

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): June 8, 2022

Foghorn Therapeutics Inc.
(Exact name of registrant as specified in its charter)

Delaware
(State or other jurisdiction of incorporation)

001-39634
(Commission
File Number)

47-5271393
(IRS Employer Identification No.)

500 Technology Square, Ste 700
Cambridge, MA
(Address of principal executive offices)

02139
(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 144-12 under the Exchange Act (17 CFR 240.144-12)
- 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.



FCGHORN[®]

THERAPEUTICS

CORPORATE OVERVIEW

Leveraging unique insights into the chromatin regulatory system to pioneer a new class of precision therapies in oncology and beyond

June 2022

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 the Collaboration Agreement with Lilly; the initiation, timing, progress and results of our research and development programs and preclinical and clinical trials, including the potential resolution of the partial clinical hold and anticipated timing of release of initial clinical data; our ability to advance product candidates that we may develop and 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 the COVID-19 pandemic on our and our collaborators' business operations, including our research and development programs and preclinical 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, FHD-609 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 and FHD-609, 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 CANCER AND OTHER DISEASES



LARGE MARKET POTENTIAL

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

Potential applications in virology, autoimmune diseases and neurology



WELL-FUNDED

\$424.7 million in cash and equivalents
(as of 3/31/2022)



UPCOMING MILESTONES

FHD-286: Initial clinical data (TBD) - mUM study enrolling; AML/MDS study on partial clinical hold

FHD-609: Initial clinical data expected in 2023



SIGNIFICANT GLOBAL PARTNERSHIPS

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

Merck collaboration to drug single specified transcription factor target; \$15 million upfront and up to \$410 million in milestones



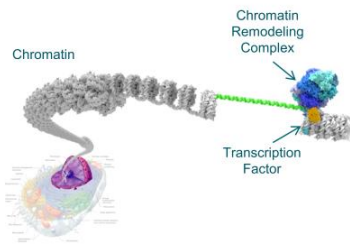
EXPERIENCED LEADERSHIP TEAM

Expertise across drug discovery, clinical development and commercialization

UNIQUE INSIGHTS INTO CHROMATIN BIOLOGY

Untapped Area for Novel Targets and Therapeutics

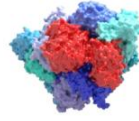
CHROMATIN REGULATORY SYSTEM CRITICAL FOR GENE EXPRESSION



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

NOVEL TARGETS GUIDED BY GENETIC DEPENDENCIES

Chromatin Remodeling Complex Mutations / Overexpression



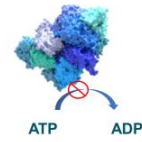
Transcription Factor Mutations / Overexpression

Mutations That Impinge on the Chromatin Regulatory System



TAILORED DRUGGING APPROACHES

Targeted Protein Degradation: Bi-functional protein degraders for targets with no enzymatic activity



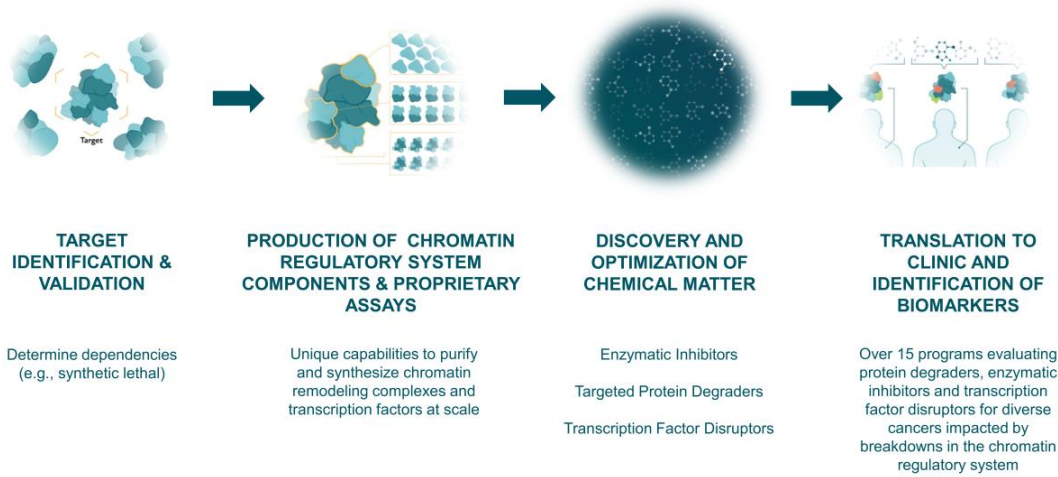
Enzymatic Inhibitors: Highly selective and allosteric small molecule inhibitors

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



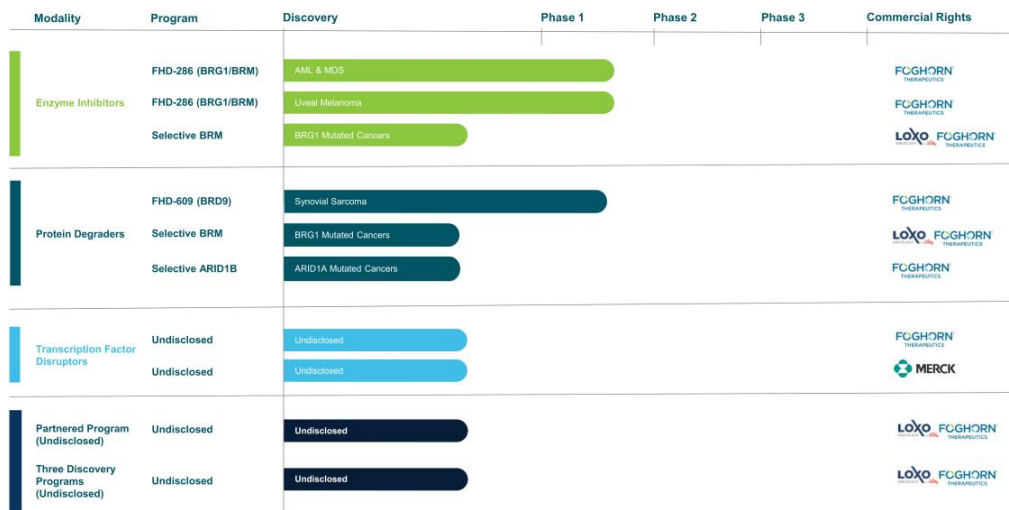
FOGHORN'S GENE TRAFFIC CONTROL® PLATFORM

Integrated, Scalable, Efficient – Repeatable Paradigm



BROAD PIPELINE ACROSS A RANGE OF TARGETS AND MODALITIES

Precision Oncology / Breadth and Depth / Over 15 Programs



FHD-286

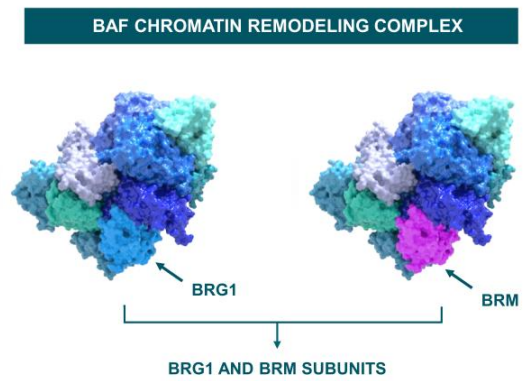
In Phase 1 Dose Escalation for AML / MDS & Uveal Melanoma

FHD-286 is a Potent, Selective, Allosteric, Small Molecule Inhibitor of the BRG1 and BRM subunits of the BAF complex

FHD-286 TARGETS ABNORMAL DEPENDENCIES ON BAF IN CANCER

Target / Approach	<ul style="list-style-type: none"> BRG1 / BRM ATPase Small molecule, allosteric, oral enzymatic inhibitor
Indications	<ul style="list-style-type: none"> Acute myelogenous leukemia (AML) and myelodysplastic syndromes (MDS) Uveal melanoma Indication expansion work ongoing in multiple solid tumors
Mutation / Aberration	<ul style="list-style-type: none"> AML: Elevated BRG1-BAF / TF activity in AML blast cells Uveal melanoma: GNAQ / GNA11 mutated UM is driven by dependency on BAF / TF activity
Program Status / Milestones	<ul style="list-style-type: none"> Phase 1 studies enrolling in mUM; partial clinical hold for AML/MDS study Initial clinical data (TBD)
New Patients Impacted / Year*	<ul style="list-style-type: none"> AML: Over 20,000 relapsed and / or refractory patients MDS: Over 7,000 higher risk MDS patients Uveal melanoma: Over 5,000 patients

* U.S., EU5, Japan



- BRM / BRG1 is the engine (ATPase) of the BAF chromatin remodeling complex
- BRG1 & BRM are highly similar proteins

FHD-286 CLINICAL DEVELOPMENT PLAN

Two Parallel Phase 1 Studies

PHASE 1 DOSE ESCALATION STUDIES

- Relapsed / Refractory AML & MDS
- Metastatic Uveal Melanoma

Phase 1 Study Designs

- Single patient accelerated titration (n=1)
- Convert to 3+3 once relevant PK / PD, safety or clinical activity observed
- Retrospective biomarker analysis to further evaluate safety and efficacy
- Assess safety, PK, biomarkers and efficacy

PHASE 1 EXPANSION STUDIES

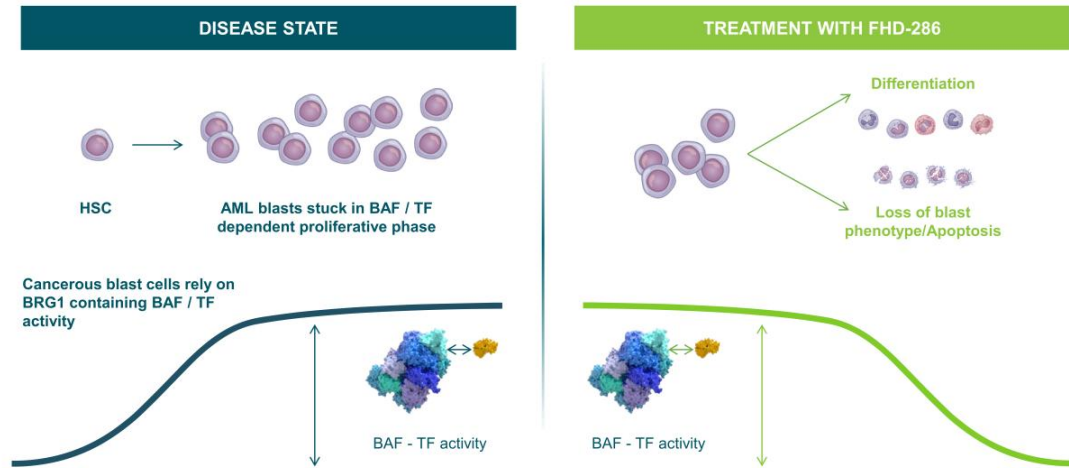
- Expansion cohorts in AML, UM and potentially other indications

POTENTIAL DEFINITIVE EFFICACY TRIALS & INDICATION EXPANSION

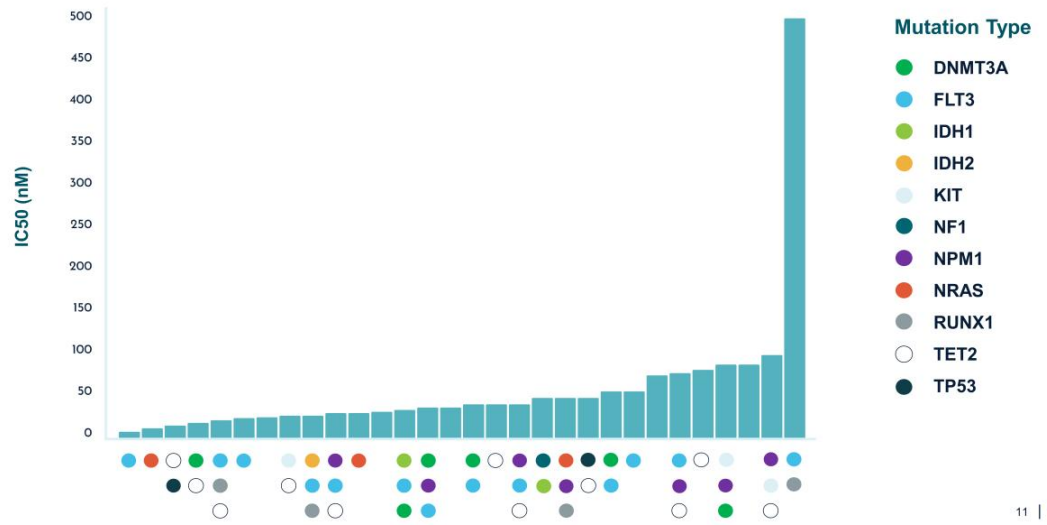
- Potential for entry into definitive efficacy trials in AML
- Potential for entry into definitive efficacy trials in metastatic UM
- Potential for indication expansion beyond AML and UM

Initial Clinical Data: Timing Dependent on Resolution of AML/MDS Study Partial Clinical Hold

AML: DEPENDENCY ON BRG1 / LINEAGE TF INTERACTIONS



FHD-286 SHOWS EFFECT ACROSS A BROAD RANGE OF MUTATIONS IN AML PATIENT-DERIVED SAMPLES



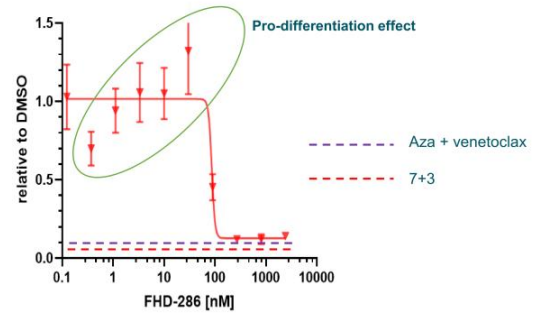
PRECLINICAL FHD-286 DATA SHOWS EFFICACY ACROSS AML PATIENT-DERIVED SAMPLES

Notable Patient ID	Deep Response	Pathology Review	Disease Status
1690AML1	Y	AML	Secondary
1695AML1	Y	AML/MDS	Secondary
1696AML1	Y	AML	Secondary
1701AML1	Y	AML	Secondary
1893AML1	Y	AML	R/R
1899AML1	Y	AML	R/R
1990pAML1	Y	AML	R/R
1991pAML1	Y	AML	de novo
2041AML1	Y	N/A	de novo
2043pAML1	Y	AML	R/R
2059AML1	Y	AML	R/R
1682AML1	~	N/A	N/A
1689AML1	~	AML/MDS	de novo
1684AML1	N	CML	R/R
1924AML1	N	AML/MDS	R/R

Y = Deep reduction in blast cells ~ = Partial reduction N = No response



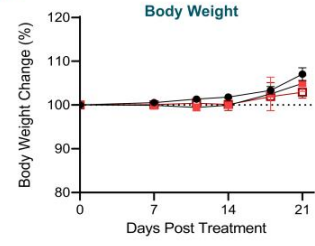
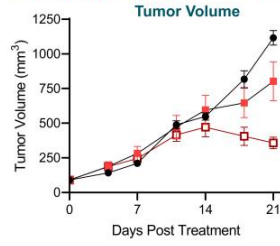
1695AML1 – BM-secondary AML



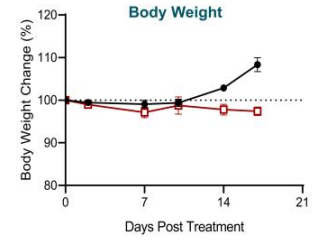
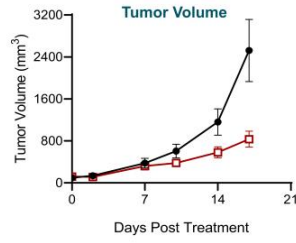
- Response observed in a majority of primary AML samples, irrespective of prior treatment or disease stage
- Additional data set from patient-derived samples demonstrate mutation-agnostic responses

DOSE-DEPENDENT TUMOR GROWTH INHIBITION OBSERVED WITH FHD-286 TREATMENT IN AML CDX MODELS

MV4-11 CDX Model
(FLT3 ITD, MLL-AF4)



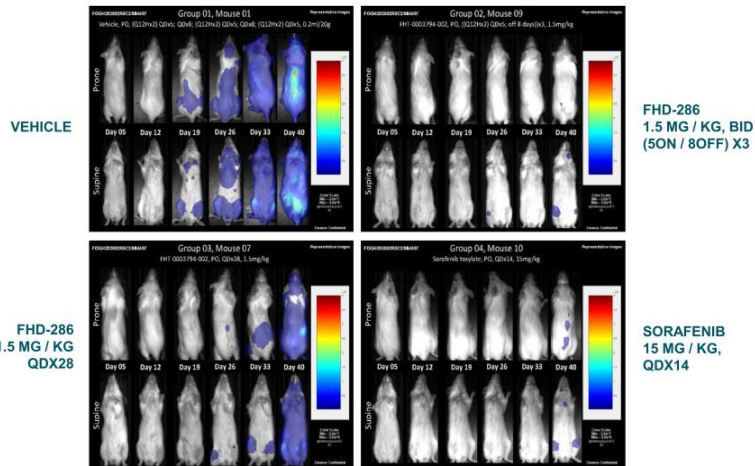
OCI-AML2 CDX MODEL
(MII-AF6, DNMT3A MUT.)



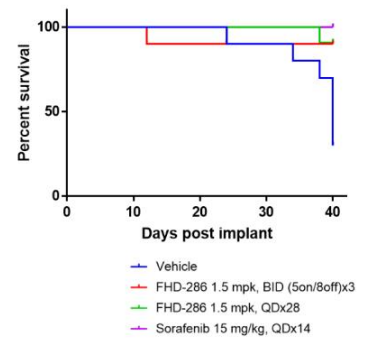
● Vehicle ■ FHD-286, 0.5 mg/kg, PO, QD ■ FHD-286, 1.5 mg/kg, PO, QD

TUMOR GROWTH INHIBITION WITH FHD-286 TREATMENT OBSERVED BY BIOLUMUMINESCENCE

Imaging in a Disseminated AML Model

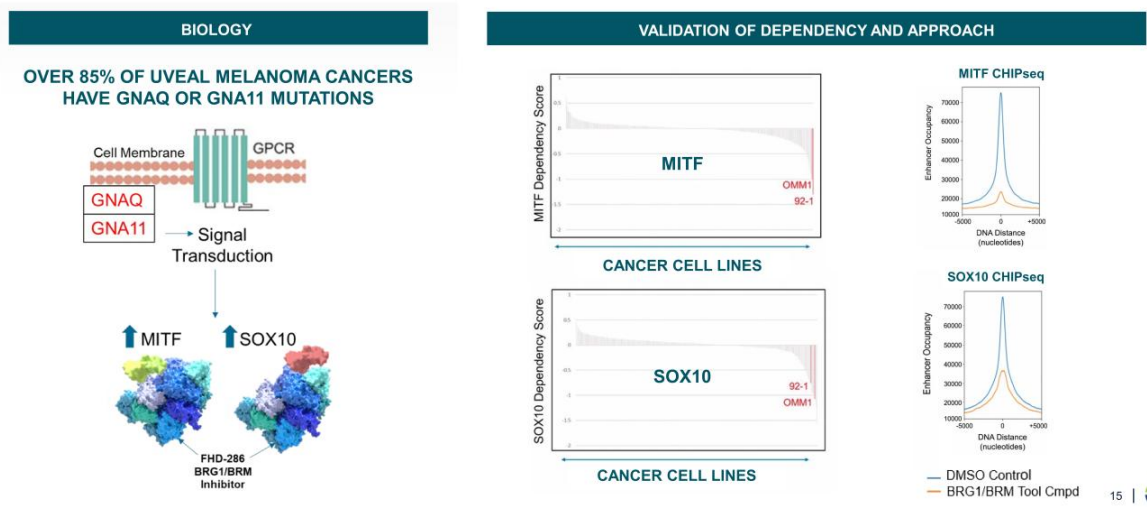


FHD-286 SURVIVAL ADVANTAGE IN DISSEMINATED AML MODEL



THERAPEUTIC RATIONALE FOR UVEAL MELANOMA

Dependency on Two Lineage Transcription Factors MITF / SOX10

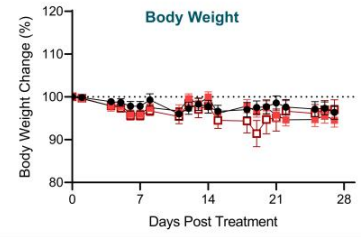
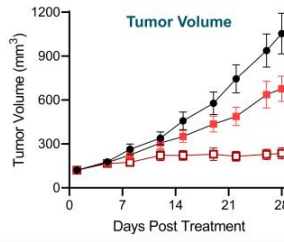


FHD-286 WAS ASSOCIATED WITH DOSE-DEPENDENT TUMOR REGRESSION IN UVEAL MELANOMA CDX MODELS AT TOLERATED DOSES

MP-46 UVEAL MELANOMA CDX MODEL

Dose-dependent tumor growth inhibition

Well-tolerated

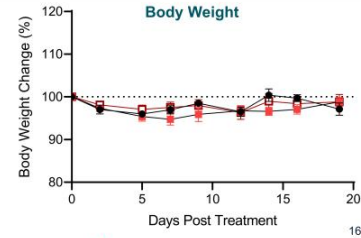
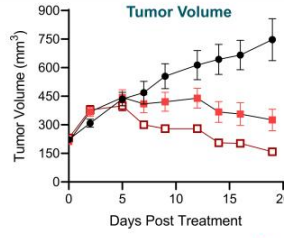


92-1 UVEAL MELANOMA CDX MODEL

Dose-dependent tumor growth inhibition

Tumor regression at 1.5 mg / kg, PO, QD

Well-tolerated

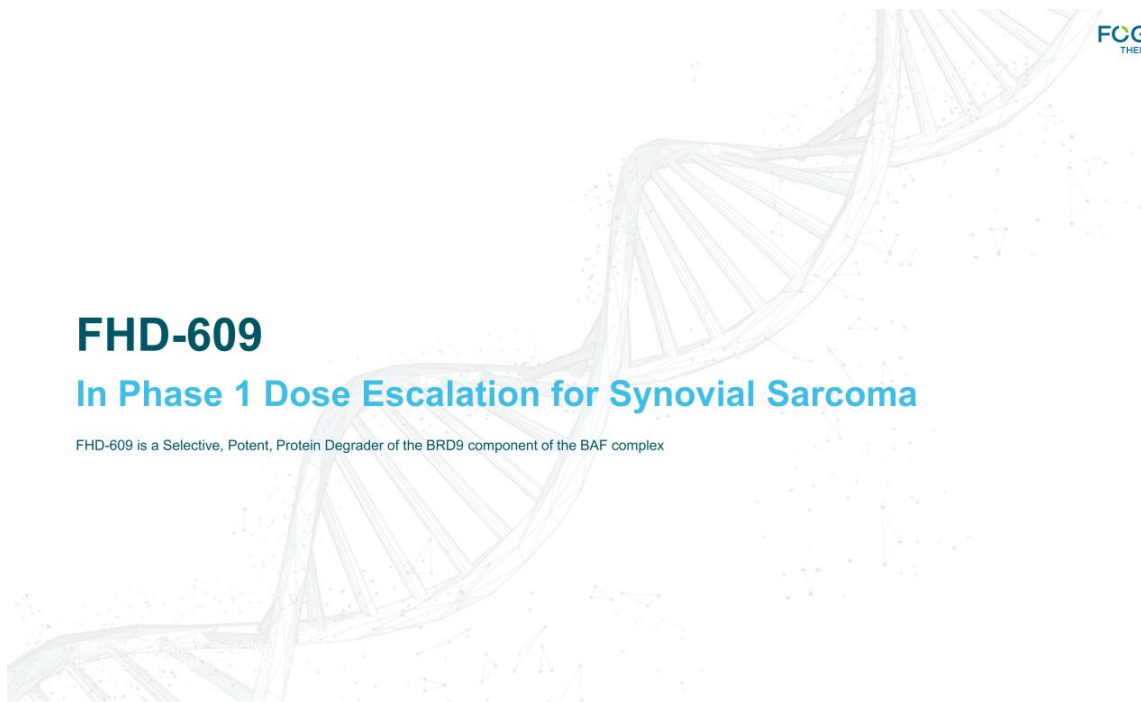


● Vehicle ■ FHD-286, 0.5 mg/kg, PO, QD ■ FHD-286, 1.5 mg/kg, PO, QD

FHD-609

In Phase 1 Dose Escalation for Synovial Sarcoma

FHD-609 is a Selective, Potent, Protein Degradator of the BRD9 component of the BAF complex

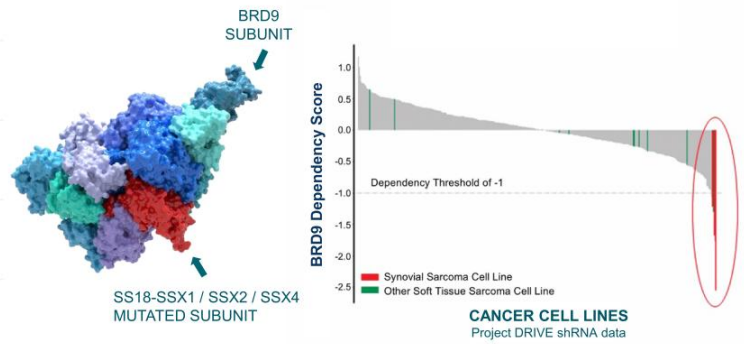


FHD-609 TARGETS AND DEGRADES THE BRD9 SUBUNIT OF BAF WHICH IS REQUIRED FOR SYNOVIAL SARCOMA CELLS TO SURVIVE

Selective, Potent BRD9 Targeted Protein Degradator

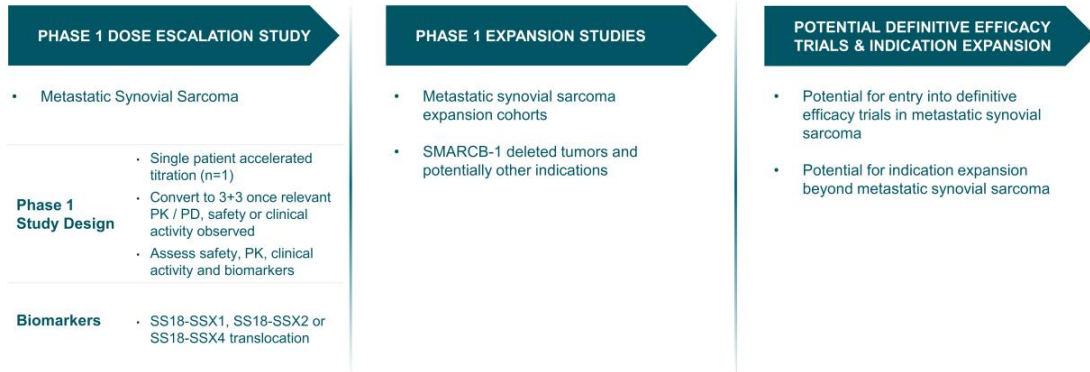
Target / Approach	<ul style="list-style-type: none"> BRD9 Intravenous protein degrader
Initial Indication	<ul style="list-style-type: none"> Synovial sarcoma
Mutation / Aberration	<ul style="list-style-type: none"> SS18-SSX1 / SSX2 / SSX4 protein fusions
Program Status / Milestones	<ul style="list-style-type: none"> Initial clinical data expected 2023
New Patients Impacted / Year*	<ul style="list-style-type: none"> Synovial sarcoma: Over 1,800

BRD9 IS REQUIRED FOR THE SURVIVAL OF SYNOVIAL SARCOMA CELLS



* U.S., EU5, Japan

FHD-609 CLINICAL DEVELOPMENT PLAN



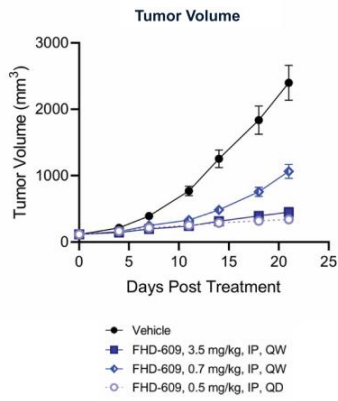
Initial Clinical Data: Expected in 2023

ROBUST *IN VIVO* ACTIVITY OBSERVED IN SYNOVIAL SARCOMA MODEL AND BRD9 DEGRADATION ASSOCIATED WITH FHD-609 TREATMENT

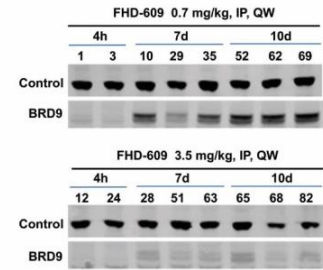
Weekly Dosing of FHD-609 Achieved Sustained BRD9 Degradation

SY01 SYNOVIAL SARCOMA CDX MODEL

- Mutation: **SS18-SSX2**
- Inhibited tumor growth
- Dose-dependent BRD9 degradation correlated with anti-tumor activity



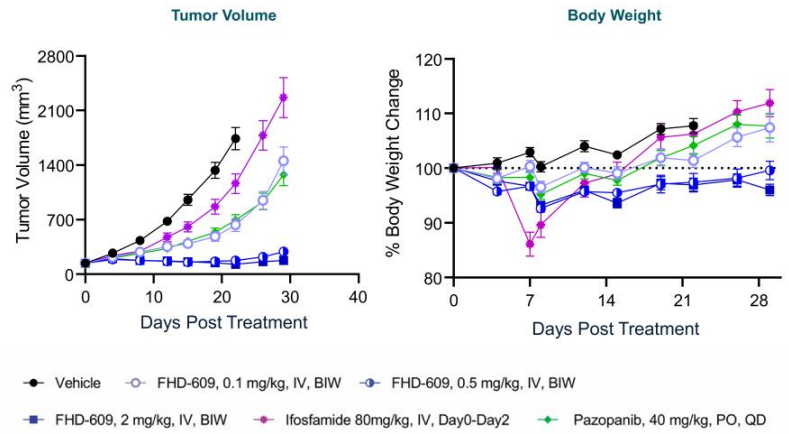
SUSTAINED BRD9 DEGRADATION



SUPERIOR TUMOR GROWTH INHIBITION WITH FHD-609 IN A SYNOVIAL SARCOMA MODEL AS COMPARED TO IFOSFAMIDE AND PAZOPANIB

ASKA CDX MODEL

- Mutation: **SS18-SSX1**
- Superior tumor growth inhibition compared to ifosfamide and pazopanib
- Complete suppression observed over 30 days at 2 mg / kg of FHD-609



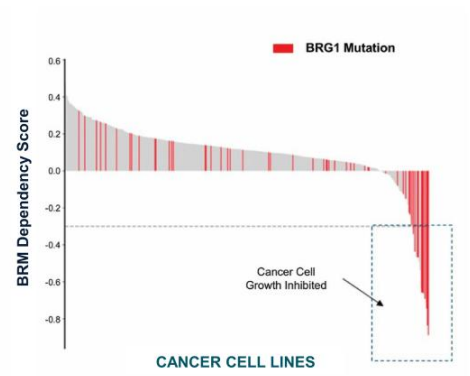
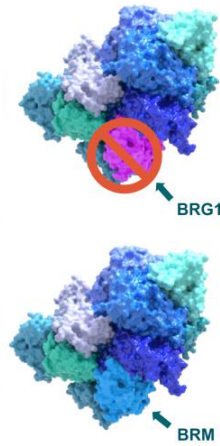
SELECTIVE BRM MODULATORS FOR BRG1 MUTATED CANCERS

Enzymatic Inhibitor and Protein Degradation Programs targeting BRG1 mutated cancers (e.g., NSCLC), 30+ cancers with BRG1 mutations

BRG1 MUTATIONS CREATE A GENETIC DEPENDENCY ON BRM

Selective BRM Modulators Overview

Target / Approach	<ul style="list-style-type: none"> BRM Enzymatic inhibitor Targeted protein degrader
Indications	<ul style="list-style-type: none"> BRG1 mutated cancers (e.g., NSCLC), 30+ cancers with BRG1 mutations
Mutation / Aberration	<ul style="list-style-type: none"> BRG1
Stage	<ul style="list-style-type: none"> Pre-clinical
New Patients Impacted / Year*	<ul style="list-style-type: none"> > 100,000
Economics of Lilly Collaboration	<ul style="list-style-type: none"> 50/50 U.S. economics Tiered ex-U.S. royalties starting in the low double-digit range and escalating into the twenties

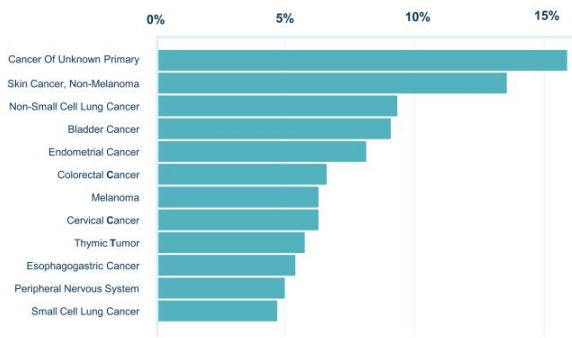


* U.S., EU5, Japan

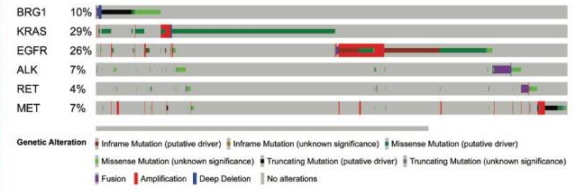
BRG1 MUTATED IN ~5% OF ALL TUMORS

Broad Addressable Patient Population

BRG1 MUTATED ACROSS RANGE OF TUMORS

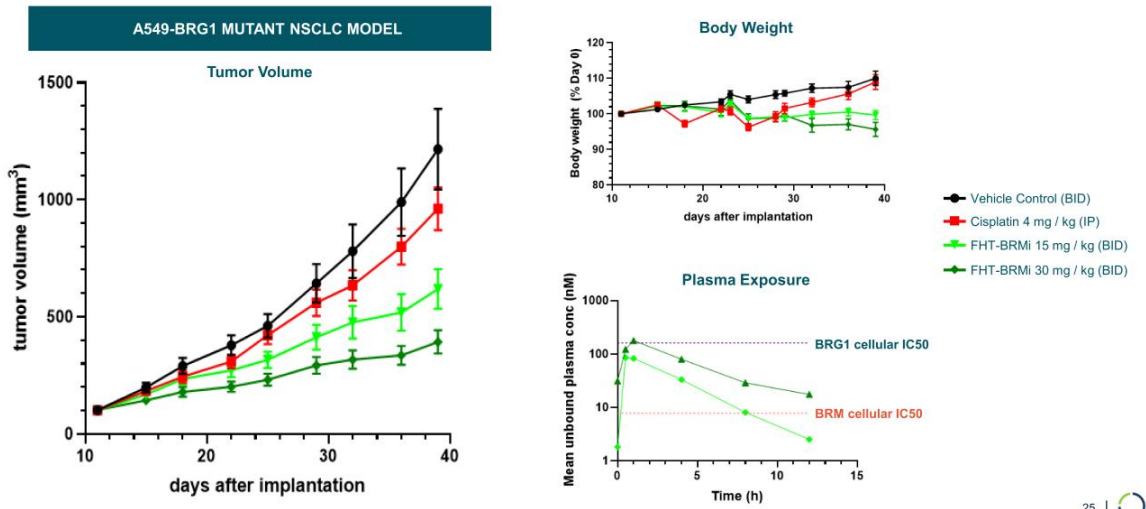


BRG1 MUTATED IN UP TO 10% OF NSCLC TUMORS, MINIMAL OVERLAP WITH OTHER MUTATIONS

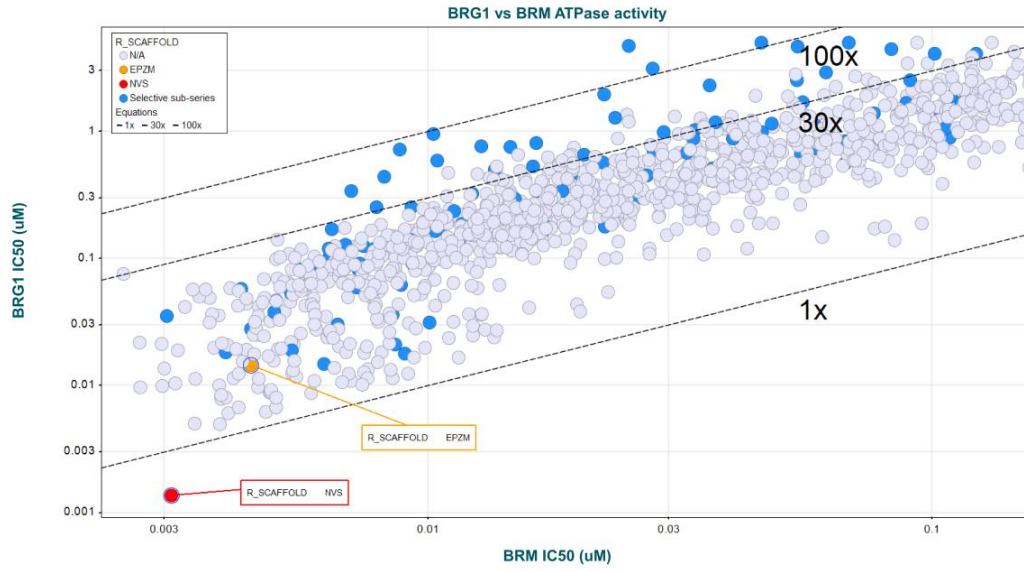


BRM SELECTIVE INHIBITOR *IN VIVO* EFFICACY

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



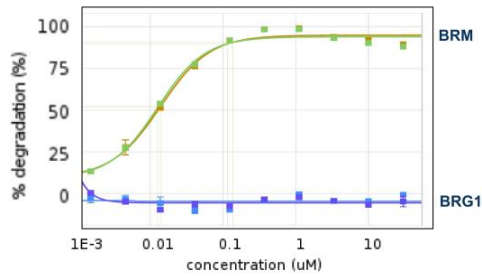
ENZYMATIC SELECTIVITY APPROACHING 200X ACHIEVED



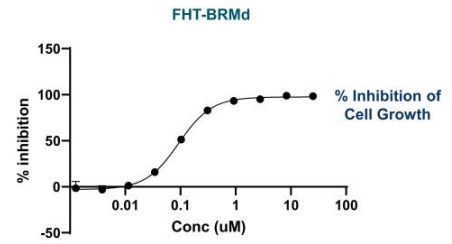
ADVANCING BRM SELECTIVE DEGRADERS

Achieving Complete BRM Degradation

BRM / BRG1 HIBIT DATA



A549 TEN-DAY PROLIFERATION ASSAY



DEGRADERS CAUSE TIME- AND DOSE-DEPENDENT BRM DEGRADATION, ANTIPROLIFERATIVE EFFECTS IN A549 BRG1 MUTANT NSCLC LUNG MODEL



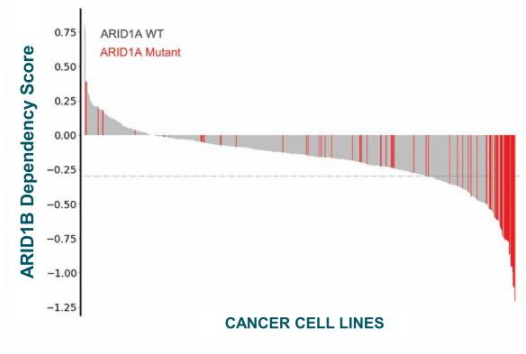
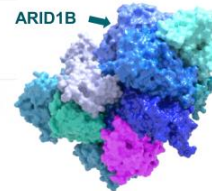
SELECTIVE ARID1B PROTEIN DEGRADER FOR ARID1A MUTATED CANCERS

Protein Degradator targeting ARID1A mutated cancers, the most mutated subunit in the BAF complex (e.g., ovarian, endometrial, colorectal, bladder and other cancers)

ARID1A: MOST MUTATED SUBUNIT IN BAF COMPLEX – CREATES DEPENDENCY ON ARID1B

Selective ARID1B Protein Degradation Overview

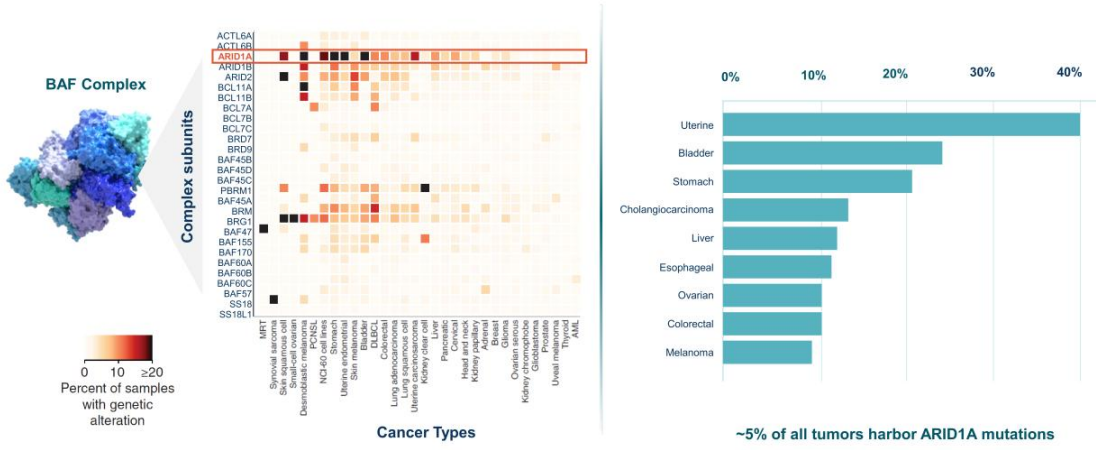
Target / Approach	<ul style="list-style-type: none">ARID1BTargeted protein degrader
Initial Indication	<ul style="list-style-type: none">ARID1A mutated cancers
Mutation / Aberration	<ul style="list-style-type: none">ARID1A mutations (e.g., ovarian, endometrial, colorectal, bladder and other cancers)
Stage	<ul style="list-style-type: none">Pre-clinical
New Patients Impacted / Year*	<ul style="list-style-type: none">> 175,000



* U.S., EU5, Japan

ARID1A MUTATED CANCERS: SIGNIFICANT OPPORTUNITY

ARID1A Mutated Across Range of Tumors



Hodges et al. 2017

TARGETING ARID1A MUTATED CANCERS: ARID1B PROTEIN DEGRADER

Advantaged by Gene Traffic Control Platform and Protein Degradation 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

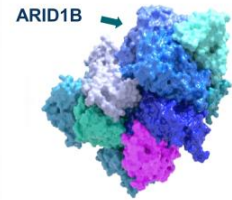
- Utilize protein degrader toolbox to create ARID1B hetero-bifunctional degraders

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

A NEW APPROACH TO DRUGGING TRANSCRIPTION FACTORS

Enabled by Proprietary Ability to Purify and Synthesize Chromatin Regulatory System Components

TFS ARE COMPELLING DRUG TARGETS...

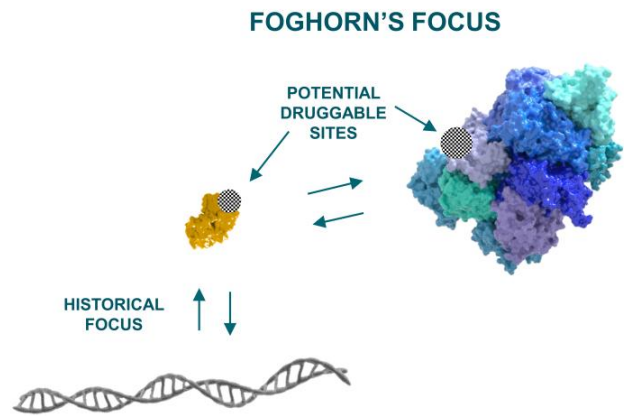
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

...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

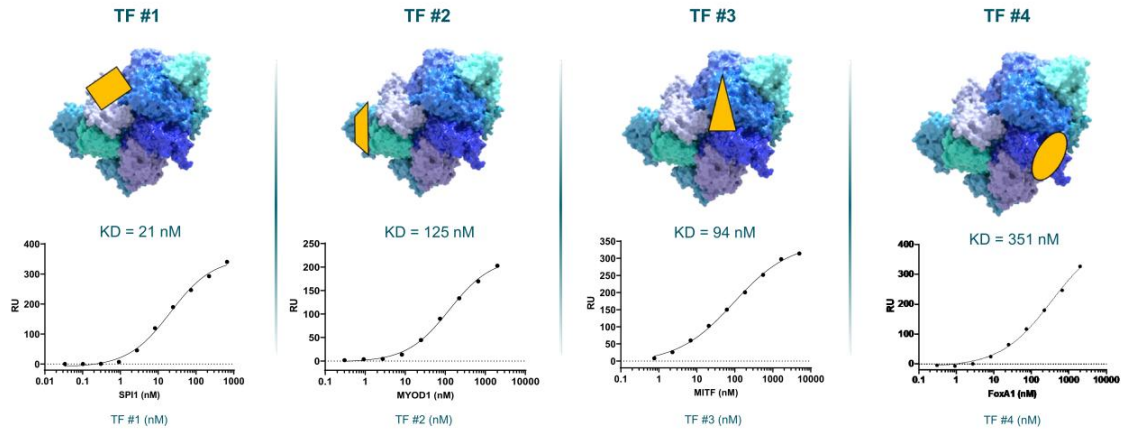
- Druggable binding pockets
- Druggable affinities



TRANSCRIPTION FACTOR-CHROMATIN REMODELING COMPLEX INTERACTIONS

Unique Insights in Where and How Transcription Factors Bind

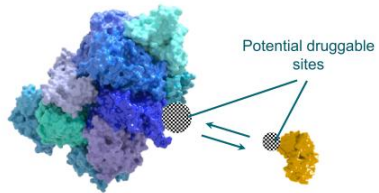
Transcription Factors (TF): 



HIGHLY SCALABLE APPROACH TO ADDRESS SIGNIFICANT UNMET MEDICAL NEED DRIVES MERCK COLLABORATION

Potential to Drug > 100 TFs Associated with BAF

TRANSCRIPTION FACTOR DISRUPTORS



- >100 TFs estimated associated with BAF
- Foghorn pursuing multiple TFs in parallel
- Approach highly scalable and potential broad application – other chromatin remodeling complexes and other diseases



- Merck collaboration to drug single specified transcription factor target
- \$15 million upfront; up to \$410 million in research, development, regulatory and sales-based milestones
- Up to low double-digit royalties on product sales

BROAD PIPELINE ACROSS A RANGE OF TARGETS AND MODALITIES

Precision Oncology / Breadth and Depth / Over 15 Programs



FIRST-IN-CLASS PRECISION MEDICINES TARGETING CANCER AND OTHER DISEASES



LARGE MARKET POTENTIAL

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

Potential applications in virology, autoimmune diseases and neurology



WELL-FUNDED

\$424.7 million in cash and equivalents
(as of 3/31/2022)



UPCOMING MILESTONES

FHD-286: Initial clinical data (TBD) - mUM study enrolling; AML/MDS study on partial clinical hold

FHD-609: Initial clinical data expected in 2023



SIGNIFICANT GLOBAL PARTNERSHIPS

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

Merck collaboration to drug single specified transcription factor target; \$15 million upfront and up to \$410 million in milestones



EXPERIENCED LEADERSHIP TEAM

Expertise across drug discovery, clinical development and commercialization

APPENDIX



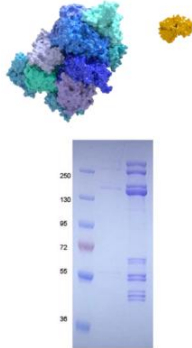


**PLATFORM &
DRUGGING CAPABILITIES**

PLATFORM IS POWERED BY ABILITY TO PRODUCE COMPONENTS AT SCALE

Drives Drug Discovery Pipeline with Cutting Edge Technology

PRODUCTION OF CHROMATIN REGULATORY SYSTEM COMPONENTS



	FEATURES	BENEFITS
	Surface Mapping	Characterize TF / BAF Binding Sites
	Assembly	Synthesize subcomplexes to enable drug discovery
	Affinity Screening & Validation	ASMS on full complex to yield novel degraders
	HTS	Multiple screening options with full complex
	Biophysics/SPR	Validation of novel small molecule binders

PROTEIN DEGRADER PLATFORM

CURRENT APPROACH

- A leader in developing heterobifunctional degraders for clinical evaluation in oncology
- Employing PROTAC and non-CRBN based molecular glue degradation approaches

DEGRADER CHEMICAL TOOLBOX

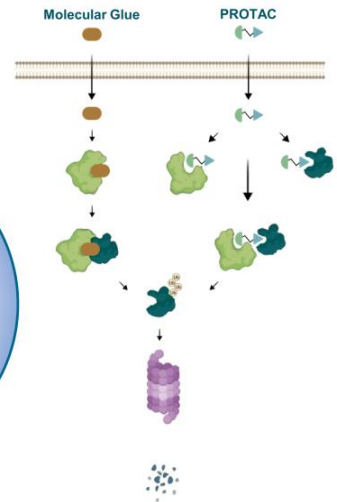
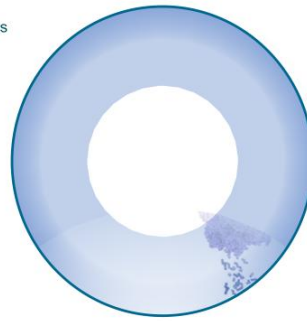
- Proprietary library of drug-like linkers, E3 ligase binders and potential glues
- Chemistry to rapidly identify and optimize degraders

ADVANCED MECHANISTIC CHARACTERIZATION

- Native target turnover understanding
- Cellular degradation kinetics and rates
- Structural, biochemical and cellular ternary complex characterization
- Global proteomics and ubiquitination studies
- Computational modeling of degraders
- Degradation efficacy across multiple cell types

OPTIMIZATION OF DEGRADER DRUG PROPERTIES

- Guidelines for both of oral and IV-administered degraders
- PK / PD, efficacy and safety modeling to optimize dosing and scheduling



STRATEGIC PARTNERSHIP
LOXO ONCOLOGY AT LILLY

STRATEGIC COLLABORATION WITH LOXO ONCOLOGY AT LILLY

Foghorn to Lead Discovery and Research Activities



\$380 MILLION UPFRONT

\$300 million cash payment

\$80 million investment 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 BRM-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

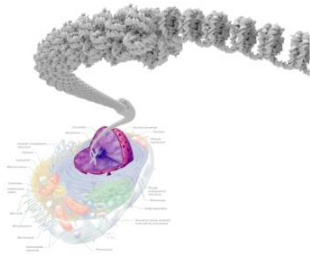


THE CHROMATIN REGULATORY SYSTEM

Orchestrates Gene Expression

THE CHROMATIN REGULATORY SYSTEM ORCHESTRATES GENE EXPRESSION

Two Major Components Work in Concert: Chromatin Remodeling
Complexes and Transcription Factors

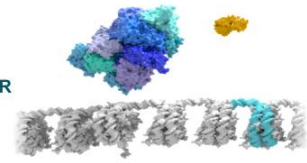


CHROMATIN

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

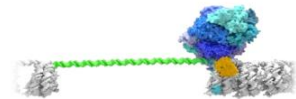
1 | CHROMATIN REMODELING COMPLEX AND TRANSCRIPTION FACTOR

Work together to orchestrate
gene expression



2 | RIGHT GENES

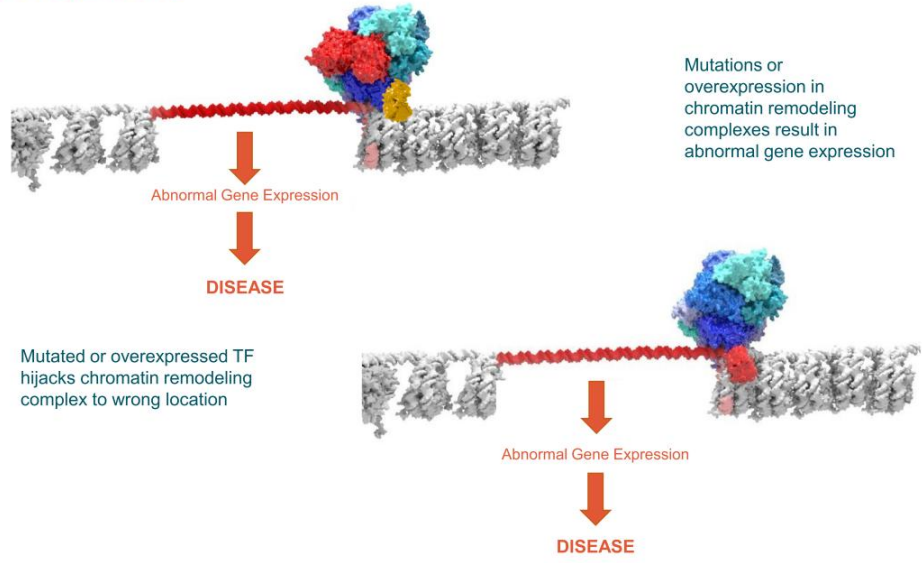
TFs guide chromatin
remodeling complexes to the
right locations



3 | NORMAL GENE EXPRESSION

Once chromatin unpacked,
gene expression can occur

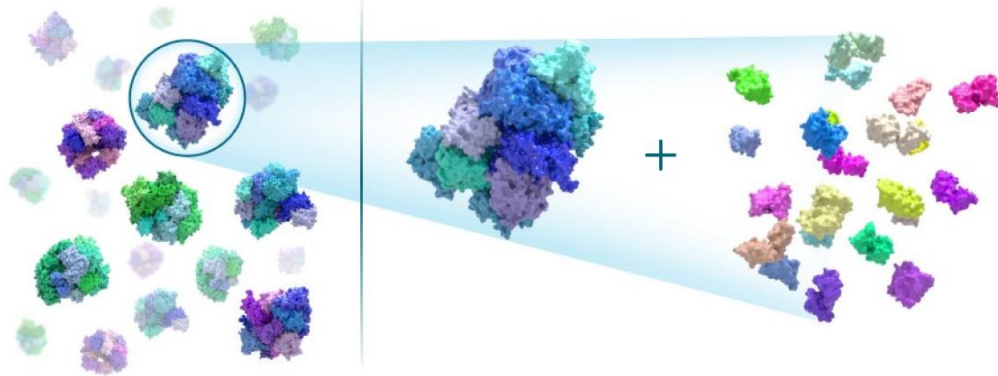
BREAKDOWNS IN THE CHROMATIN REGULATORY SYSTEM CAN LEAD TO DISEASE



CHROMATIN REGULATORY SYSTEM

Abundance of Targets within the BAF Complex

BAF COMPLEX AND ASSOCIATED TRANSCRIPTION FACTORS



28 Chromatin Remodeling
Complexes and >1,000 TFs

BAF Complex Subunits Mutated
and Dysregulated in Cancer

Estimate >100
Transcription Factors
Associated with Just
the BAF Complex



Leadership Team, Board & Advisors

Expertise across drug discovery, clinical development
and commercialization

PROVEN LEADERSHIP TEAM



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