NSCLC) and prostate cancer.
  - **AML Update.** Foghorn commenced a Phase 1 study of FHD-286 in combination with decitabine or low-dose cytarabine (LDAC) in relapsed and/or refractory AML patients, with the first patient dosed during the third quarter of 2023. The first clinical data are expected in the second half of 2024.
  - **TKI Resistance.** Recently published data, along with Foghorn’s work, suggest that FHD-286 may play an important role in overcoming resistance in EGFR/KRAS tumors. The company is conducting preclinical work to further explore the opportunity.

- **Differentiated Pipeline Advancement.** Foghorn continues to expand its platform and pipeline. The Company anticipates the potential for six new investigational new drug (IND) applications in the next four years. The Company continues to progress programs for multiple targets that include chromatin remodeling complexes, transcription factors, helicases and other chromatin-related factors. These targets include selective BRM* and wholly owned programs including CBP, EP300, and ARID1B, as well as other undisclosed targets, which combined could address more than 20 tumor types impacting more than 500,000 new patients annually.
    - **Selective EP300 and Selective CBP programs.** Foghorn presented new preclinical data for its EP300 and CBP selective degrader programs at Hanson Wade's 6th Annual Targeted Protein Degradation Summit on October 31, 2023.
        - EP300 selective degraders showed potent cellular antiproliferation and in vivo tumor growth inhibition in an AR+ enzalutamide prostate in vivo model.
        - CBP selective degraders demonstrated significant tumor growth inhibition in a colorectal cancer in vivo model. Antiproliferative effects were also observed for numerous cancer cell lines, including colorectal, gastric and bladder cancers.
        - At preclinical efficacious doses, neither the EP300 nor the CBP selective degraders caused thrombocytopenia, a commonly observed safety liability for dual CBP/EP300 inhibitors.
        - Additional preclinical data will be presented in the first half of 2024.
- ***Loxo@Lilly Collaboration.** Foghorn is engaged in a strategic collaboration with Loxo@Lilly and continues to advance the BRM selective inhibitor and degrader programs along with other undisclosed programs.
- **Strong Balance Sheet and Cash Runway**. As of September 30, 2023, the Company had $259.9 million in cash, cash equivalents and marketable securities providing cash runway into the first half of 2026.

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 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 are 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, including statements regarding potential combination trials involving FHD-286, the progress of our Loxo@Lilly collaboration, and our proprietary pre-clinical programs. 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, 2022, 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.

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Exhibit 99.2

FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

January 2024

Forward Looking Statements

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “could,” “may,” “might,” “will,” “likely,” “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” or the negative of these terms or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements include, but are not limited to, statements concerning: the potential outcomes from our collaboration agreements with Lilly; the initiation, timing, progress and results of our research and development programs and pre-clinical studies and clinical trials, including with respect to our Phase 1 study of FHD-286 in combination with decitabine or cytarabine in relapsed and/or refractory AML patients and anticipated timing of release of clinical data; our ability to advance product candidates that we may develop and to successfully complete preclinical and clinical studies; our ability to leverage our initial programs to develop additional product candidates using our Gene Traffic Control Platform; the impact of exogeneous factors, including macroeconomic and geopolitical circumstances, on our and our collaborators’ business operations, including our research and development programs and pre-clinical studies; developments related to our competitors and our industry; our ability to expand the target populations of our programs and the availability of patients for clinical testing; our ability to obtain regulatory approval for FHD-286 and any future product candidates from the FDA and other regulatory authorities; our ability to identify and enter into future license agreements and collaborations; our ability to continue to rely on our CDMOs and CROs for our manufacturing and research needs; regulatory developments in the United States and foreign countries; our ability to attract and retain key scientific and management personnel; the scope of protection we are able to establish, maintain and enforce for intellectual property rights covering FHD-286, our future products and our Gene Traffic Control Platform; and our use of proceeds from capital-raising transactions, estimates of our expenses, capital requirements, and needs for additional financing. Any forward-looking statements represent the Company’s views only as of today and should not be relied upon as representing its views as of any subsequent date. The Company explicitly disclaims any obligation to update any forward-looking statements. The Company’s business is subject to substantial risks and uncertainties.

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

Broad pipeline across a range of targets and modalities

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

Well-Funded

$259.9 million in cash and equivalents
(as of 09/30/2023)

Provides **runway into H1'26**

Value Drivers

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

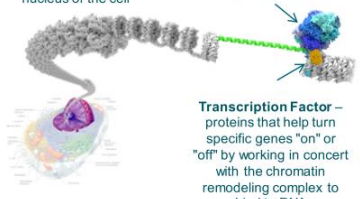
Advancement of preclinical assets (BRM-Selective, CBP, EP300, ARID1B) towards INDs

Major Strategic Collaboration

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

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

Chromatin Regulatory System Critical for Gene Expression



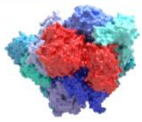
Chromatin – compacted form of DNA inside the nucleus of the cell

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

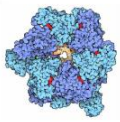
Novel Targets Guided by Genetic Dependencies

Chromatin Remodeling Complex Mutations / Overexpression



Transcription Factor Mutations / Overexpression

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



Tailored Drugging Approaches

Enzymatic Inhibitors
Highly selective and allosteric small molecule inhibitors

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

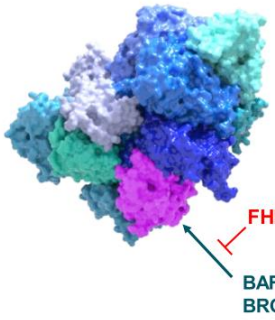
Enzymatic Inhibitors	Targeted Protein Degraders	Transcription Factor Disruptors

Broad and Deep Pipeline Across a Range of Targets and Modalities

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

Significant Opportunity

- ~27,000 drug treated relapsed and/or refractory (R/R) AML patients*
- No broad differentiation agent approved in AML
- Significant combination potential

*U.S., EU5, Japan

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

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

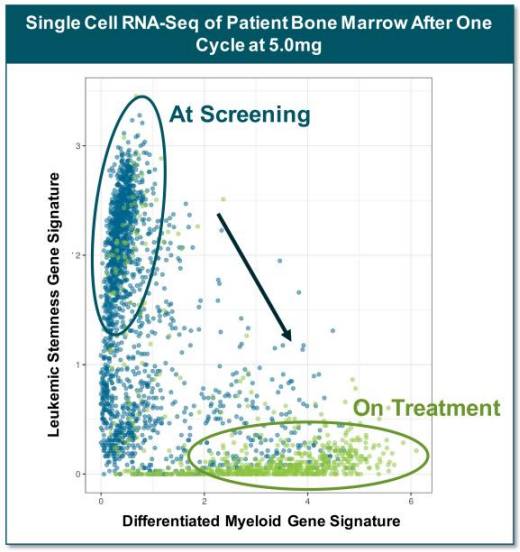
Dose Level	Mutations	Cytogenetics Risk	Starting CD11b%	Max CD11b%	CD11b+ Fold Change	Starting CD34%	Min CD34%	CD34+ % Decrease
10mg	N/A	Adverse	7	62	9.2x	94	27	(71%)
7.5mg	CBFB (locus at 16q22)		2	94	59.4x	70	2	(97%)
7.5mg	KMT2A rearrangement	Adverse	3	58	21.4x	85	9	(90%)
7.5mg	RUNX1, KRAS, ASXL1, JAK2, 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 (leukemic stem cell marker) decreases

CD11b (marker of differentiation) increases

Clinical Patient Samples Show Loss of Leukemic Stem Cell Identity and Transformation to Differentiated Marrow

- **At Screening:** Bone marrow heavily infiltrated with leukemic stem cell-like blasts.
- **On Treatment:** Bone marrow has lost leukemic stem cell phenotype and shifted to a more mature phenotype.
- Supportive & recapitulated pre-clinical data of FHD-286's impact on leukemic stem cell potential.
- Similar effects observed across dose levels (i.e., 5.0mg, 7.5mg and 10.0mg).



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

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

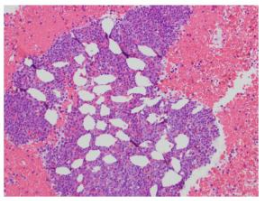
Prior non-AML treatment:

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

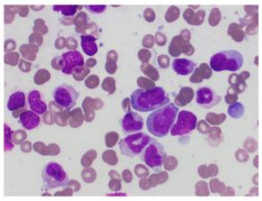
Initiation of FHD-286 at 10 MG Dose:

- 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



Meaningful Clinical Benefit in Heavily Pre-Treated Patient

Patient Background:

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

Prior AML Treatment:

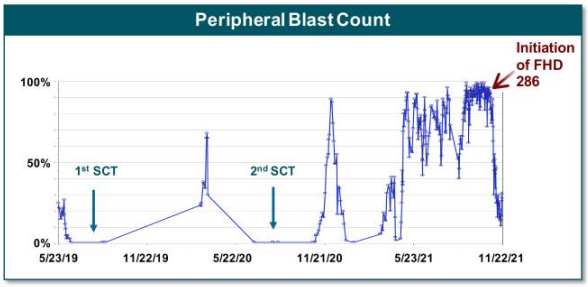
- Progressive disease with CNS Leukemia: 7 lines prior treatment and 2 bone marrow transplants

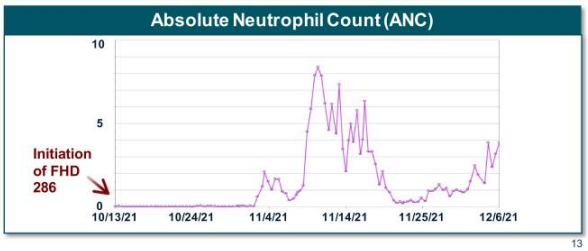
Prior non-AML treatment:

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

Initiation of FHD-286 at 10 MG Dose:

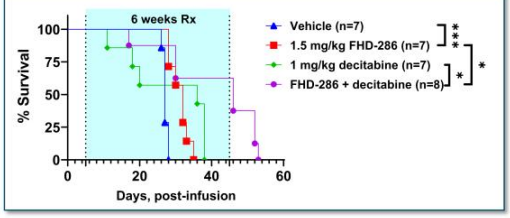
- Drop in peripheral blast, 97% to 5%
- Bone marrow reduction from 89% to 48%, with ANC recovery



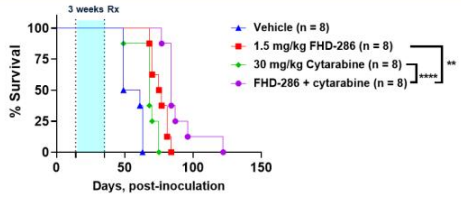


Pre-Clinical Data Demonstrate Significant Combination Potential with Multiple Agents in AML

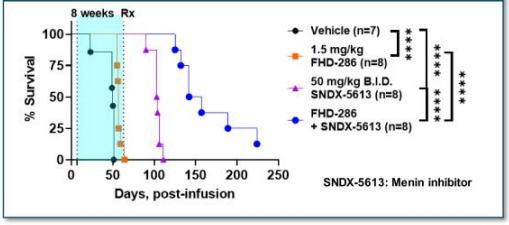
FHD-286 + decitabine
(MLL-AFP + FLT3 TKD Luc/GFP PDX)



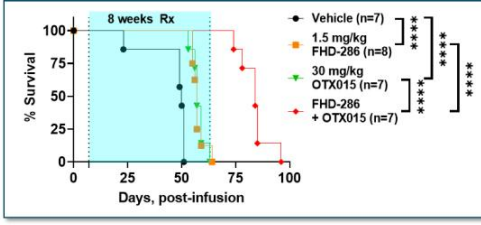
FHD-286 + cytarabine
(MV4, 11 FLT3 ITD CDX)



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

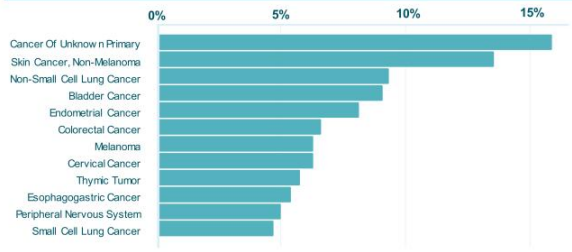
Summary: Advancing Selective BRM Inhibitor and Degrader Implicated in Up to 5% of All Solid Tumors with BRG1 Mutations

Partner / Approach	• BRM Selective Programs part of the Loxo@Lilly collaboration
	• Two drugging approaches:
	  • Selective Inhibition
	  • Selective Degradation

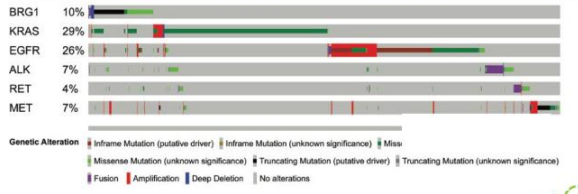
Opportunity	• BRG1 mutated cancer • ~8-10% of NSCLC, bladder, endometrial, colorectal • > 100,000 patients per year*
Stage	• Transitioned the BRM Selective inhibitor program to Loxo@Lilly in Q3'23
Economics of Lilly Collaboration	• 50/50 U.S. economics • Tiered ex-U.S. royalties starting in the low double-digit range and escalating into the twenties

* Per year incidence in the U.S., EU5, Japan

Large Commercial Opportunity

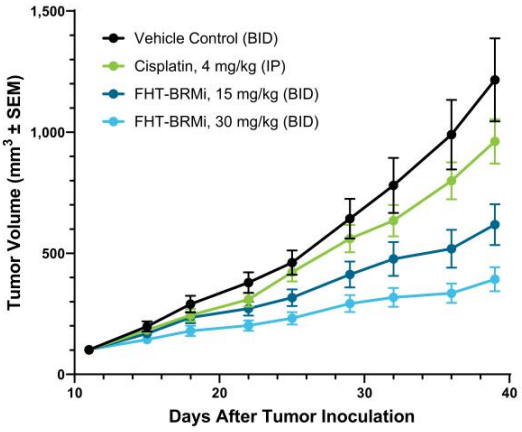


10% of NSCLC Tumors, Minimal Overlap with Other Mutations

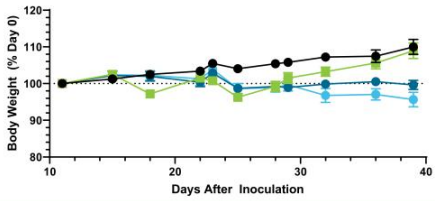


BRM Selective Inhibitor Demonstrates PK/PD and *In Vivo* Efficacy in a BRG1 Mutant Lung CDX Model

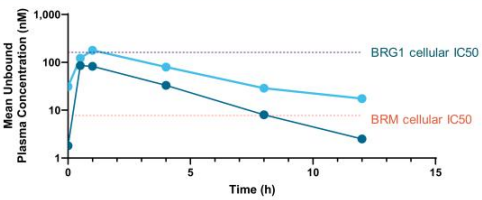
A549-BRG1 Mutant NSCLC Model



Body Weight Loss

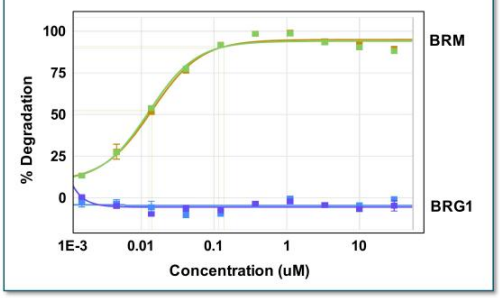


PK/PD

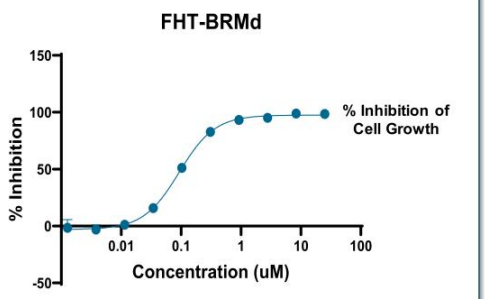


BRM Selective Degrader Achieves Complete BRM Degradation and Cell Growth Inhibition

BRM/BRG1 HIBIT Data



A549 Ten-Day Proliferation Assay



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

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

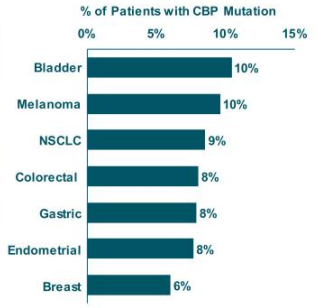
* Per year incidence in the U.S., EU5, Japan

Commercial Opportunity

EP300 Dependent Cancers

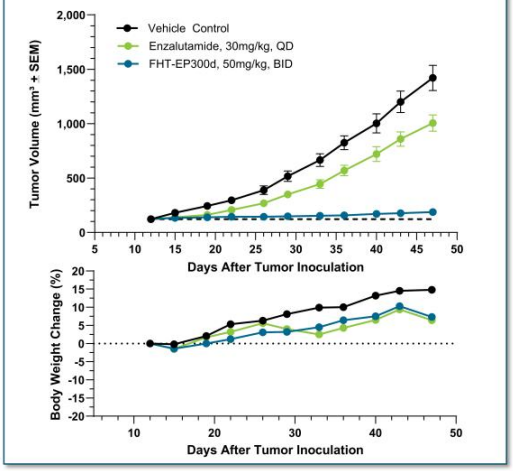
- **Solid Tumors**
  - AR+ mCRPC
  - HR+ breast
- **Hematologic malignancies**
  - DLBCL
  - Multiple Myeloma

CBP Mutant Cancers

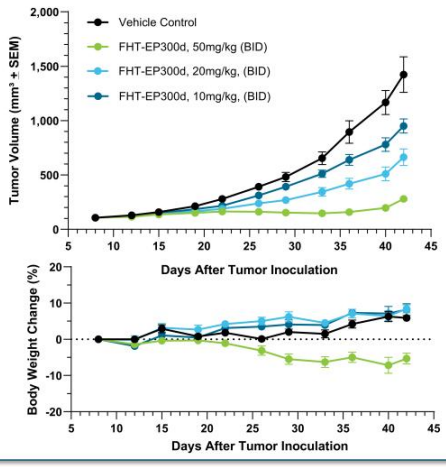


EP300 Degradation Results in Significant Tumor Growth Inhibition in AR+ VCAP Prostate and KARPAS422 DLBCL Models

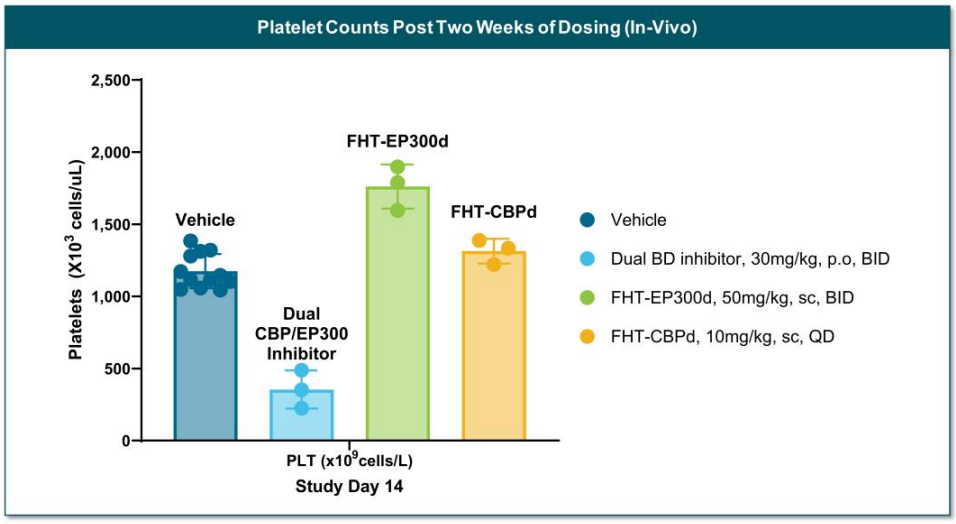
Prostate
AR+ VCAP Model



DLBCL
KARPAS422 (CBP Null) Model



Selective Degradation of EP300 and CBP Does Not Show Thrombocytopenia in Mice at Relevant Doses



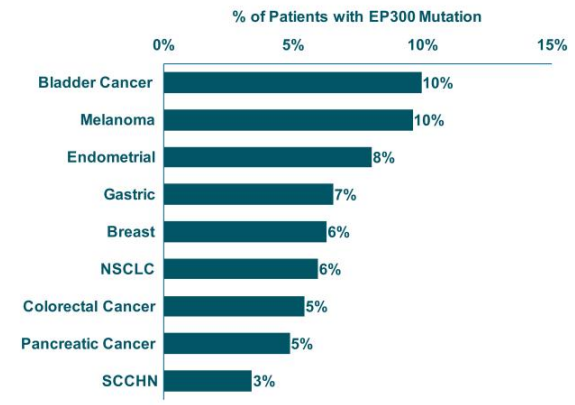
Selective CBP Protein Degrader

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

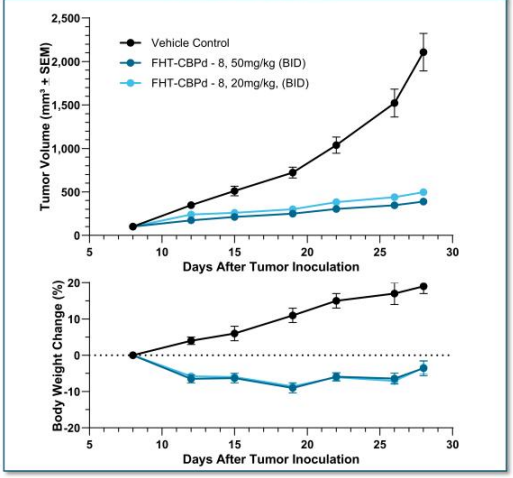
Commercial Opportunity



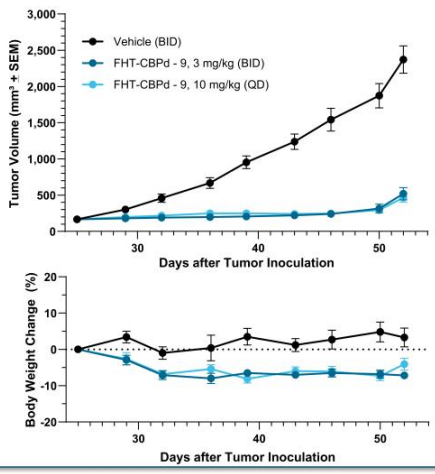
* Per year incidence in the U.S., EU5, Japan

Selective CBP Protein Degrader Result in Significant Tumor Growth Inhibition in Colorectal and Bladder EP300 Null Models

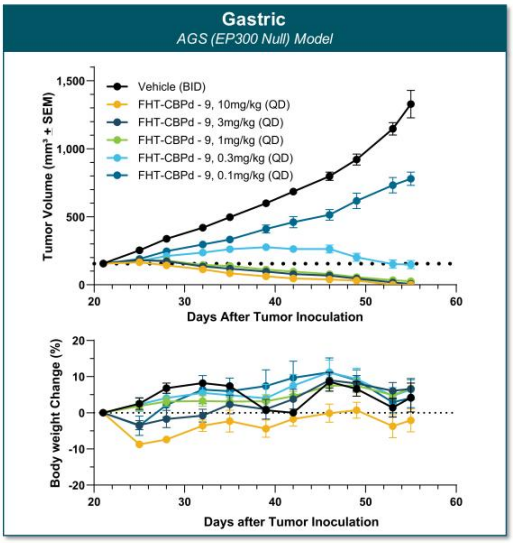
Colorectal
RKO (EP300 Null) Model



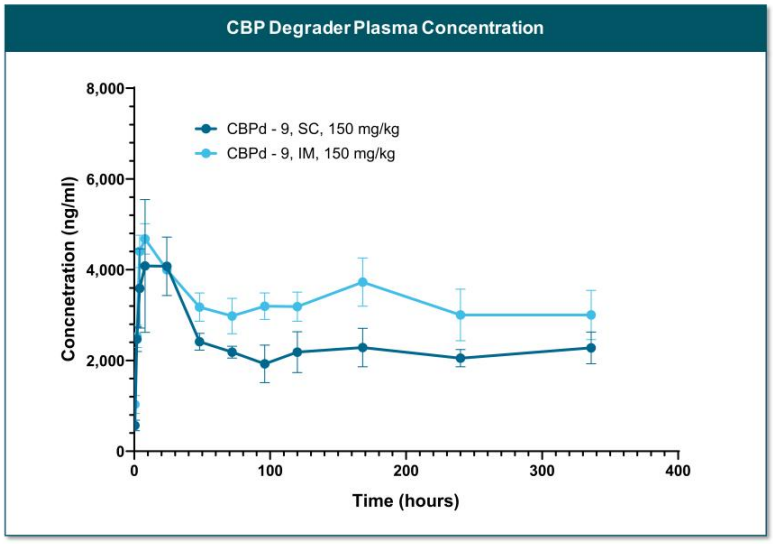
Bladder
639V (EP300 Null) Model



Selective CBP Protein Degrader Result in Tumor Regression in Gastric EP300 Null Models



Long-Acting Injectable Formulations of CBP Degrader Could Enable Once Every 2 Weeks (or Better) Dosing Frequency

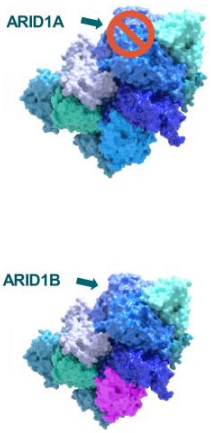


Selective ARID1B Protein Degrader

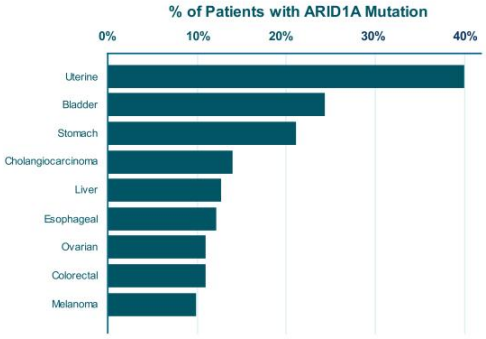
For ARID1A Mutated Cancers

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

Targeting ARID1B for ARID1A Mutated Cancers is Enabled by Foghorn's Unique Biology and Discovery Capabilities

Gene Traffic Control Platform

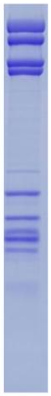
- Platform produces BAF complexes and subcomplexes containing either ARID1A or ARID1B at scale
- Enables proprietary screens against ARID1B

Protein Degrader Capabilities

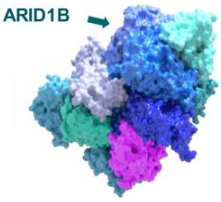
- Utilize protein degrader toolbox to create ARID1B hetero-bifunctional degraders

Program Status

- Validated selective chemical binders of ARID1B
- In process of expanding binders into novel selective protein degraders
- Assessing outcomes of ARID1B degradation and impact on BAF complex formation



Highly purified ARID1B / BAF complex



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…

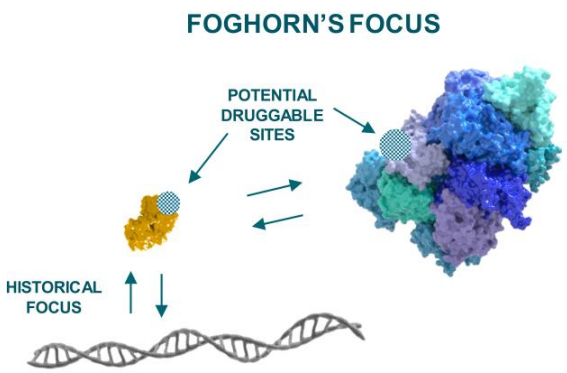
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…But Historically Difficult to Target…

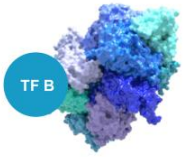
- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities

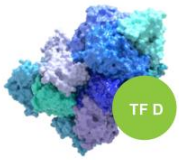
Foghorn Has a New Approach Focusing on Interaction with BAF

- Druggable binding pockets
- Druggable affinities



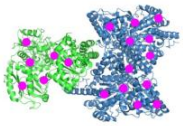
Transcription Factors Bind to BAF Directly with High Degree of Specificity; Unique Insights into Where and How Transcription Factors Bind



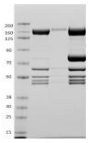


Mapping the TF-BAF Interaction

Mass spec. foot-printing



Pull-down assays

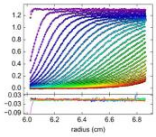


Foghorn's collection of BAF sub-complexes and domains

Validating the TF-BAF Interaction

Biophysical

AUC / SPR / ITC

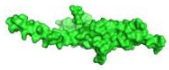


Biochemical

TR-FRET / FP

Structural

Crystal / NMR



Broad and Deep Pipeline Across a Range of Targets and Modalities

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

Broad pipeline across a range of targets and modalities

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

Well-Funded

$259.9 million in cash and equivalents

(as of 09/30/2023)

Provides **runway into H1'26**

Value Drivers

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

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

Major Strategic Collaboration

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