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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): September 8, 2023
_____

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.

The Company is furnishing as Exhibit 99.1 to this Current Report on Form 8-K a presentation, dated September 2023, which the Company intends to use 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 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	Investor Presentation dated September 2023

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: September 8, 2023

Exhibit 99.1

FCGHORN®
THERAPEUTICS

CORPORATE OVERVIEW

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

September 2023

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 NEW AREA OF CANCER BIOLOGY

Foghorn is a **leader in targeting chromatin biology,** which has unique potential to address underlying dependencies of many genetically defined cancers

Broad pipeline of **over 15 programs** 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

$284.3 million in cash and equivalents

(as of 06/30/2023)

Provides **runway into H2'25**

SIGNIFICANT UPCOMING VALUE DRIVERS

Selective BRM program on track to transition to Loxo@Lilly in **H2'23**

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

MAJOR STRATEGIC COLLABORATION

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

FOGHORN: SIGNIFICANT VALUE CREATION OPPORTUNITIES

Potential Impact in >500K Patients Across More Than 20 Tumor Types with 6 Potential New INDs by 2026

- Validated platform with first-in-class target in the clinic (FHD-286), with Phase 1 combination data expected in H2 2024

- At least **6** additional potential NME **INDs** by 2026

- **>20** genetically defined tumor types in **over 500K** patients – includes lung, prostate, bladder, ovarian, colorectal, breast

- Opportunity for additional partnerships

	POTENTIAL PATIENTS IMPACTED BY PIPELINE	NEW MOLECULAR ENTITIES TO IND / CLINIC
Today	>500K PATIENTS	2 NME INDs
By 2026	>2M PATIENTS	8 NME INDs
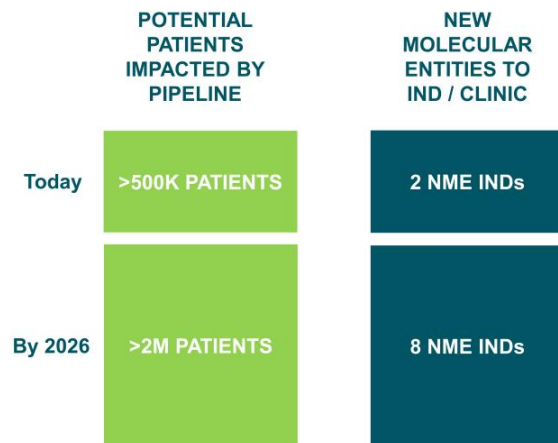
UNIQUE INSIGHTS INTO CHROMATIN BIOLOGY
Untapped Area for Novel Targets and Therapeutics

CHROMATIN REGULATORY SYSTEM CRITICAL FOR GENE EXPRESSION	NOVEL TARGETS GUIDED BY GENETIC DEPENDENCIES	TAILORED DRUGGING APPROACHES
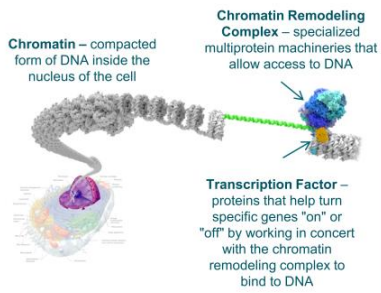
Chromatin – compacted form of DNA inside the nucleus of the cell

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

Chromatin Remodeling Complex Mutations / Overexpression
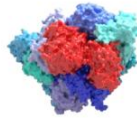
Transcription Factor Mutations / Overexpression

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

Enzymatic Inhibitors Highly selective and allosteric small molecule inhibitors

ATP   ADP

Targeted Protein Degradation Molecular glue and bi-functional protein degraders

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

Potential druggable sites

FOGHORN'S VALIDATED GENE TRAFFIC CONTROL® PLATFORM

Integrated, Scalable, Efficient – Repeatable Paradigm

UNIQUE TARGETS → **SPECIALIZED APPROACH** → **SELECTIVE THERAPEUTICS**
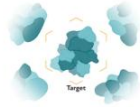
UNIQUE TARGETS	SPECIALIZED APPROACH	SELECTIVE THERAPEUTICS
Deep Mechanistic Understanding of the Chromatin Regulatory System	Biochemistry, Biophysics and Assays of Large Complexes and Proteins	Small Molecule and Degrader Platform
What to Drug:	**Where to Drug:**	**How to Drug:**
Identify disease dependencies	Engineer selectivity via unique assays and protein capabilities	Biology first - small molecule modality agnostic

Enzymatic Inhibitors	Targeted Protein Degraders	Transcription Factor Disruptors

BROAD PIPELINE ACROSS A RANGE OF TARGETS AND MODALITIES

Precision Oncology / Breadth and Depth / Over 15 Programs

Modality	Program	Discovery	Phase 1	Phase 2	Phase 3	Commercial Rights	Patient Population*
Enzyme Inhibitors	FHD-286 (BRG1/BRM)	AML Combination Study				FOGHORN THERAPEUTICS	Over 27,000
	Selective BRM	BRG1 Mutated Cancers, e.g. , NSCLC & Bladder				LOXO FOGHORN THERAPEUTICS	Over 100,000
	FHD-286 (BRG1/BRM)#	Uveal Melanoma				FOGHORN THERAPEUTICS	
Protein Degraders	Selective BRM	BRG1 Mutated Cancers, e.g., NSCLC & Bladder				LOXO FOGHORN THERAPEUTICS	Over 100,000
	Selective ARID1B	ARID1A Mutated Cancers, e.g., Ovarian, Endometrial & Colorectal				FOGHORN THERAPEUTICS	Over 175,000
	Selective CBP	EP300 Mutated Cancers, e.g., Prostate, Bladder, Colorectal, Breast				FOGHORN THERAPEUTICS	Over 100,000
	Selective EP300	CBP Mutated & Subsets of EP300 Dependent Cancers				FOGHORN THERAPEUTICS	Over 100,000
	FHD-609 (BRD9)^#	Synovial Sarcoma & SMARCB1-Loss Tumors				FOGHORN THERAPEUTICS	
Transcription Factor Disruptors	Undisclosed	Undisclosed				FOGHORN THERAPEUTICS	
Partnered Program	Undisclosed	Undisclosed				LOXO FOGHORN THERAPEUTICS	
3 Discovery Programs	Undisclosed	3 Undisclosed Programs				LOXO FOGHORN THERAPEUTICS	

* Per year incidence in the U.S., EU5, Japan | ^ On partial clinical hold | # Not advancing independently

Inhibition of the BRG1 and BRM Subunits of the BAF Complex

PHASE 1 COMBINATION STUDY FOR AML

FHD-286 Is a Potent, Selective, Allosteric, Small Molecule Inhibitor of the BRG1 and BRM
Subunits of the BAF Complex

TARGETING BAF DEPENDENCY IN CANCER

BAF CHROMATIN REMODELING COMPLEX

BRG1

BRM

BRG1 AND BRM SUBUNITS
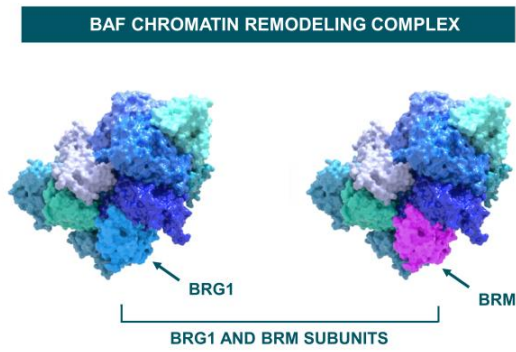
- BRM / BRG1 is the engine (ATPase) of the BAF chromatin remodeling complex

- Dependency on BRM / BRG1 is **well-established with multiple tumor types,** including uveal melanoma, AML / MDS, NSCLC and prostate

- Foghorn's lead asset targeting BRM / BRG1, **FHD-286, is a potent, selective, allosteric, small molecule inhibitor of the BRG1 and BRM subunits** of the BAF complex

- Phase 1 in combination with decitabine or low dose cytarabine for AML initiated; **data anticipated in H2'2024**

FHD-286: FIRST-IN-CLASS BROAD-BASED DIFFERENTIATION AGENT WITH SIGNIFICANT COMBINATION POTENTIAL IN AML

SIGNIFICANT OPPORTUNITY	PRE-CLINICAL AND CLINICAL DATA DEMONSTRATE BROAD-BASED DIFFERENTIATION	INITIATED PHASE 1 COMBINATION STUDY
~27,000 drug treated relapsed and/or refractory (R/R) AML patients in G7, with significant unmet need	First-in-class mechanism	Phase 1 in combination with decitabine or low dose cytarabine for AML initiated; data anticipated in H2'2024
No broad differentiation agent approved in AML	Differentiation observed in heavily pre-treated patients, regardless of mutational status	Focusing on first-line R/R AML patients
Significant opportunity for FHD-286	Peripheral blood and bone marrow blast reductions leading to absolute neutrophil count (ANC) recoveries in a subset of patients	
	Strong combination potential observed in pre-clinical models with multiple agents	

FHD-286 HAS THE POTENTIAL TO DIFFERENTIATE LEUKEMIC STEM CELLS WHICH ARE DRIVERS OF RELAPSE IN AML
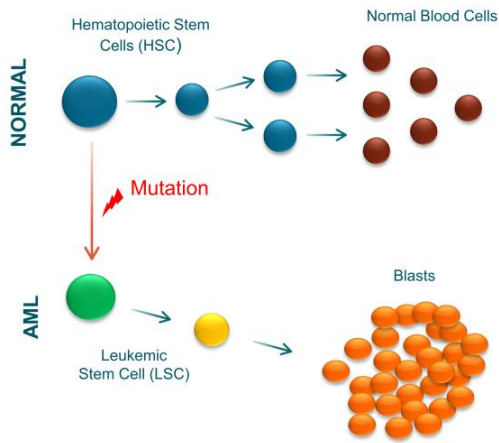
- Current AML therapies generally target proliferative blasts

- LSCs are drivers of AML relapse

- Combined approach of targeting both LSCs and blasts can theoretically prevent relapse
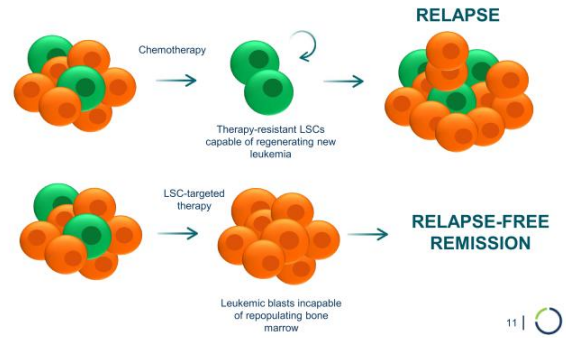
FHD-286 PHASE 1 MONOTHERAPY DOSE ESCALATION OVERVIEW

DESIGN
• Oral daily dosing of FHD-286 as monotherapy
• R/R AML and R/R MDS patients who exhausted all treatment options
• Doses tested: 2.5mg, 5.0mg, 7.5mg, 10.0mg once daily

PATIENTS
• 40 patients enrolled: 36 R/R AML and 4 R/R MDS
• 67.5% had 3+ prior lines
• Majority with abnormal karyotype (82.5%) and poor genetic risk factors (65% with adverse genetic status)
• Broad range of mutations

STUDY OBJECTIVES
• Safety and tolerability, MTD and/or RP2D
• Pharmacokinetics and pharmacodynamics, clinical activity, biomarker analysis

FHD-286 PHASE 1 DOSE ESCALATION SAFETY SUMMARY

Overall, adverse event profile consistent with a highly relapsed and/or refractory AML population

MOST COMMON TRAES

- Dry mouth, increased blood bilirubin, increased ALT, rash

MOST COMMON ≥ GRADE 3 TRAES

- Increased blood bilirubin, hypocalcemia, DS, stomatitis, increased ALT

EXPERT ADJUDICATION COMMITTEE ASSESSMENT OF DIFFERENTIATION SYNDROME

- Number of subjects with DS
  - 1 R/R AML subject adjudicated as having definitive DS; this subject also had 2 events of Investigator-reported DS
  - 5 R/R AML subjects adjudicated as indeterminate for DS; 2 of these 5 subjects had at least one event of Investigator-reported DS

- Range of Initial Onset: 4 to 42 days

- Potential DS Symptom include:
  - Pleural effusion, pericardial effusion, volume overload, weight gain, elevated WBC counts, hypotension, ground glass opacities and/or pulmonary infiltrates on imaging without documentation of positive cultures, hypoxia, pyrexia, and/or multi-organ involvement (lung, heart, and/or kidneys)

PERIPHERAL BLOOD AND BONE MARROW BLAST COUNT REDUCTION LEADING TO ANC RECOVERY IN A SUBSET OF PATIENTS

Diagnosis	Dose Level	Mutations	Cytogenetic Risk	Cycles on Study	ANC Recovery	Peripheral Starting Blast %	Peripheral Min Blast %	Peripheral Blast % Change	BMA Starting Blast %	BMA Min Blast %	BMA Blast % Change
AML	10mg	N/A	Adverse	2.2	**YES**	15	0	(100)	40	6	(85)
AML	10mg	DNMT3A, U2AF1, DDX41, CUX1, TP53	Adverse	0.5	N	20	0	(100)	13	2	(85)
AML	10mg	NRAS, SF3B1	Intermediate	7.3	N	2	0	(100)	12	5	(58)
AML	10mg	NRAS, BRCA1, MEN1, CDKN1Ap	Adverse	0.3	N	80	11	(86)	52	-	-
AML	10mg	D17Z1, TP53	Intermediate	0.6	N	9	1	(89)	9	-	-
AML	10mg	GATA2, ETV6, KDR	Intermediate	1.4	N	2	2	0	5	-	-
AML	7.5mg	RUNX1, KRAS, ASXL1, JAK2, TET2, EZH2, ETNK1	Intermediate	2.9	N	83	1	(99)	83	2	(98)
AML	7.5mg	ASXL1, TP53, U2AF1	Adverse	1.3	N	-	5	-	36	14	(61)
AML	7.5mg	KMT2A rearrangement	Adverse	2.8	**YES**	97	5	(95)	89	48	(46)
AML	7.5mg	N/A	Adverse	4.1	**YES**	28	4	(86)	25	15	(40)
* MDS	7.5mg	DNMT3A, TP53	Adverse	1.4	N	-	0	-	8	5	(38)
AML	7.5mg	DNMT3A, KRAS, NRAS	Adverse	1.8	N	32	2	(94)	47	49	4
AML	7.5mg	CBFB (locus at 16q22)	Favorable	1.7	**YES**	32	0	(100)	27	29	7
AML	7.5mg	N/A	Adverse	0.1	N	35	19	(46)	72	-	-
AML	7.5mg	ASXL1, BCOR, FLT3ITD, NF1, CBL, H1-B, NFE2	Adverse	0.7	N	8	7	(13)	25	-	-
AML	7.5mg	N/A	-	0.5	N	0	0	0	8	-	-
AML	7.5mg	NRAS, ASXL2, SRSF2	Adverse	0.1	N	93	-	-	17	-	-
AML	7.5mg	ASXL1, DNMT3A, TET2, TP53	Adverse	0.5	N	-	4	-	-	-	-
AML	7.5mg	FLT3ITD	Favorable	0.8	N	0	39	-	12	-	-

* MDS Patient

14 |

PERIPHERAL BLOOD AND BONE MARROW BLAST COUNT REDUCTION LEADING TO ANC RECOVERY IN A SUBSET OF PATIENTS

Diagnosis	Dose Level	Mutations	Cytogenetic Risk	Cycles on Study	ANC Recovery	Peripheral Starting Blast %	Peripheral Min Blast %	Peripheral Blast % Change	BMA Starting Blast %	BMA Min Blast %	BMA Blast % Change
AML	5mg	RUNX1, NRAS, ASLX1	Adverse	3.1	**YES**	29	0	(100)	35	12	(66)
AML	5mg	N/A	Adverse	8.0	N	-	2	-	11	7	(36)
AML	5mg	N/A	Adverse	1.8	**YES**	6	0	(100)	24	16	(33)
AML	5mg	ASXL1, DNMT3A, KRAS, PTPN11, WT1, GRIN2AWT1	Adverse	2.0	N	32	38	19	49	52	6
* MDS	5mg	RUNX1, NRAS, KRAS, SF3B1, ASXL2, CSF3R, GATA2	Adverse	1.0	**YES**	5	13	160	11	14	27
* MDS	5mg	DNMT3a, TET2	Intermediate	1.9	**YES**	0	0	0	1	2	100
AML	5mg	TET2, WT1, GATA2, PLCG2, ARHGEF28, BRD4, CDK12, DDX41, KMT20, PARP1, ZRSR2	Intermediate	1.7	**YES**	9	0	(100)	18	46	156
AML	5mg	KRAS, PTNP11, IRF8, MSH6, RUNX1	-	1.3	N	17	7	(59)	-	80	-
AML	5mg	TP53	Adverse	0.7	N	41	20	(51)	18	-	-
AML	5mg	TP53	Adverse	0.5	N	44	35	(20)	55	-	-
AML	5mg	PPM1D, TP53	Adverse	0.5	N	15	12	(20)	18	-	-
AML	5mg	KRAS, TET2	Adverse	0.6	N	37	32	(14)	56	-	-
* MDS	5mg	ASXL1, DNMT3A, IDH1, SRSF2, SF3B1, TET2	-	0.4	N	0	0	0	0	-	-
AML	5mg	N/A	Adverse	0.5	N	10	11	13	-	-	-
AML	5mg	ASXL1, NRAS, EP300, STAG2, RUNX1, TET2	Adverse	0.1	N	25	32	25	11	-	-
AML	5mg	CEBPA, KMT2C, NCOR1, CBL	-	0.3	N	48	75	56	64	-	-
AML	2.5mg	NRAS, WT1	Adverse	1.4	N	36	62	72	45	74	64
AML	2.5mg	BCR/ABL, PMLRARA, RUNX1, TET2	-	2.4	N	68	28	(59)	30	-	-
AML	2.5mg	N/A	Adverse	0.8	N	7	0	(100)	22	-	-
AML	2.5mg	DNMT3A, KRAS, TP53	Adverse	0.8	N	28	40	46	45	-	-
AML	2.5mg	DNMT3A, TP53	Adverse	1.0	N	4	-		25	-	

* MDS Patient

PATIENT 5: 25-YEAR-OLD WITH AML OBSERVED MEANINGFUL CLINICAL BENEFIT

25-YEAR-OLD MALE WITH TREATMENT-RELATED AML WITH A 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 7.5 MG Dose:**
  - Drop in peripheral blast, 97% to 5%
  - Bone marrow reduction from 89% to 48%, with ANC recovery

PERIPHERAL BLAST COUNT

ABSOLUTE NEUTROPHIL COUNT

16
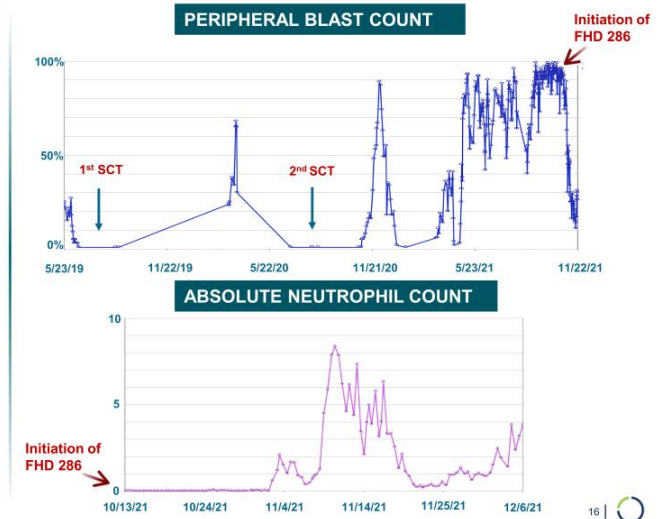
PATIENT 7: 47-YEAR-OLD WITH SECONDARY AML SHOWED CLEAR SIGNS OF DIFFERENTIATION

47-YEAR-OLD MALE WITH SAML WITH AN 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 BLAST REDUCTION FROM 40% TO 6%
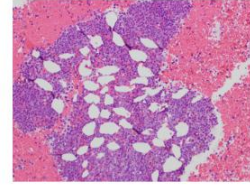
BONE MARROW ASPIRATE DEMONSTRATING CLEAR EVIDENCE OF DIFFERENTIATION
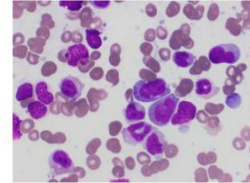
FHD-286 DEMONSTRATED DIFFERENTIATION ACROSS A BROAD RANGE OF GENETIC BACKGROUNDS

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%)

CD11b (marker of differentiation) increases

CD34 (leukemic stem cell marker) decreases

18

CLINICAL PATIENT SAMPLES SHOW LOSS OF LEUKEMIC STEM CELL IDENTITY AND TRANSFORMATION TO DIFFERENTIATED MARROW

- Single-cell RNA-seq of patient bone marrow aspirates show that marrow is heavily infiltrated with leukemic stem cell-like blasts at screening

- On treatment aspirates demonstrate that the bone marrow has lost leukemic stem cell phenotype and shifted to a more mature phenotype

- These samples recapitulate pre-clinical data of FHD-286's impact on leukemic stem cell potential

- Similar effects observed across 5.0mg, 7.5mg and 10.0mg dose levels

PATIENT BONE MARROW SHIFTS FROM LEUKEMIC STEM CELL-LIKE TO DIFFERENTIATED PHENOTYPE DURING FHD-286 THERAPY

SINGLE CELL RNA-SEQ OF PATIENT BONE MARROW AFTER ONE CYCLE AT 5.0MG

Screening

On Tx

LEUKEMIC STEMNESS GENE SIGNATURE

DIFFERENTIATED MYELOID GENE SIGNATURE
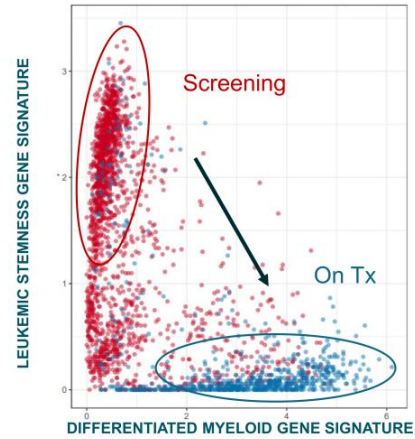
PRE-CLINICAL DATA DEMONSTRATE BROAD SINGLE AGENT AML ACTIVITY WITH SIGNIFICANT POTENTIAL FOR COMBINATION
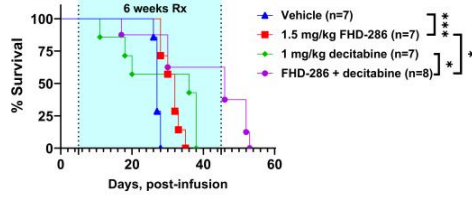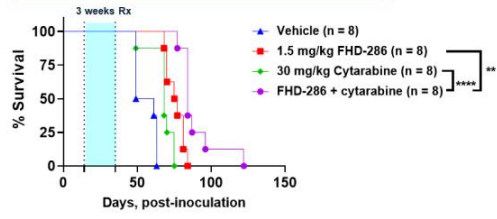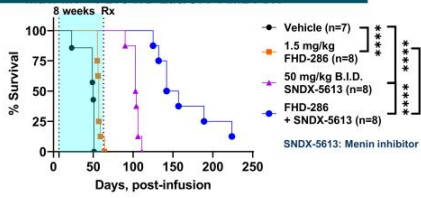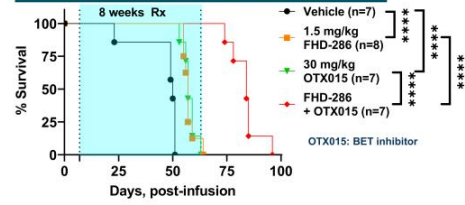
FHD-286 PHASE 1 COMBINATION STUDY OVERVIEW
Data anticipated in H2'2024

DESIGN	DS MANAGEMENT
· Standard 3+3 dose escalation design · Oral daily dosing of FHD-286 in combination with either fixed dose decitabine or fixed dose cytarabine · R/R AML patients 　· Allows for first-line relapsed and/or refractory AML patients	· Combination of FHD-286 with decitabine or cytarabine may mitigate the risk for differentiation syndrome given the cytoreductive properties of these agents · Adjudication committee · Enhanced monitoring and guidelines

STUDY OBJECTIVES
· Safety, tolerability and efficacy of the combination regimens · Pharmacokinetics and pharmacodynamics, biomarker analysis

SELECTIVE BRM MODULATORS
FOR BRG1 MUTATED CANCERS

Enzymatic Inhibitor and Protein Degrader 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	• BRM • Enzymatic inhibitor • Targeted protein degrader
Indications	• BRG1 mutated cancers (e.g., NSCLC), 30+ cancers with BRG1 mutations
Mutation / Aberration	• BRG1
Stage	• Pre-clinical
New Patients Impacted / Year*	• > 100,000
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
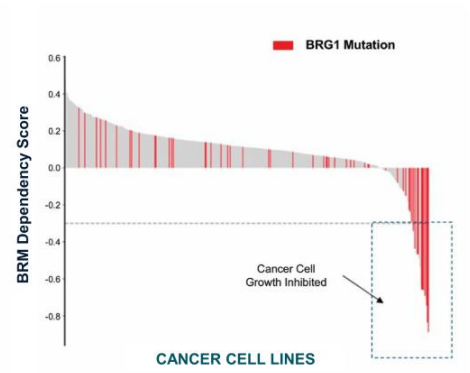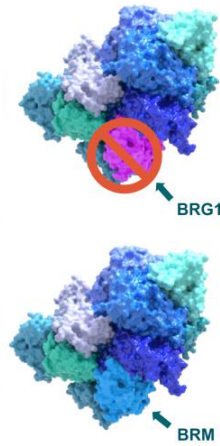
* Per year incidence in the U.S., EU5, Japan

BRG1 MUTATED IN ~5% OF ALL TUMORS
Broad Addressable Patient Population

BRG1 MUTATED ACROSS RANGE OF TUMORS
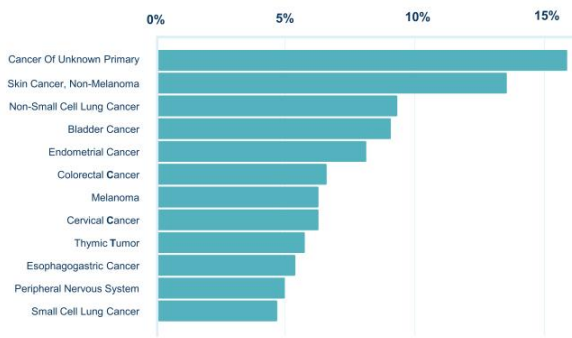
BRG1 MUTATED IN UP TO 10% OF NSCLC TUMORS, MINIMAL OVERLAP WITH OTHER MUTATIONS
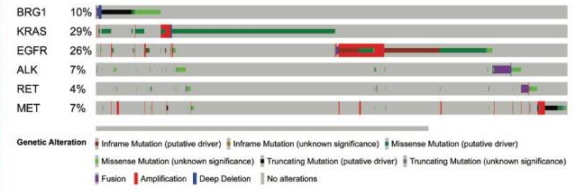
BRM SELECTIVE INHIBITOR *IN VIVO* EFFICACY
Demonstrates PK / PD and *In Vivo* Efficacy in a BRG1 Mutant Lung CDX Model

A549-BRG1 MUTANT NSCLC MODEL

Body Weight

- Vehicle Control (BID)
- Cisplatin 4 mg / kg (IP)
- FHT-BRMi 15 mg / kg (BID)
- FHT-BRMi 30 mg / kg (BID)

BRG1 cellular IC50

BRM cellular IC50
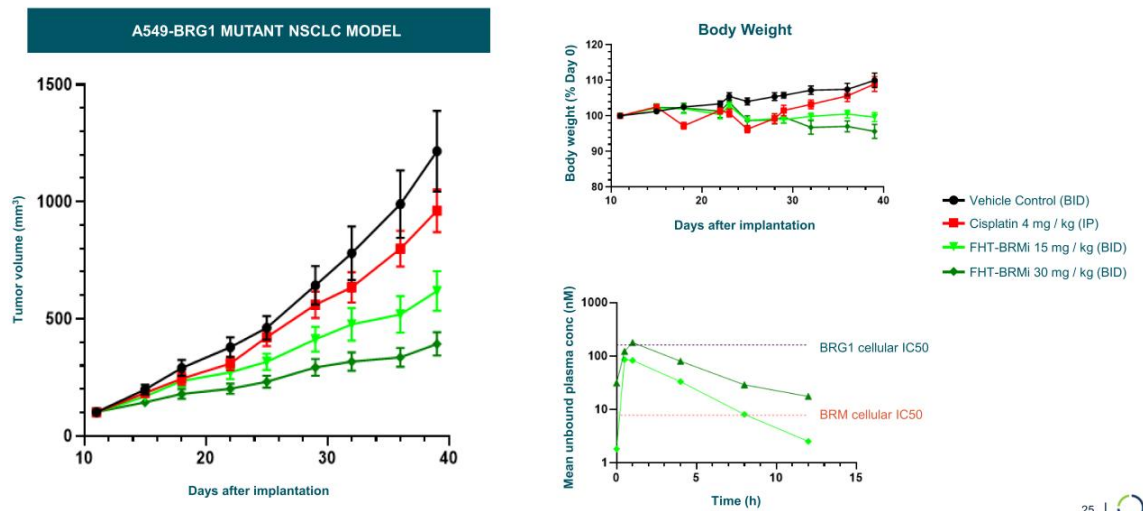
ENZYMATIC SELECTIVITY APPROACHING 200X ACHIEVED

BRG1 vs BRM ATPase activity
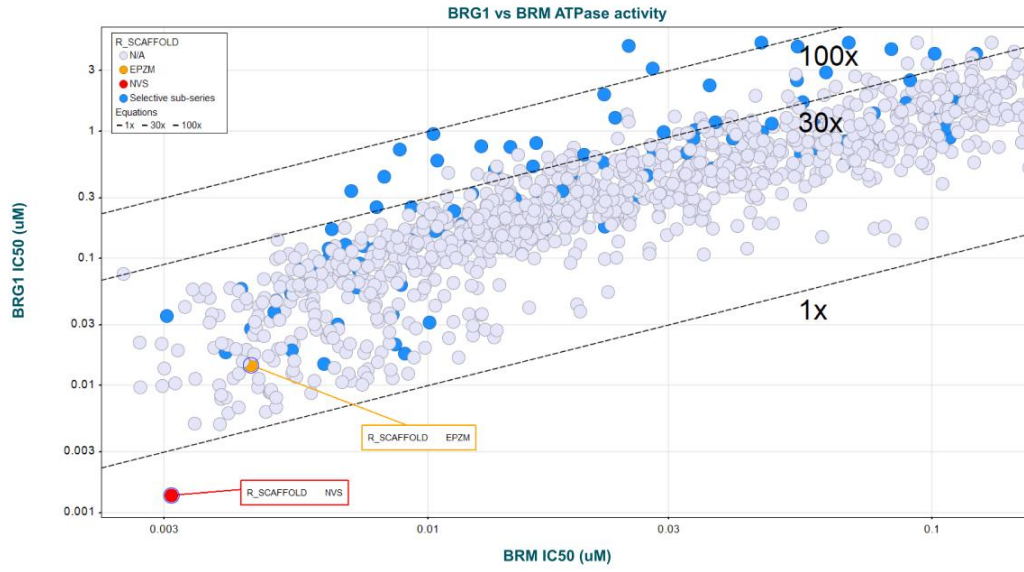
ADVANCING BRM SELECTIVE DEGRADERS
Achieving Complete BRM Degradation
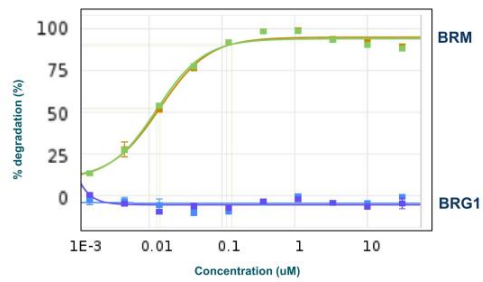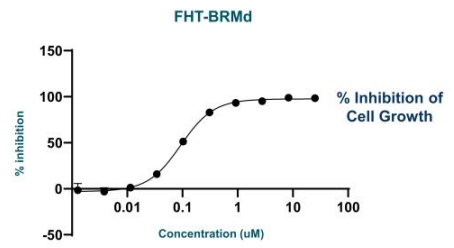
DEGRADERS CAUSE TIME- AND DOSE-DEPENDENT BRM DEGRADATION, ANTIPROLIFERATIVE EFFECTS IN A549 BRG1 MUTANT NSCLC LUNG MODEL

SELECTIVE CBP PROTEIN DEGRADER
FOR EP300 MUTATED CANCERS

Implicated in Subsets of Cancers Including Bladder, Colorectal, Breast, Gastric and Lung

ADVANCING HIGHLY SELECTIVE CBP PROTEIN DEGRADER FOR EP300 MUTATED CANCERS

Selective CBP Protein Degrader Overview

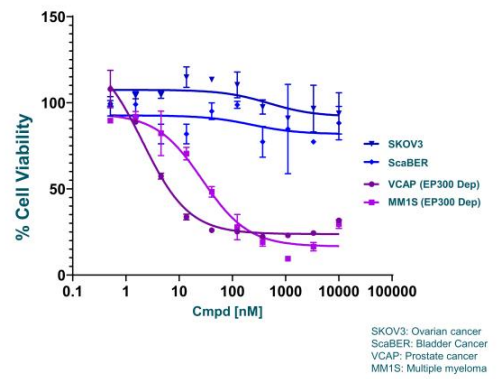|	
Target / Approach	· CREB binding protein (CBP) · Targeted protein degrader
Initial Indication	· EP300 mutated cancers (e.g., subsets of bladder, colorectal, breast, gastric and lung cancers)
Mutation / Aberration	· EP300 mutated cancers
Stage	· Pre-clinical
New Patients Impacted / Year*	· Over 100,000
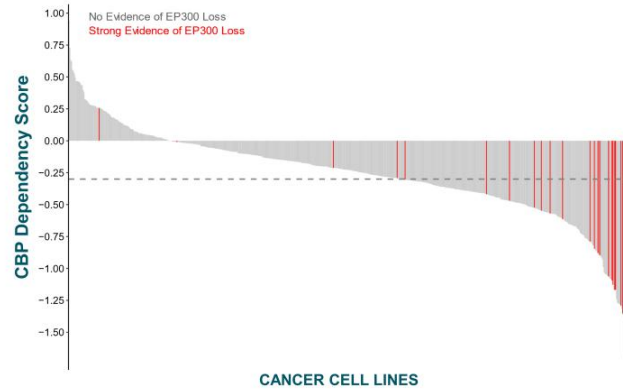
* Per year incidence in the U.S., EU5, Japan

ADVANCEMENT OF HIGHLY SELECTIVE CBP DEGRADERS

SELECTIVE CBP DEGRADATION
Osteosarcoma Cell Line

Selective CBP Degrader
U2OS

Endogenous IF

HiBiT-CBP
U2OS

Dmax: 88%

HiBiT-EP300
U2OS
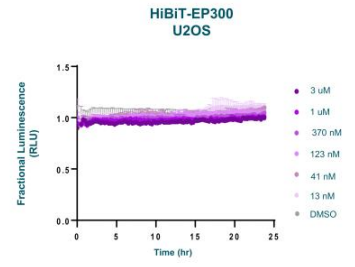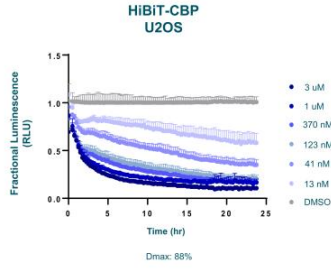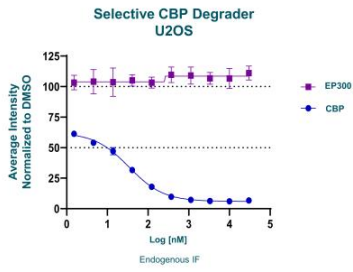
HIGHLY SELECTIVE DEGRADER OF CBP DEMONSTRATES CBP-DEPENDENT CELL KILLING ACROSS MULTIPLE CANCERS
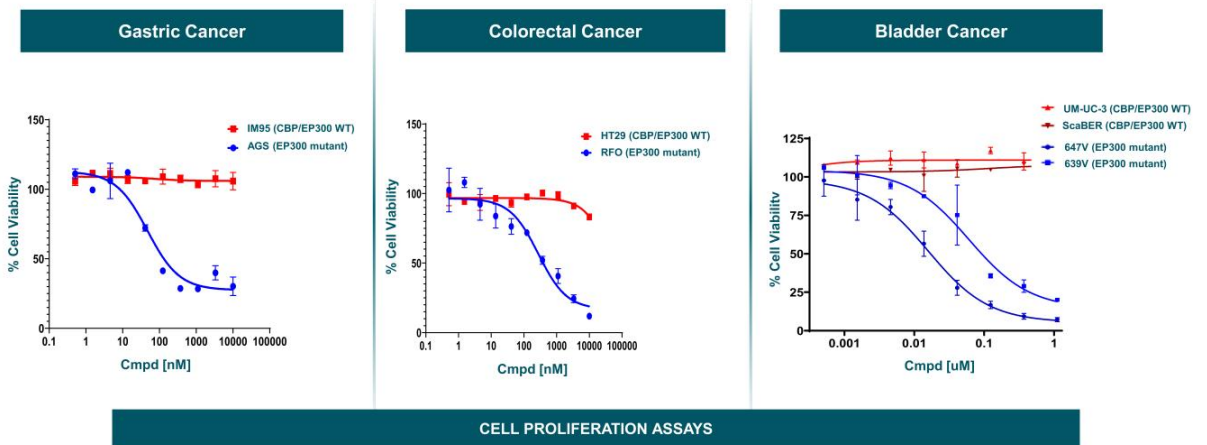
SELECTIVE EP300 PROTEIN DEGRADER
FOR CBP MUTANT CANCERS & EP300 DEPENDENT MALIGNANCIES

Implicated in CBP Mutated Cancers and Subsets of EP300 Dependent Malignancies (e.g., Bladder, NSCLC, Various Lymphomas and Leukemias)

ADVANCING HIGHLY SELECTIVE EP300 PROTEIN DEGRADER FOR CBP MUTANT CANCERS & EP300 DEPENDENT MALIGNANCIES

Selective EP300 Protein Degrader Overview

Target / Approach	· E1A binding protein p300 (EP300) · Targeted protein degrader
Initial Indication	· CBP mutant cancers and subsets of EP300 dependent malignancies (e.g., prostate, bladder, NSCLC, various lymphomas and leukemias)
Mutation / Aberration	· CBP mutant cancers · EP300 dependent malignancies
Stage	· Pre-clinical
New Patients Impacted / Year*	· Over 100,000

No Evidence of CREBBP Loss
Strong Evidence of CREBBP Loss

EP300 Dependency Score

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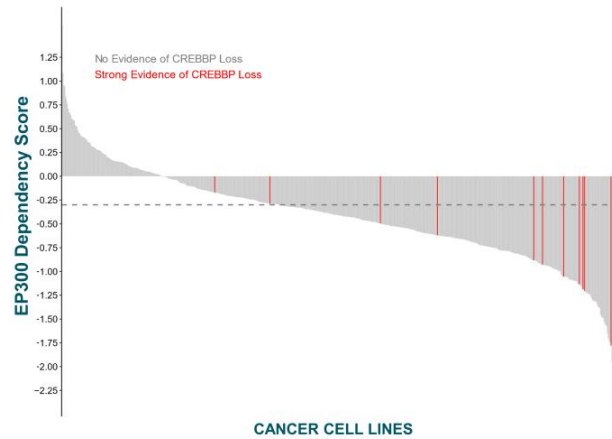
* Per year incidence in the U.S., EU5, Japan

ADVANCEMENT OF HIGHLY SELECTIVE EP300 DEGRADERS

SELECTIVE ARID1B PROTEIN DEGRADER
FOR ARID1A MUTATED CANCERS

Protein Degrader 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
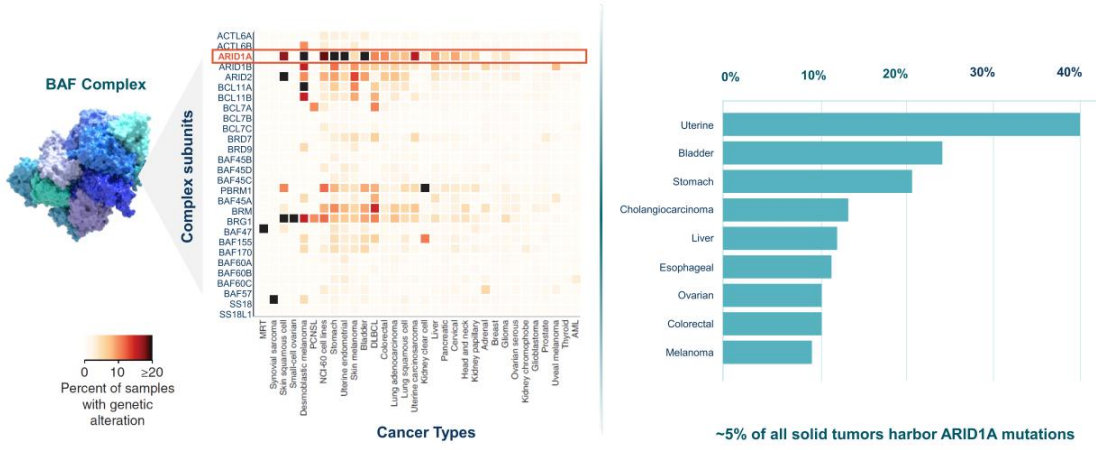
Selective ARID1B Protein Degrader Overview

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
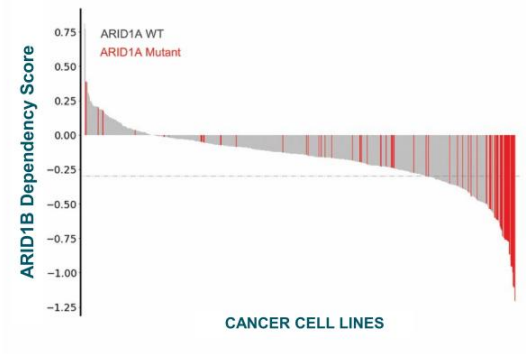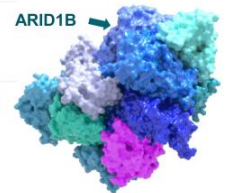
ARID1A MUTATED CANCERS: SIGNIFICANT OPPORTUNITY
ARID1A Mutated Across Range of Tumors

BAF Complex

Complex subunits

Percent of samples with genetic alteration

0 10 ≥20

Cancer Types

~5% of all solid tumors harbor ARID1A mutations

TARGETING ARID1A MUTATED CANCERS: ARID1B PROTEIN DEGRADER

Advantaged by Gene Traffic Control Platform and Protein Degrader 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

ARID1B
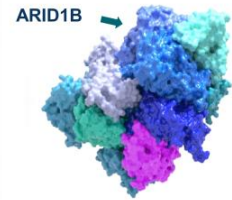
TRANSCRIPTION FACTORS
A NOVEL APPROACH

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

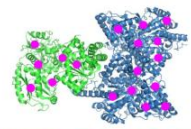- Druggable binding pockets
- Druggable affinities

FOGHORN'S FOCUS

POTENTIAL DRUGGABLE SITES

HISTORICAL FOCUS
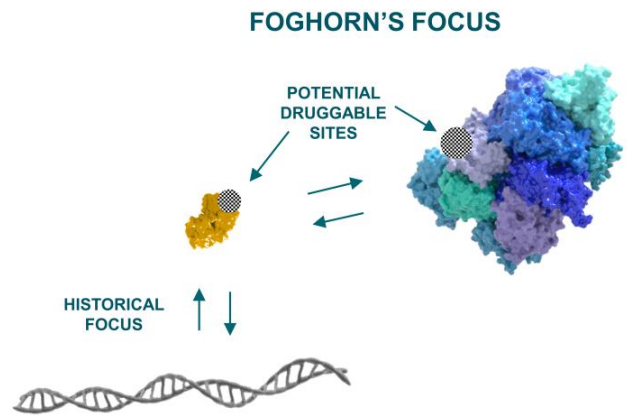
TRANSCRIPTION FACTORS BIND TO BAF DIRECTLY WITH HIGH DEGREE OF SPECIFICITY
Unique Insights into Where and How Transcription Factors Bind

SPI1

TF B

TF C

TF D
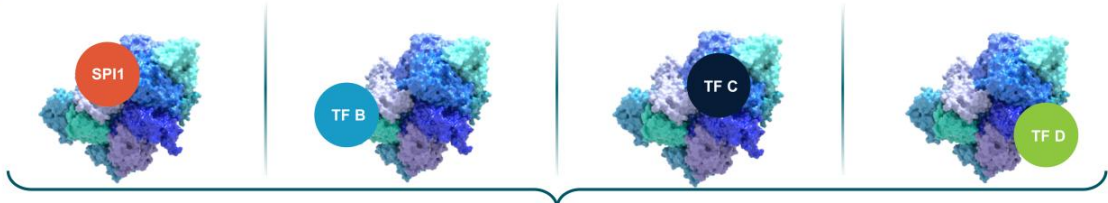
MAPPING THE TF-BAF INTERACTION

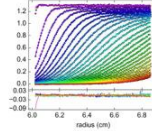**MASS SPEC. FOOT-PRINTING**
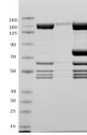
PULL-DOWN ASSAYS

Foghorn's collection of BAF sub-complexes and domains
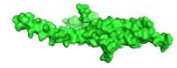
VALIDATING THE TF-BAF INTERACTION

BIOPHYSICAL
AUC / SPR / ITC

BIOCHEMICAL
TR-FRET / FP

SPI1

STRUCTURAL
Crystal / NMR

FIRST-IN-CLASS PRECISION MEDICINES TARGETING MAJOR UNMET NEEDS IN CANCER

LEADER IN NEW AREA OF CANCER BIOLOGY

Foghorn is a **leader in targeting chromatin biology,** which has unique potential to address underlying dependencies of many genetically defined cancers

Broad pipeline of **over 15 programs** 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

$284.3 million in cash and equivalents

(as of 06/30/2023)

Provides **runway into H2'25**

SIGNIFICANT UPCOMING VALUE DRIVERS

Selective BRM program on track to transition to Loxo@Lilly in **H2'23**

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

MAJOR STRATEGIC COLLABORATION

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

BROAD PIPELINE ACROSS A RANGE OF TARGETS AND MODALITIES

Precision Oncology / Breadth and Depth / Over 15 Programs

Modality	Program	Discovery	Phase 1	Phase 2	Phase 3	Commercial Rights	Patient Population*
Enzyme Inhibitors	FHD-286 (BRG1/BRM)	AML Combination Study				FOGHORN THERAPEUTICS	Over 27,000
	Selective BRM	BRG1 Mutated Cancers, e.g., NSCLC & Bladder				LOXO FOGHORN THERAPEUTICS	Over 100,000
	FHD-286 (BRG1/BRM)#	Uveal Melanoma				FOGHORN THERAPEUTICS	
Protein Degraders	Selective BRM	BRG1 Mutated Cancers, e.g., NSCLC & Bladder				LOXO FOGHORN THERAPEUTICS	Over 100,000
	Selective ARID1B	ARID1A Mutated Cancers, e.g., Ovarian, Endometrial & Colorectal				FOGHORN THERAPEUTICS	Over 175,000
	Selective CBP	EP300 Mutated Cancers, e.g., Prostate, Bladder, Colorectal, Breast				FOGHORN THERAPEUTICS	Over 100,000
	Selective EP300	CBP Mutated & Subsets of EP300 Dependent Cancers				FOGHORN THERAPEUTICS	Over 100,000
	FHD-609 (BRD9)^#	Synovial Sarcoma & SMARCB1-Loss Tumors				FOGHORN THERAPEUTICS	
Transcription Factor Disruptors	Undisclosed	Undisclosed				FOGHORN THERAPEUTICS	
Partnered Program	Undisclosed	Undisclosed				LOXO FOGHORN THERAPEUTICS	
3 Discovery Programs	Undisclosed	3 Undisclosed Programs				LOXO FOGHORN THERAPEUTICS	

* Per year incidence in the U.S., EU5, Japan | ^ On partial clinical hold | # Not advancing independently

43

APPENDIX

FHD-286

PHASE 1 COMBINATION STUDY FOR AML

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

SIGNIFICANT UNMET NEED REMAINS IN R/R AML REGARDLESS OF GENOMIC ALTERATIONS

~27,000 DRUG TREATED RELAPSED AND/OR REFRACTORY AML PATIENTS IN G7

US: ~ 11,500 patients
G6: ~ 16,800 patients

FIT

INTENSIVE CHEMO (IC)
30%
1LRR
FHD-286 OPPORTUNITY

UNFIT

VEN/HMA
30%
1LRR
FHD-286 OPPORTUNITY

PHASE 1 MONOTHERAPY DOSE ESCALATION PATIENT CHARACTERISTICS

Highly Relapsed and Refractory, Abnormal Karyotype, and Poor Genetic Risk Factors

		2.5 mg QD N=5	5 mg QD N=16	7.5 mg QD N=13	10 mg QD N=6	Total N=40
Age (years)						
	Median (min, max)	73.0 (61, 84)	67.5 (43, 80)	66.0 (25, 75)	45.0 (27, 79)	65.5 (25, 84)
ECOG status at baseline, n (%)						
	0	0	5 (31.3)	5 (38.5)	3 (50.0)	13 (32.5)
	1	4 (80.0)	8 (50.0)	7 (53.8)	3 (50.0)	22 (55.0)
	2	1 (20.0)	3 (18.8)	1 (7.7)	0	5 (12.5)
Type of AML/MDS, n (%)						
	De novo AML	0	8 (50.0)	6 (46.2)	3 (50.0)	17 (42.5)
	Secondary AML	5 (100)	5 (31.3)	6 (46.2)	3 (50.0)	19 (47.5)
	MDS	0	3 (18.8)	1 (7.7)	0	4 (10.0)
Number of prior lines of systemic anti-cancer therapy for AML/MDS						
	Median (min, max)	3 (1, 5)	3 (1, 6)	4 (1, 7)	3 (1, 5)	3 (1, 7)
Risk stratification by genetics at screening, n (%)						
	Favorable	0	0	2 (15.4)	0	2 (5.0)
	Intermediate	0	1 (6.3)	0	3 (50.0)	4 (10.0)
	Adverse	4 (80.0)	10 (62.5)	9 (69.2)	3 (50.0)	26 (65.0)
	Unknown/missing	0 / 1 (20.0)	5 (31.3) / 0	2 (15.4) / 0	0 / 0	7 (17.5) /1 (2.5)

AML=acute myeloid leukemia; ECOG=Eastern Cooperative Oncology Group; Max=maximum; MDS=myelodysplastic syndrome; Min=minimum; MPD=myeloproliferative disease; QD=once daily.

PHASE 1 MONOTHERAPY DOSE ESCALATION SAFETY SUMMARY

Any Grade Treatment-Related Adverse Events Occurring in >10% of Subjects

|	2.5 mg QD N=5	5 mg QD N=16	7.5 mg QD N=13	10 mg QD N=6	Total N=40
Any grade TRAE	5 (100)	15 (93.8)	10 (76.9)	4 (66.7)	34 (85.0)
Dry mouth	0	8 (50.0)	3 (23.1)	0	11 (27.5)
Increased blood bilirubin	0	4 (25.0)	3 (23.1)	2 (33.3)	9 (22.5)
ALT increased	1 (20.0)	4 (25.0)	2 (15.4)	1 (16.7)	8 (20.0)
Rash	1 (20.0)	4 (25.0)	1 (7.7)	2 (33.3)	8 (20.0)
Diarrhea	0	4 (25.0)	2 (15.4)	1 (16.7)	7 (17.5)
Nausea/vomiting	0	5 (31.3)	0	2 (33.3)	7 (17.5)
Fatigue	1 (20.0)	5 (31.3)	0	1 (16.7)	7 (17.5)
Dysgeusia	0	4 (25.0)	0	2 (33.3)	6 (15.0)
Decreased appetite	1 (20.0)	3 (18.8)	0	1 (16.7)	5 (12.5)
AST increased	0	4 (25.0)	1 (7.7)	0	5 (12.5)
Hypocalcemia	2 (40.0)	1 (6.3)	0	1 (16.7)	4 (10.0)
Differentiation syndrome	1 (20.0)	1 (6.3)	2 (15.4)	0	4 (10.0)
Mucosal inflammation	1 (20.0)	1 (6.3)	2 (15.4)	0	4 (10.0)
Peripheral edema	1 (20.0)	2 (12.5)	1 (7.7)	0	4 (10.0)

PHASE 1 MONOTHERAPY DOSE ESCALATION SAFETY SUMMARY

Grade 3 or Higher Treatment-Related Adverse Events Occurring in >5% of Subjects

	2.5 mg QD N=5	5 mg QD N=16	7.5 mg QD N=13	10 mg QD N=6	Total N=40
Grade ≥3 TRAEs	1 (20.0)	9 (56.3)	8 (61.5)	2 (33.3)	20 (50.0)
Increased blood bilirubin	0	2 (12.5)	2 (15.4)	1 (16.7)	5 (12.5)
Hypocalcemia	1 (20.0)	1 (6.3)	0	1 (16.7)	3 (7.5)
Differentiation syndrome	0	1 (6.3)	2 (15.4)	0	3 (7.5)
Stomatitis	0	2 (12.5)	1 (7.7)	0	3 (7.5)
ALT increased	0	1 (6.3)	2 (15.4)	0	3 (7.5)
Rash	0	1 (6.3)	1 (7.7)	0	2 (5.0)
Fatigue	0	1 (6.3)	0	1 (16.7)	2 (5.0)
Mucosal Inflammation	0	0	2 (15.4)	0	2 (5.0)
Diarrhea	0	2 (12.5)	0	0	2 (5.0)

FHD-286 SIGNIFICANTLY REDUCES LEUKEMOGENIC POTENTIAL IN *IN VIVO* TRANSPLANT MODEL

LEUKEMOGENIC POTENTIAL TRANSPLANT MODEL
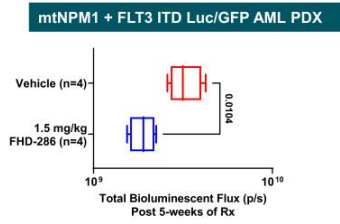
Tumor bearing animals treated for 5 weeks and then sacrificed
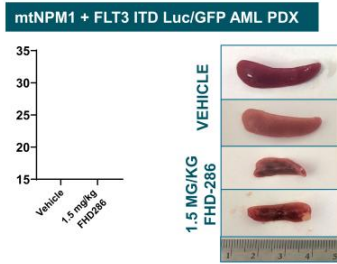
Spleens and bone marrow removed and assessed
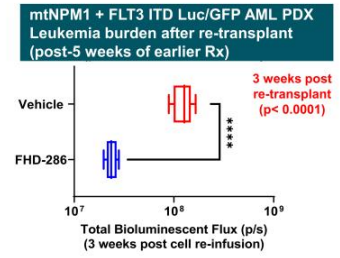
Bone marrow from sacrificed animals transplanted into new, non-tumor bearing animals; monitored for relapse

INCREASES IN CD11b+ CELLS AND DECREASES IN CD34+ CELLS ARE ASSOCIATED WITH DIFFERENTIATION AGENTS

- Mature differentiated cells are functionally specialized and compose the majority of cells in the body

- Cancer cells often revert to a more stem-like state in order to gain self-renewal and resistance phenotypes

- CD34 is a marker of hematopoietic stem cells that can differentiate into CD11b+ mature myeloid cells

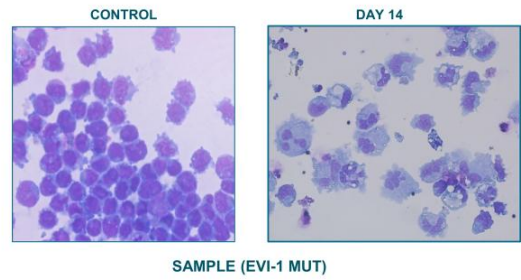- During the differentiation process, CD11b+ cells increase and CD34+ cells decrease
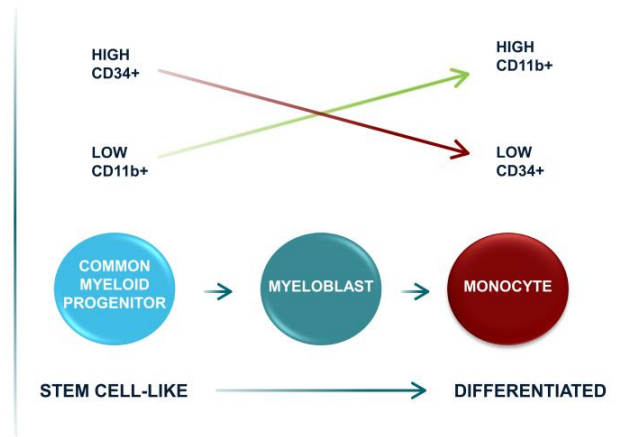
ROBUST DIFFERENTIATION EFFECT OBSERVED IN AML PRE-CLINICAL MODELS

FHD-286 CAUSES DIFFERENTIATION IN AML CELL LINES
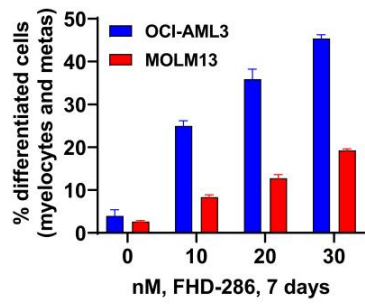
MORPHOLOGIC CHANGES OBSERVED IN PATIENT DERIVED CELL MODELS TREATED WITH FHD-286

CONTROL — DAY 14

SAMPLE (EVI-1 MUT)

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

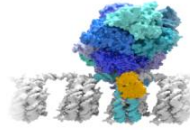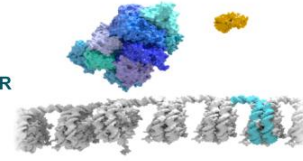Two Major Components Work in Concert: Chromatin Remodeling Complexes and Transcription Factors
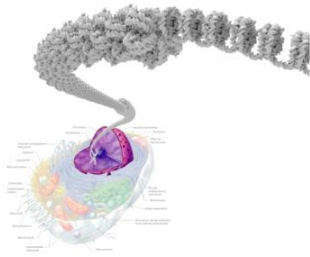
CHROMATIN

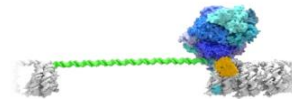Chromatin – compacted form of DNA inside the nucleus of the cell

1 | CHROMATIN REMODELING COMPLEX AND TRANSCRIPTION FACTOR

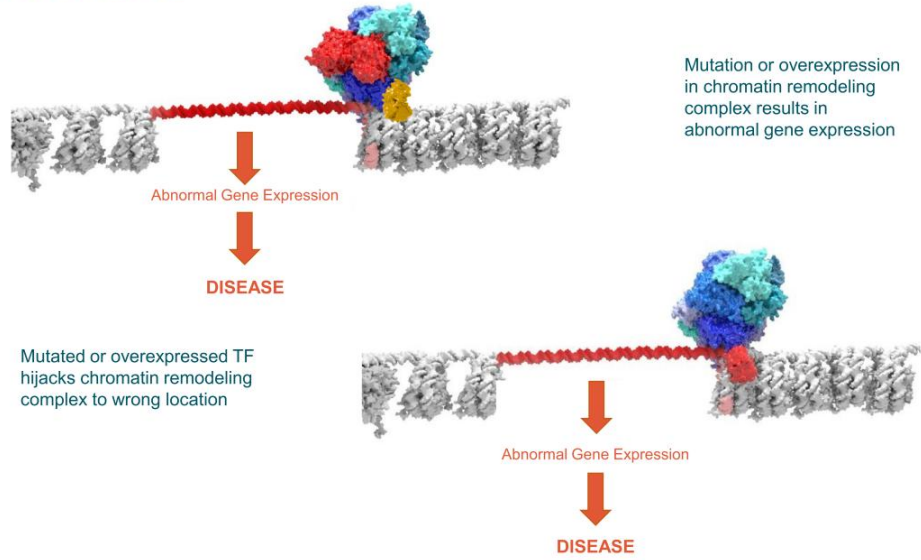Work together to orchestrate gene expression

2 | RIGHT GENES

TFs guide chromatin remodeling complexes to the right locations

3 | NORMAL GENE EXPRESSION

Once chromatin is unpacked, gene expression can occur

BREAKDOWNS IN THE CHROMATIN REGULATORY SYSTEM CAN LEAD TO DISEASE

Mutation or overexpression in chromatin remodeling complex results in abnormal gene expression

Abnormal Gene Expression

DISEASE

Mutated or overexpressed TF hijacks chromatin remodeling complex to wrong location

Abnormal Gene Expression

DISEASE

CHROMATIN REGULATORY SYSTEM
Abundance of Targets within the BAF Complex

BAF COMPLEX AND ASSOCIATED TRANSCRIPTION FACTORS
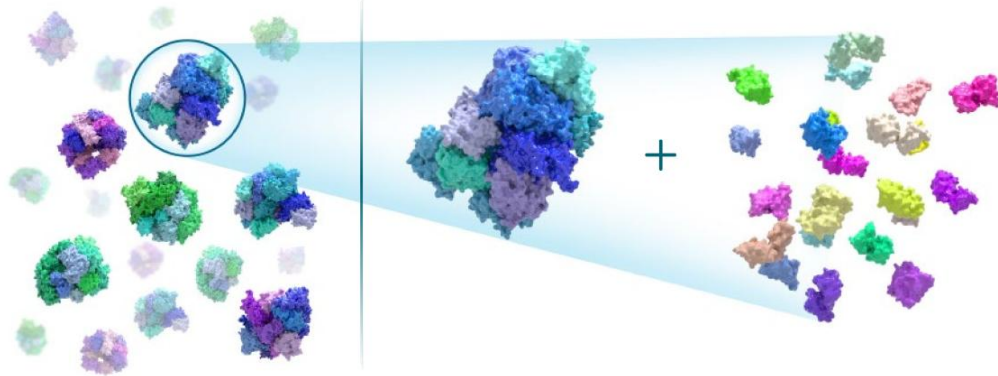
28 Chromatin Remodeling Complexes and >1,000 TFs

BAF Complex Subunits Mutated and Dysregulated in Cancer

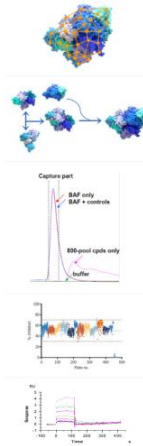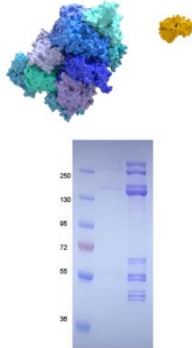Estimate >100 Transcription Factors Associated with Just the BAF Complex

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

60

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
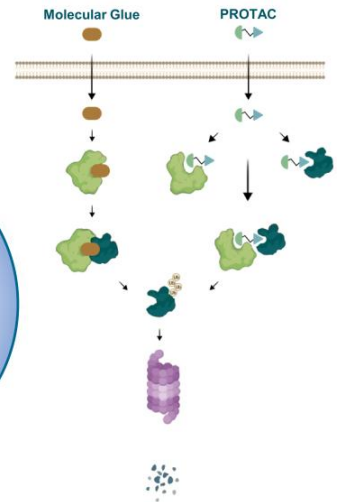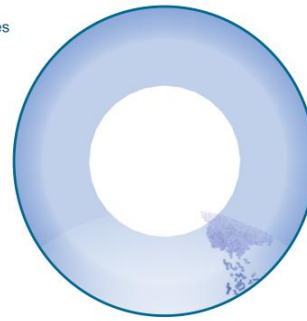
Leadership Team, Board & Advisors

EXPERTISE ACROSS DRUG DISCOVERY, CLINICAL DEVELOPMENT AND COMMERCIALIZATION

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VP, Investor Relations & Corporate Communications

KEVIN WILSON
VP, Chemistry

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