Building A New Protein Degrader Company For Unmet Medical Needs

Gyre THERAPEUTICS

August 2026

Forward-looking Statements

This presentation contains "forward-looking statements" within the meaning of the federal securities laws, including Section 27A of the United States Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, regarding the current plans, expectations and strategies of Gyre Therapeutics, Inc. and its subsidiaries ("Gyre"), which statements are subject to substantial risks and uncertainties and are based on management's estimates and assumptions. All statements, other than statements of historical facts included in this presentation, are forward-looking statements, including Gyre's ability to leverage China operations for discovery, validation and development of therapeutics, clinical development plans, anticipated timelines and milestones of Gyre's degrader antibody-conjugate platform, CG923308, CG620953, CG009301, CG001419, F351 and ETUARY™, including the geographic location and timing of anticipated regulatory submissions and approvals, and the potential therapeutic benefits, efficacy, safety and differentiation of such product candidates, and market size and commercial opportunity estimates, Gyre's plans, objectives, goals, strategies, future events, or intentions relating to Gyre's products and markets, the safety, efficacy and clinical benefits of Gyre's product candidates, the anticipated timing and design of any planned and ongoing preclinical studies and clinical trials, Gyre's research and development efforts, plans and objectives of management for future operations and future results of anticipated product development efforts, potential addressable market size and Gyre's liquidity and capital resources and business trends. In some cases, you can identify forward-looking statements by terms such as "believe," "can," "could," "anticipate", "design," "estimate," "expect," "forecast," "intend," "may," "might," "plan," "target", "seek", "goal", "assume", "milestones", "potential," "predict," "objective," "should," "strategy," "will," "would," "forthcoming," or the negative of these terms, and similar expressions that are predictions of or indicate future events and future trends. These forward-looking statements may include express or implied statements relating to: the estimated future financial performance and financial position of Gyre; the therapeutic potential and utility, efficacy and clinical benefits of the product candidates of Gyre; the risk/benefit profile of Gyre's product candidates; expectations regarding Gyre's research and development efforts, including geographic location and timing of initiation of clinical trials for Gyre's product candidates; Gyre's expectations regarding the advancement of product candidates into IND-enabling studies; and Gyre's expectations, hopes, beliefs, intentions and strategies; and other statements that are not historical fact. These statements involve known and unknown risks, uncertainties and other factors that could cause Gyre's actual results to differ materially from the forward-looking statements expressed or implied in this presentation, in addition to those risks and uncertainties, such as the uncertainties inherent in the clinical drug development process, the regulatory approval process, the timing of any regulatory filings, the potential for substantial delays, the risk that earlier study results may not be predictive of future study results, manufacturing risks, competition from other therapies or products and the impacts of current macroeconomic and geopolitical risks. A discussion of these and other factors, is set forth in Gyre's Annual Report on Form 10-K for the year ended December 31, 2025 filed with the Securities and Exchange Commission (the "SEC") on March 13, 2026 and elsewhere in such other filings and in Gyre's periodic reports and subsequent disclosure documents filed with the SEC. Gyre cannot assure you that it will realize the results, benefits or developments that it expects or anticipates or, even if substantially realized, that they will result in the consequences or affect Gyre or its business in the way expected. Forward-looking statements are not historical facts and reflect management's current views with respect to future events. Given the significant uncertainties, you should evaluate all forward-looking statements in the context of these risks and uncertainties and not place undue reliance on these forward-looking statements as predictions of future events. All forward-looking statements in this presentation apply only as of the date made and are expressly qualified in their entirety by the cautionary statements included in this presentation. Gyre has no intention to publicly update or revise any forward-looking statements to reflect subsequent events or circumstances, except as required by law.

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Gyre Is A Global Development Engine Fueled by China Discovery and Commercialization Platform

Value Drivers	Details	Key Features
Growth Opportunities	• Diversified R&D programs spanning across multiple indications • Both early- and late-stage pipeline assets provide near- and long-term catalysts	**Degrader platform for the development of TPDs & DACs**
Established Innovation Engine	• Support our next-generation targeted protein degrader (TPD) and Degrader Antibody-Conjugate (DAC) platform development • Fully-integrated R&D capabilities	**Dual degraders for oncology/I&I DACs in Pre-IND studies**
Global Infrastructure	• Leveraging cost-efficient China operations for accelerated discovery, preclinical validation, and early clinical-stage development	**San Diego: HQ** **Shanghai: Discovery** **Beijing: Manufacturing & Sales**
Commercial Footprint	• ETUARY™ has been the market-leading pirfenidone brand in China for treatment of idiopathic pulmonary fibrosis (IPF); FY26 ETUARY™ Total Revenue Guidance of $100.5-$111MM • F351 adds a near-term revenue growth catalyst for the China commercial portfolio	**Established market for ETUARY™** **F351 potential launch in early 27' post-NMPA decision for CHB-induced liver fibrosis**

A Commercial Foundation and Integrated U.S.– China R&D Engine Support Capital-Efficient Advancement of a Diversified Pipeline

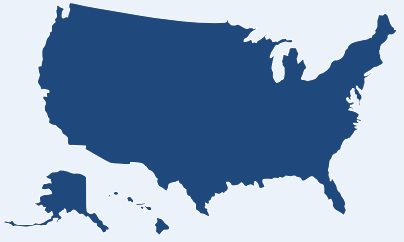
Gyre's Flexible, Capital-Efficient Global Development Strategy

China Revenue Builds & De-Risks the Early Pipeline

- **Cash-flow Generation:** Established commercial operations in China to help offset cash burn.
- **Reinvestment to R&D Engine:** China-generated cash funds preclinical development quickly, at scale, and cost-efficiently, leveraging Gyre's already-established China infrastructure for multiple shots-on-goal.
- **Translational De-risking:** Conduct broader preclinical and translational studies to select strongest candidates and build IND-enabling packages.

U.S. Capital Advances Priority Assets Globally

- **Focused Capital Allocation:** U.S.-raised capital invests into the highest-priority U.S. and global clinical programs.
- **Flexible Clinical Routing:** Initiate studies in the U.S., China or both based on the most efficient development path.
- **Pivotal Development and Launch:** Advance selected assets through U.S. and global clinical studies, pivotal trials, regulatory approvals and launch.



Priority Pipeline and Upcoming Catalysts

Program and Description	Indication	Pre-IND Studies	Phase 1	Phase 2	Phase 3	Regulatory	Catalyst
DAC Platform / Next-Gen Degrader Technologies	**Oncology / Inflammatory Disease Discovery Platform**	*Discovery & lead selection in China*					Lead DAC selection / IND-enabling advancement
CDK2 / Cyclin E Degrader CG923308	**CDK4/6i-resistant Breast Cancer and Other Solid Tumors**	*IND-enabling development in China*					Q1'27 IND-application submission in U.S. and/or China; Phase 1 start in 2027
TYK2 / JAK 1 Degrader CG620953	**Systemic Lupus Erythematosus (SLE) & Rheumatoid Arthritis (RA)**	*IND-enabling development in China*					Q1'27 IND-application submission in U.S. and/or China; Phase 1 start in 2027
GSPT1 Degrader CG009301	**R/R AML, HR-MDS, R/R ALL; Solid Tumor Cancers**		*Phase 1 dose escalation ongoing in China*				Phase 1 data in patients with high-risk hematologic malignancies expected at trial completion
TRK Degrader CG001419	**Cancer-induced Bone Pain (CIBP)**		*Phase 1 in healthy volunteers completed*				Planning Phase 2 initiation in U.S. or China
Hydronidone F351	**Chronic Hepatitis B (CHB)-induced Liver Fibrosis**						NDA-filing accepted by China NMPA in May 2026; Potential launch post-NMPA decision in early 2027

Cancer Inflammation / Fibrosis Degrader DAC

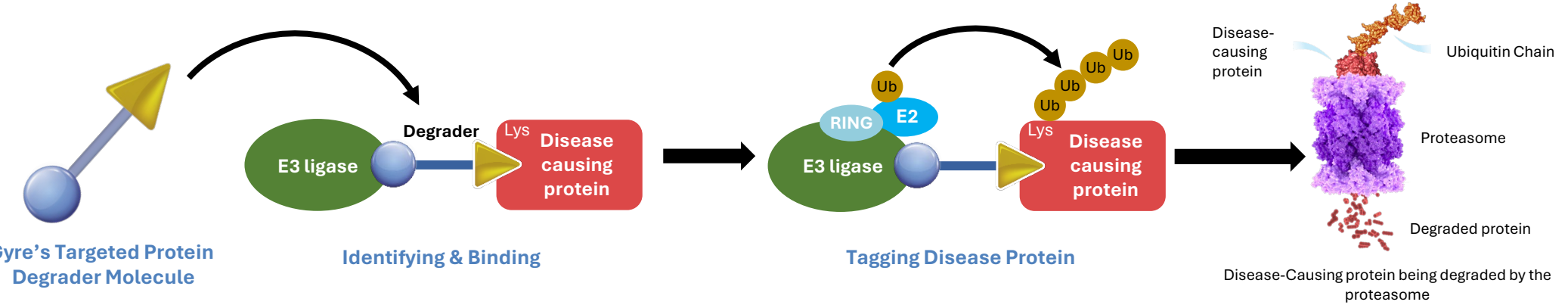
China R&D incubator programs in cancer, pain, immunology & inflammatory (I&I) diseases and others enables the company to continue shifting new promising clinical programs to global development

Gyre THERAPEUTICS

Targeted Protein Degraders (TPDs)

A Modular Degrader Platform: From Discovery to Clinic

Ubiquitin Proteasome System (UPS) – Our Engine for Targeted Protein Degradation



Gyre's Proprietary Targeted Protein Degraders' Catalytic Mechanism of Action Enables:

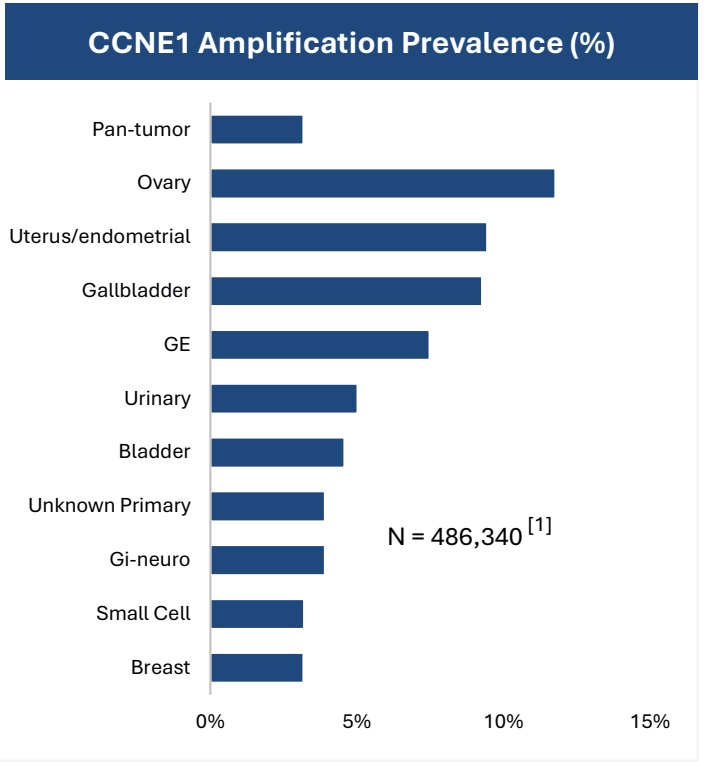
- ✓ High Potency
- ✓ Access to Undruggable Targets
- ✓ Modular Design to Create Multiple Degraders

Our expertise also translates well into implementing TPD technology into Antibody-drug conjugates (ADCs) to make next-gen ADCs, called Degrader Antibody Conjugates (DACs)

One Platform. Multiple Degraders. Built for Broad Impact Across Oncology and Immunology.

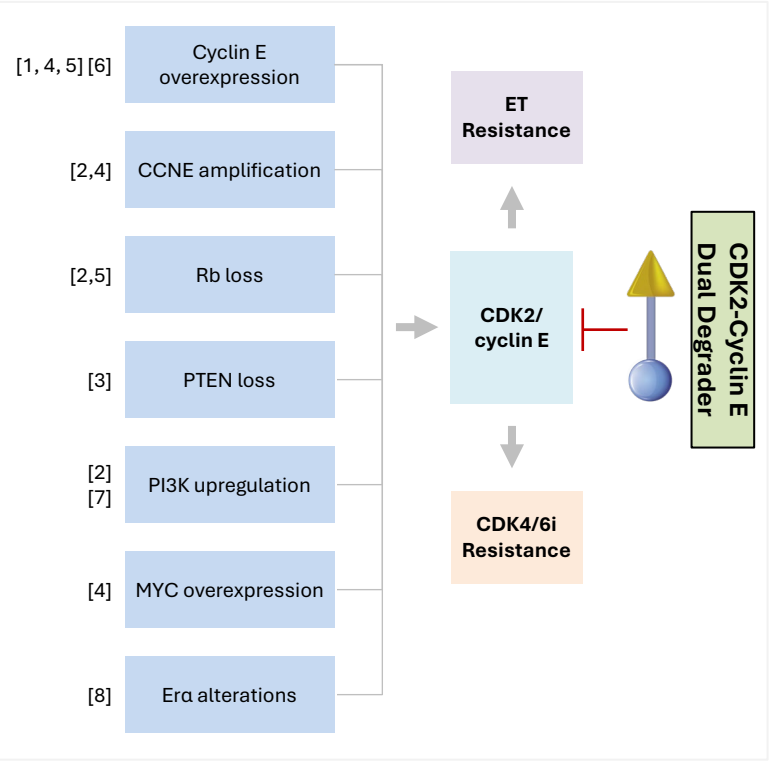
CDK2-Cyclin E Dual Degrader: Novel Strategy for Multiple Solid Cancer Types

Cyclin E Is Functionally Amplified or Overexpressed Across Multiple Cancer Types



:[1] Lee et al (2026) Clin Cancer Res. PMID:41870274

Diverse CDK4/6i or Endocrine Therapy Resistance Converge on Activation of CDK2/Cyclin E



[1] Turner NC (2019) J Clin Oncol. PMID: 30807234; [2] Herrera-Abreu MT (2016) Cancer Res. PMID: 27020857; [3] Costa C (2020) Cancer Discov. PMID: 31594766
[4] Freeman-Cook (2021) Cancer Cell. PMID: 34520734; [5] Wander SA (2020) Cancer Discov. PMID: 32404308; [6] Al-Qasem AJ(2022) NPJ Precis Oncol. 2022 PMID: 36153348
[7] Josefine B (2012) Breast Cancer Res Treat. PMID: 23242584; [8] Caldon CE (2012) Mol. Cancer Ther. PMID: 22564725

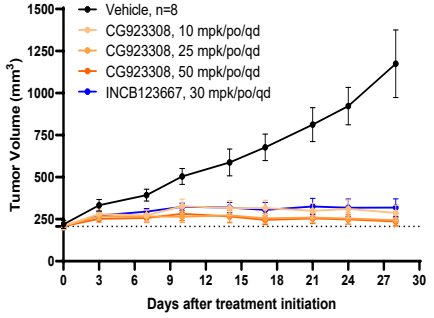
Market Opportunity & Unmet Medical Need

- Verzenio, Kisqali, and Ibrance
- ~$14.6B Total Global Revenues in 2025
- Projected total global revenues between $25.8B - $35.7B by early 2030s
- ~50% of patients develop resistance to CDK4/6 inhibitors within 2 years of starting therapy

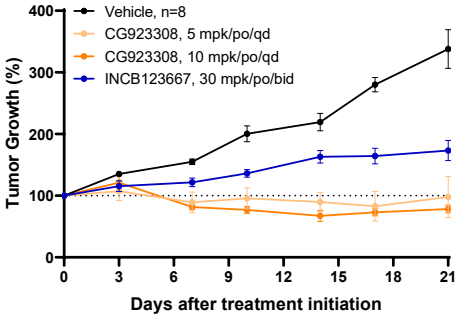
Strategic Market Research, July 2026

CG923308 Demonstrates Anti-Cancer Efficacy in Resistant Preclinical Models

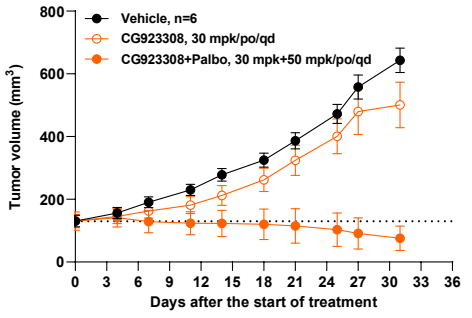
OVCAR3 Ovarian CDX



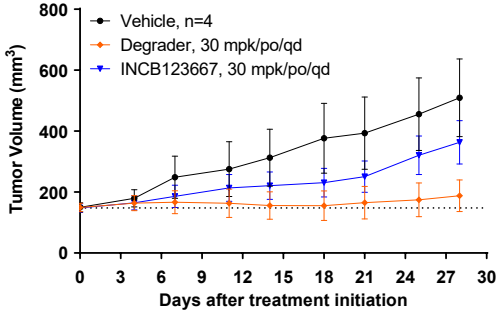
MKN1 Gastric CDX



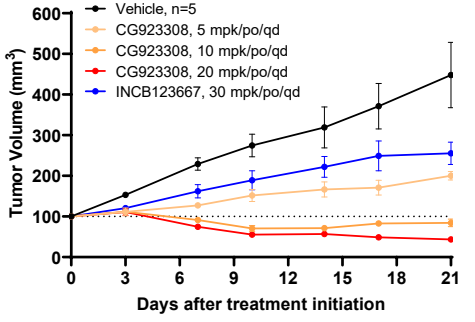
CDK4/6i-resistant MCF7 CDX



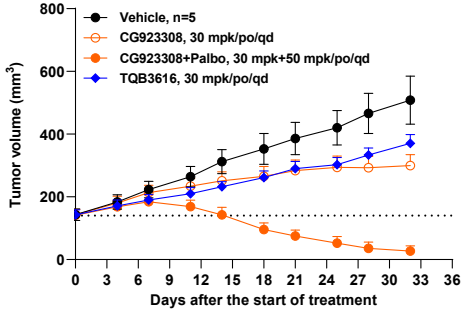
Chemo-resistant TNBC PDX



HCC1599 TNBC CDX



CDK4/6i-resistant breast PDX

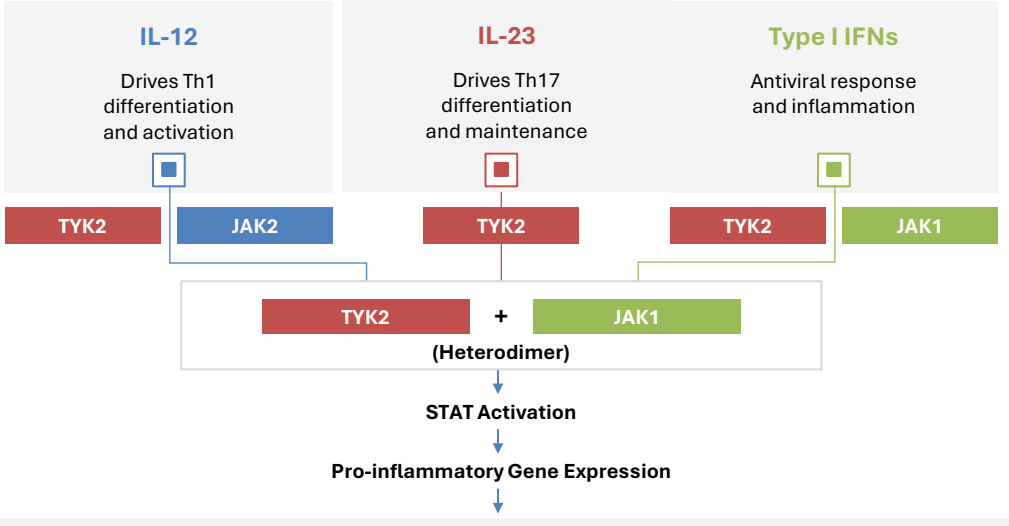


INCB123667: A phase 2/3 CDK2 inhibitor by Incyte; **Palbo**: Palbociclib (Ibrance), CDK4/6 inhibitor developed by Pfizer; **TQB3616**: CDK2/4/6 inhibitor developed by Chia Tai Tianqing

Minimum Effective Dose is 5~30 mg/kg QD (Human Equivalent Dose: 25~160 mg/day) Estimated therapeutic Window: >20-fold (based on dosage); >10-fold (based on exposure)

TYK2-JAK1 Dual Degrader: Next-Generation Therapy for Autoimmune Diseases

Cytokine signaling mediated by TYK2 and JAK1



Chronic Inflammation and Autoimmune Diseases

Advantages of Dual Targeting TYK2 and JAK1 with a Degrader

- **Broader Cytokine Coverage**
- **Higher and Broader Efficacy Potential**
- **JAK2/3 Sparing and Improved Safety Profile**
- **Best-in-Class Opportunity**

Significant Unmet Need and Market Opportunity Across Autoimmune Diseases

125MM+ Patients Worldwide[1] Psoriasis	**18MM** Patients Worldwide[2] Rheumatoid Arthritis	**204K+** Patients U.S.[3] Systemic Lupus Erythematosus	Other Inflammatory and Autoimmune Indications

Notes:
1. https://www.psoriasis.org/psoriasis-statistics/;
2. https://www.who.int/news-room/fact-sheets/detail/rheumatoid-arthritis;
3. https://www.niams.nih.gov/health-topics/lupus/basics/symptoms-causes;

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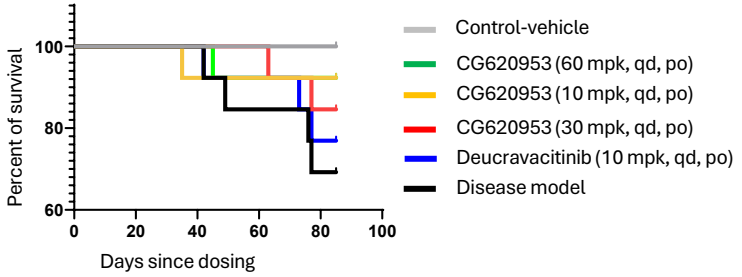
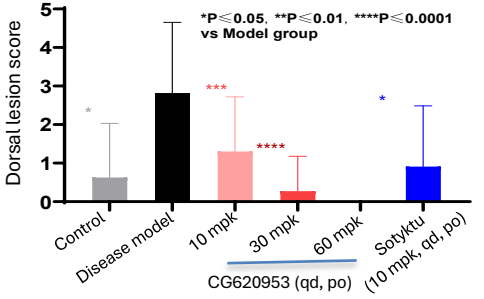
CG620953 Demonstrates Efficacy in Lupus and RA Preclinical Models

Lupus

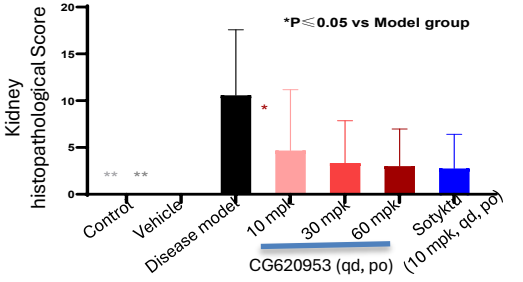
Survival rate



Dorsal skin lesion score

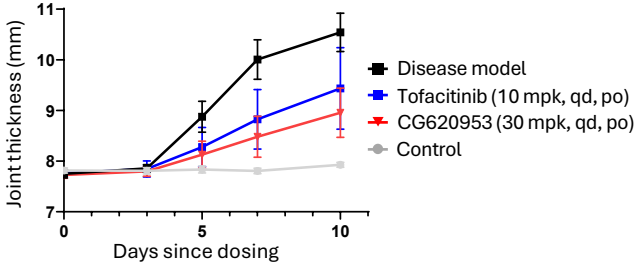


Kidney injury histopathology

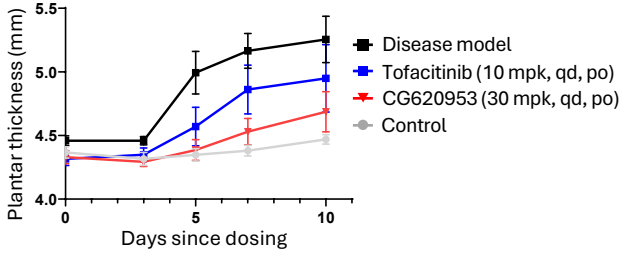


RA

Joint inflammation & cartilage destruction



Hind paw edema



CG620953 exhibit consistent or improved efficacy outcomes to standard-of-care in preclinical models

Pan-TRK Degrader: Best-in-Class, Non-Opioid Franchise in Cancer-Induced Bone Pain

Cancer-Induced Bone Pain U.S. Market Opportunity

280K – 330K

New cases of malignant bone metastasis in the U.S. annually [3,4]

Significant Unmet Need

70-90% develop symptomatic pain with limited current treatment options [5]

193-225 Days

Average treatment duration for cancer-induced bone pain [6,7]

Our Differentiated Approach

CG001419

First-in-class Oral PAN-TRK Degrader

Differentiated Mechanism

Degrades TrkA at the source

Early Clinical Validation

Phase 1a target engagement in high single-digit / low double-digit nanomolar ranges.
Well tolerated up to highest dose tested [(8)]

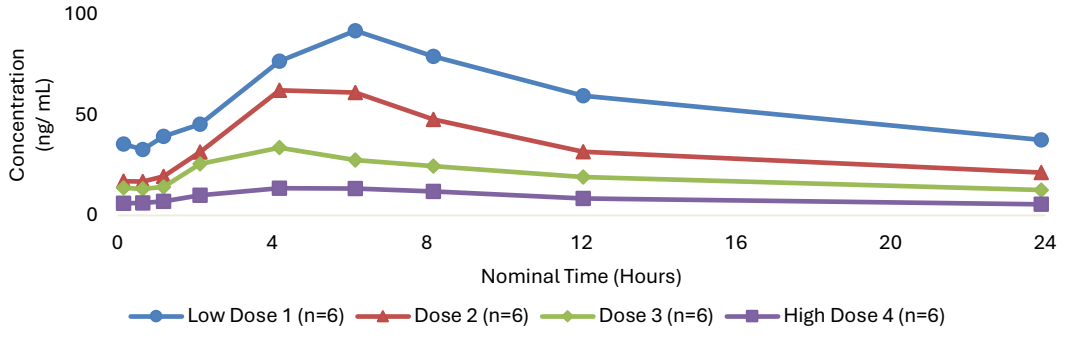
Speed Advantage

U.S.–China clinical development enables parallel execution and faster timelines

1. https://pubmed.ncbi.nlm.nih.gov/30627511
2. https://pubmed.ncbi.nlm.nih.gov/37343145
3. https://pubmed.ncbi.nlm.nih.gov/22570568
4. https://pubmed.ncbi.nlm.nih.gov/26229504
4. https://pubmed.ncbi.nlm.nih.gov/23344095
6. https://pubmed.ncbi.nlm.nih.gov/25919474
7. https://pubmed.ncbi.nlm.nih.gov/37343145
8. Estimates based on treatment pricing of $10 / day

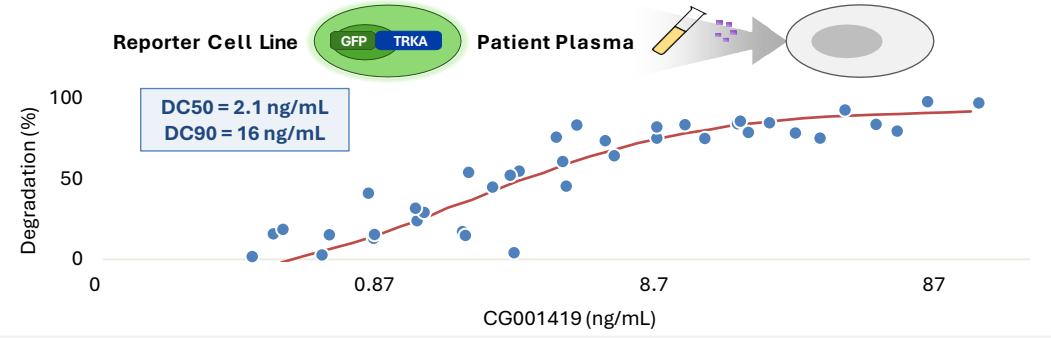
CG001419 Phase 1 Results: PD Target Engagement and Safety

Multiple Ascending Dose (MAD) PK – Day 7



Pharmacodynamics: Potential Target Engagement

Surrogate PD Assay Demonstrated Degradation Potency in the Low-nM Range.



Safety / Tolerability from Phase 1

Pharmacodynamics: potential target engagement demonstrated

- Plasma-to-reporter cell assay showed **TRKA degradation**, supporting potential target engagement
- Potent surrogate PD activity observed: **DC50 = 2.1 ng/mL; DC90 = 16 ng/mL**

Favorable safety / tolerability profile

- Single and multiple oral doses were well tolerated up to the highest tested dose levels
- Most TEAEs were **mild or moderate; no Grade 4 TEAEs** reported
- Common TEAEs were largely general / administration-site events, likely related to blood collection procedures

Predictable exposure profile supports continued development

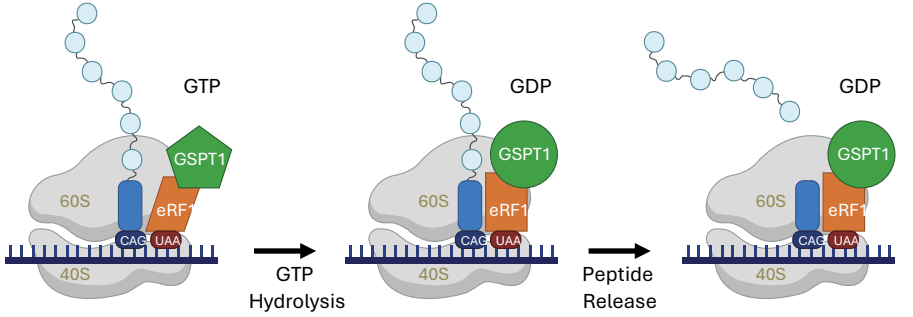
- Single-dose CG001419 exposure increased **dose-proportionally**
- Fed condition increased systemic exposure; 7-day MAD exposure increased **less than dose-proportionally** for CG001419 and metabolites M2/M8

Completed Phase 1 studies in healthy volunteers; currently planning Phase 2 for cancer-induced bone pain to commence in U.S. or China

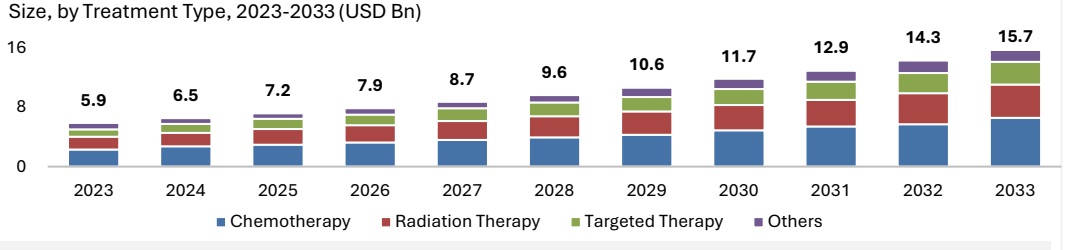
Source: Company data; CG001419-101 (NCT06636500).

CG009301: Targeting GSPT1 for AML and Solid Tumor Cancers

- GSPT1 controls protein translation termination and plays important function for leukemia stem cells and tumor cells with MYC overproduction
- GSPT1 lacks an active site and is often considered “undruggable”
- Other programs targeting GSPT1 to develop a therapeutic showed insufficient therapeutic index. CG009301 demonstrated encouraging safety profile in our preclinical studies and early clinical development.



Global Blood Cancer Treatment Market



The Market will Grow At the CAGR of:	**10.3%**	The Forecasted Market size for 2033 in US$:	**15.7Bn**

market.us

U.S. Patient Population

AML [1]	MDS [1]	ALL [1]	MYC-amplified Solid Tumors [2],[3]
~20,800 new cases	**~10,000** new cases	**~6,500** new cases	**28%**
11,220 mortality	**30-40%** MDS progress to AML [4]	**1,330** mortality	

Notes:
1. 2024 by American Cancer Society estimates
2. The Cancer Genome Atlas (TCGA) estimates
3. Schaub et al (2018) *Cell Syst* PMID: 295967830
4. Volpe et al. (2022) *Clin Lymphoa Myelom Leuk*, PMID: 34544674

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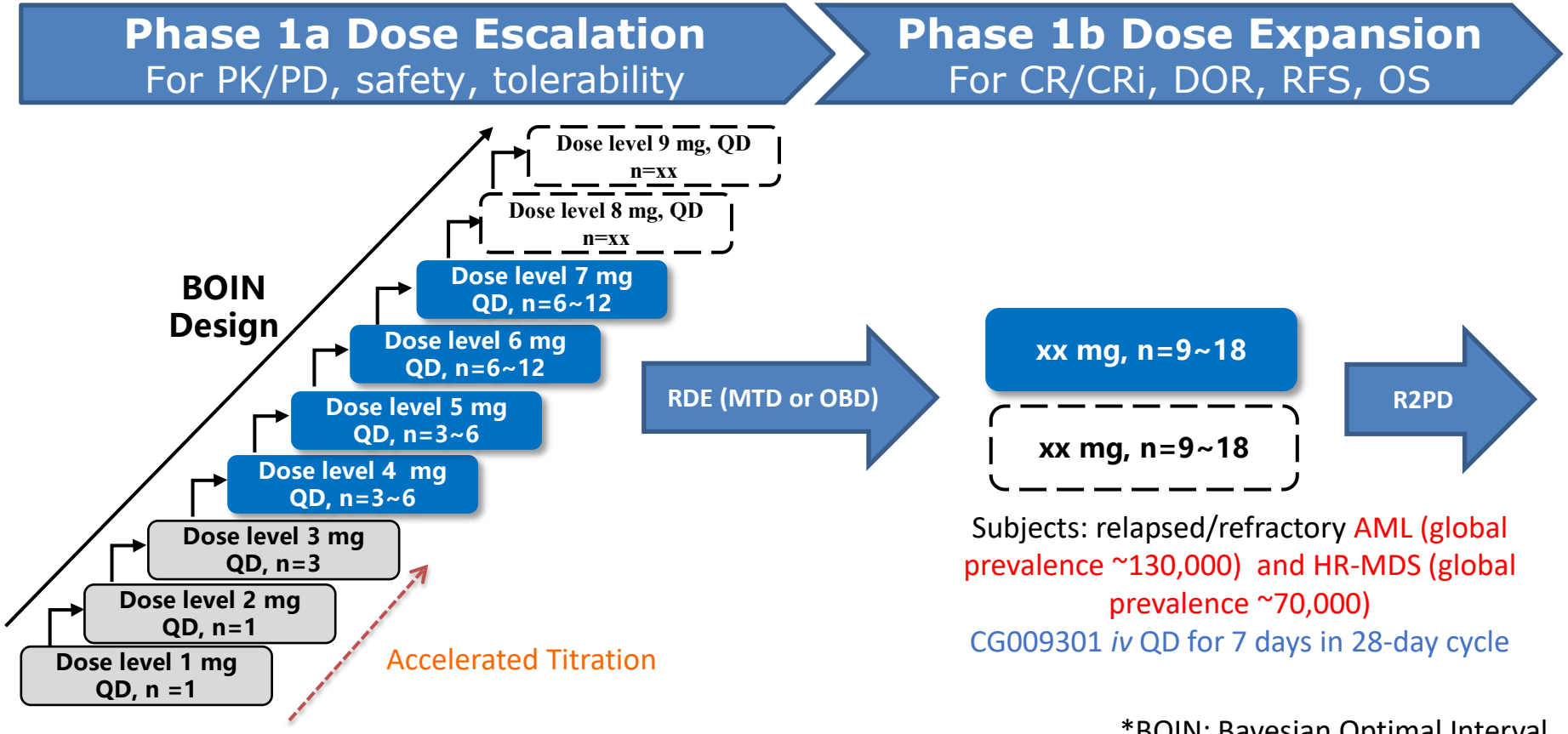
CG009301: Ongoing Phase 1 in China for patients with high-risk heme malignancies



Subjects: all relapsed/refractory hematological malignancies
CG9301 *iv* QD 7 days in 28-day cycle

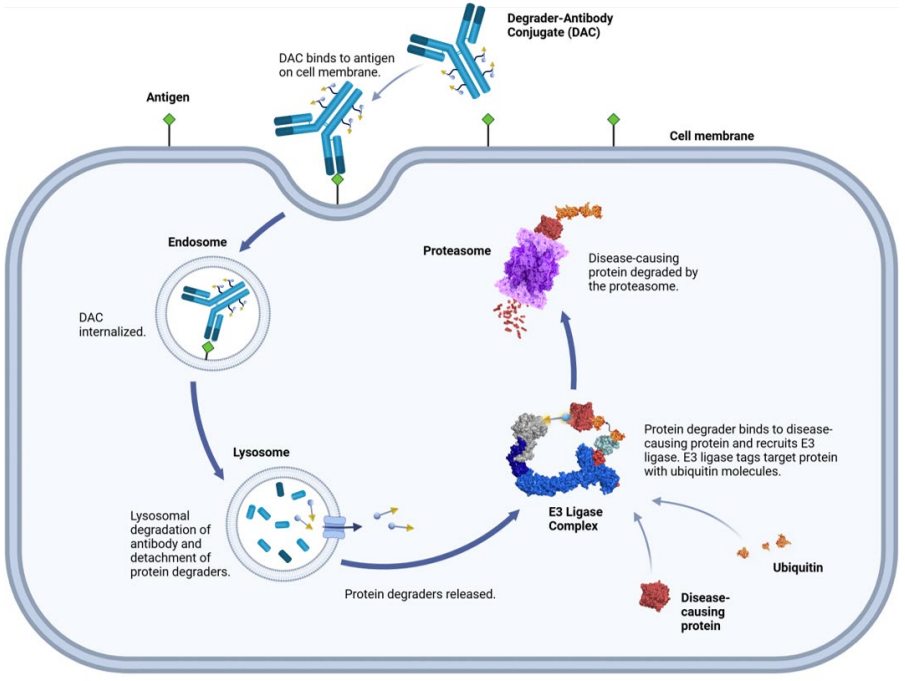
Subjects: relapsed/refractory AML (global prevalence ~130,000) and HR-MDS (global prevalence ~70,000)
CG009301 *iv* QD for 7 days in 28-day cycle

*BOIN: Bayesian Optimal Interval
Target DLT rate: 0.25
Acceptable DLT range [0.197, 0.298]

Degrader-Antibody Conjugates (DACs)

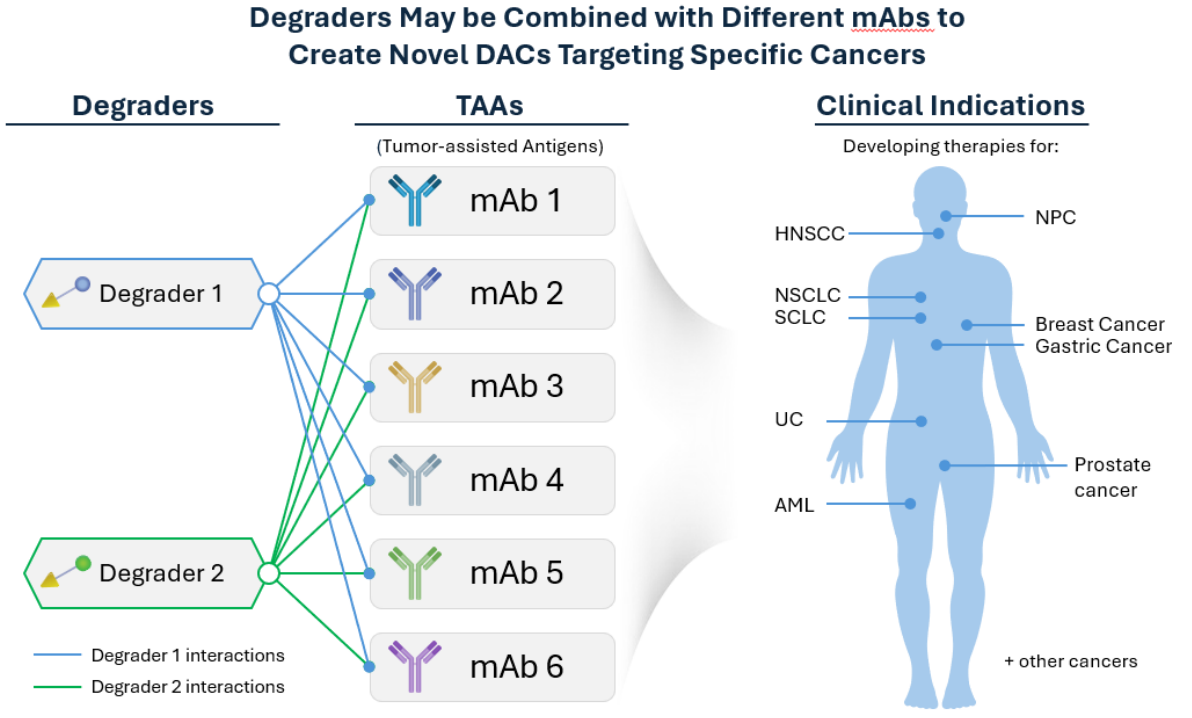
Leveraging Expertise in TPDs and China ADC Platform to Develop Degrader-Antibody Conjugates (DACs)

Gyre's DAC Mechanism of Action & Benefits



HIGH POTENCY | **IMPROVED PK** | **IMPROVED SAFETY**

Gyre's DAC Platform for Multiple Cancer Types



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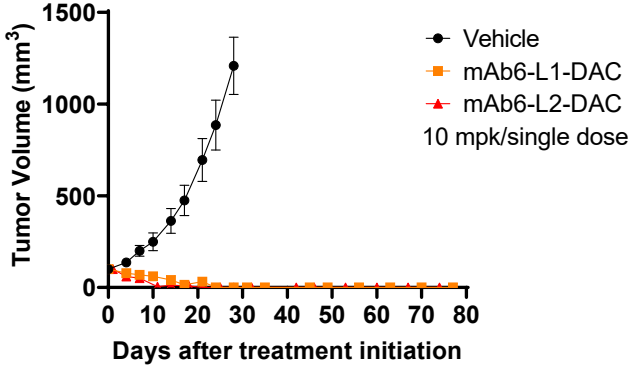
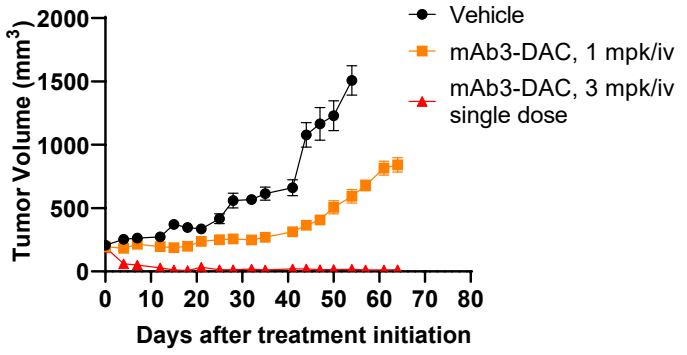
Gyre's DACs Demonstrate Broad Efficacy Across Diverse Tumor Types

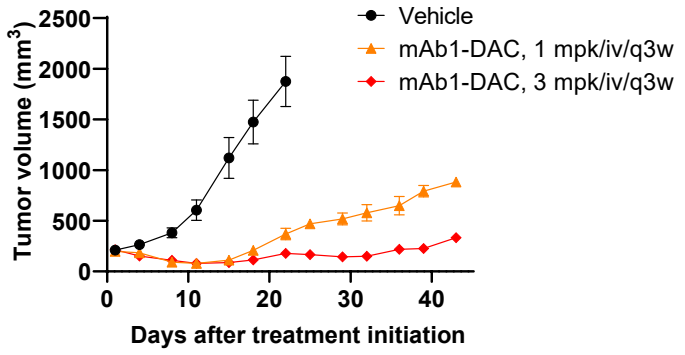
MV4;11 AML CDX model



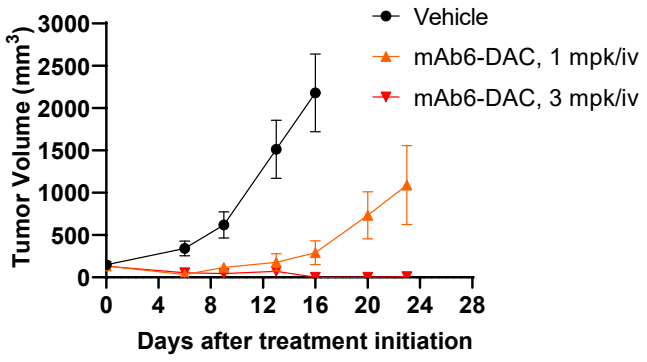
MDA-MB-453 Breast Cancer Model



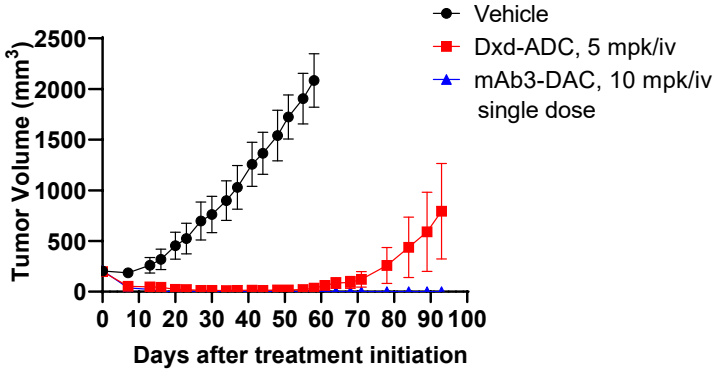
VCaP Prostate Cancer CDX model



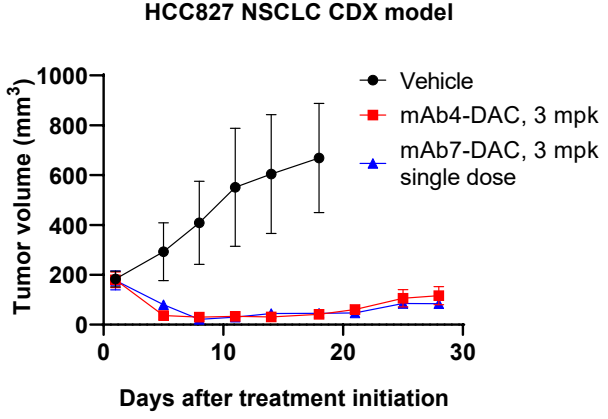
NOMO-1 AML CDX model



BT474 Breast CancerCDX model



HCC827 NSCLC CDX model

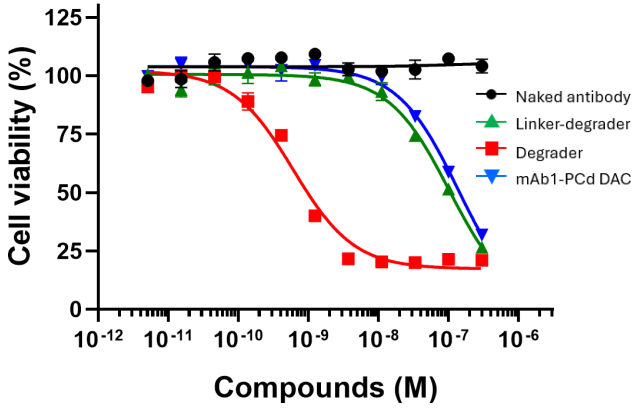


mAb: monoclonal antibody recognizing a specific antigen; **DXd-ADC**: HER2-DXd ADC (Enhertu) Developed by Daiichi Sankyo and AstraZenec

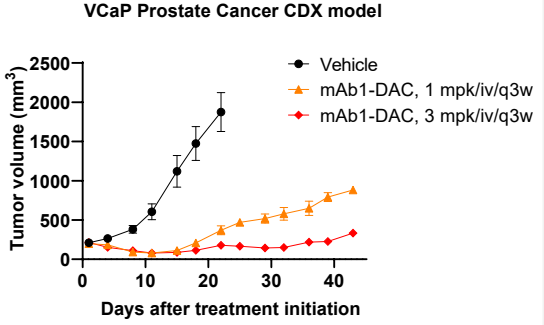
DAC for Prostate Cancer and Solid Tumors With An Epigenetic-Targeting Payload

- Prostate cancer causes 35,000 – 36,500 death each year and is the second-leading cause of cancer death in the US. The Androgen Receptor (AR) acts as a critical oncogenic engine, driving prostate tumor growth, survival, and therapy resistance even after castration.
- Epigenetic factors are frequently mutated across human malignancies, including prostate cancer.
- Prior developments targeting epigenetic factors for prostate cancer has been limited by the toxicity.

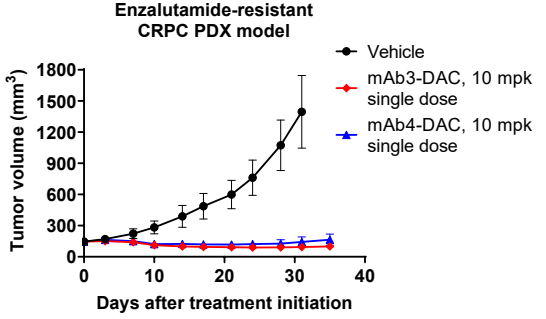
Protein degradation & cytotoxicity



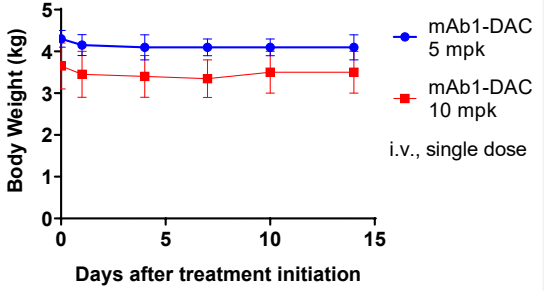
Prostate cancer CDX model



Therapeutic-resistant PDX model



Non-human primate pilot toxicity



Commercial Foundation and Growth Opportunities with F351 (Hydronidone)

ETUARY™: The Commercial Anchor of Our Fibrosis Franchise Since 2011

A Durable Revenue Base that Funds Innovation and Supports Our Next-generation Degrader / DAC Platform

Established Leadership

ETUARY™ has anchored our IPF franchise in China through consistent execution and broad physician reach, supported by ~370 commercial personnel across 3,000+ hospitals and pharmacies.

Durable Market Position

ETUARY™ was approved in 2011 prior to the Reference Listed Drug (RLD) requirement. Without RLD, generic competitors cannot conduct the required bioequivalence studies – creating a market exclusivity beyond patent protection.

Funds the Innovation Pipeline

ETUARY™'s recurring commercial revenue supports our next-generation degrader / DAC platform without dilution dependence.

1 ETUARY™ (Pirfenidone) (1) → **2 Etorel ™ – Launched in 2025** → **3 F351 (Hydronidone)**

China's Leading Pirfenidone Brand
For the Treatment of IPF (1)

Complementary PF option that extends the fibrosis toolkit alongside ETUARY™.

NDA accepted for priority review by NMPA in May 2026.

A One-stop Shop for Fibrosis – Building Physician Reach Ahead of F351

Notes:
1. ETUARY market position based on IQVIA CHPA data for pirfenidone in China [2014-2026]: legal review required. Financial data inclusive of pro forma data prior to GNI Group and Catalyst Biosciences business combination. 2017 sales in audited China GAAP; 2025 in audited U.S. GAAP

F351 Phase 3 Results in CHB-associated Liver Fibrosis Supports Regulatory Filing

F351 poised to be the next-generation of fibrosis therapy

Breakthrough Therapy Designation Priority Review of NDA
(China NMPA, 2021, 2026)

NDA accepted by NMPA in May 2026

Primary Endpoint Met with High Statistical Significance

≥1-stage fibrosis regression at Week 52:

- F351: 52.85% (n=123) vs.
- Placebo: 29.84% (n=124)
- **Delta: 23.01%**
- **p = 0.0002** (ITT [1] analysis with central blinded pathology review)
- Consistent with fibrosis regression rates observed in Phase 2

Key Secondary Endpoint Reduction in Liver Inflammation

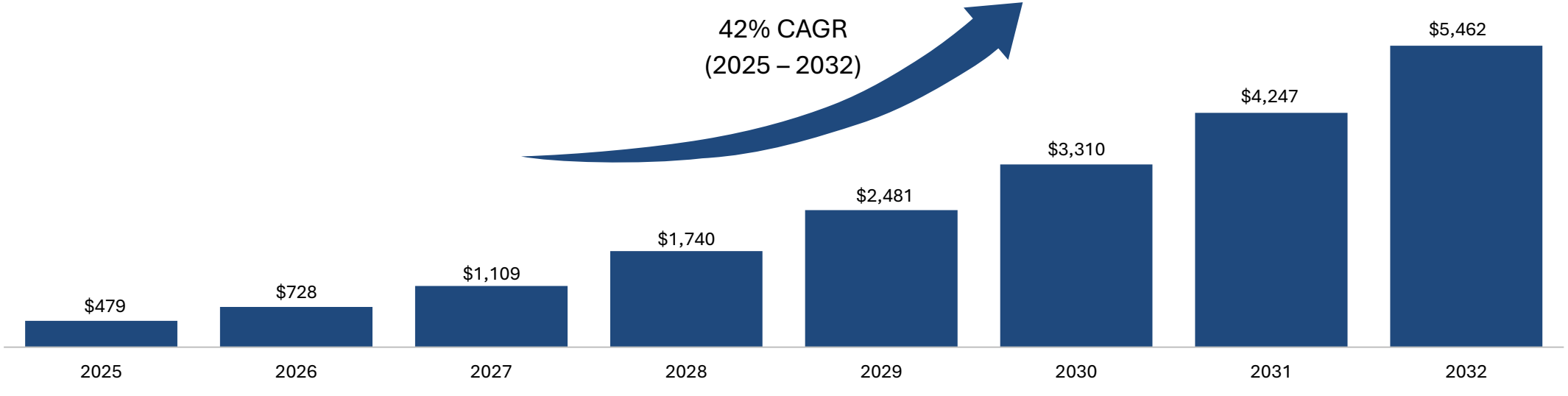
≥1-grade inflammation improvement without fibrosis progression at Week 52:

- F351: 49.57% (n=123) vs.
- Placebo: 34.82% (n=124)
- **Delta: 14.75%**
- **p = 0.0246**
- Reinforces anti-inflammatory activity

MASH Fibrosis Market Provides Significant Upside Opportunity For F351

China-First Strategy Provides Accelerated POC

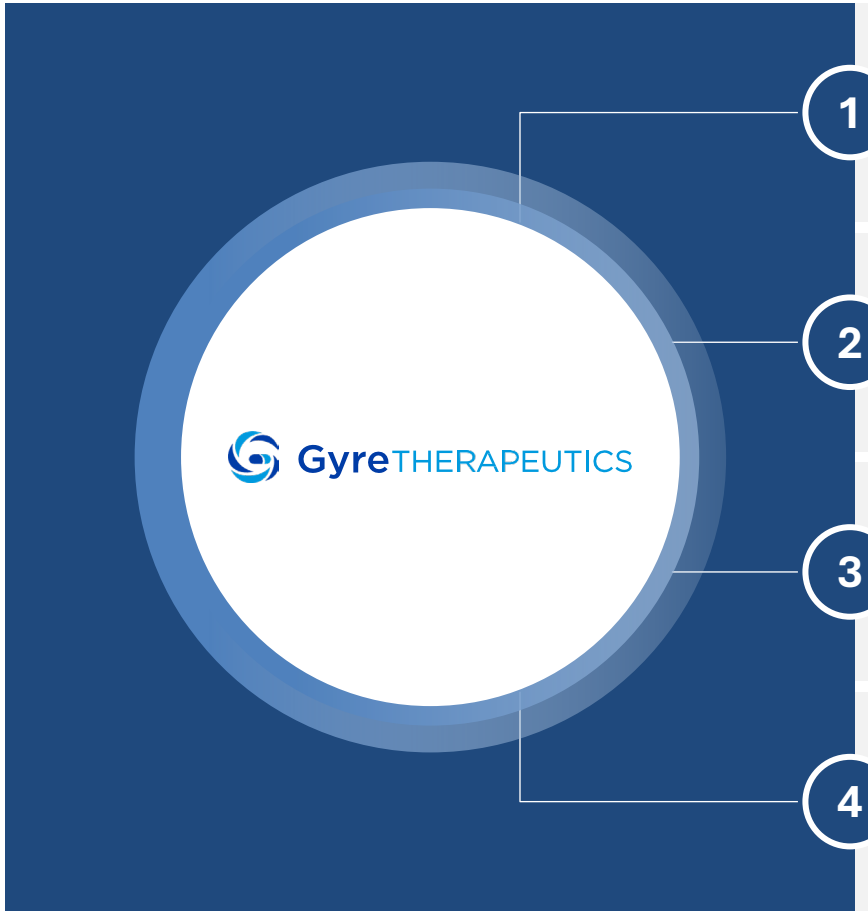
Forecasted Market Size of MASH Fibrosis Therapies in the USA ($MM USD)



- Current U.S. MASH prevalence is estimated at ~14MM [1]
- MASH represents tremendous growth opportunity due to very low current MASH diagnosis rate (5-10%)
- Rising obesity and diabetes increase MASH progression via liver inflammation

USA MASH Therapeutics Sales forecast from Evaluate Pharma as of 5-13-2026
Assumes 50% of all MASH patients progress to MASH fibrosis (Stage ≥F2) based on Luthra & Sheth (2025).
Note:
1. Le et al. (2025) JAMA; AJMC; MASH Awareness; Estes et al. (2018) AASLD; L.E.K. interviews

Key Value Drivers



Lead TPD Assets

- Opportunities in cancer and autoimmune diseases with dual degraders
- TPD portfolio highly validated for large unmet need indications

TPD / DAC R&D Engine

- Multiple shots on goal
- DACs paint a promising picture as the next-gen to ADCs / TPDs alone

Global Exposure

- Lean operations to de-risk assets
- U.S. HQ and integrated U.S. and China R&D

Revenues to Support Operations

- ETUARY™ and anticipated F351 sales in China expected to cover operation expenses to support early-stage R&D

Thank You!

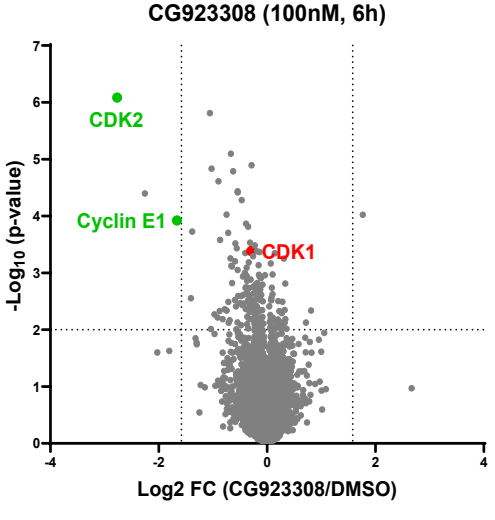
Gyre THERAPEUTICS

Appendix

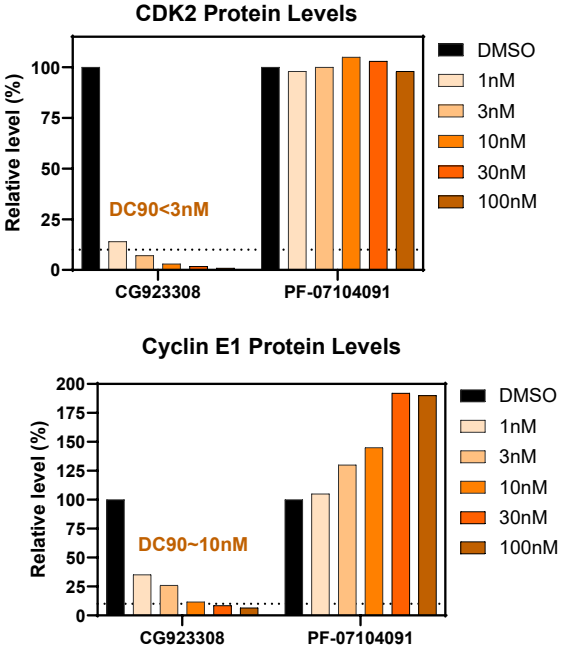
Gyre THERAPEUTICS

CG923308: CDK2-Cyclin E Dual Degrader On-Target Activity Promotes Growth Inhibition

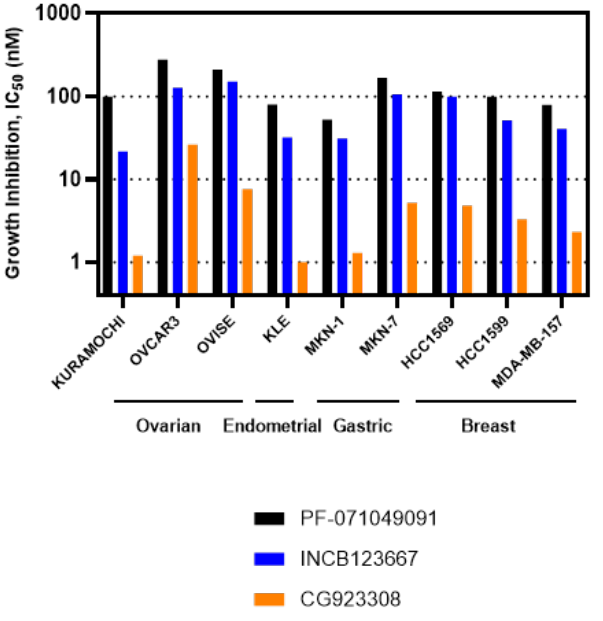
Potent and Selective Degradation Profile



Overcome Feedback Induction of Cyclin E1



Induced Potent Growth Inhibition

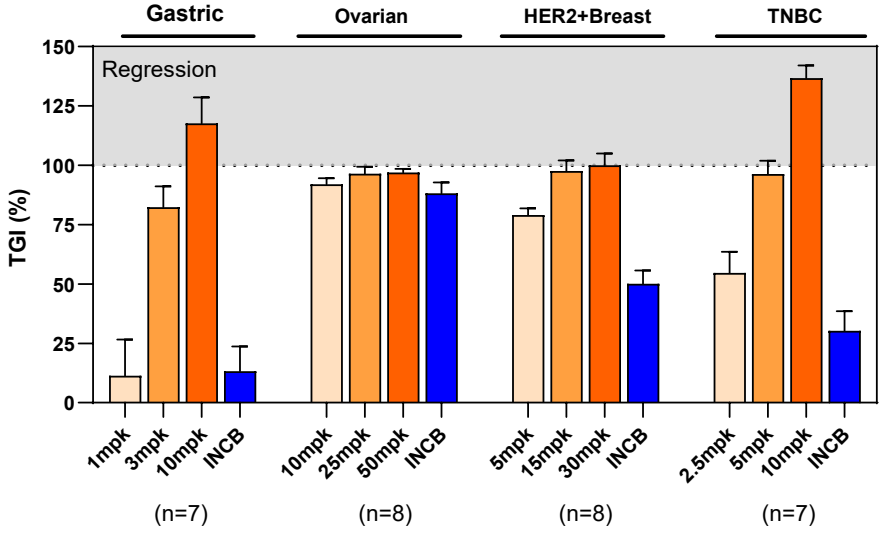


A next-generation CDK2 targeted strategy designed to overcome CDK4/6i or ET resistance in breast cancer

Gyre THERAPEUTICS

CG923308 Demonstrates Anti-Cancer Efficacy in Resistant Preclinical Models

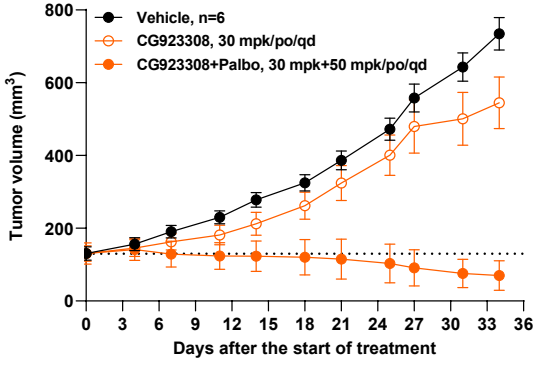
A CCNE1-Amplified Solid Tumors



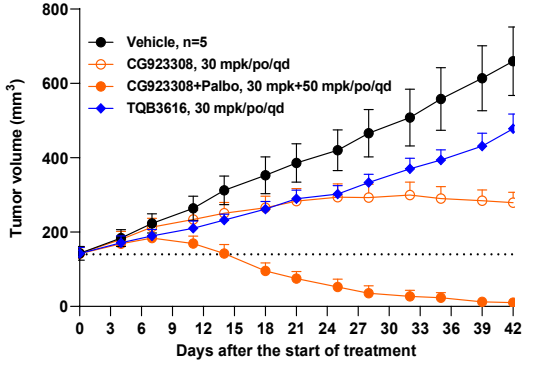
INCB: *INCB123667 at 30 mg/kg, a CDK2 inhibitor at Phase III*
CG923308 ： 5-50 mg/kg

B CDK4/6i-Resistant HR+HER2- Breast Cancers

HR+HER2- breast CDX



HR+HER2- breast PDX



4/5 CR

TQB3616: *a CDK2/4/6 inhibitor approved by NMPA*

All doses were well tolerated in animals, with no significant body weight loss observed during the studies

TGI (%) = [1-(Ti-T0)/ (Vi-V0)] ×100% (If Ti>T0); TGI (%) = [1-(Ti-T0)/ (T0)] ×100% (If Ti<T0)

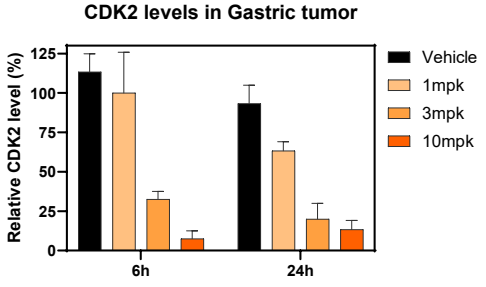
Minimum Effective Dose is 5~30 mg/kg QD (Human Equivalent Dose: 25~160 mg/day)

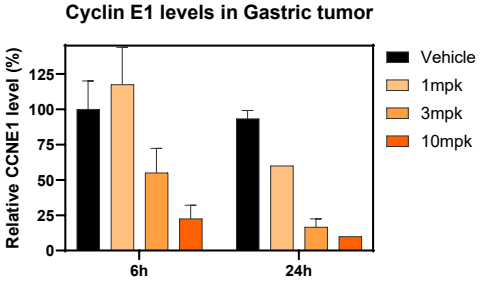
Gyre THERAPEUTICS

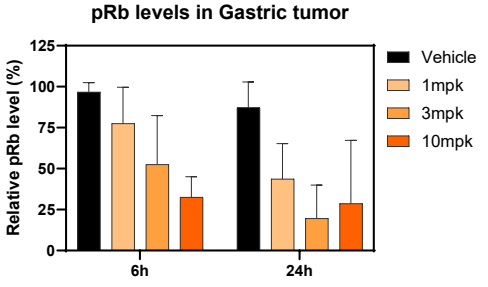
TGI (%) = [1-(Ti-T0)/ (Vi-V0)] ×100% (If Ti>T0); TGI (%) = [1-(Ti-T0)/ (T0)] ×100% (If Ti<T0)
Ti is the tumor volume of treatment group, T0 is the tumor volume of the treatment group on the first day of treatment; Vi is the tumor volume of vehicle control group, V0 is the tumor volume of the vehicle group on the first day of treatment.

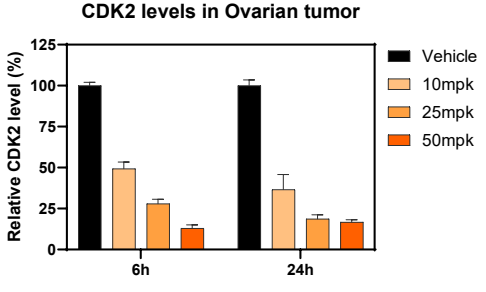
CDK2-Cyclin E Protein Degradation in Preclinical Model Tumors

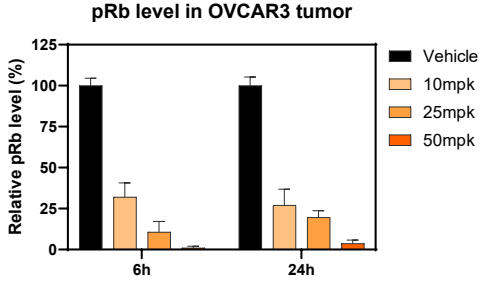
Orally administrated CG923308 induces CDK2-cyclin E degradation in tumors











Gyre THERAPEUTICS

CG620953 Demonstrates Favorable Safety and PK

FAVORABLE SAFETY PROFILE

Preliminary safety data from 4 species (rodent and non-rodent) reveal a wide safety margin.

Favorable Safety Signals
- No genotoxicity, no hERG inhibition, and no cardiotoxicity in in vitro and in vivo studies

Low CNS Liability
- Minimal brain penetration

No Off-target Liabilities Identified
- Clean KinomeScan and proteomics profile with No neosubstrate degradation

FAVORABLE PK PROPERTIES

Oral Administration

Species	Dose (mg/kg)	AUC(last) (hr*ng/mL)	F (%)
Mouse	30	27,232	~100
Rat	10	2,754	31.9
Dog	10	2,536	37.2
Non-human primates	25	8,826	25.4

- Moderate-to-high oral bioavailability
- Low-to-moderate clearance rate
- No CYP inhibition

Gyre THERAPEUTICS

Non-Opioid Franchise in Cancer Pain

Large, Underserved Market

>$11Bn

Global Cancer Pain Market by 2028 (1)

70% of patients treated with opioids for cancer-induced bone pain report continued bone pain (2)

40–80% of cancer patients experience breakthrough cancer pain (3)

~3MM new cases of chemotherapy-induced neuropathic pain annually worldwide (4)

Validated Target In Cancer Pain

The NGF / TrkA Pathway is a Key Driver of Cancer Pain

1 Cancer cells produce NGF, activating TrkA on sensory nerves in bone

2 TrkA mutations cause congenital insensitivity to pain (CIPA) - genetic validation (5)

3 Blocking the NGF / TrkA pathway reduces cancer bone pain (6)

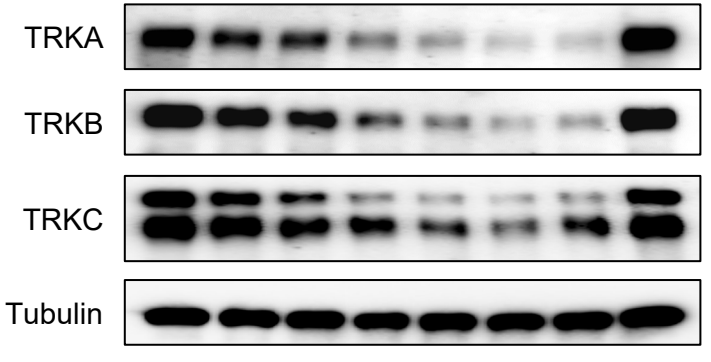
Tanezumab (anti-NGF antibody) Showed Pain Reduction in Phase III Cancer Bone Pain Trials (7)

Notes:
1. Custom Market Insights, 2024
2. Gyre/Cullgen 8-K, Jun 2, 2026
3. Nexalis Investor Announcement, Apr 19, 2026
4. Dogwood 10-K, Mar 18, 2026
5. Cocco, Scaltriti & Dri/on, Nat Rev Clin Oncol, 2018
6. Gyre/Cullgen 8-K, Jun 2, 2026
7. Pfizer tanezumab Phase III cancer bone pain trials
8. Gyre/Cullgen 8-K, Mar 2, 2026

CG001419 Degrades TRK Proteins in Neurons and Neuron-like Cells

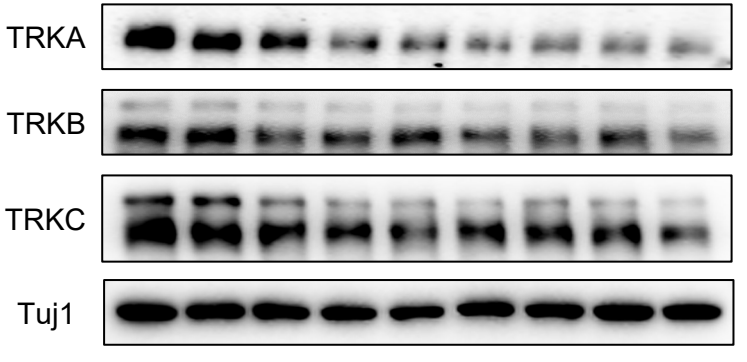
A. CG001419 degrades TRKs in mouse neurons

CG001419 (nM)	0	2.5	5	10	20	40	80	Ent.
Lane	1	2	3	4	5	6	7	8



B. CG001419 degrades TRKs in NT2-derived human neurons

CG001419 (nM)	0	0.6	1.25	2.5	5	10	20	40	80
Lane	1	2	3	4	5	6	7	8	9

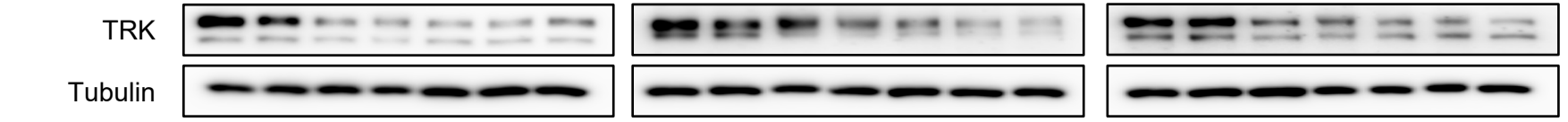


C. CG001419 degrades ectopically expressed dog TRKs in dog MDCK cells

Cell line	MDCK-TRKA						
CG001419 (nM)	0	2.1	6.2	18.5	55.6	166.7	500
Lane	1	2	3	4	5	6	7

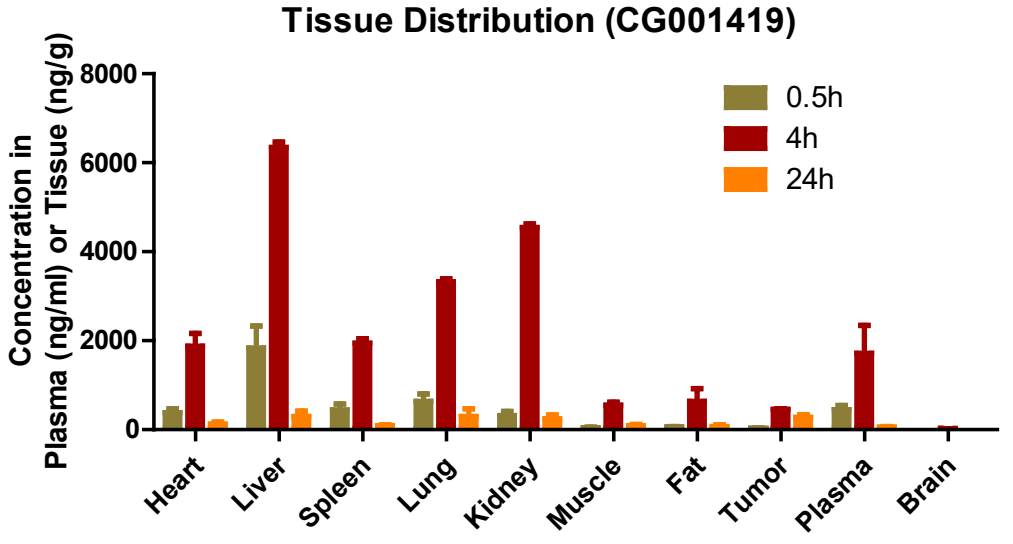
MDCK-TRKB						
0	2.1	6.2	18.5	55.6	166.7	500
1	2	3	4	5	6	7

MDCK-TRKC						
0	2.1	6.2	18.5	55.6	166.7	500
1	2	3	4	5	6	7



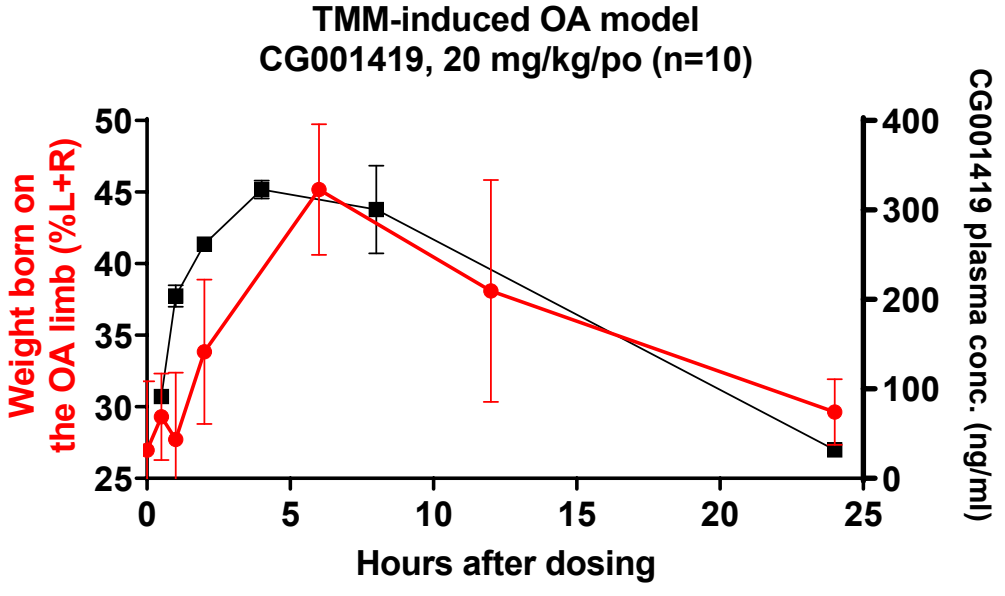
Gyre THERAPEUTICS

CG001419 Does Not Penetrate Into the Brain



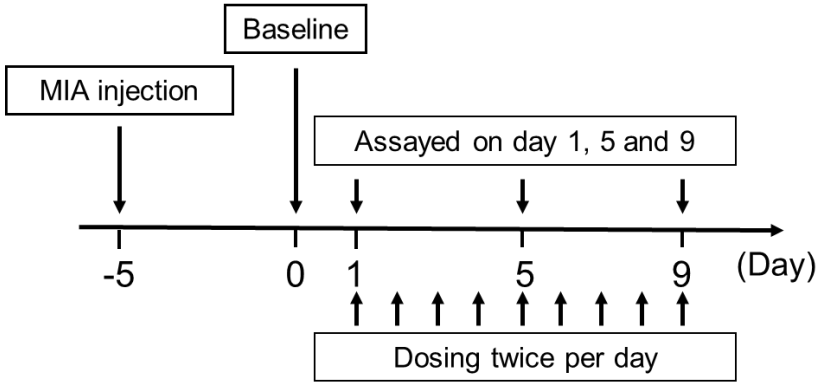
Conc. (ng/ml)	Heart	Liver	Spleen	Lung	Kidney	Muscle	Fat	Brain	Tumor	Plasma	Brain/ Plasma	Tumor/ Plasma
0.5 h	384	1835	443	637	318	40	64	5	32	447	0.011	0.072
4 h	1875	6340	1945	3320	4545	552	635	18	456	1715	0.010	0.266
24 h	124	299	90	295	246	92	73	4	277	58	0.066	4.775

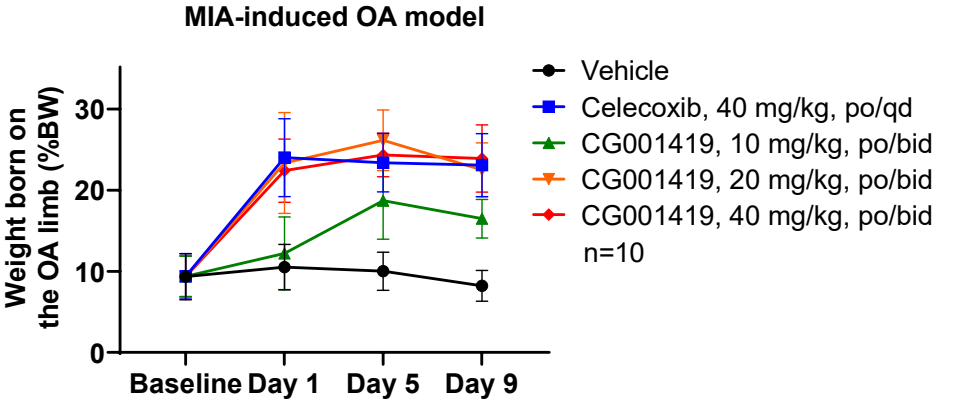
CG001419 Shows PK-driven Analgesic Activity



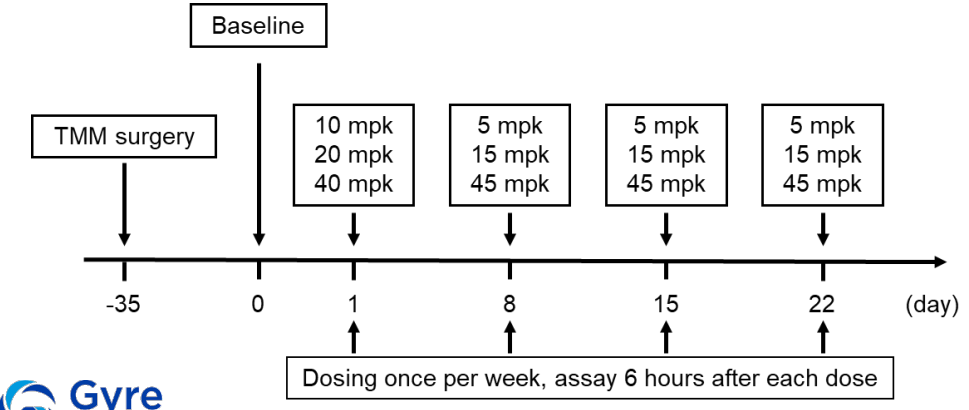
CG001419 Shows Significant Analgesic Activity in Two Rat Models of OA Pain

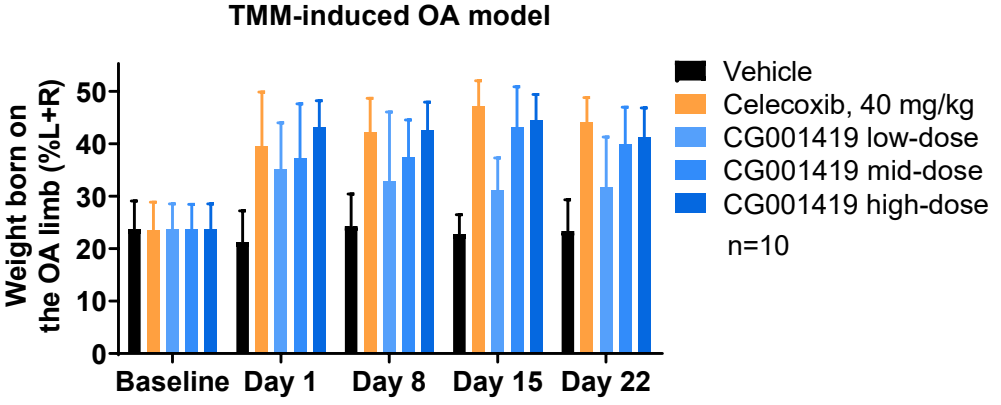
A. Monoiodoacetate (MIA)-induced OA model, continuous dosing





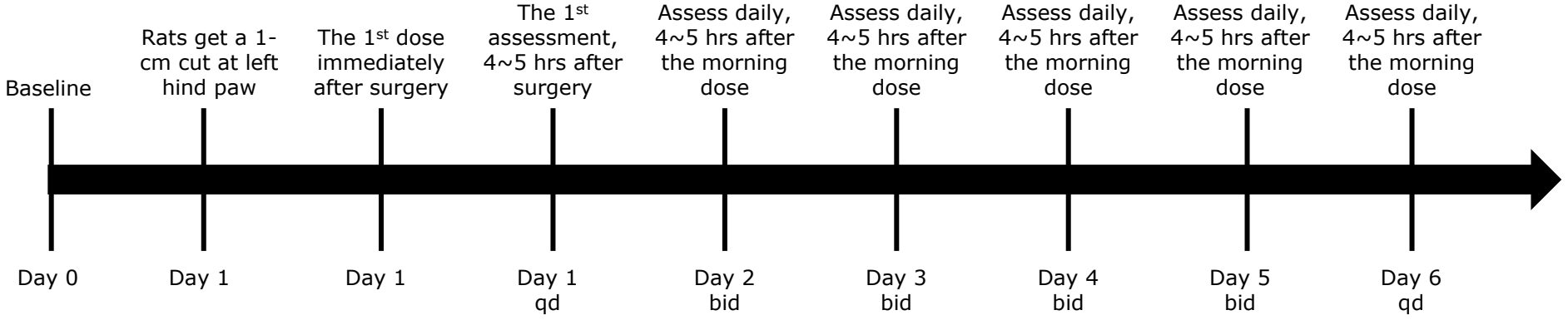
B. Total medial meniscectomy (TMM)-induced OA model, once per week dosing

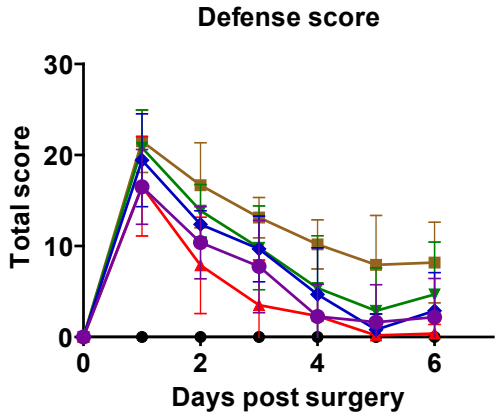


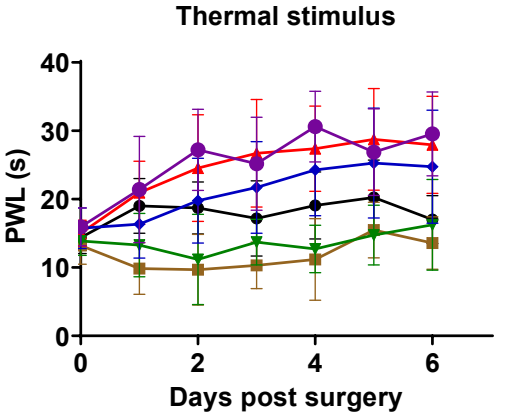


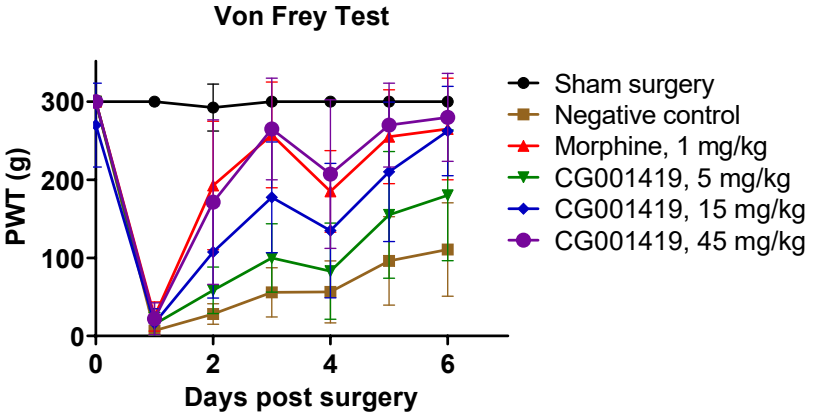
Gyre THERAPEUTICS

CG001419 Shows Significant and Dose-Dependent Analgesic Activity in a Surgical Pain Model





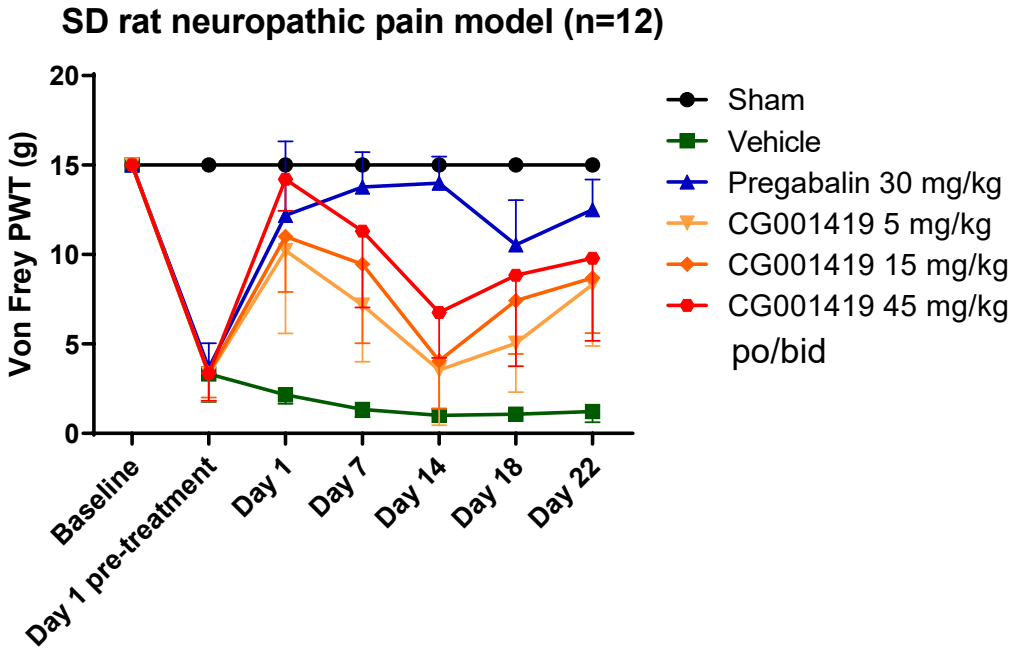




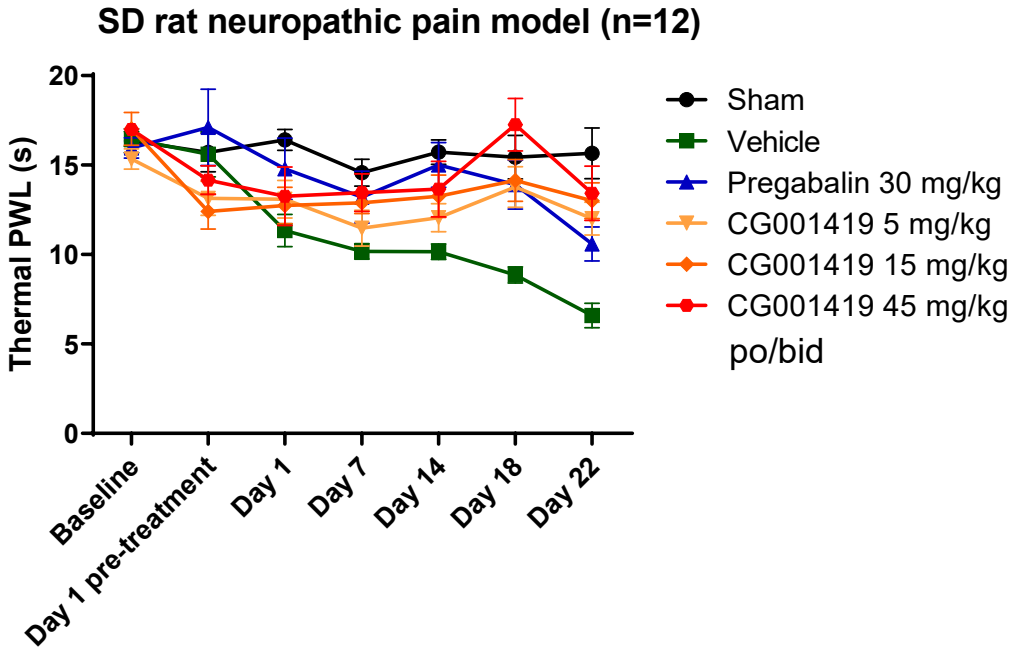
Gyre THERAPEUTICS

CG001419 Shows Significant Analgesic Activity in a Rat Neuropathic Pain Model (sciatic nerve incision)

A. Mechanical allodynia



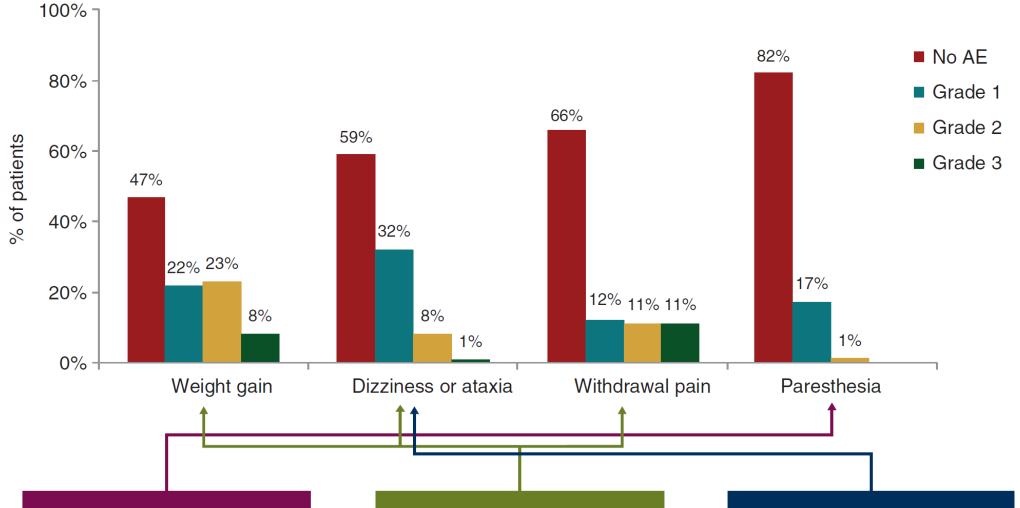
B. Heat stimulus



Gyre THERAPEUTICS

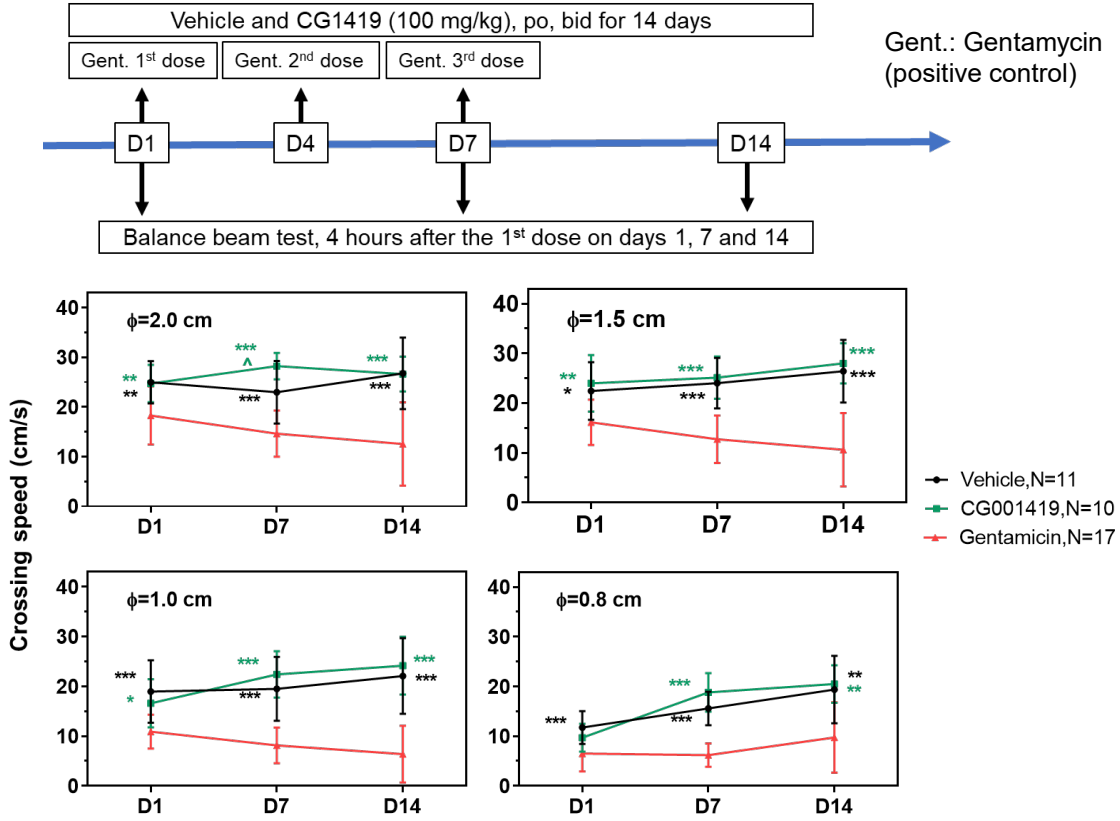
CG001419 Does Not Affect Mouse Mobility in a Balance Beam Test

A. TRK inhibition may induce ataxia via impacting on the peripheral nerve system in human



Liu et al., (2020) *Ann. Oncol.* PMID: 32422171

B. CG1419 does not impair mouse crossing through balance beams of 4 different diameters (0.8-2.0 cm)

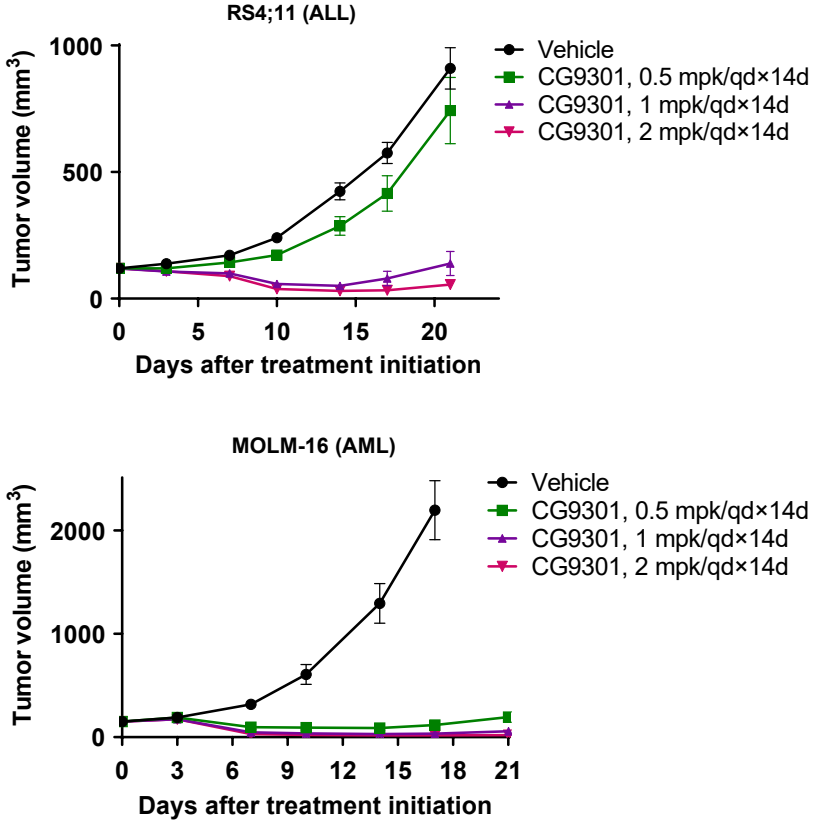


Data shown as mean±SD, One-way ANOVA with Turkey's test; * compared with gentamicin group, *, **, ***: P<0.05, <0.01, <0.001 repectively; ^:compare with vehicle group, P<0.05.

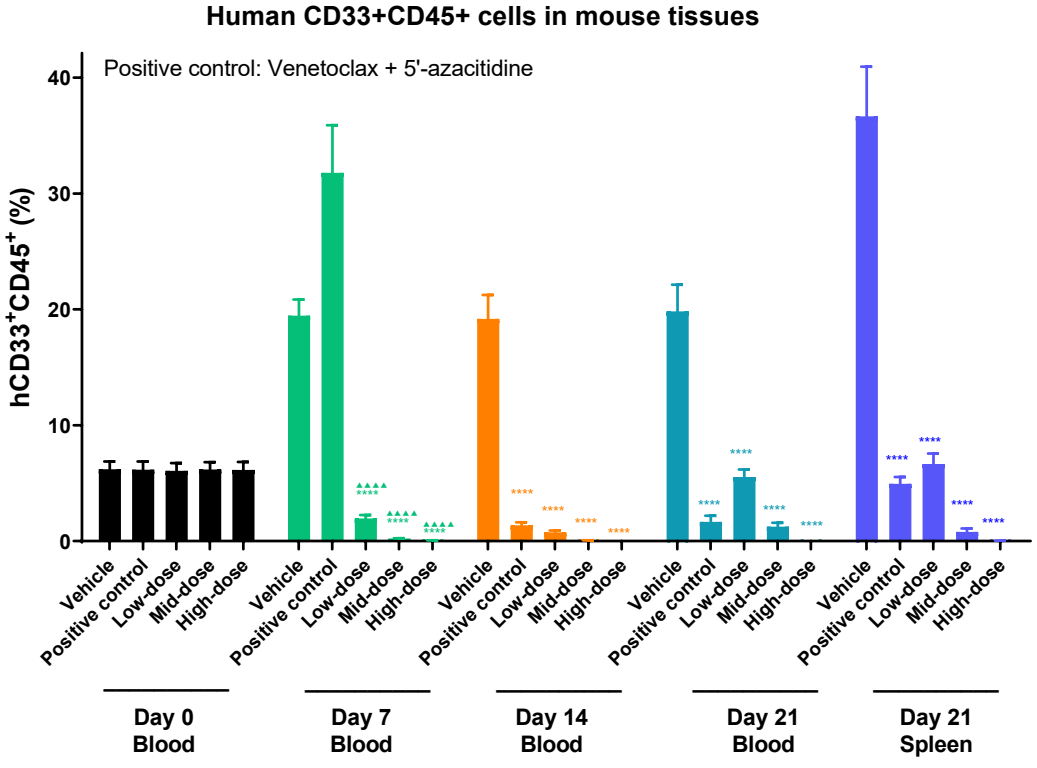
Gyre THERAPEUTICS

IV Administered CG9301 Shows Efficacy in Leukemia CDX and Orthotopic PDX Models

A. CG9301 induces significant regression in subcutaneous leukemia xenograft models

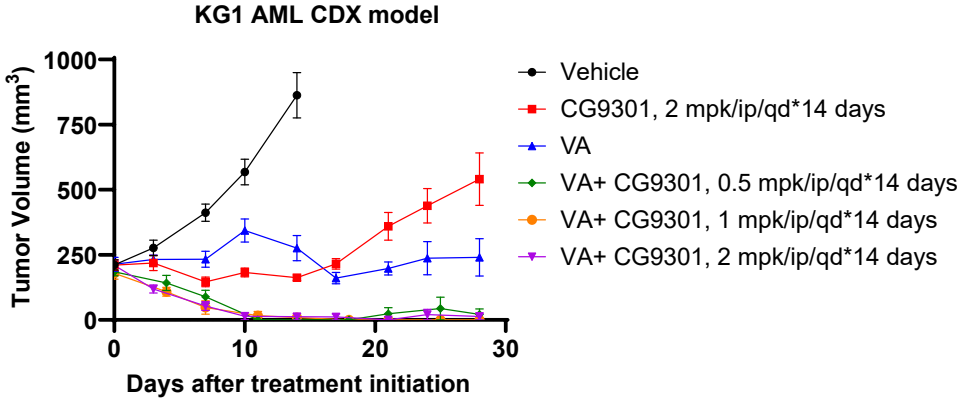


B. CG9301 induces significant reduction of tumor burden in an orthotopic AML PDX model

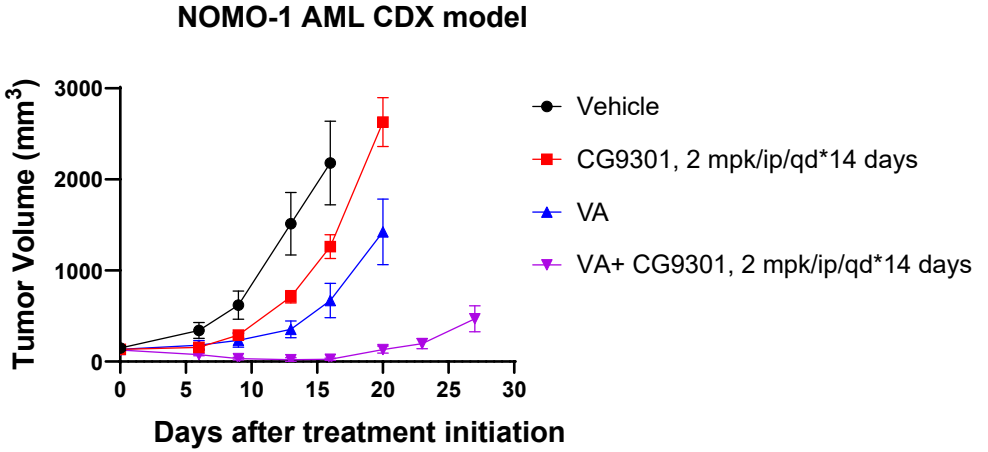


Gyre THERAPEUTICS

CG9301 shows synergistic interaction with SOC venetoclax and azacitidine in AML models



VA: Venetoclax 50 mpk/po/qd +Azacitidine 2.5mpk/ip/4 days on 3 days off for 2 cycles

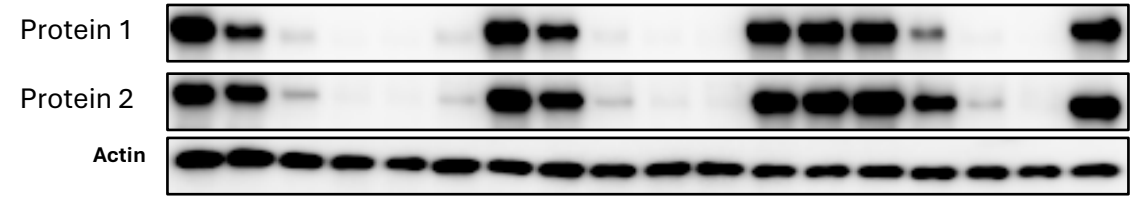


Gyre THERAPEUTICS

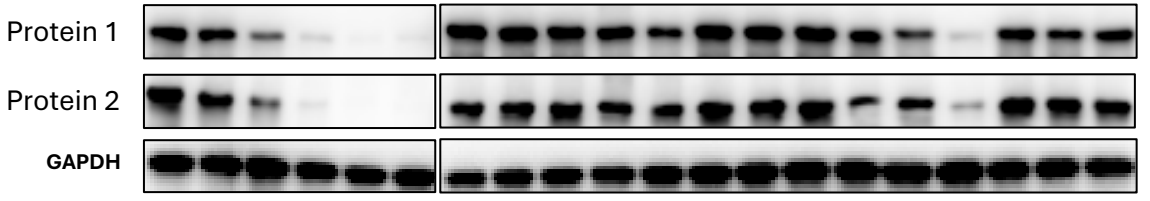
Epigenetic Factor DAC Demonstrates Potent and TAA-dependent Target Degradation and Cell Killing

A. Gyre's epigenetic factor DAC induces potent protein target degradation in a TAA-dependent manner *in vitro*

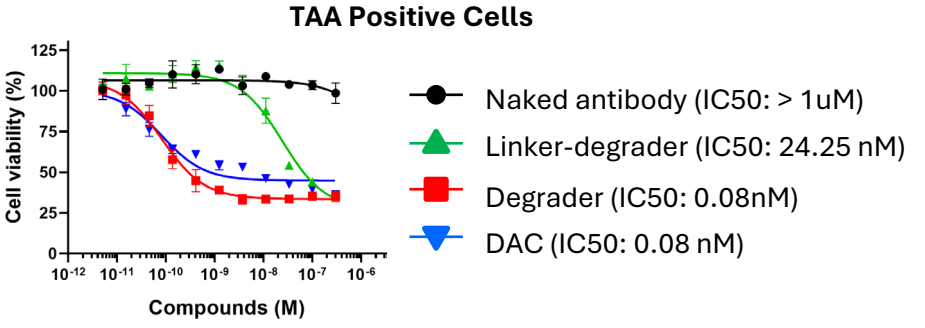
Cell Line	TAA Positive Cell Line																	
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

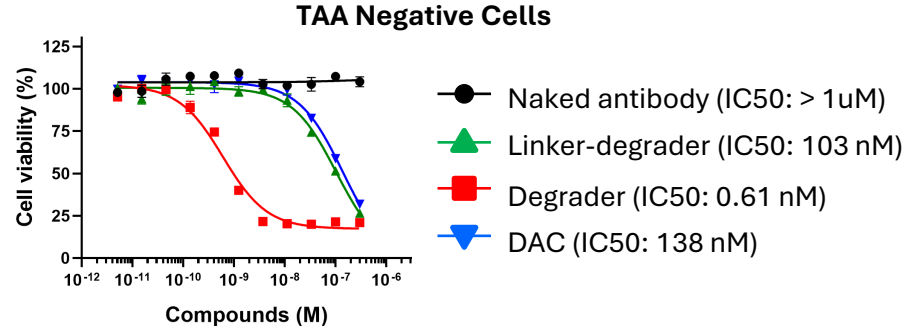


Cell Line	TAA Negative Cell Line																		
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					mAb		DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20



B. Gyre's epigenetic factor DAC kills cancer cells in a TAA-dependent manner

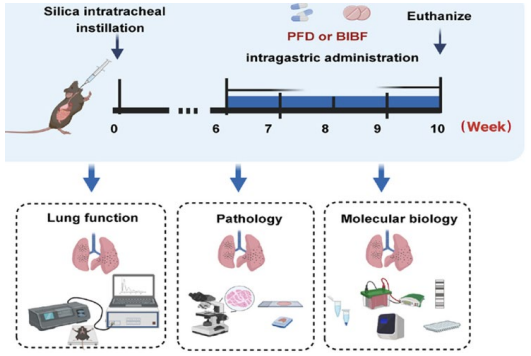




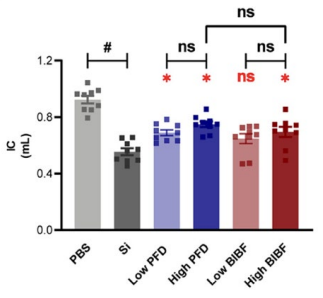
Gyre THERAPEUTICS

ETUARY™ Lifecycle Management: Potential to Drive Market Expansion Via Label Expansion

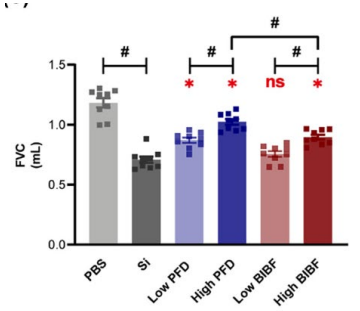
A. Preclinical studies demonstrate efficacy of ETUARY™ in treating pneumoconiosis[1]



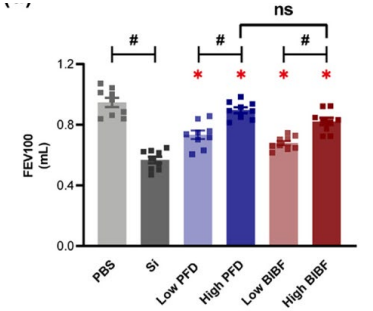
Operating Lung Volume



Gross Vital Capacity

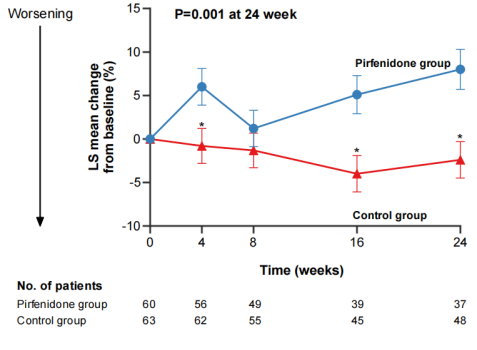


Airway Resistance / Flow

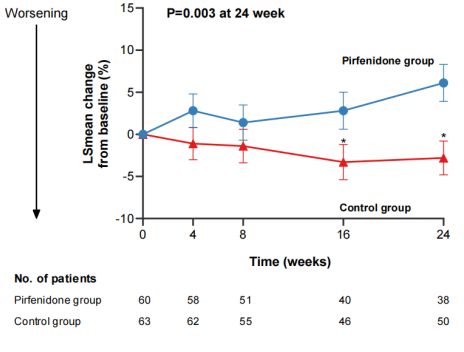


B. A phase 2 trial demonstrates efficacy of ETUARY™ in treating radiation-induced lung injury (RILI)[2]

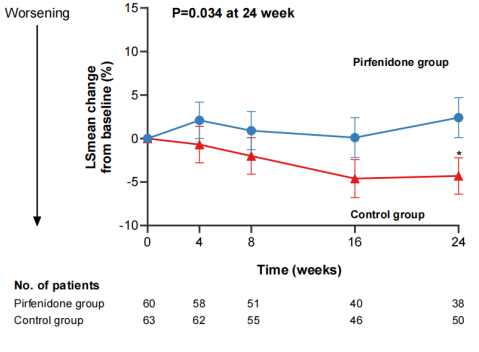
Gas Transfer Efficiency



Airway Resistance / Flow



Gross Vital Capacity



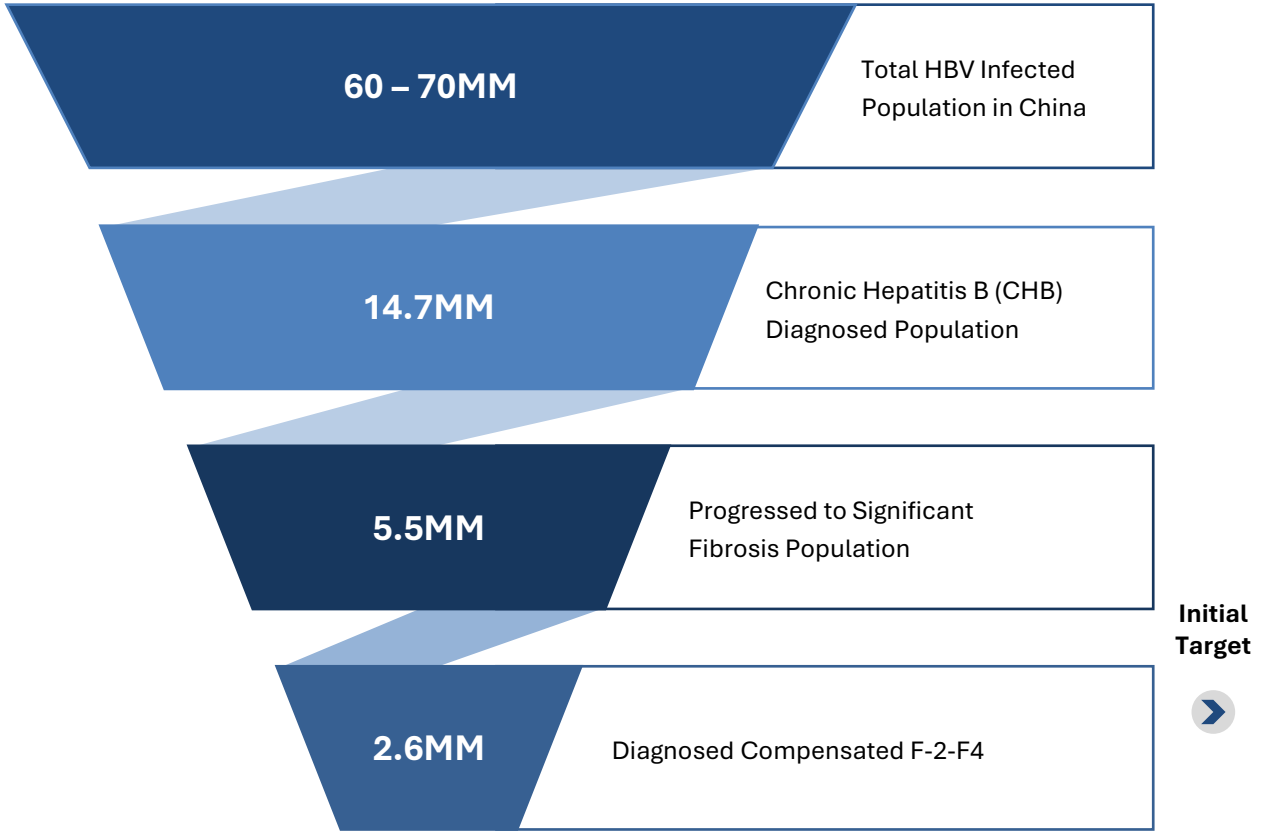
Gyre THERAPEUTICS

Ref 1. Bai et al. (2025) https://pubmed.ncbi.nlm.nih.gov/39546810/

Ref 2. Hou et al (2025) https://pubmed.ncbi.nlm.nih.gov/41207313/

BIBF: nintedanib; DLCO: Diffusing Capacity of the Lung for Carbon Monoxide ; IC: Inspiratory Capacity; FEV: Forced Expiratory Volume; FVC: Forced Vital Capacity; PFD: Pirfenidone; Si: Silicosis

F351 / Hydronidone Targets CHB Fibrosis – an Untapped Market in China



The Market for **CHB-associated liver fibrosis** is significantly **unmet**

Current standard of treatment, e.g. entecavir, tenofovir, focuses on only **reducing liver inflammation**

Patients with **F2 – F4** Fibrosis are at a **high risk of progression** to **cirrhosis** and **HCC**, major causes of liver-related mortality

Hydronidone, a structural analog of pirfenidone, reverses fibrosis by modulating **TGF- β / P38γ / Smad7** signaling pathway – a key driver of fibrosis progression. It received **Breakthrough Therapy designation** from PRC's NMPA in 2021, enabling expedited review

Notes: The Fourth National Serological Survey on HBV in China (2020) provided baseline HBV prevalence data. The 60-70MM total HBV cases and F2-F4 fibrosis estimates are derived using internal modeling based on this survey's fibrosis prevalence rates and awareness levels.

F351: Anti-fibrotic Activity Unlike Existing and Advanced Clinical MASH Therapies

Drug	MOA	Status	MASH Stage	Fibrosis Mechanism
Resmetirom (Rezdiffra®)	THR-β agonist	FDA/EMA Approved	F2-F3	Indirect – reduces lipotoxicity and improves mitochondrial function in hepatocytes
Semaglutide (Wegovy®)	GLP-1 receptor agonist	FDA Approved	F2-F3	Primarily indirectly through systemic weight loss
Tirzepatide (Zepbound®)	Dual GIP/GLP-1 receptor agonist	Phase 3	F2-F3	Primarily indirectly through systemic weight loss
Efruxifermin (AKR-001)	FGF21 analog (Fc-fusion protein)	Phase 3	F2-F3; F4 (cirrhosis arm)	Dual direct + indirect antifibrotic
Lanifibranor	Pan-PPAR (α/δ/γ) agonist	Phase 3	F2-F3	Partial direct + indirect antifibrotic
Survodutide (BI 456906)	Dual glucagon/GLP-1 agonist	Phase 3	F1-F3	Primarily indirectly through systemic weight loss
F351	TGF-β / p38γ / Smad7 modulator	NDA under review by NMPA (CHB-associated liver fibrosis); Phase 1 complete (MASH)	F2-F3 or F4	Direct antifibrotic targets the primary drivers of collagen deposition and fibrotic scarring in the liver.

F351 is the only therapeutic that has met the primary endpoint for reduction of fibrosis in a phase 3 clinical trial