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): February 16, 2021

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) 245-0399

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.

Foghorn Therapeutics Inc. (the "Company") is furnishing as Exhibit 99.1 to this Current Report on Form 8-K a presentation, dated February 16, 2021, which the Company intends to use from time to time in meetings with or presentations to investors.

The information in this Item 7.01 (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 in any filing under the Securities Act of 1933, as amended, 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	Investor Presentation, dated February 16, 2021

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/ Allan Reine
Allan Reine, M.D.
Chief Financial Officer

Date: February 16, 2021

Exhibit 99.1

Targeting the Chromatin Regulatory System

A Product Platform with Potential to Impact Millions of Patients

February 16, 2021

Precision Oncology: Novel Biology with Breadth and Depth

Large Market Potential / Precision Approach

- Biology implicated in up to 50% of cancer potentially impacting ~2.5 million patients

Experienced Leadership Team

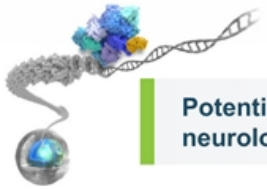
- Expertise across drug discovery, clinical development and commercialization
- Over 220 drug candidates into the clinic and over 30 drugs approved

Novel Biology and Targets

- Targeting the chromatin regulatory system
- Integrated and scalable platform
- Chromatin remodeling complexes, transcription factors, and other components

Multiple Drugging Approaches

- Synthetic lethality
- Protein degradation
- Transcription factor disruptors

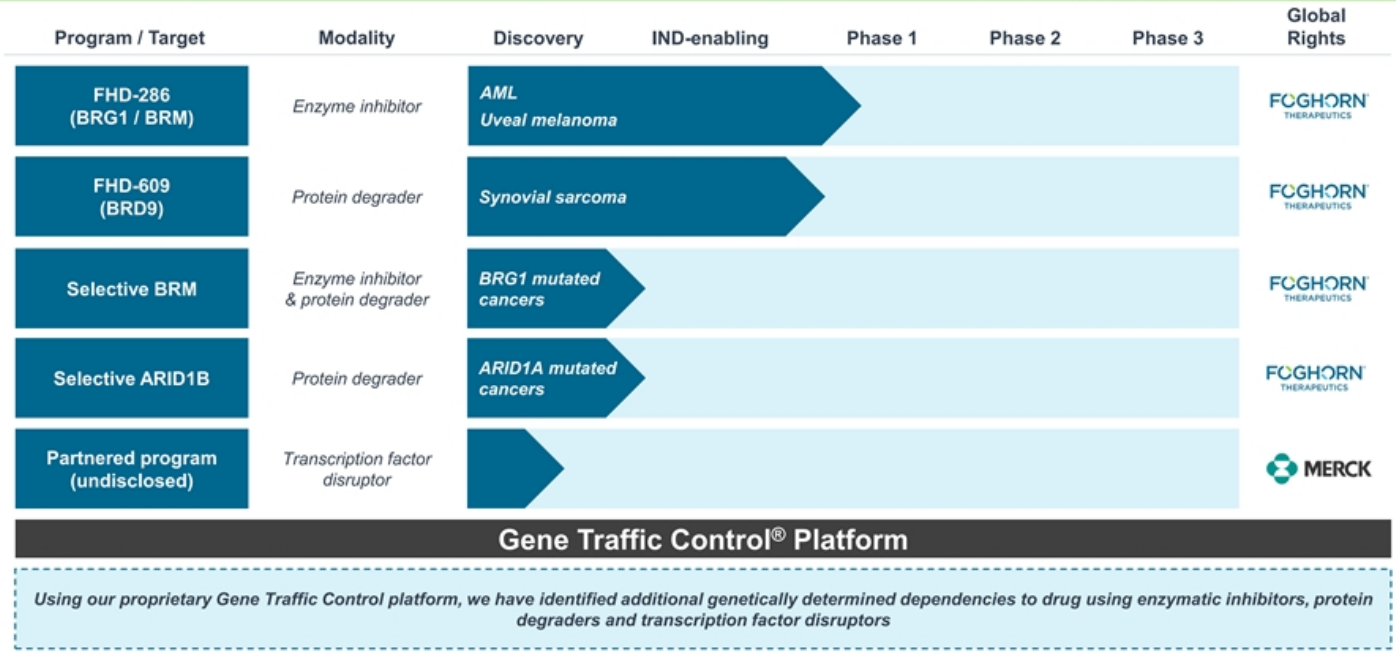


Potential applications beyond oncology in diseases including virology, autoimmune disease and neurology

FOGHORN THERAPEUTICS

On Track for Entry Into the Clinic with First Two Programs

Precision Oncology / Breadth and Depth



Using our proprietary Gene Traffic Control platform, we have identified additional genetically determined dependencies to drug using enzymatic inhibitors, protein degraders and transcription factor disruptors

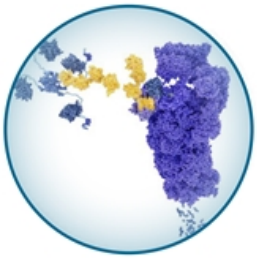
Our Gene Traffic Control Platform Makes It Possible to Understand and Drug Genetic Dependencies within the Chromatin Regulatory System

Integrated, Scalable, Efficient – Repeatable Paradigm

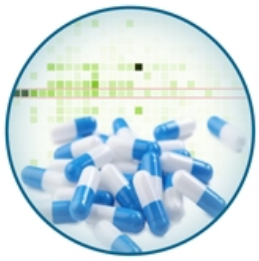
Target Identification And Validation

Production of Chromatin Regulatory System Components at Scale & Proprietary Assays

Discovery and Optimization of Chemical Matter



Targeted Protein Degradation



Translation to Clinic and Identification of Biomarkers

Experienced Leadership Team with Industry Leading Advisors and Investors

SENIOR LEADERSHIP TEAM

Adrian Gottschalk, President & CEO

Development through commercialization in multiple therapeutic areas across >25 drug programs

Biogen

Carl Decicco, Ph.D., CSO

>200 drugs transitioned to the clinic, 20 approvals

Bristol-Myers Squibb

Sam Agresta, M.D., M.P.H., CMO

Global drug approval experience, 3 drug approvals

agios Genentech Infinity

Michael LaCascia, CLO

Former GC, Vertex Pharmaceuticals, global law and compliance experience, 2 drug approvals

VERTEX WilmerHale

Steve Bellon, Ph.D., SVP, Drug Discovery

20+ years in industry drugging novel targets, incl. bromo platform at Constellation

Constellation AMGEN VERTEX

Allan Reine, M.D., CFO

15 years biotech investor and public company CFO

pieris

Carlos Costa, SVP, HR

20+ years worldwide experience across multiple countries and regions

Biogen Roche P&G

Fanny Cavalie, SVP, Business & Operations

Global development through launch for >20 drugs, portfolio and BD strategy experience

Biogen McKinsey &Company

BOARD OF DIRECTORS

Doug Cole, M.D.
Flagship Pioneering – Board Chair; Founder

Adrian Gottschalk
Foghorn President & CEO

Jose Baselga, M.D., Ph.D.
AstraZeneca R&D Oncology

Cigall Kadoch, Ph.D.
Dana-Farber, Broad, HMS; Founder

Scott Biller, Ph.D.
Former CSO and Strategic Advisor, Agios

Adam Koppel, M.D., Ph.D.
Bain Capital Life Sciences

Simba Gill, Ph.D.
Evelo Biosciences, Partner at Flagship Pioneering

Michael Mendelsohn, M.D.
Cardurion Pharmaceuticals

SCIENTIFIC AND OTHER ADVISORS

Charles Sawyers, M.D.
MSKCC, HHMI – SAB Chair

David Schenkein, M.D.
General Partner, GV

Gerald Crabtree, M.D.
Stanford, HHMI; Founder

Tony Kouzarides, Ph.D.
Gurdon Institute – University of Cambridge

Faheem Hasnain
Gossamer Bio, Chair of Mirati

Ian Smith
Former COO Vertex Pharmaceuticas, Chair of Solid Bio., Chair of ViaCyte

Craig Peterson, Ph.D.
Professor UMass Medical School

FOGHORN THERAPEUTICS

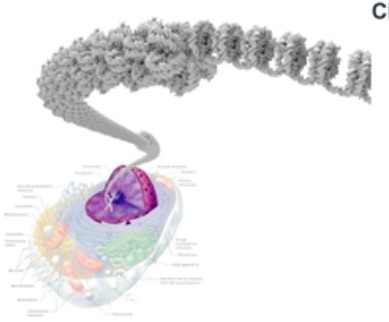
The Chromatin Regulatory System

Orchestrates Gene Expression

The Chromatin Regulatory System Orchestrates Gene Expression

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

Chromatin

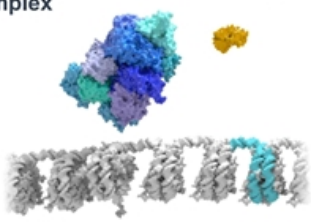


Chromatin – compacted form of DNA inside the nucleus of the cell

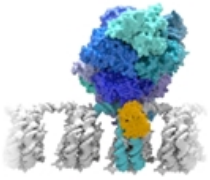
1 Work together to orchestrate gene expression

Chromatin remodeling complex

Transcription Factor

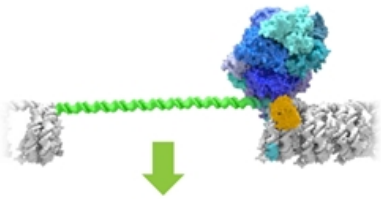


2 TF's guide chromatin remodeling complexes to the right locations



Right genes

3 Once chromatin unpacked, gene expression can occur



Normal gene expression

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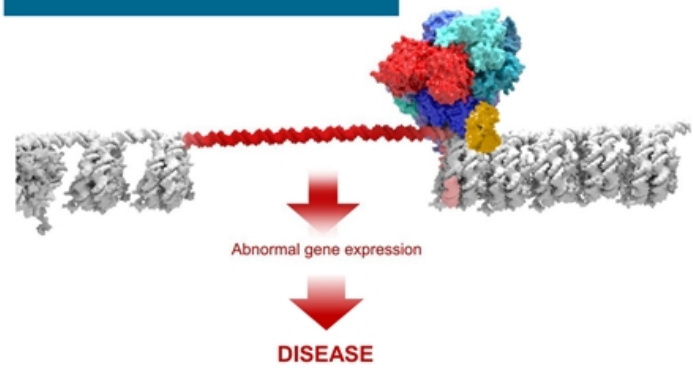
Breakdowns in the Chromatin Regulatory System Lead to Disease

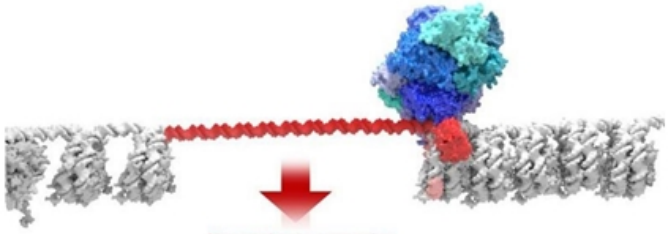
Mutations in Chromatin Remodeling Complex Result in Abnormal Gene Expression



Abnormal gene expression

DISEASE

Mutated or Overexpressed TF Hijacks Chromatin Remodeling Complex to Wrong Location



Abnormal gene expression

DISEASE

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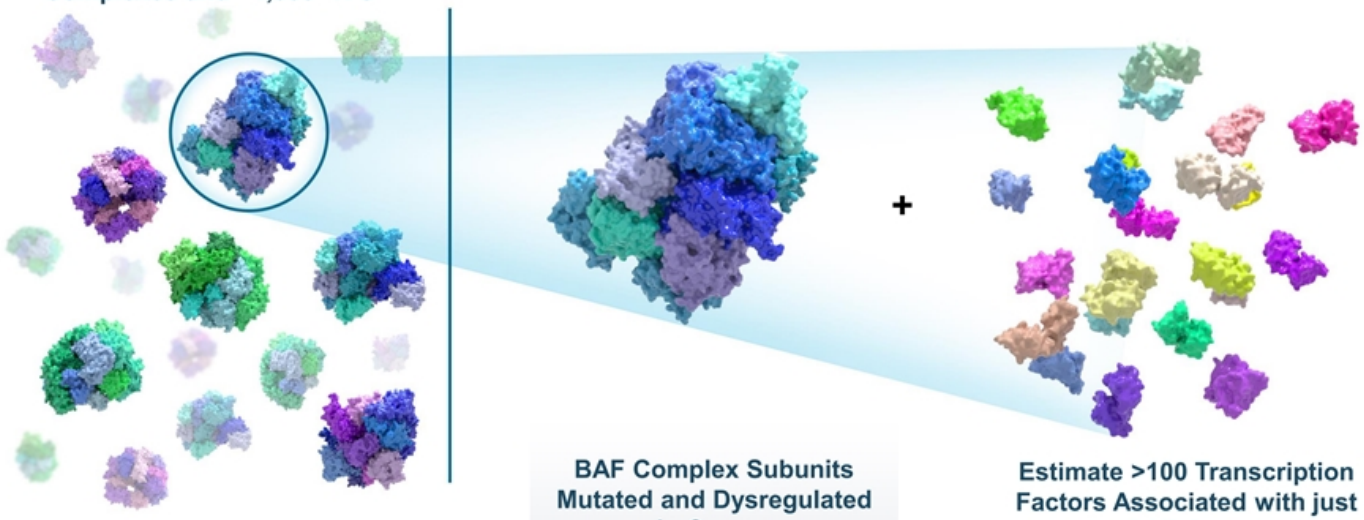
Chromatin Regulatory System Implicated in Over 50% of Cancers Potentially Impacting Over 2.5M Patients

28 Chromatin Remodeling Complexes and >1,000 TFs

BAF Complex and Associated Transcription Factors



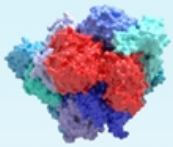
BAF Complex Subunits Mutated and Dysregulated in Cancer

Estimate >100 Transcription Factors Associated with just the BAF Complex

Mutations Lead to Disease Specific Genetic Dependencies on the Chromatin Regulatory System

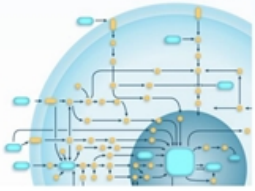
Novel Targets

Mutations in Chromatin Remodeling Complexes



Transcription Factor Mutations / Overexpression

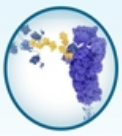
Mutations that Impinge on the Chromatin Regulatory System



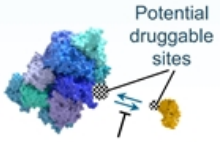
Tailored Drugging Approaches

Enzymatic Inhibitors:
Highly selective and allosteric small molecule inhibitors

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



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



Pursuing a Precision Oncology Approach

Each Program is Based on a Genetically Defined Dependency

Program	Mutation / Abberation	Genetic Dependency	Target Patient Population*	Drug Approach
FHD-286	Elevated BRG1 expression	BRG1	AML (20,000)	Enzymatic Inhibitor
	GNAQ/GNA11	SOX10 / MITF / BAF complex	Uveal Melanoma (5,000)	
FHD-609	SS18-SSX1, SSX2, SSX4	BRD9	Synovial Sarcoma (>1,800)	Protein Degrader
Selective BRM	BRG1	BRM	BRG1 mutated cancers (>100K)	Enzymatic Inhibitor / Protein Degrader
Selective ARID1B	ARID1A	ARID1B	ARID1A mutated cancers (>175K)	Protein Degrader
Transcription Factors	Various	Specific TF – Chromatin Remodeling Complex	Various	Transcription Factor Disruptor

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*U.S., EU5, Japan

FHD-286: Clinical Entry Point - AML and Uveal Melanoma

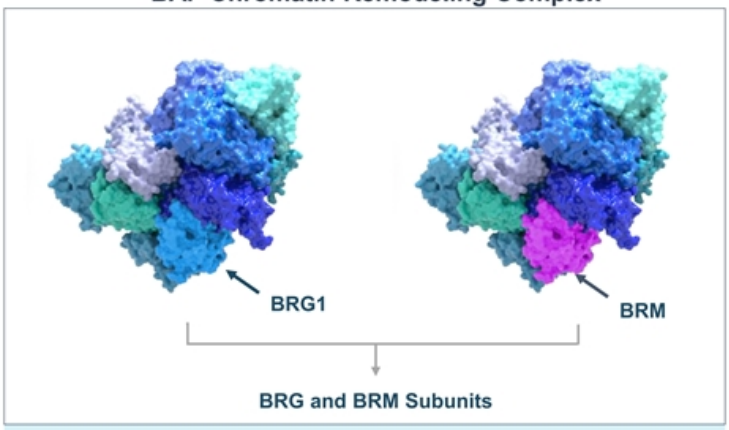
FHD-286 is a Potent, Selective, Allosteric, Small Molecule Inhibitor of the BRG1 and BRM subunits of the BAF complex

FHD-286 Targets Abnormal Dependencies on BAF in Cancer

Target / Approach	• BRG1/BRM ATPase • Small molecule, allosteric, oral enzymatic inhibitor
Indications	• Acute myelogenous leukemia (AML) • Uveal melanoma • Indication expansion work ongoing in multiple solid tumors
Mutation / Aberration	• **AML**: BRG1 elevated in blast cells • **Uveal Melanoma**: GNAQ/GNA11 mutated UM is driven by an abnormal dependency on BAF
Program Status	• On track for clinical data as early as Q4'21
New Patients Impacted / year*	• **AML: Over 20,000 relapsed and/or refractory patients** • **Uveal melanoma: Over 5,000 patients**

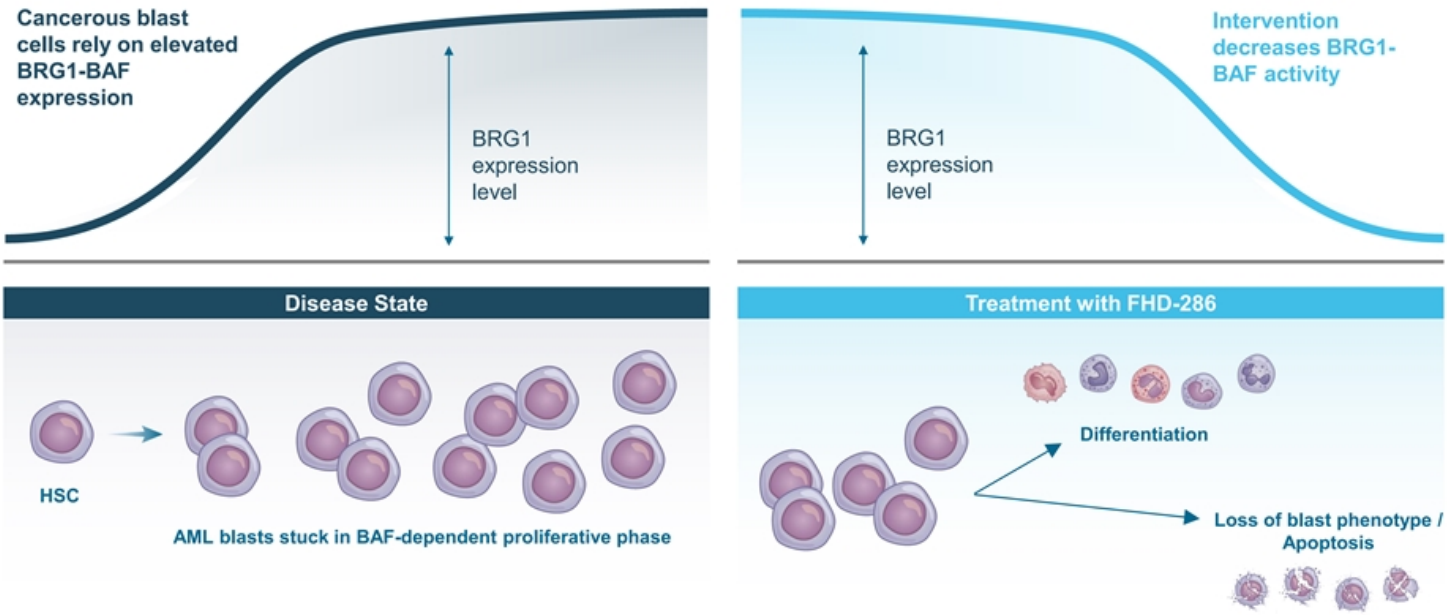
* US, EU5, Japan

BAF Chromatin Remodeling Complex



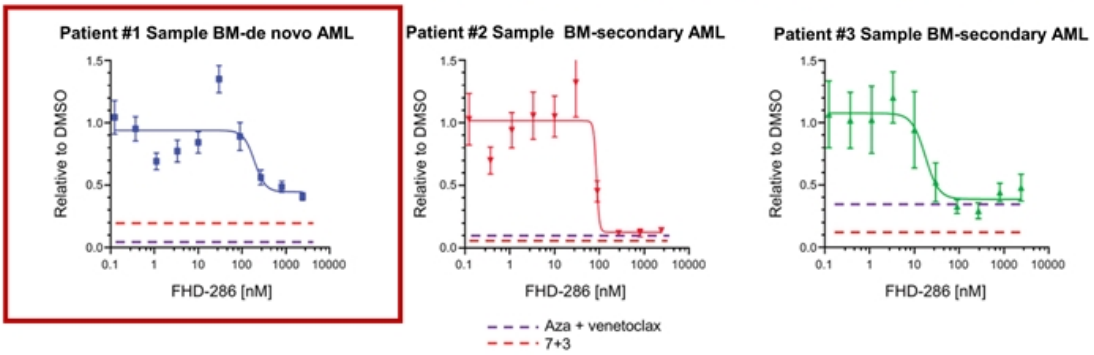
- **BRM/BRG1 is the engine (ATPase) of the BAF chromatin remodeling complex**
- **BRG1 & BRM are highly similar proteins**

Therapeutic Rationale for AML: Blast Cells Dependent on BRG1-BAF

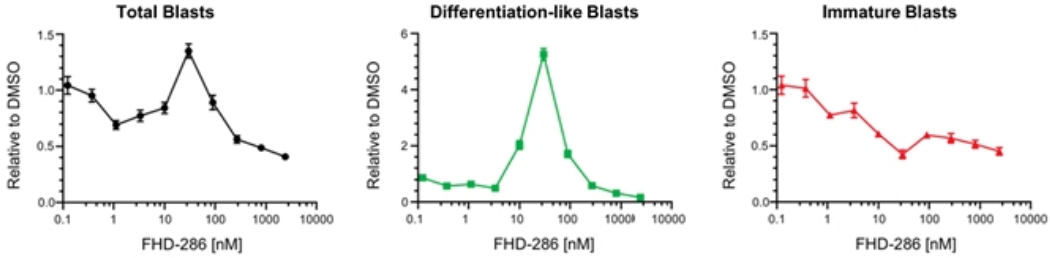


Treatment with FHD-286 of Patient-Derived AML Tumor Samples was Associated with both Differentiation and Cytoreduction

Exposure-dependent differentiation effect and cytoreduction (Cytoreduction is equivalent to standard of care)



Patient #1 Sample: Example of exposure-dependent differentiation effect



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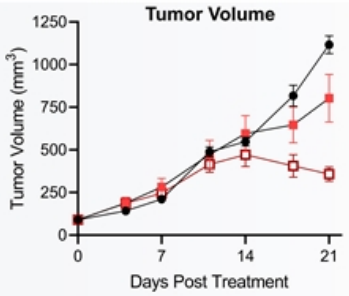
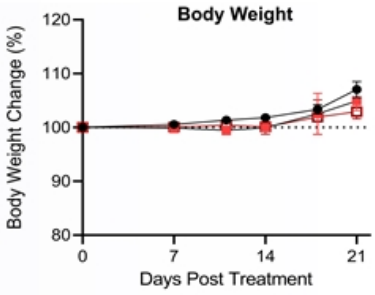
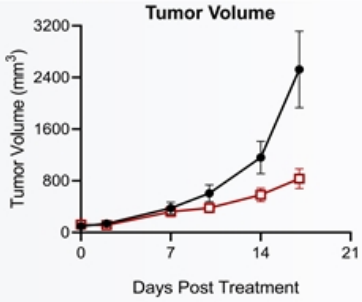
Dose-Dependent Tumor Growth Inhibition Observed with FHD-286 Treatment in AML CDX Models

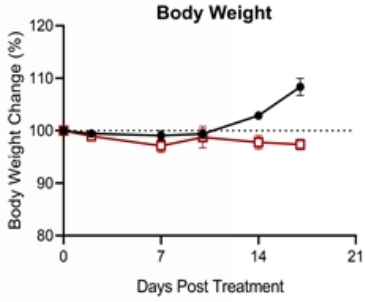
MV4-11 CDX Model
(FLT3 ITD, MLL-AF4)

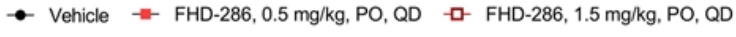




OCI-AML2 CDX Model
(MII-AF6, DNMT3a mut.)







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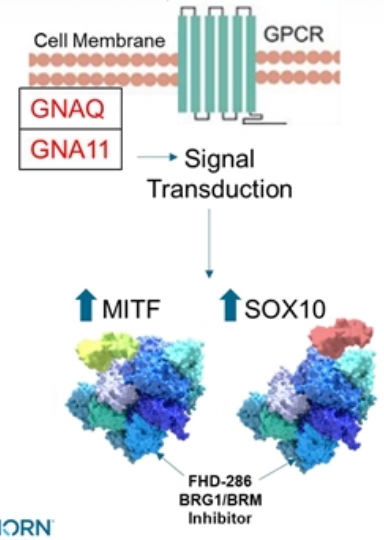
Therapeutic Rationale for Uveal Melanoma: Dependency on Overexpression of the MITF / SOX10 Transcription Factors and the BAF Complex

Inhibiting BRG1/BRM to shut down the abnormal TF interaction with the BAF complex

Biology

Over 85% of uveal melanoma cancers have GNAQ or GNA11 mutations



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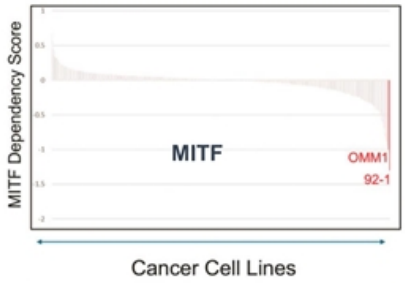
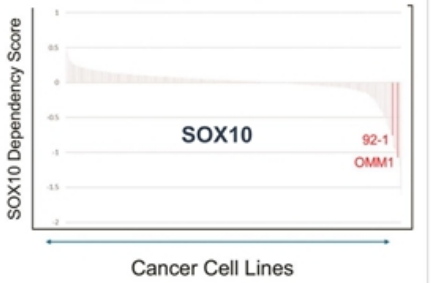
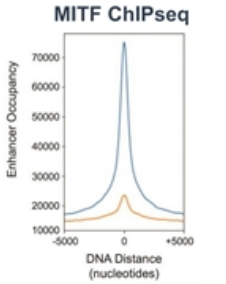
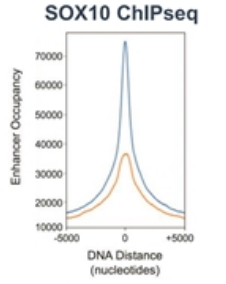
Validation of Dependency and Approach









DMSO Control
BRG1/BRM Tool Cmpd

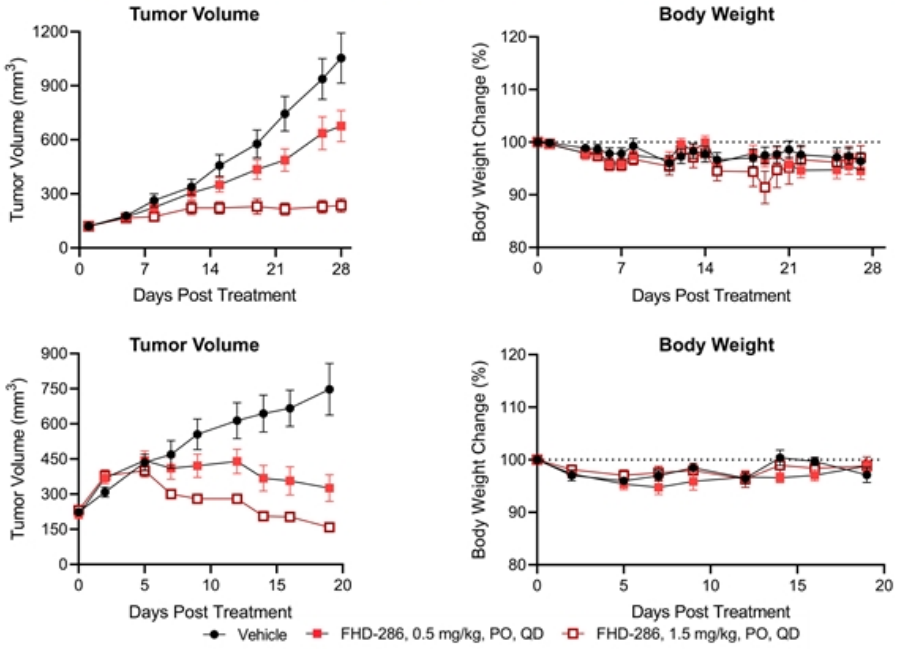
FHD-286 was Associated with Dose-Dependent Tumor Regression in Uveal Melanoma CDX Models at Tolerated Doses

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



FHD-286 Clinical Development Plan

Our Understanding of the Biology and Target allows for the Selection of Focused Patient Populations

CLINICAL PLAN

AML & Uveal Melanoma FIH Phase 1 Studies

Relapsed / Refractory AML

Metastatic Uveal Melanoma

Trial Design

- "3 + 3" accelerated titration design
- Retrospective biomarker analysis to further evaluate safety and efficacy
- Assess safety, PK, biomarkers and efficacy

Expansion cohorts in AML, UM and potentially other indications

Potential for entry into definitive efficacy trials in AML

Potential for entry into definitive efficacy trials in metastatic uveal melanoma

Potential for Indication Expansion Beyond AML and UM

Early clinical data as early as Q4 2021

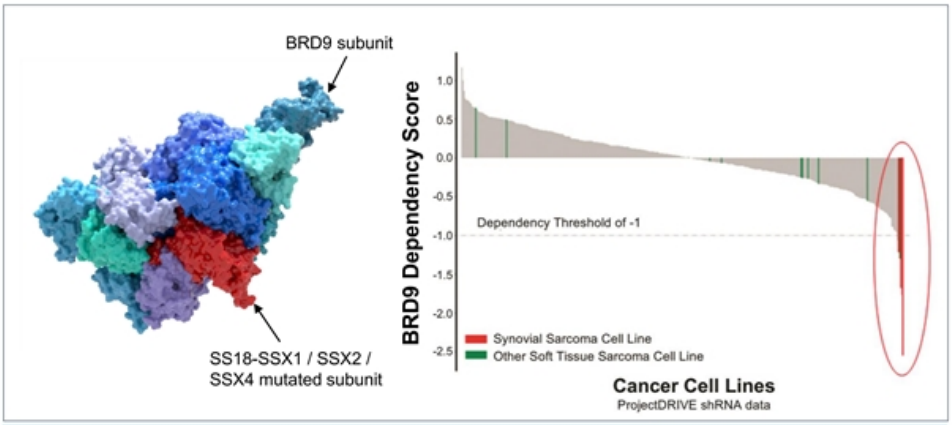
FHD-609: Clinical Entry Point – Synovial Sarcoma

FHD-609 is a Selective, Potent, Protein Degrader 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 Degrader

Target / Approach	• BRD9 • Intravenous Protein Degrader
Initial Indication	• Synovial Sarcoma
Mutation / Aberration	• SS18-SSX1 / SSX2 / SSX4 protein fusions
Upcoming Milestones	• IND submission Q2 2021
New Patients Impacted / year*	• **Synovial Sarcoma: Over 1,800 patients / year**



- **BRD9 is required for the survival of synovial sarcoma cells**

* US, EU5, Japan

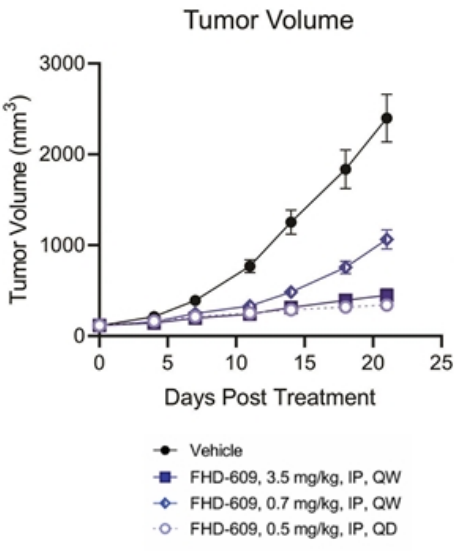
FOGHORN THERAPEUTICS

Robust *in vivo* Activity Observed in Synovial Sarcoma Model and BRD9 Degradation Associated with FHD-609 Treatment

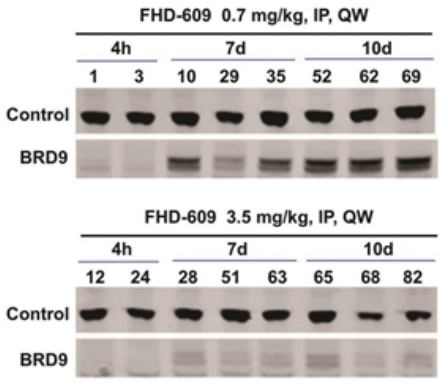
Weekly Dosing of FHD-609 Achieved Sustained BRD9 Degradation

SY01 Synovial Sarcoma CDX Model

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



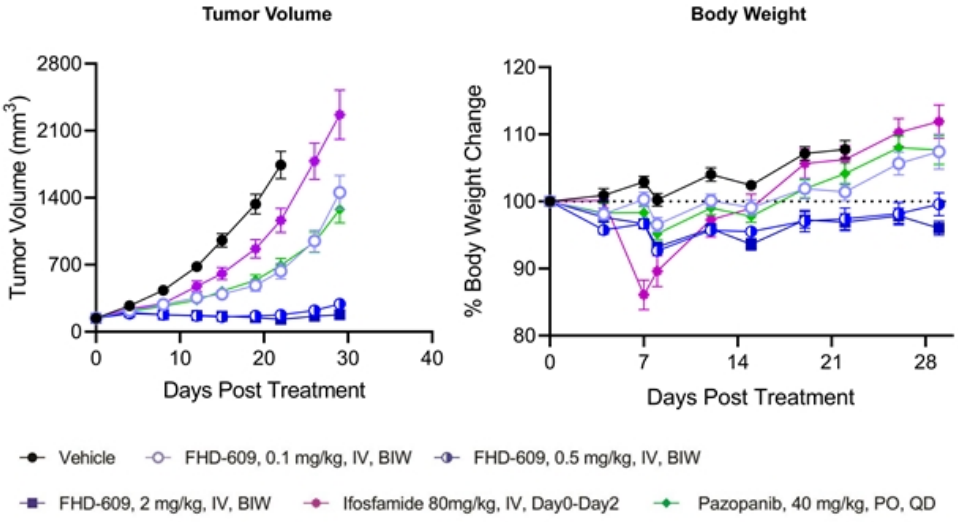
- **Sustained BRD9 Degradation**



Superior Tumor Growth Inhibition of 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



FHD-609 Clinical Development Plan

Our Understanding of the Biology and Target allows for the Selection of Focused Patient Populations

CLINICAL PLAN

Synovial Sarcoma FIH Phase 1

Metastatic Synovial Sarcoma

Synovial Sarcoma expansion cohorts

SMARCB-1 deleted tumors and potentially other indications

Potential for entry into definitive efficacy trials in synovial sarcoma

Trial design:

- "3+3" Design
- Patients: Synovial cell sarcoma
- Primary endpoints: Assess safety, PK, efficacy and biomarkers

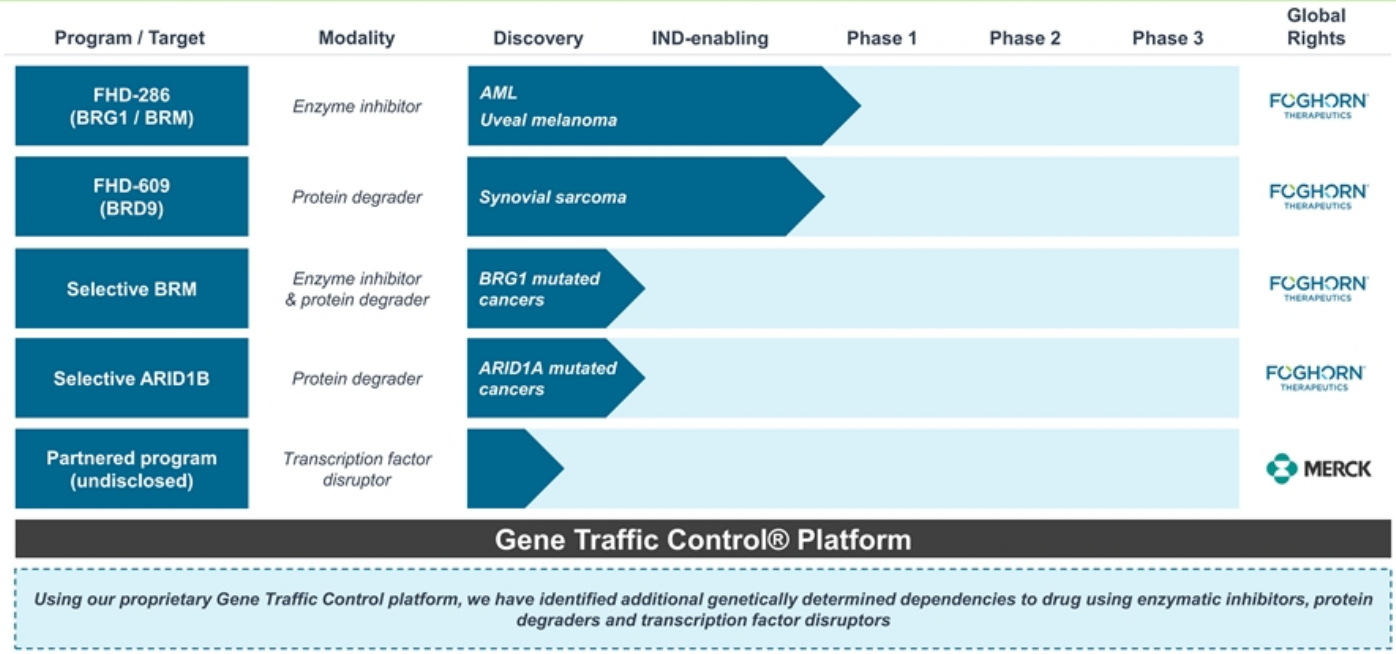
Biomarkers:

- SS18-SSX1, SS18-SSX2 or SS18-SSX4 translocation

IND submission expected in Q2 2021

On Track for Entry Into the Clinic with First Two Programs

Precision Oncology / Breadth and Depth



Gene Traffic Control® Platform

Using our proprietary Gene Traffic Control platform, we have identified additional genetically determined dependencies to drug using enzymatic inhibitors, protein degraders and transcription factor disruptors

Selective BRM Modulators for BRG1 Mutated Cancers

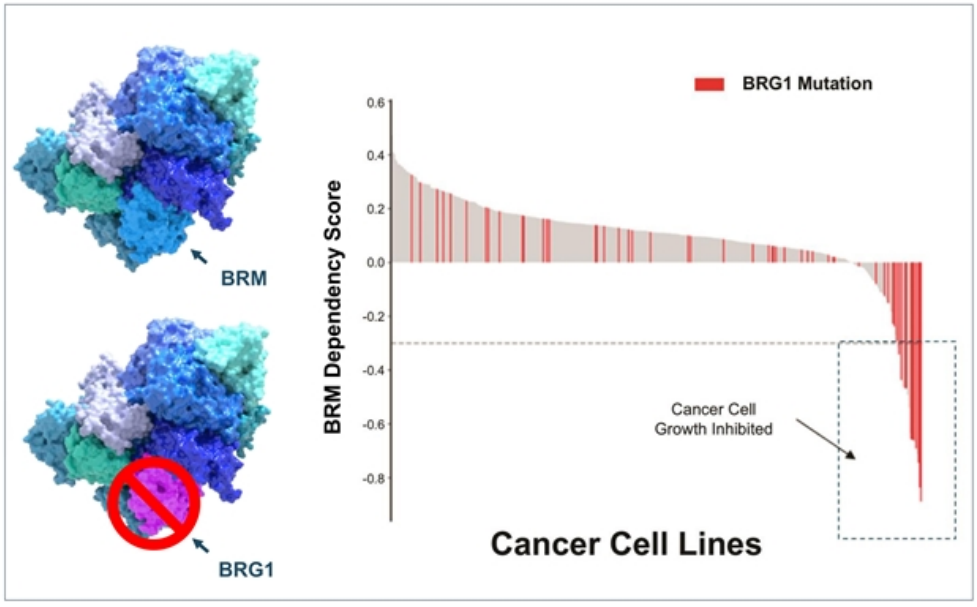
Enzymatic Inhibitor and Protein Degrader Programs

BRG1 Mutations Create a Genetic Dependency on BRM

Selective BRM Modulators Overview

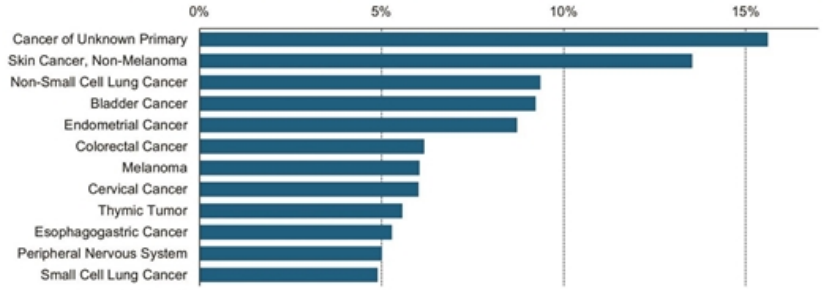
Target / Approach	• BRM • Enzymatic inhibitor • Targeted protein degrader
Indication	• BRG1 mutated cancers (e.g., NSCLC), 30+ cancers with BRG1 mutations
Mutation / Aberration	• BRG1
Stage	• Pre-clinical
New Patients Impacted / year*	• **> 100,000**

* US, EU5, Japan



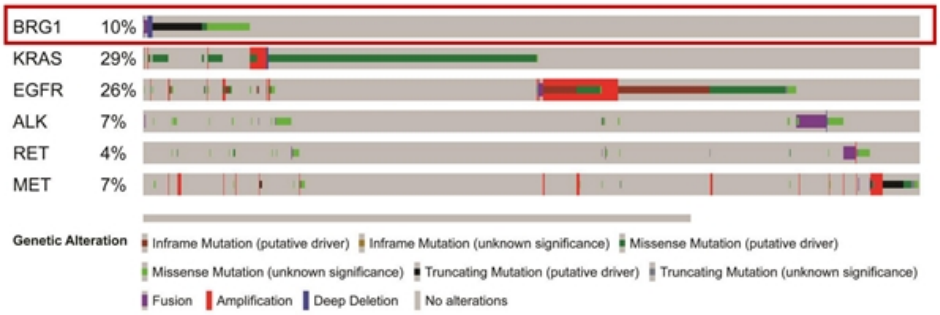
FOGHORN THERAPEUTICS

BRG1 Mutated in ~5% of All Tumors – Potential Broad Addressable Patient Populations



BRG1 mutated across range of tumors

Accounts for ~5% of all tumors



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

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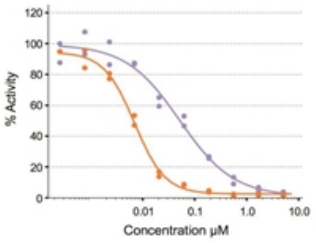
20X Selective BRM Inhibitor and Targeted Protein Degrader Discovered from Gene Traffic Control Platform

Selective BRM Modulators

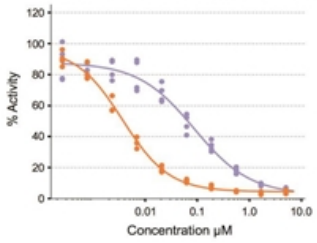
BRM Selective Inhibitor Program

- Panel showing biochemical selectivity of a 20X more selective inhibitor of BRM vs. BRG1

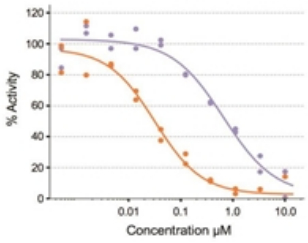
Enzyme assay using BRG1 and BRM subunits



Enzyme assay using BRG1 and BRM containing full BAF complexes

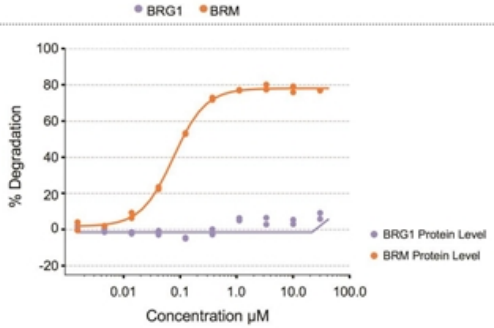


Cellular assays for BRG1 and BRM



BRM Selective Degrader Program

- Selective BRM degrading molecules led to the degradation of over 75% of BRM while leaving BRG1 unchanged



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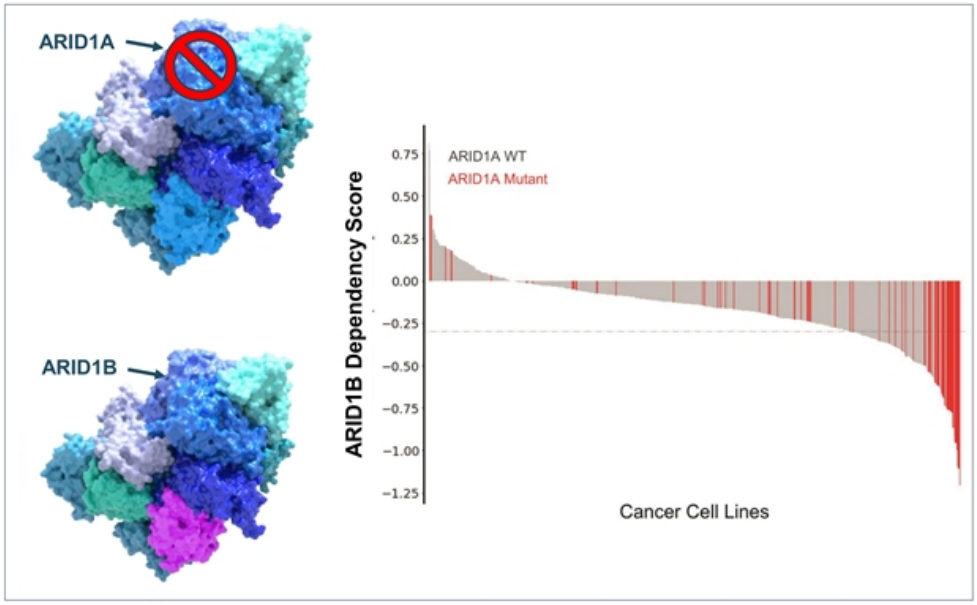
Selective ARID1B Protein Degrader for ARID1A Mutated Cancers

ARID1A – Most Mutated Subunit in BAF Complex – Creates Dependency on ARID1B

Selective ARID1B Protein Degrader Overview

Target / Approach	• ARID1B • Targeted Protein Degrader
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**

* US, EU5, Japan



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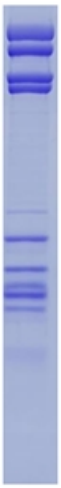
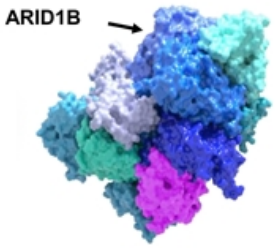
ARID1B Highlights Broad Potential of Foghorn Gene Traffic Control Platform

Biology

- ARID1A is the most highly mutated subunit of the BAF complex in cancer
- ~5% of all tumors harbor ARID1A mutations including; endometrial cancer (~40%), bladder cancer (~25%) and ovarian (~15%)
- Synthetic lethal relationship with ARID1B
- Developing protein degraders to ARID1B



Highly purified ARID1B-BAF Complex



Drugging Strategy

- Platform produces BAF complexes and subcomplexes containing either ARID1A or ARID1B at scale
- Status: Validating hits from multiple High Throughput Screens

Novel Approach to Targeting Transcription Factors

Disrupting Transcription Factor – Chromatin Remodeling Complex Interactions

A New Approach to Drugging Transcription Factors

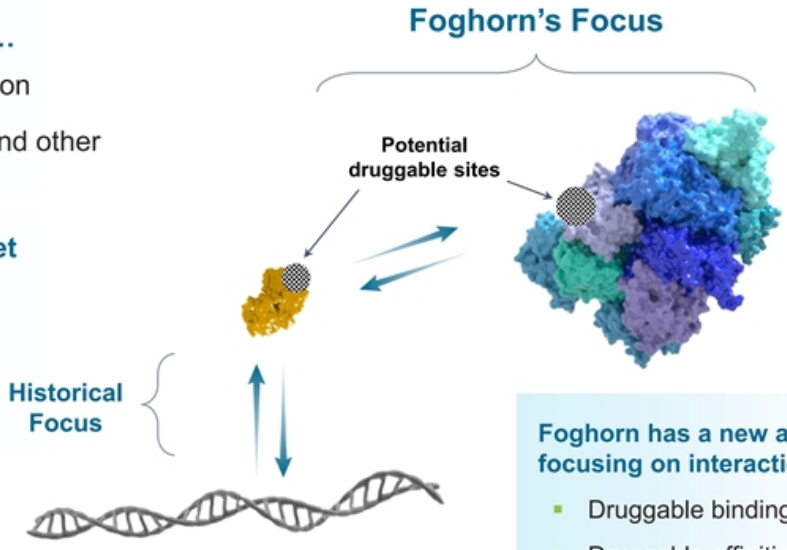
Enabled by Proprietary Ability to Purify and Synthesize Chromatin Regulatory System Components

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



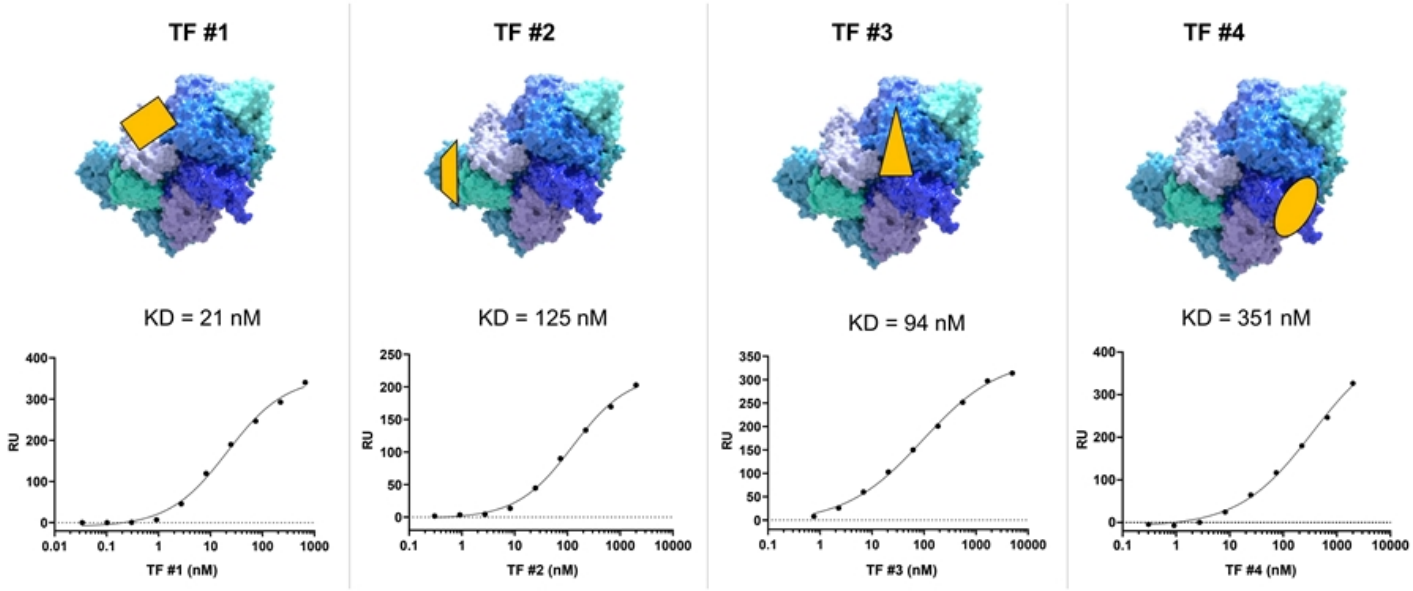
Foghorn has a new approach focusing on interaction with BAF

- Druggable binding pockets
- Druggable affinities

FOGHORN THERAPEUTICS

Transcription Factor-Chromatin Remodeling Complex Interactions

Unique Insights in Where and How Transcription Factors Bind



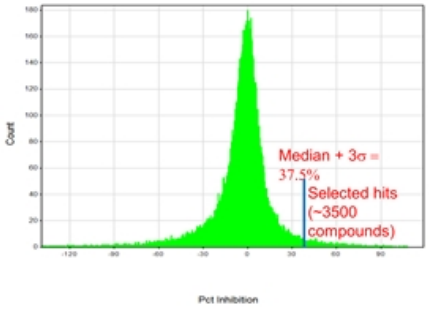
Transcription Factors (TF):

FOGHORN THERAPEUTICS

Highly Scalable Approach and Significant Unmet Medical Need

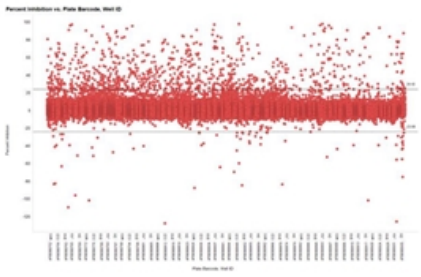
Potential to drug > 100 TFs associated with BAF

TF #X Primary HTS of >330K compounds



- >100 TFs estimated associated with BAF
- Foghorn pursuing multiple TFs in parallel
- HTS on full BAF complex + TF target
- Approach highly scalable and potential broad application – other chromatin remodeling complexes and other diseases

TF #Y Primary HTS of >250K compounds

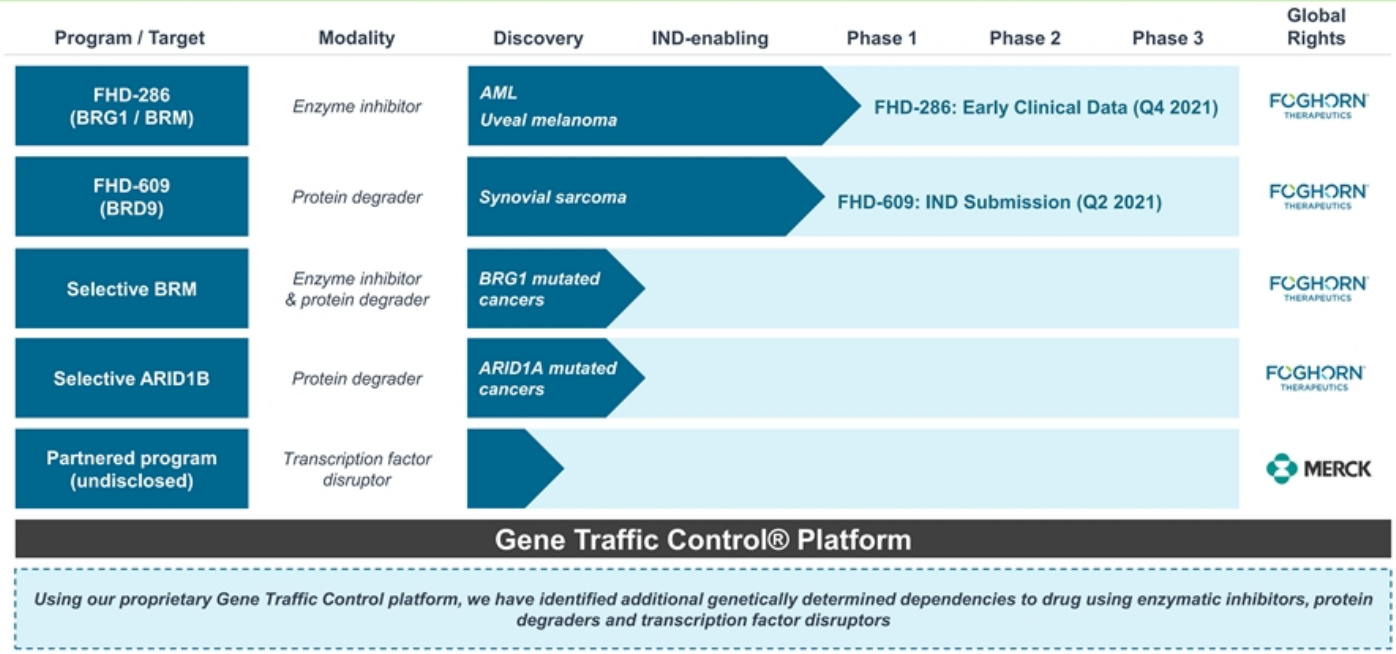


- Merck collaboration to drug single specified transcription factor target
- $425 million in up-front, research, development and sales-based milestones
- Up to low double-digit royalties on product sales

MERCK

On Track for Entry Into the Clinic with First Two Programs

Precision Oncology / Breadth and Depth



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