A Fully-Integrated Biopharmaceutical Company Focused on Fibrosis, Inflammatory Diseases and Cancer

Gyre THERAPEUTICS

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. ("Gyre") and its subsidiaries, including Cullgen Inc. ("Cullgen"), 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 CG923308, F528, CG620953, F351, CG001419, CG009301, and ETUARY™, including 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. 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Gyre Therapeutics (Nasdaq: GYRE): At-A-Glance

Following Recent Acquisition of Cullgen

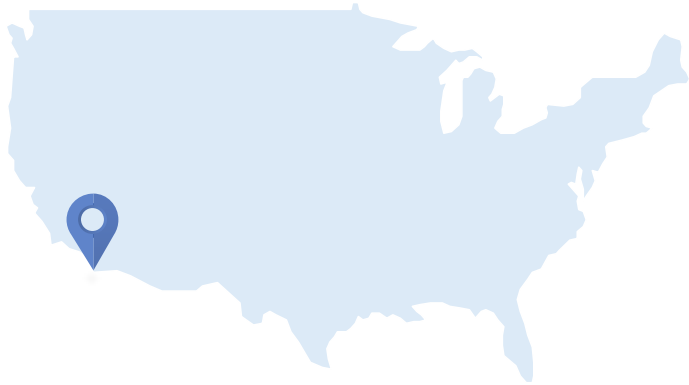
Pipeline ranges from discovery stage to marketed products with programs covering multiple therapeutic areas including fibrosis, inflammatory diseases and cancer

~740 Employees WW

~170	R&D
~85	Manufacturing
~370	Sales & Marketing
~115	G&A

Combined entity intends to **leverage established and cost-efficient China operations** for accelerated discovery, early validation, and development of next generation therapeutics based on degraders and DACs

San Diego, CA
Corporate HQ
- G&A, Clinical Development



Beijing, China
Manufacturing, Clinical Development and Commercialization

Shanghai, China
Drug Discovery, Clinical Development

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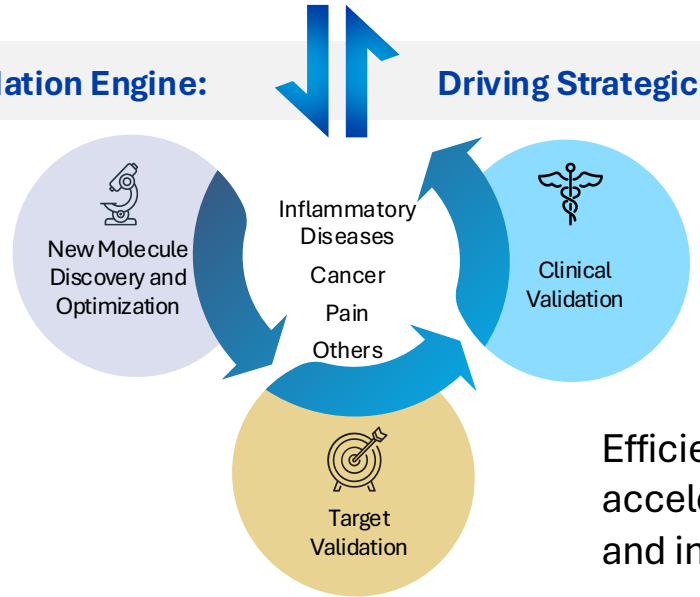
Fibrosis, Inflammatory Diseases, Cancer Portfolio Based on US-China Innovation

IND Enabling	Phase 1	Phase 2	Phase 3	NDA Filed	Marketed
CG620953 TYK2/JAK1 Degrader for Inflammatory Diseases	**F351 (hydronidone)** MASH-Associated Liver Fibrosis		**ETUARY™ (pirfenidone)** Pneumoconiosis Line Extension	**F351 (hydronidone)** CHB-associated Liver Fibrosis	**ETUARY™ (pirfenidone)** Idiopathic Pulmonary Fibrosis (IPF)
CG923308 CDK2/Cyclin E Degrader for Solid Cancers			**ETUARY™ (pirfenidone)** Radiation Induced Lung Injury (Phase 2/3) Line Extension		
F528 Chronic Obstructive Pulmonary Disease (COPD)					

Degrader

China Innovation and Validation Engine: **Driving Strategic Value and Efficiency**



Efficient and cost-effective operations accelerate new pipeline development and indication expansion

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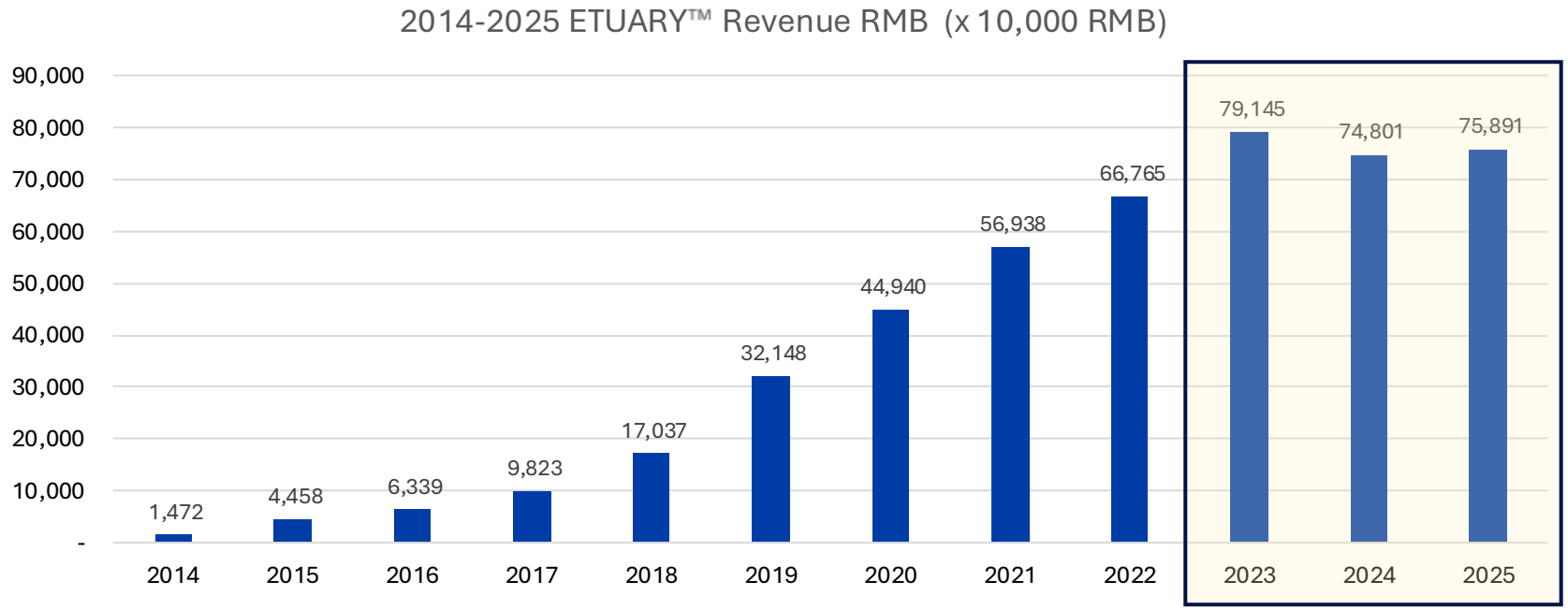
Gyre's ETUARY™ is the Market Leader of Pirfenidone for Treatment of Organ Fibrosis

First Product ETUARY™ Demonstrates Gyre's Capability from Innovation to Commercialization and Managing Life Cycle of Innovative Drugs



Upcoming line extensions for ETUARY™ are expected to catalyze sales growth

Third party pirfenidone sales outside of China fell substantially during same period and never recovered due to generic competition.

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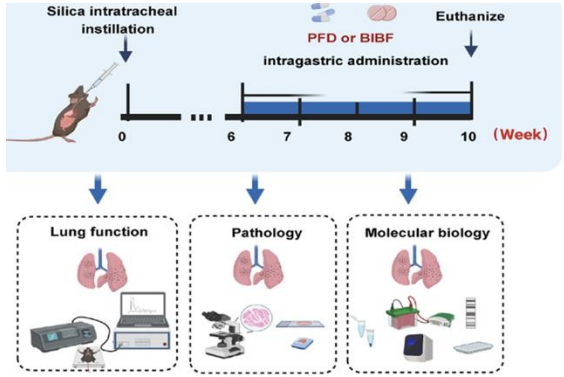
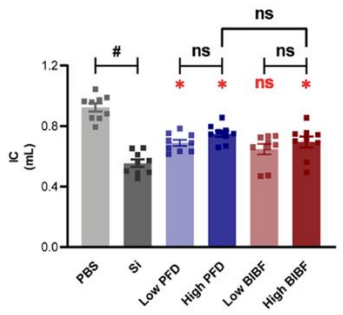
Promising Preclinical and Clinical Results for Two ETUARY™ Line Extensions

Potential to Drive Market Expansion

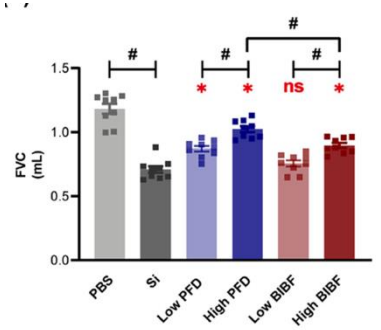
A. Preclinical studies demonstrate efficacy of Etuary™ in treating pneumoconiosis[1]



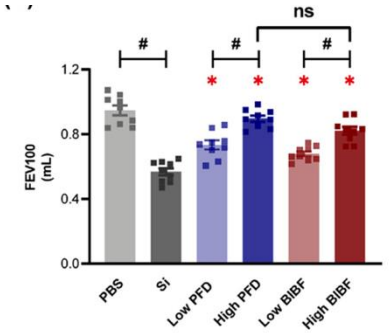
Operating Lung Volume



Gross Vital Capacity

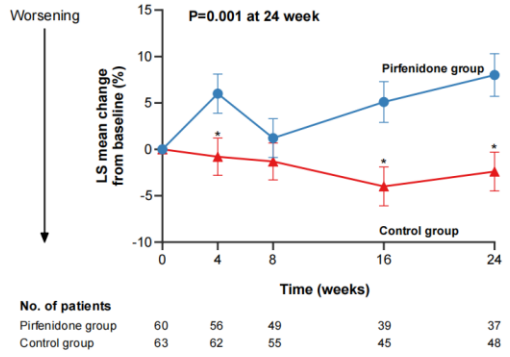


Airway Resistance / Flow

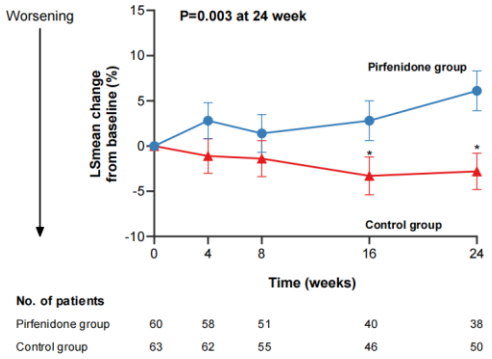


B. A phase 2 trial demonstrates efficacy of Etuary™ in treating radiation-induced lung injury (RILI)[2]

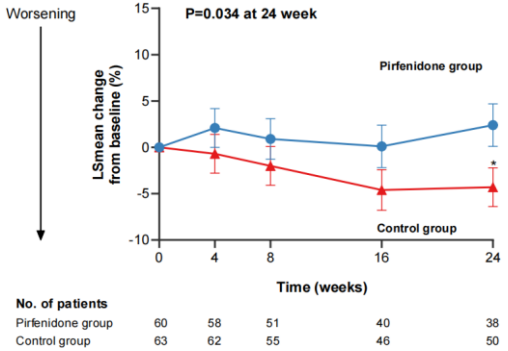
Gas Transfer Efficiency



Airway Resistance / Flow



Gross Vital Capacity



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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) A Next Generation Liver Fibrosis Therapy

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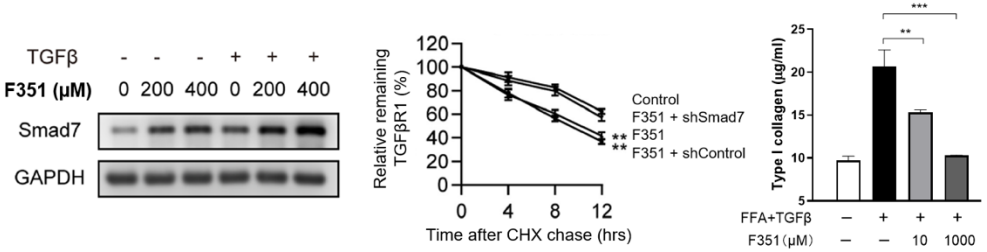
F351 (hydronidone): An NDA Stage Next-Generation Fibrosis Therapy

Primary Indication	Liver fibrosis caused by Chronic Hepatitis B (CHB) and Metabolic dysfunction-Associated Steatohepatitis (MASH)
Summary	Next-Generation Pirfenidone: An Antifibrotic Agent with Enhanced Potency, Improved Safety, and Favorable Metabolism.
Clinical Rationale	F351 modulates the Smad7-TGF-β and p38γ signalling pathways, preventing the activation of hepatic stellate cells (HSCs)—the primary drivers of collagen deposition and fibrotic scarring in the liver. ***F351 will be positioned as a complementary therapy to agents targeting metabolic drivers of fibrosis such as agonists of GLP-1 and THR-β receptors and FGF21 analog.***
Current Status	Phase 3 trial of CHB-associated liver fibrosis was completed in China; Last patient completed treatment Oct 2024; Reported positive topline data in Q2 2025 — met primary endpoint. NDA accepted by NMPA in May 2026
Regulatory	Breakthrough Therapy designation from NMPA (March 2021) for CHB-induced liver fibrosis by NMPA and CDE. U.S. IND for MASH filed, with anticipated Phase 2 start in 2027.
Opportunity	China: Largest burden of hepatitis B world-wide, with an estimated 79 – 86 million cases of chronic HBV infections[1] USA: 14.9 million MASH patients in 2020 and increases to 23.2 million by 2050[2]

F351 Upregulates Smad7, Downregulates TGF-β receptor I, and Reduces Collagen Secretion[3,4]



F351 Development Highlights

Phase 1 China & USA

- ✓ Well tolerated as a single agent and upon repeated oral dosing, with no SAEs reported.
- ✓ Consistent safety and PK profile in US trial

Phase 2 China

- ✓ Met primary endpoint of improvement of liver fibrosis score (Ishak decrease of ≥1 point)
- ✓ Good tolerability
- ✓ The 90 mg/tid dose selected for Phase 3

Phase 3 China

- ✓ Positive Phase 3 topline results in CHB-associated liver fibrosis.
- ✓ NDA accepted by China NMPA in May 2026

1. https://pmc.ncbi.nlm.nih.gov/articles/PMC11806133/
2. https://pubmed.ncbi.nlm.nih.gov/39821400/
3. Xu et al (2023) PMID:37641479
4. Internal unpublished results

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F351 Phase 3 Results Demonstrate New Global Potential in Liver Fibrosis and Cirrhosis

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

Favorable Safety Profile

≥1-stage fibrosis regression at Week 52:

- F351: 52.85% (n=123) vs.
- Placebo: 29.84% (n=124)
- **Delta: 23.01%**
- **p = 0.0002** (ITT1 analysis with central blinded pathology review)
- Consistent with fibrosis regression rates observed in Phase 2

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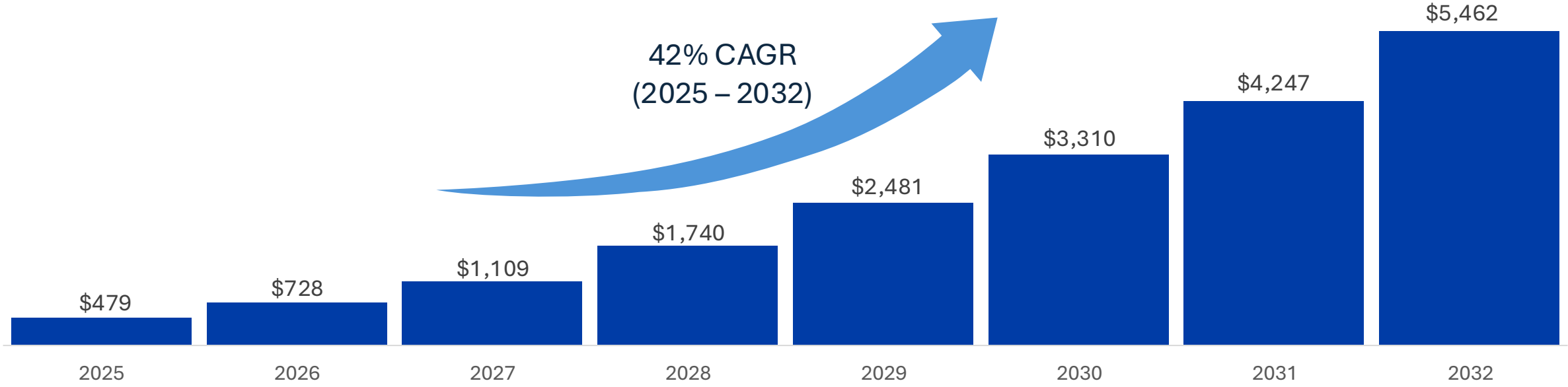
U.S. MASH Fibrosis Market Provides Significant Opportunity for F351

China First Strategy Provides Accelerated POC

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



- Current U.S. MASH prevalence is estimated at ~14 million[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).
1 Le et al. (2025) JAMA; AJMC; MASH Awareness; Estes et al. (2018) AASLD; L.E.K. interviews

Promising Path for Future Growth:

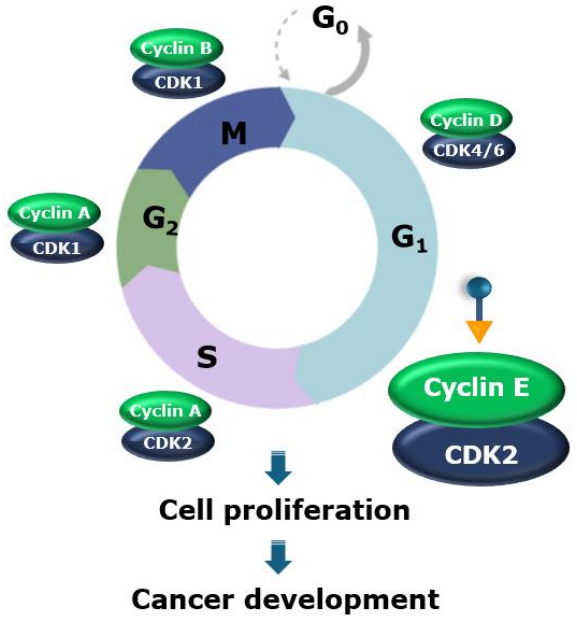
Targeted Protein Degraders (TPDs) and Degrader-Antibody Conjugates (DACs) Pipeline

- CDK2 - Cyclin E Dual-Degrader for Solid Tumors
- TYK2 - JAK1 Dual-Degrader for Inflammatory Diseases

CDK2 - Cyclin E Dual-Degrader for Solid Tumors

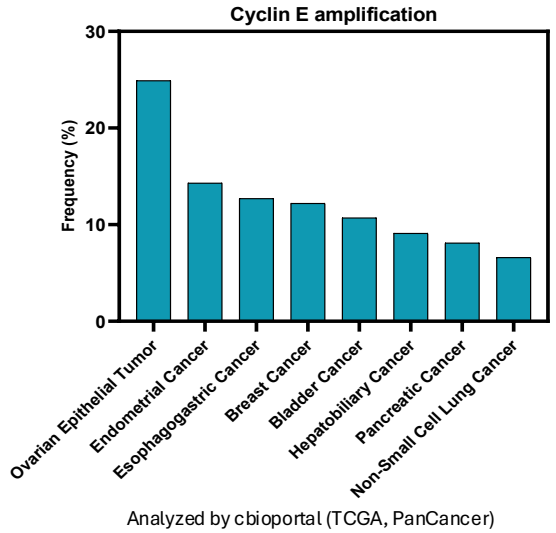
Hyperactivation of the CDK2–Cyclin E Complex Drives Solid Tumor Progression and Confers Resistance to Breast Cancer Therapy

A. CDK2-cyclin E promotes cell proliferation and cancer development



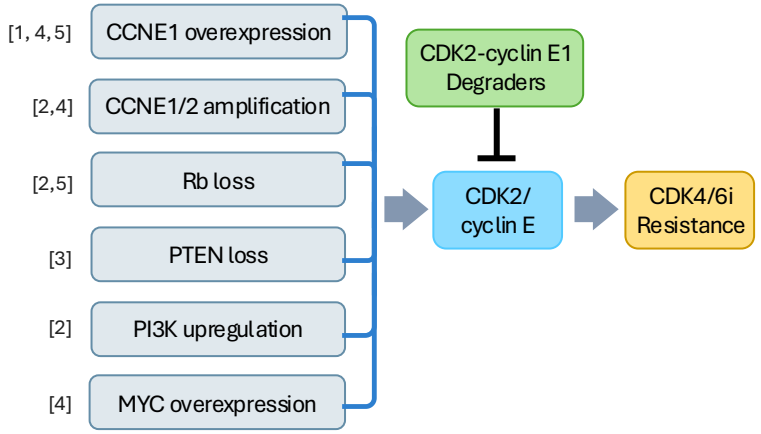
CDK2-cyclin E dual degrader blocks feedback induction of cyclin E by CDK2 inhibition and achieves sustained suppression of cell proliferation

B. Cyclin E is frequently amplified across multiple cancer types



Analyzed by cbioportal (TCGA, PanCancer)

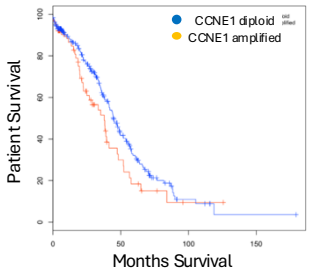
D. Diverse CDK4/6i resistance converge on activation of CDK2/cyclin E



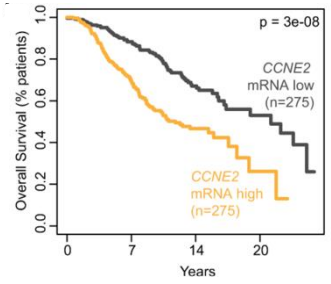
[1] Turner NC (2019) *J Clin Oncol*. PMID: 30807234
[2] Herrera-Abreu MT (2016) *Cancer Res*. PMID: 27020857
[3] Costa C (2020) *Cancer Discov*. PMID: 31594766
[4] Freeman-Cook (2021) *Cancer Cell*. PMID: 34520734
[5] Wander SA (2020) *Cancer Discov*. PMID: 32404308

C. Elevated cyclin E1 and E2 expression correlates with poor patient survival and therapy resistance

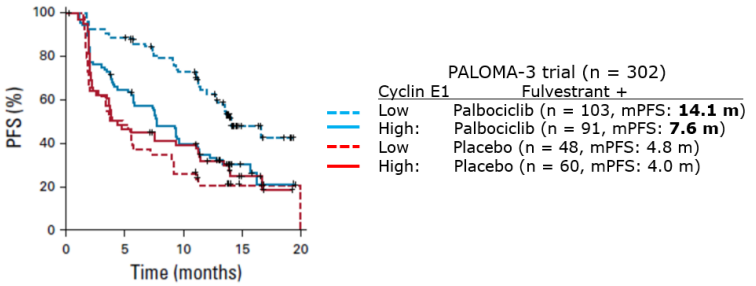
CCNE1 amplification correlates with poor survival in ovarian cancer patients[1]



CCNE2 overexpression correlates with resistance to endocrine therapy in breast cancer[2]



High CCNE1 expression is associated with resistance to CDK4/6 inhibitor in breast cancer[3]



1. TCGA (2011) *Nature* PMID: 21720365; 2. Milioli et al. (2020) *Endocr Relat Cancer* PMID: 32061162; 3. Freeman-Cook (2021) *Cancer Cell* PMID: 34520734

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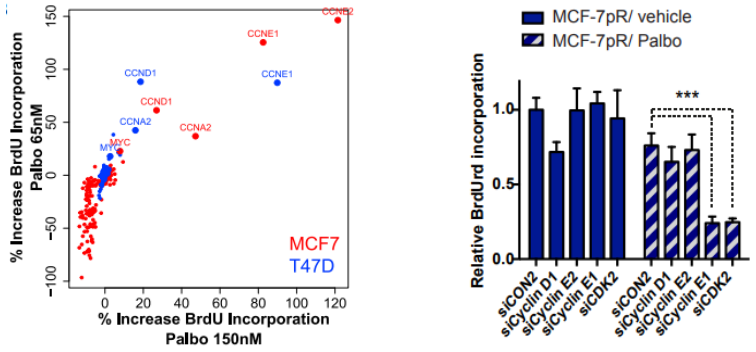
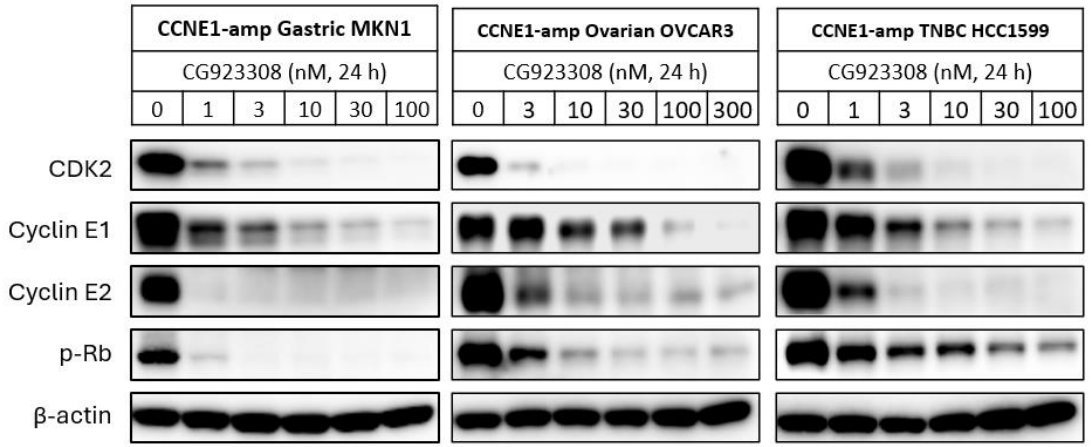
CDK2-Cyclin E Degrader for Treating *CCNE1*-amplified Solid Tumors and Breast Cancer Resistant to CDK4/6 Inhibitors; IND Anticipated Q1 2027

A. Deletion of CDK2/E1 re-sensitizes CDK4/6i-resistant cells to CDK4/6i[1,2]



1. Freeman-Cook (2021) *Cancer Cell* PMID: 34520734; 2. Herrera-Abreu et al (2016) *Cancer Res* PMID: 27020857

C. Discovery of selective CDK2 - cyclin E dual degraders



B. Target product profile

Indication	Solid tumors with *CCNE1* amplification (CCNE1^{amp}); HR+/HER2- breast cancer with CDK4/6i resistance;
Patient Population	Estimated in the US for 2024 by ACS (cancer.org) and cbioportal analysis of TCGA database **CCNE1^{amp} solid cancer: >25,000 new cases/year** • Ovarian cancer (19,680 new cases, 19% CCNE1^{amp}) • Endometrial cancer (67,880 new cases, 10.8% CCNE1^{amp}) • TNBC (62,144 new cases, 10.7% CCNE1^{amp}) • Esophagogastric cancer (49,260 new cases, 10.1% CCNE1^{amp}) • Non-small-cell lung cancer (187,664 new cases, 4% CCNE1^{amp}) **HR+HER2- metastatic breast cancer with CDK4/6i resistance: ~25,000 patients/year** • (310,720 new cases of breast cancer, 73% are HR+, 20-30% with metastatic disease; 40-50% progression rate)
Current SOC (US)	• Chemotherapy/ADCs • Hormone therapy (ovarian, breast) • Immunotherapy (breast, esophagogastric) • Targeted therapy (e.g. CDK4/6i, HER2 mAb, PARPi)
Unmet Clinical Needs	• Chemo/ADCs/hormone/targeted therapy: drug resistance, side effects • Immunotherapy: low response rate as monotherapy
Clinical Position	• Solid tumors with CCNE1^{amp} • Breast cancer with CDK4/6i resistance
Biomarker	• CCNE1^{amp} • CDK4/6i resistant
Proof-of-concept Study	Phase 1a/1b with expansion cohorts in CCNE1^{amp} ovarian, endometrial, TNBC, esophagogastric cancer as monotherapy; Phase 1a/1b with expansion cohorts in CDK4/6i resistant HR+ breast cancer as monotherapy;

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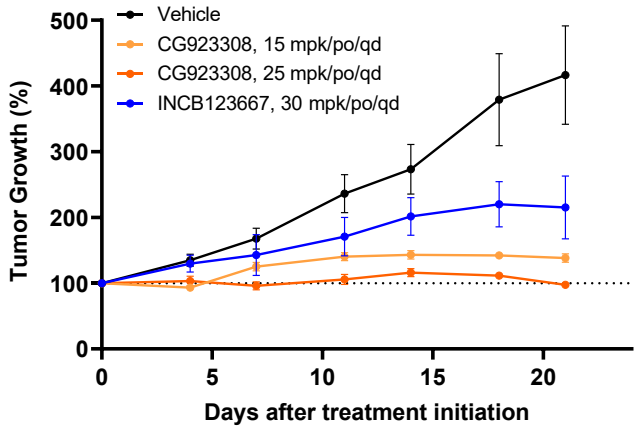
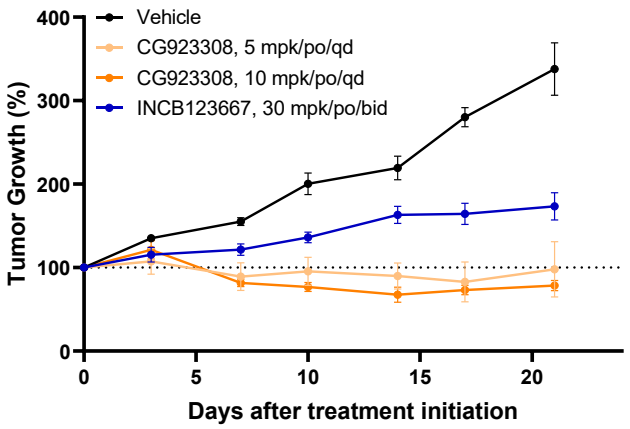
CDK2-Cyclin E Degrader Demonstrates Greater *In Vivo* Anti-Cancer Efficacy Than Phase 2/3 CDK2 Inhibitors in CDX and PDX Models

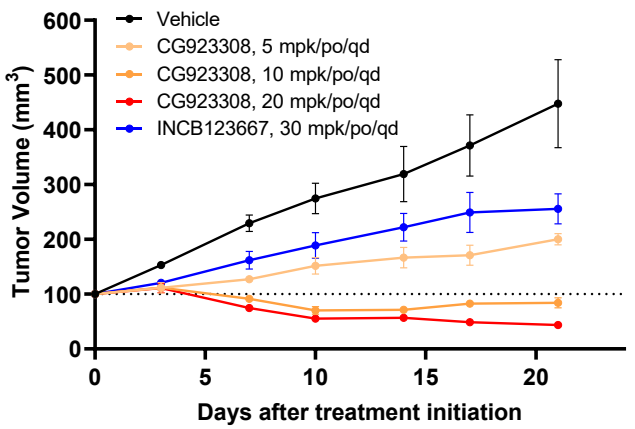
A. *CCNE1*-amp OVCAR3 Ovarian CDX



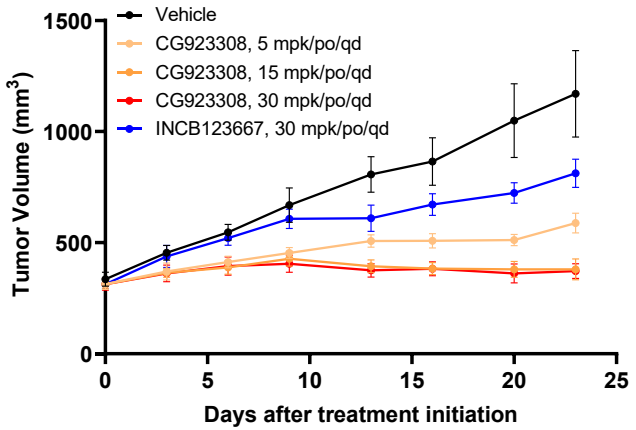
B. *CCNE1*-amp MKN1 Gastric CDX



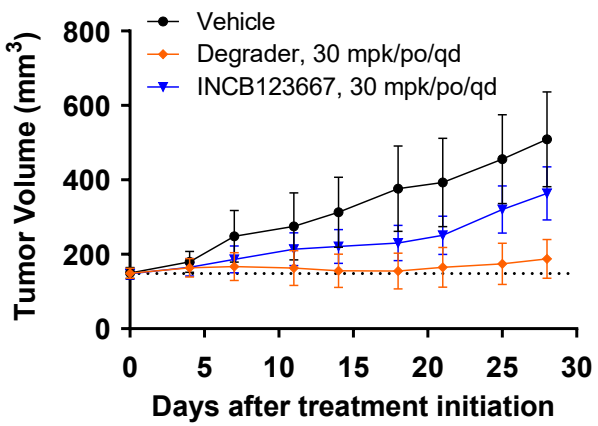
C. *CCNE1*-amp HCC1599 TNBC CDX



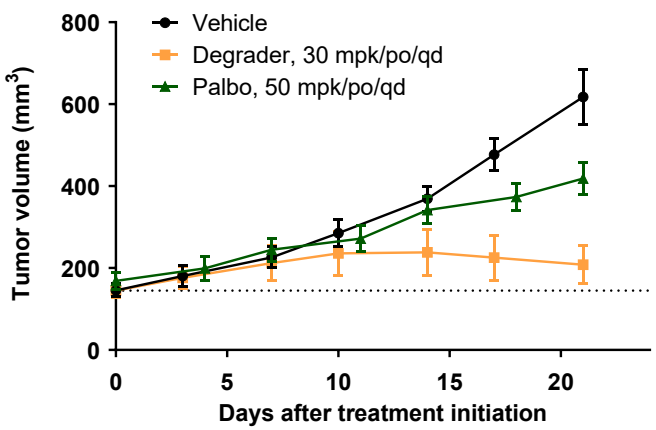
D. *CCNE1*-amp HCC1569 Breast CDX



E. Chemo-resistant *CCNE1*-amp TNBC PDX



F. *Rb*-deficient, CDK4/6i-resistant Breast PDX

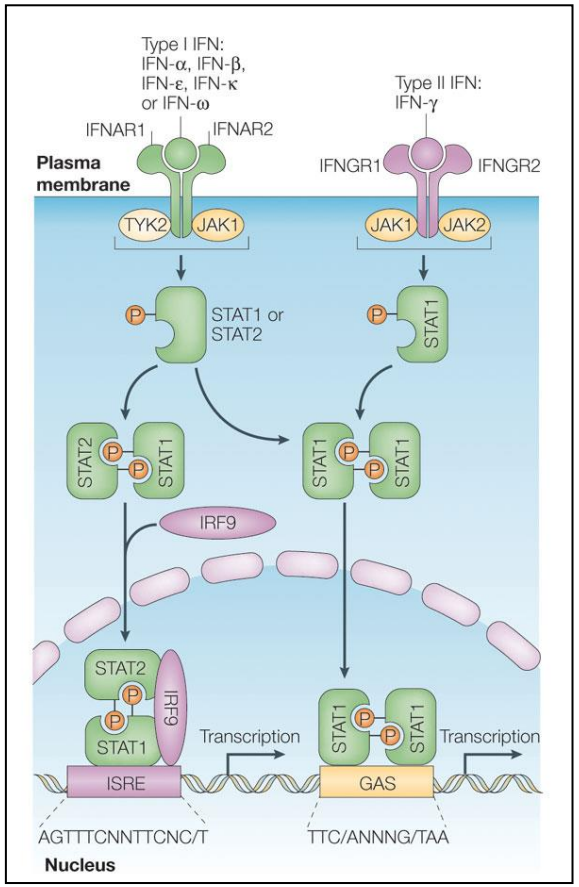


All doses were well tolerated in animals, with no significant body weight loss observed during the studies

TYK2 - JAK1
Dual-Degrader
for Inflammatory Diseases

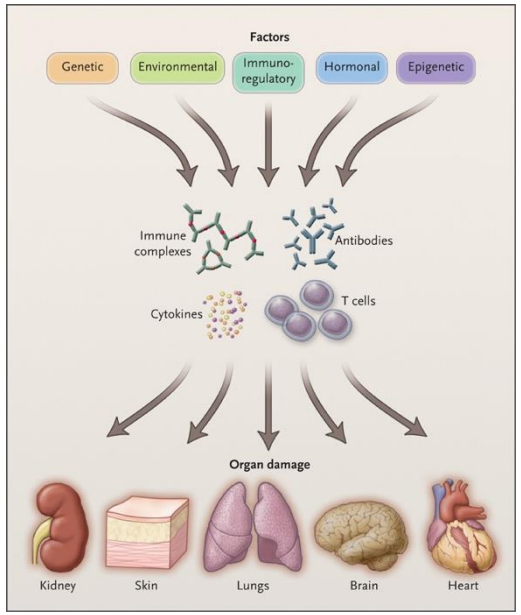
Dual Targeting of TYK2 and JAK1 for Autoimmune Diseases, Focus on Systemic Lupus Erythematosus and Rheumatoid Arthritis

A. TYK2/JAK - STAT signaling



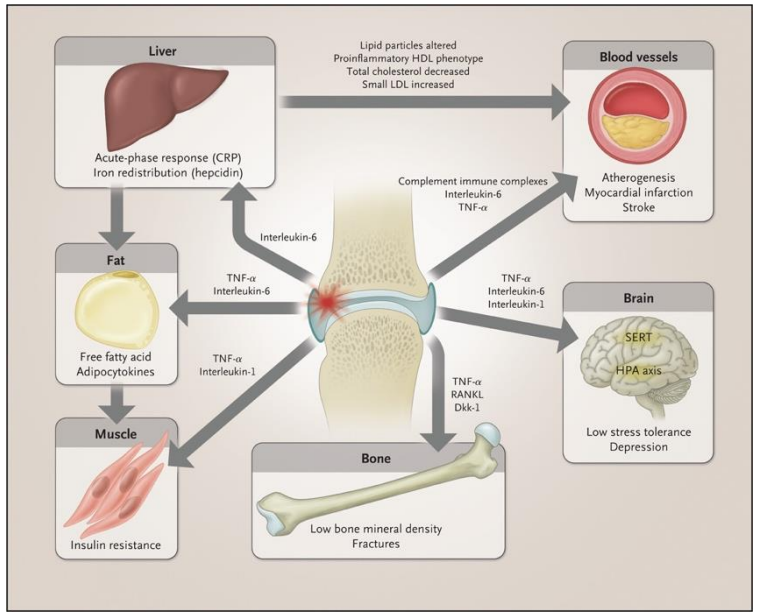
Platanias, LC. (2005) *Nat Rev Immunol* PMID:15864272

B. SLE Mechanism



Tsokos GC.(2011) *NEJM* PMID: 22129255

C. RA Mechanism



McInnes & Schett (2011) *NEJM* PMID: 22150039

Significant Opportunity

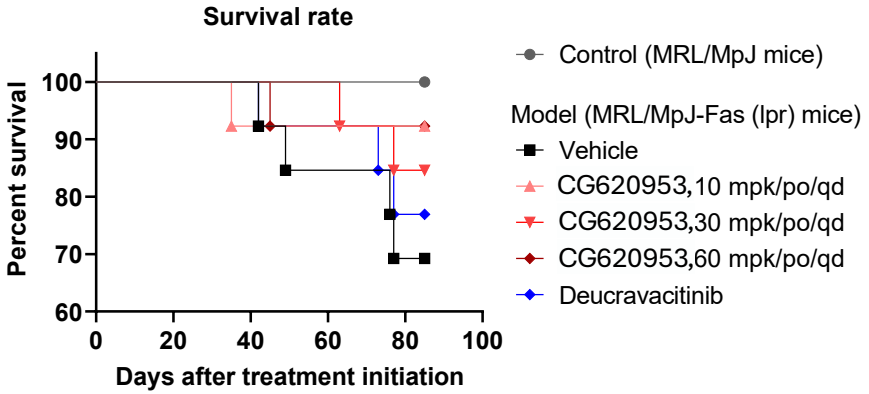
- 125,000,000 psoriasis patients worldwide[1]
- 18,000,000 rheumatoid arthritis patients worldwide[2]
- ~204,000 lupus patients in the US in 2018[3]

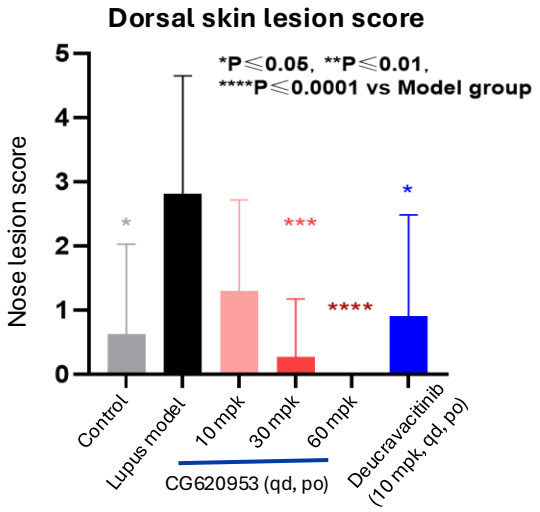
1. https://www.psoriasis.org/psoriasis-statistics/
2. https://www.who.int/news-room/fact-sheets/detail/rheumatoid-arthritis
3. https://www.niams.nih.gov/health-topics/lupus/basics/symptoms-causes

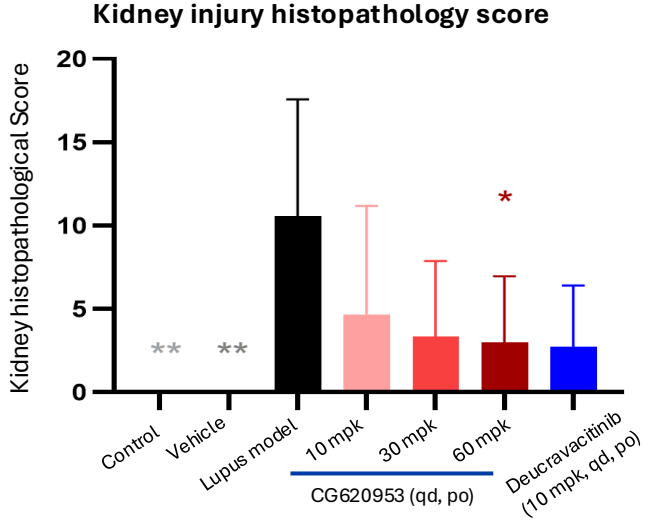
Gyre THERAPEUTICS

CG620953 Demonstrates Superior Efficacy in Preclinical Models of Lupus and Rheumatoid Arthritis (RA)

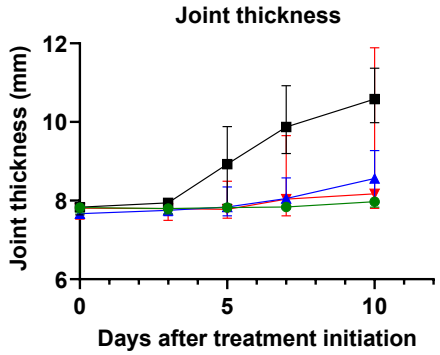
A. CG620953 is effective in a mouse model of systemic lupus erythematosus (SLE)

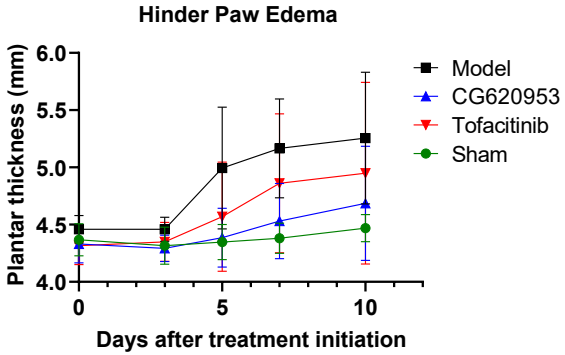




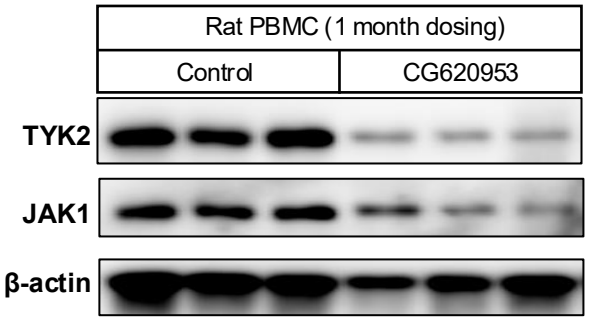


B. CG620953 shows efficacy in a rat model of rheumatoid arthritis





C. Targeted protein degradation in RA model



Gyre THERAPEUTICS

Leveraging China Innovation Advantages to Advance Pipeline Products

Gyre's China Innovation and Validation Engine Provides Ability to Leverage China's Unique Ecosystem Pillars

	Drug Discovery Capabilities	Streamlined Clinical Enrollment	Cooperative Regulatory Environment
China Ecosystem Opportunities	• Lower operational costs • Affordable STEM talent • Early-stage development up to 55% faster[1]	• Rapid enrollment (weeks vs. months)[1] • Dense urban clinical sites (Beijing, Shanghai, Guangzhou) • Treatment-naïve patient base[1]	• Similar IND approval speed as U.S. FDA • Priority review and breakthrough designation • Harmonization with int'l standards • NMPA reforms
Examples of Gyre Success	• Creation of >1,000 functional degraders each year since inception • DAC creation to NHP testing in as few as 3 months	• Established network of healthcare providers • ETUARY™ Phase 3 IPF study enrolled patients 3x faster than most recent Phase 3 IPF study in USA[2].	• F351 Breakthrough designation • F351 Priority Review status
	DISCOVERY	**CLINICAL**	**REGULATORY**

China Advantage:

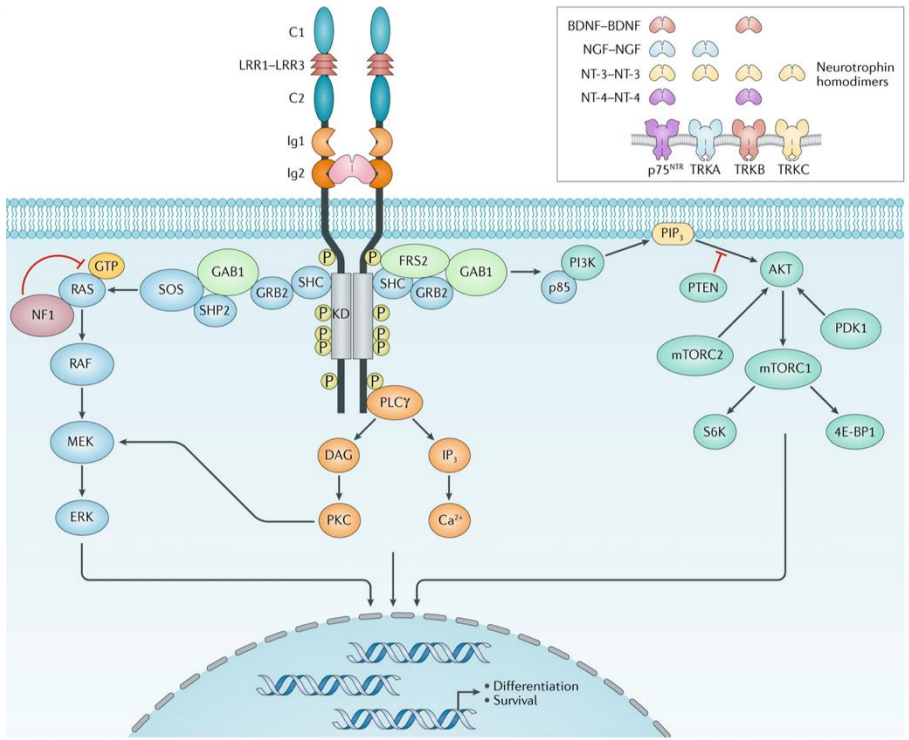
Cost-effective Operations | **Speedy Timelines** | **International Standards**

1. ARC Group Insights – July 21, 2025
2. Comparison vs. Boehringer Ingelheim nerandomilast phase 3 study

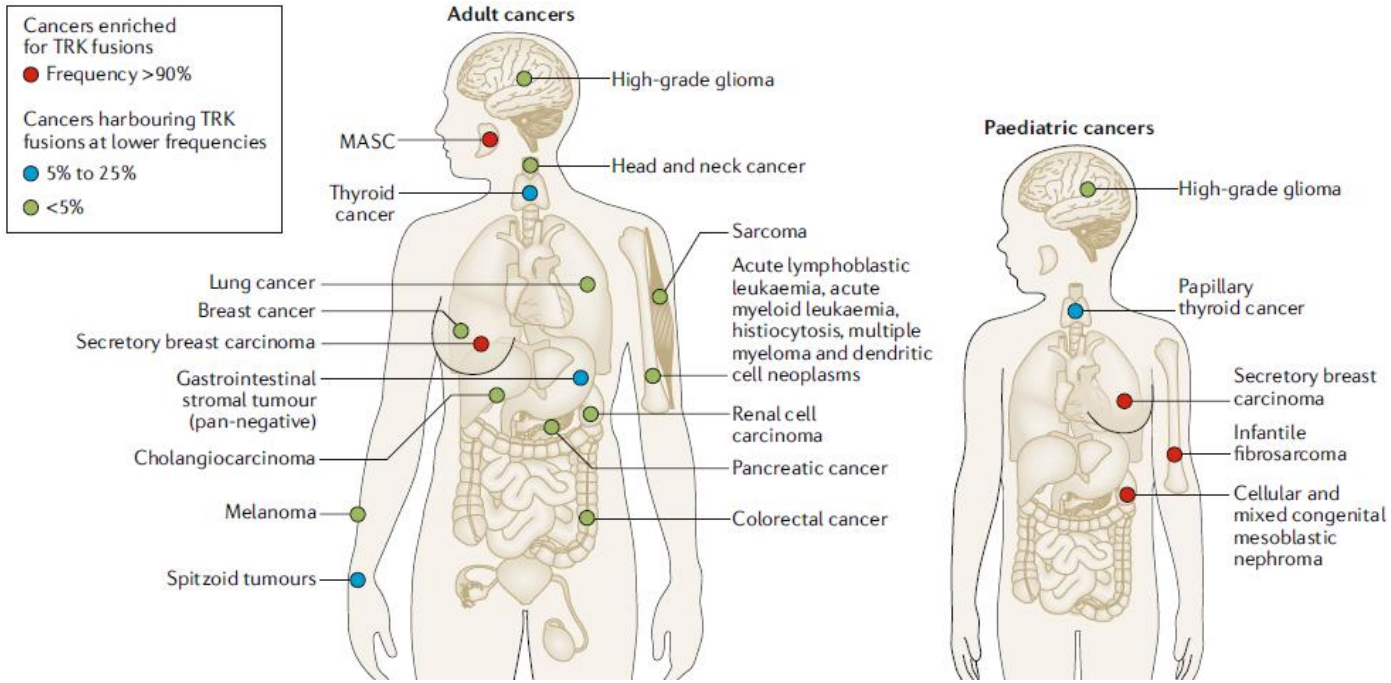
Degraders for Cancer and Cancer-induced Bone Pain
- TRK degrader (CG001419)
- GSPT1 degrader (CG009301)

Targeting TRK for Pain and Cancer

A. TRK signaling pathways

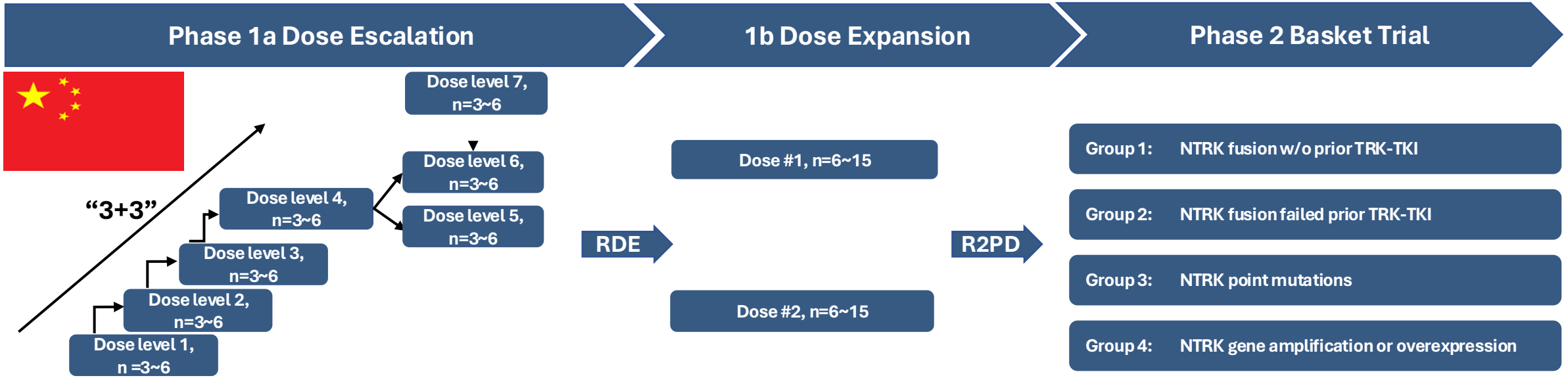


B. Activation of TRK in multiple solid tumors



Cocco, Scaltriti & Drilon (2018) *Nat Rev Clin Oncol* PMID: 30333516

Clinical Development of CG001419 for Cancer



Future Data Points

- Data from first 22 patients demonstrated no observed DLTs, treatment-related SAEs or grade ≥3 treatment related AEs.
- Enrollment in Phase 1b (dose expansion portions) began Q1 2026.

Summary of CG001419 for Cancer

1 Positioning and Differentiation

- First-in-Class, selective, oral TRK degrader for the treatment of adult cancer patients with NTRK gene abnormalities
- Potential use in cancer patients with NTRK gene fusion who acquire resistance to prior TRK kinase inhibitors via NTRK mutations

2 Clinical Strategy

- Exploratory study in cancer patients with NTRK amplification, overexpression and point mutations

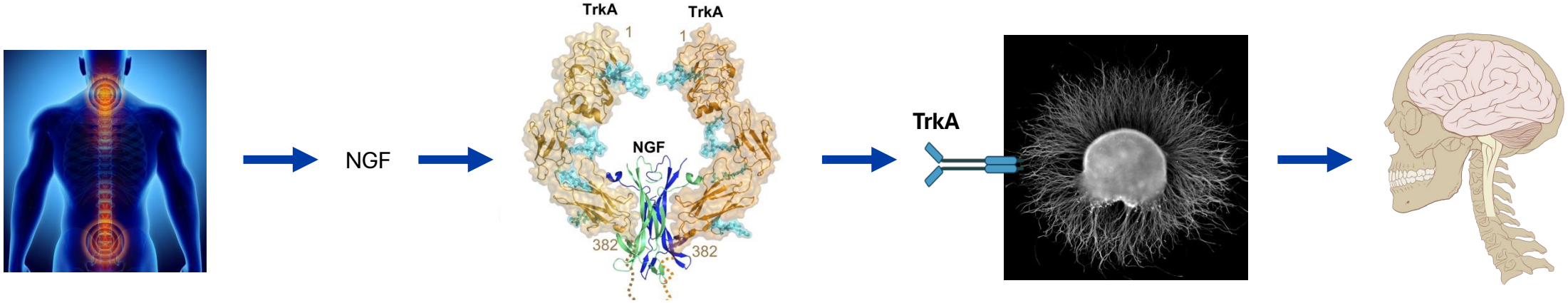
3 Clinical Development Plan

A rational, step-wise, biomarker-driven Phase 1/2 study design with expansion cohorts to provide early efficacy readout and assess the safety, PK and PK/PD relationships in selected tumors

- If successful, expected accelerated regulatory pathway toward early approval, including breakthrough designation
- Early incorporation of biomarker strategy supports development of precision treatment and associated companion diagnostic (CDx)

NGF And TRK Are Key Mediators of Acute and Chronic Pain

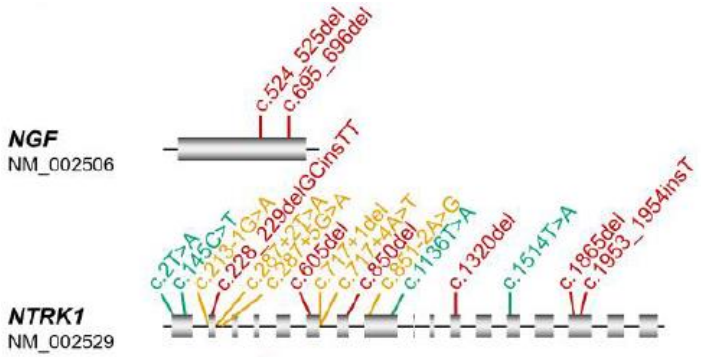
A. Nerve growth factor (NGF) stimulates the TrkA signaling pathway to transmit pain to the central nervous system



B. TRKA mutations cause congenital insensitivity to pain and anhidrosis (CIPA)

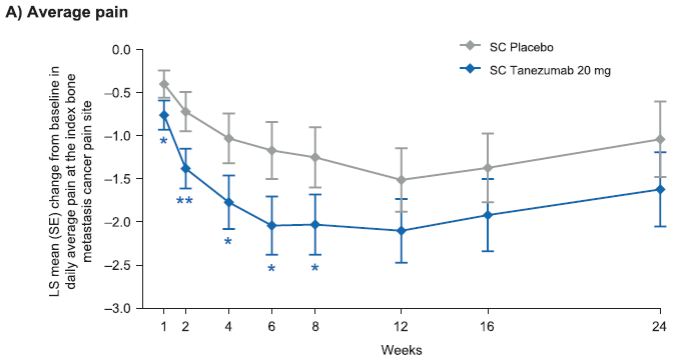
Sequencing of a cohort 78 CIPA patients

Mutations identified in:	22 genes
Mutation in *TRKA*:	20 patients
Mutation in *NGF*:	2 patients
Mutation in *Nav1.7*:	22 patients
Other 19 genes:	34 patients



Indo et al (1996) *Nat Genet*. PMID: 8696348
Lischka et al (2023) *Brain* PMID: 37769650

C. Blocking NGF reduces cancer bone pain



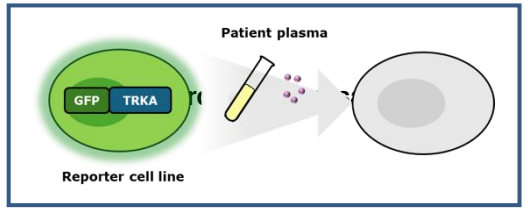
Fallon et al. (2023) *Oncologists* PMID: 37343145

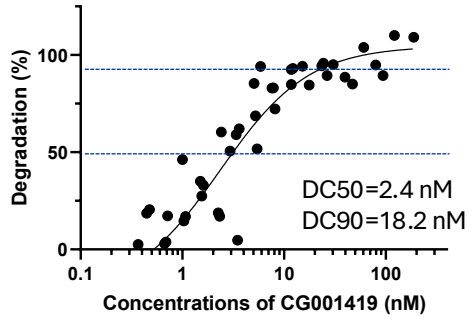
Gyre THERAPEUTICS

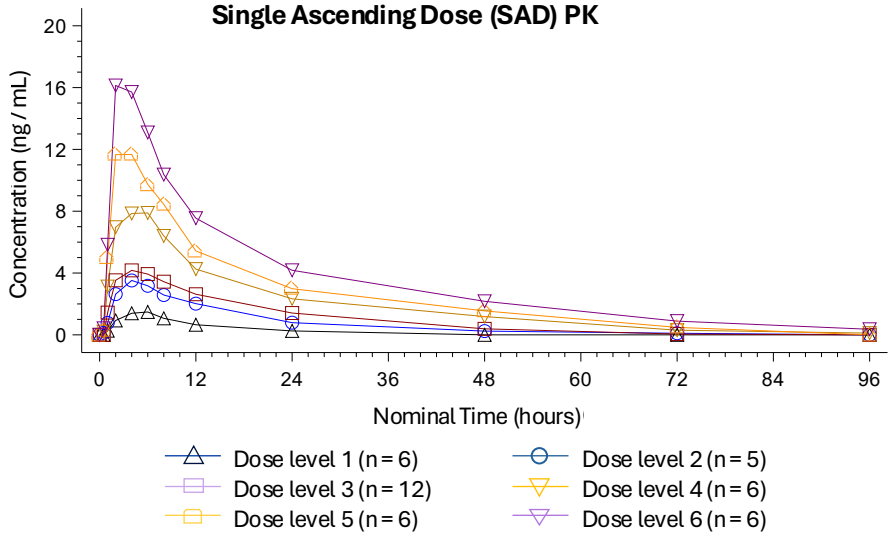
Summary of Phase 1 PD, PK and Safety Study of CG001419

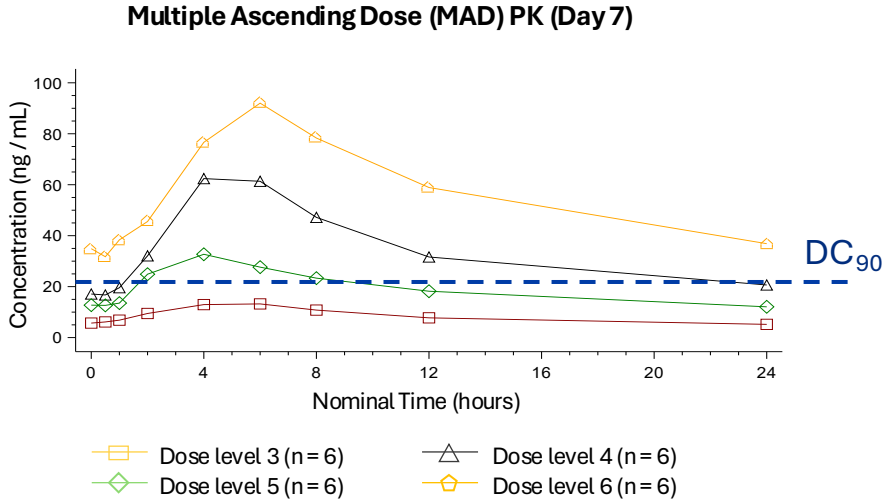
CG001419-101 (NCT06636500): a SAD/MAD/FE study in healthy subjects in Australia

- The surrogate PD assay demonstrated DC_{50} and DC_{90} values of 2.4 nM and 18.2 nM, respectively
- Single and multiple oral doses of CG001419 up to the highest dosing levels were safe and well tolerated by the healthy subjects
- In the SAD/FE of the study, 72.2% had a TEAE and in the MAD 83.9% had a TEAE
- Most TEAEs were considered mild or moderate at their maximum severity in both parts of the study. No Grade 4 (potentially life-threatening) TEAEs were reported
- The most frequently reported TEAEs by SOC were general disorders and administration site conditions. Since the drug was administered orally, these were likely due to blood collection procedures
- Following a single oral dose, the exposure to CG001419 increased in a dose-proportional manner
- The food-effect cohort demonstrated a higher systemic exposure under the fed condition
- For the MAD cohorts after multiple daily dosing for 7 days, exposure to CG001419, metabolite M2 and M8 increased in a less than dose-proportional manner









Gyre THERAPEUTICS

CG001419: Differentiated as a Potential First in Class Non-Opioid Medicine for the Treatment of Pain

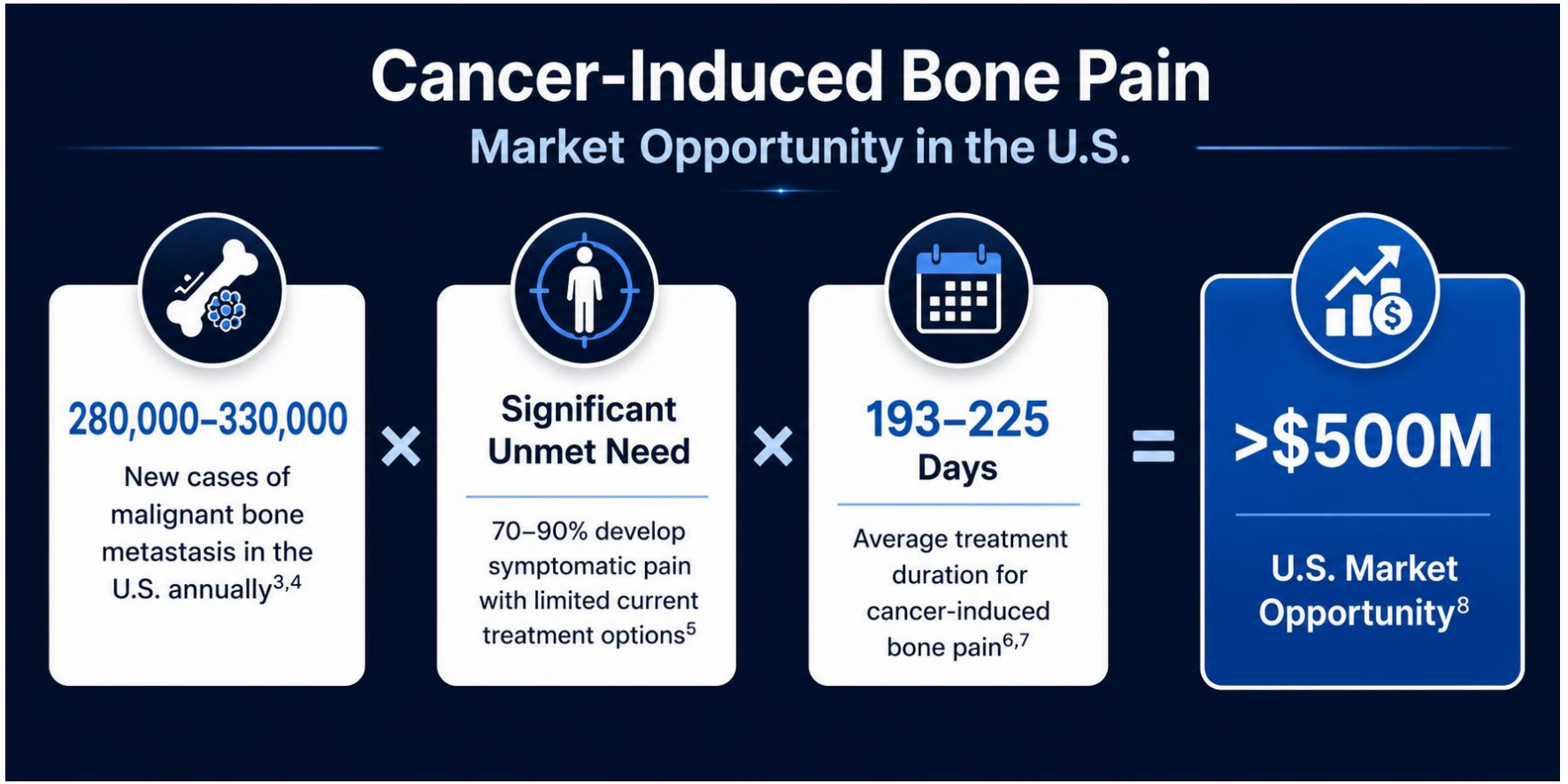
	Opioids	NSAIDs	Cebranopadol (Tris Pharma)	Journavx (Suzetrigine, VX-548) (Vertex)	VX-993 (Vertex)	LTG-001 (Latigo Bio)	STC-004 (SiteOne Therapeutics / Lilly)	CG001419 (Gyre Therapeutics)
Safety Concerns	Risk to develop dependency	GI issues, headache, dizziness	Nausea	–	–	–	–	–
Effective	✓	Moderate	Moderate	Moderate	Did not meet acute pain primary endpoint	TBD	TBD	✓ Preclinical studies
MOA	Neuron hyperpolarization	COX inhibitor	Dual-NMR (NOP and opiate receptor) agonist First-in-class	Nav1.8 inhibitor First-in-class	Nav1.8 inhibitor Fast-follower	Nav1.8 inhibitor Fast-follower	Nav 1.8 inhibitor Fast-follower	TRK degrader First-in-class
Non-addictive	Rapid development (< 5 – 14 days)	✓	TBD	✓	✓	✓	✓	✓
Phase	Approved	Approved	Phase 3 Trials Complete	Approved	Discontinued as monotherapy for acute pain	Phase 1 Complete	Phase 1 Complete	Phase 1 Complete

Cancer-induced Bone Pain Market Opportunity

Current pharmacological therapies to treat CIBP are inadequate, with 70% treated with opioids reporting continued bone pain[1]

Tanezumab, an NGF antibody, showed reduction in pain in phase III trials of cancer patients with bone pain[2]

Opioids – Marginally effective at relieving CIBP and come with significant side-effects (nausea, vomiting, constipation) especially for advanced or palliative stage cancer patients where quality of life is paramount



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

Gyre THERAPEUTICS

Summary of CG001419 for Pain

1 Positioning and Differentiation

- First-in-class TRK degrader as an analgesic for acute and chronic pain
- Potential for differentiation in efficacy and safety from NSAIDs, opioids, and NAV1.8 inhibitors via novel mechanism of action

2 Clinical Strategy

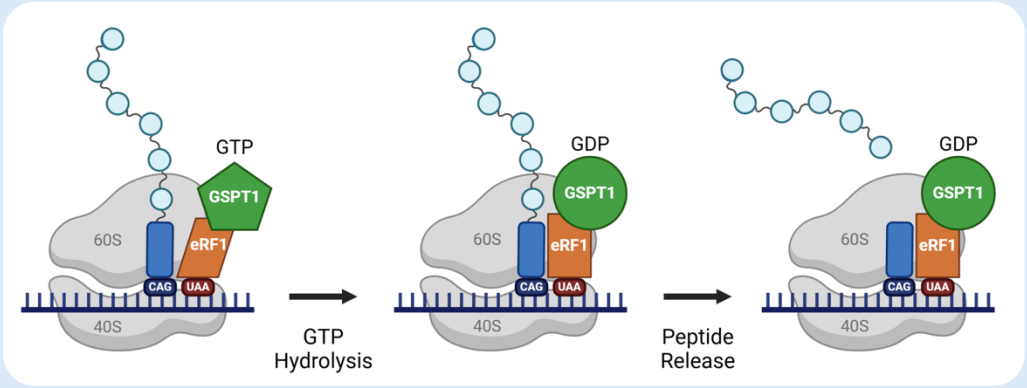
- Rational, mechanism-based selection of indications and target populations
- Planned Phase 2 study in cancer-induced bone pain applications
- Objectives:
  1) magnitude and time course of CG001419 analgesia relative to placebo
  2) safety of CG001419 compared to placebo
  3) PK characteristics of CG001419

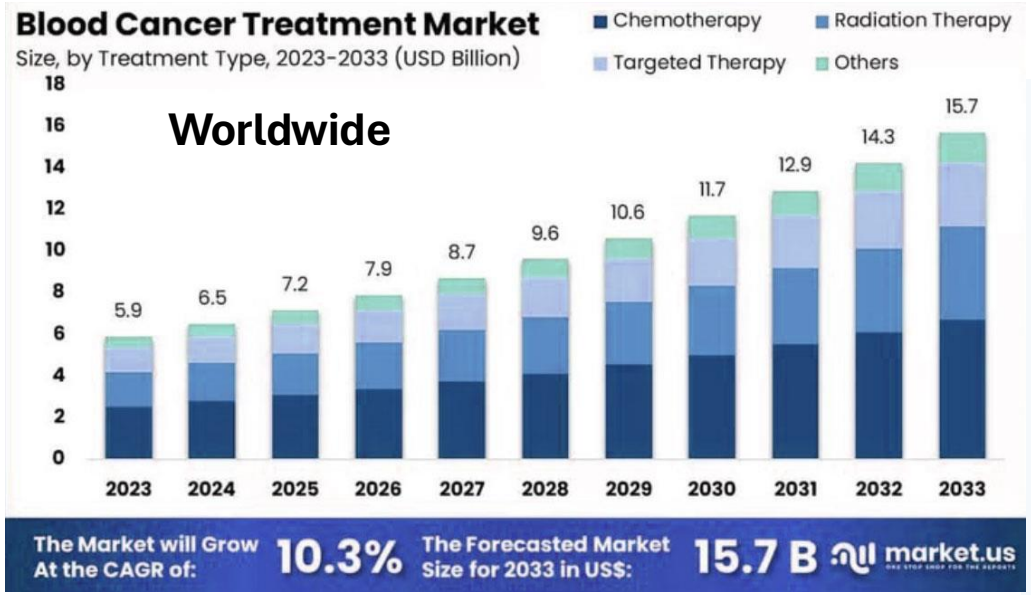
3 Clinical Development Plan

- Phase 1a trial in Australia to assess PK and safety completed Q4 2025
- Phase 2 POC trial in patients with cancer-induced bone pain or other metastatic cancer pain syndromes

Targeting GSPT1 for AML and MYC+ 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”.
- Cullgen has developed a potent and selective GSPT1 degrader, CG009301.
- Preclinical studies have validated the selectivity, potency and safety of CG009301.





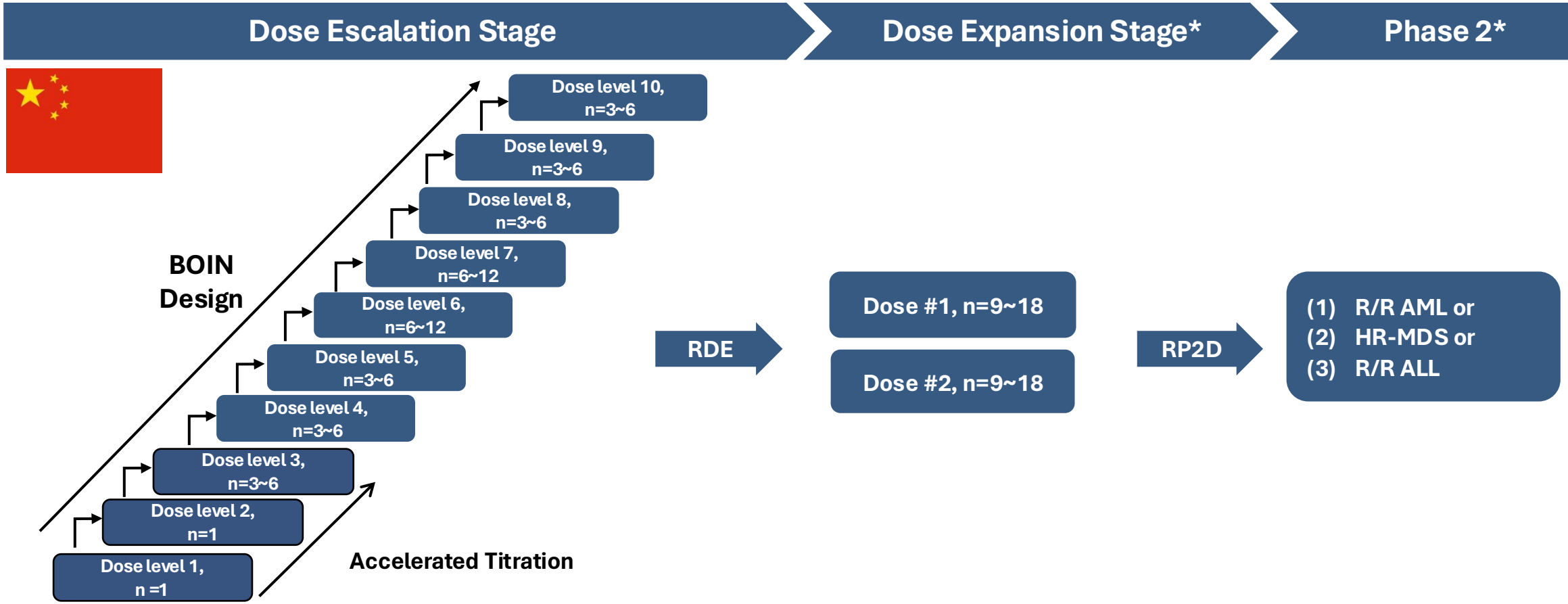
US Patient Population

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	

[1] 2024 by American Cancer Society estimates
[2] The Cancer Genome Atlas (TCGA) estimates
[3] Schaub et al (2018) *Cell Syst* PMID: 29596783
[4] Volpe et al. (2022) *Clin Lymphoa Myelom Leuk*, PMID: 34544674

Gyre THERAPEUTICS

Clinical Development of CG009301 in Patients with Recurrent or Refractory Hematologic Malignancies



- Dose escalation stage currently underway
- Data from first 8 patients demonstrated no observed DLTs
- Anticipate enrollment of approximately 30 – 45 patients

Gyre THERAPEUTICS

* Subject to regulatory alignment

Summary of CG009301 for Cancer

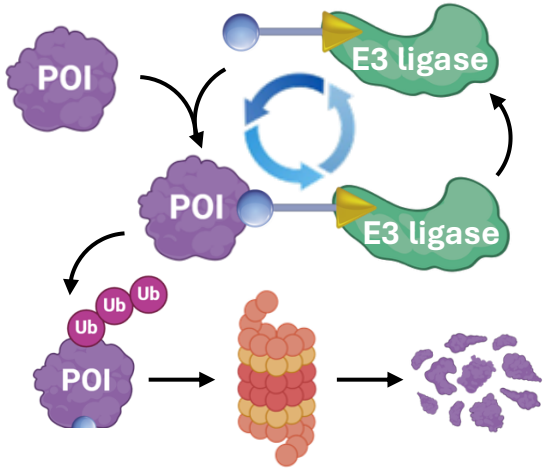
1 Positioning and Differentiation	• Relapsed / refractory AML, HR-MDS, and ALL patients • Potential to also treat relapsed / refractory MYC-driven solid tumors • Pre-clinical leukemia models indicate strong anti-tumor activity
2 Clinical Strategy	• Cullgen initiated a Phase 1 clinical trial in subjects with refractory hematologic malignancies in April 2025 in China. The expansion cohorts will focus on R/R AML, HR-MDS and ALL patients with hopes of identifying the optimal cohort for subsequent Phase 2 testing.
3 Clinical Development Plan	• The Phase 1a/1b data is expected to be submitted as the basis for an IND application to conduct the Phase 2 studies in the chosen disease population

Degrader-Antibody Conjugates (DACs)

34

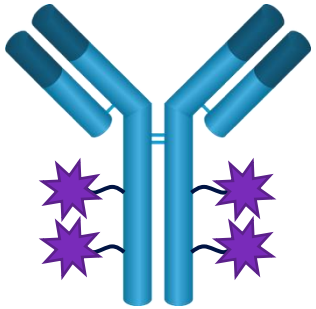
Degrader-Antibody Conjugates (DACs) Are the Next-Generation of ADCs

TPD
(Targeted protein degradation)



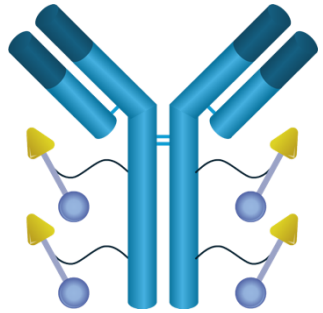
+

ADC
(Antibody drug conjugate)



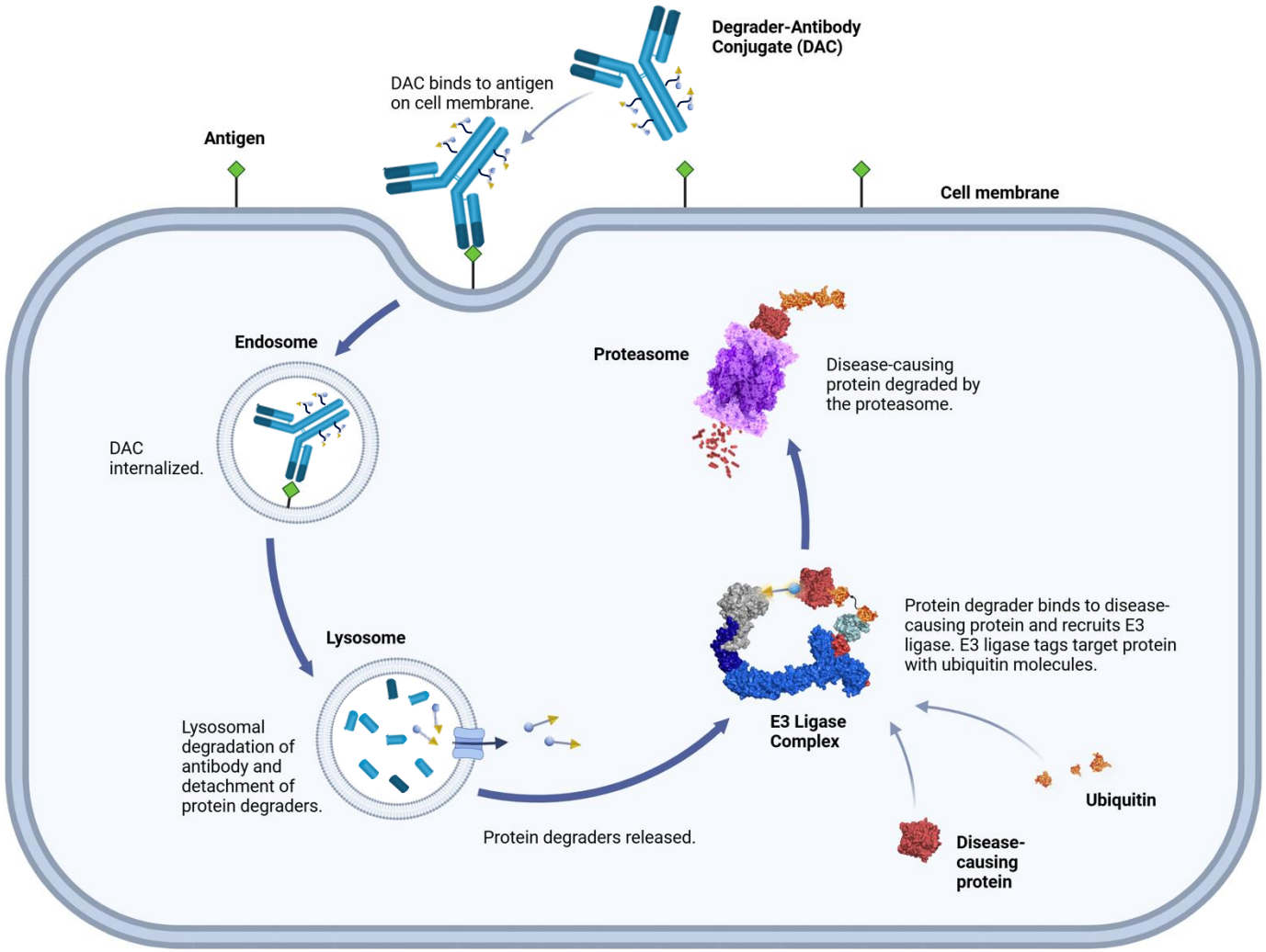
→

DAC
(Degrader antibody conjugate)



Modality	ADCs	TPD	DAC
Mechanism of Delivery	Intravenous	Oral or IV	Intravenous
Payload	Indiscriminate	**Tumor target selective**	**Target selective**
Efficacy	Requires potent payload	**Catalytic & potent**	**Catalytic & potent**
Tumor Selective Delivery	**Tumor cell selective**	Depends on E3	**Tumor cell selective**
Ability to Reduce Off Target Toxicity	No	Depends on E3	Yes
Need for oral bio-availability or cell permeability optimization	No	Yes	No

DAC Mechanism of Action Overview



HIGH POTENCY

The catalytic mechanism of action of TPDs ensures small quantity of degrader delivered by the antibody to achieve sufficient efficacy.

IMPROVED PK

Extended half-life, reduced systemic clearance, improved solubility, and bypassing the need for oral bio-availability or cell permeability optimization.

IMPROVED SAFETY

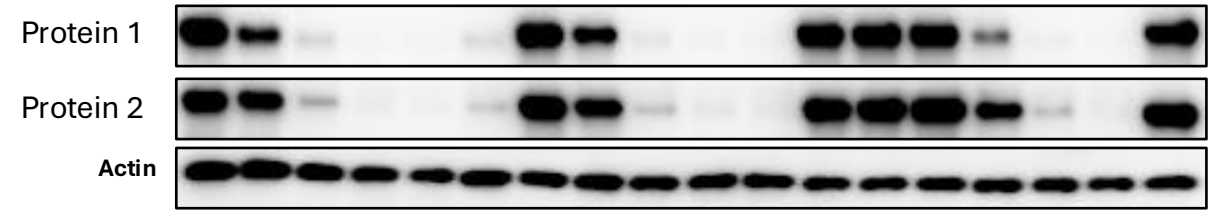
Reduced toxicity through dual target selectivity at the cell surface (antibody-tumor associated antigen) and intracellularly (degrader-target protein).

Gyre THERAPEUTICS

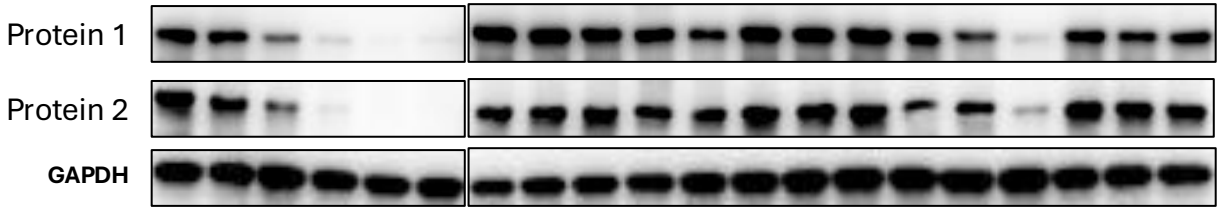
Epigenetic Factor DAC Demonstrates Potent and TAA-dependent Target Degradation and Cell Killing

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

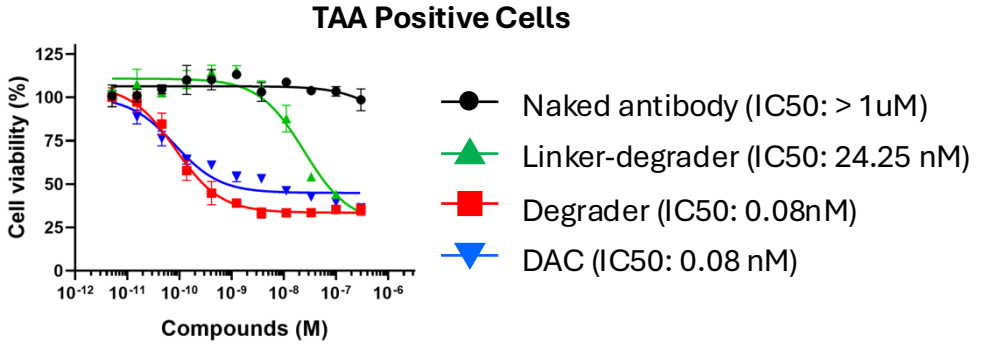
Cell Line	TAA Positive Cell Line																	
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

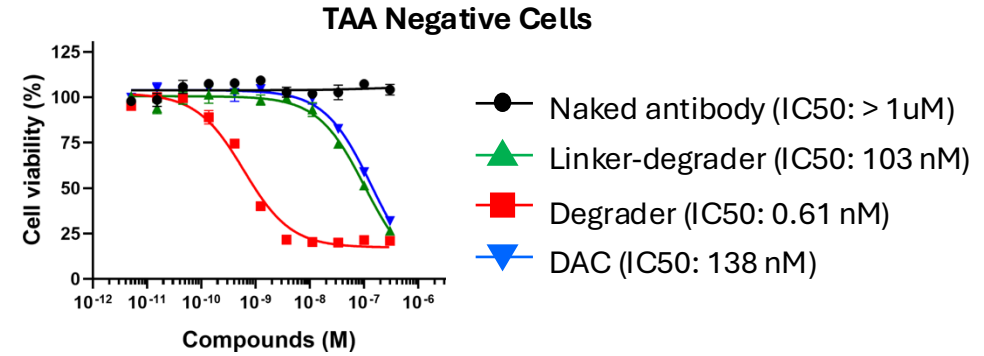


Cell Line	TAA Negative Cell Line																		
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					mAb		DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20



B. Cullgen epigenetic factor DAC kills cancer cells in a TAA-dependent manner



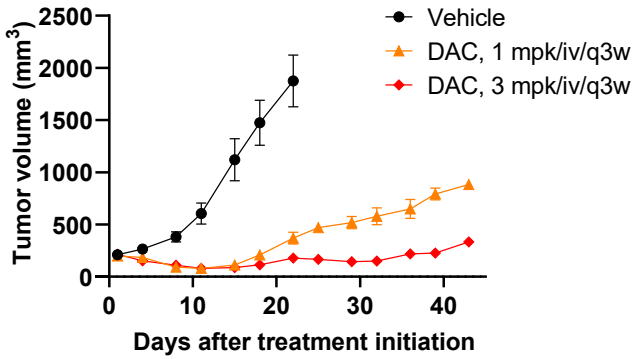


Gyre THERAPEUTICS

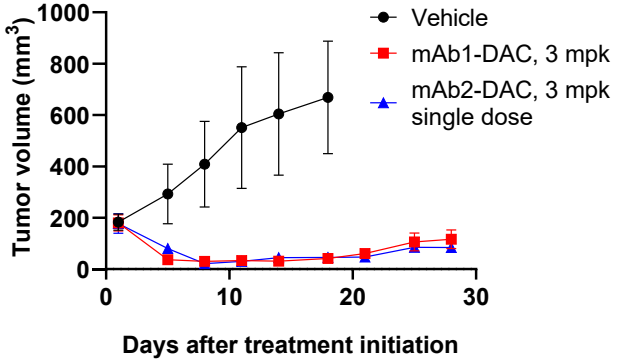
Degrader-Antibody Conjugates: Durable Tumor Regression, Superior to Dxd-ADC, and Overcoming Resistance in CDX and PDX Animal Models

A. DACs exhibit durable tumor growth inhibition in various solid tumor models

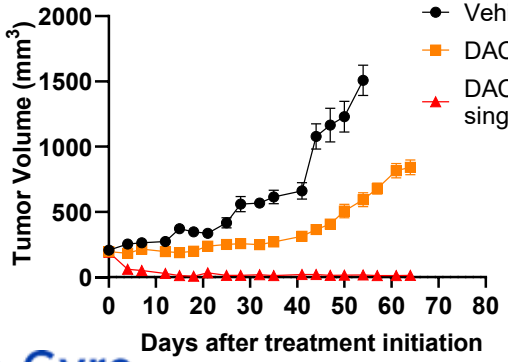
VCaP Prostate Cancer CDX model



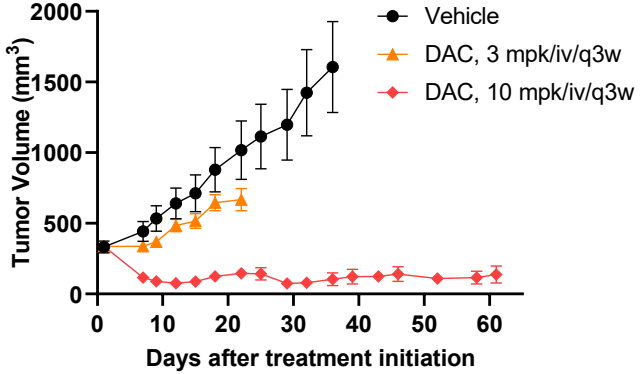
HCC827 NSCLC CDX model



MDA-MB-453 Breast Cancer Model

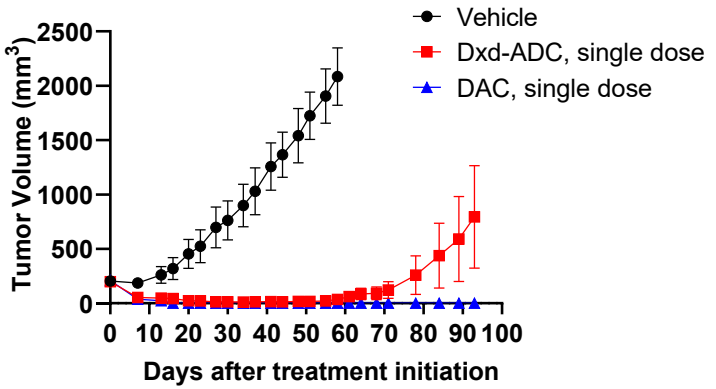


NCI-N87 Grastric Cancer CDX Model

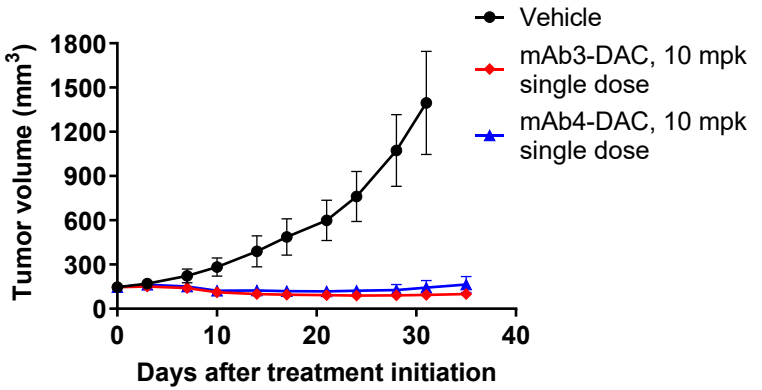


B. DACs are more effective than Dxd-based ADCs

BT474 Breast Cacner CDX model



C. DACs are effective in a CRPC PDX model resistant to enzalutamide



Gyre THERAPEUTICS

Gyre Has Established a Robust DAC Platform

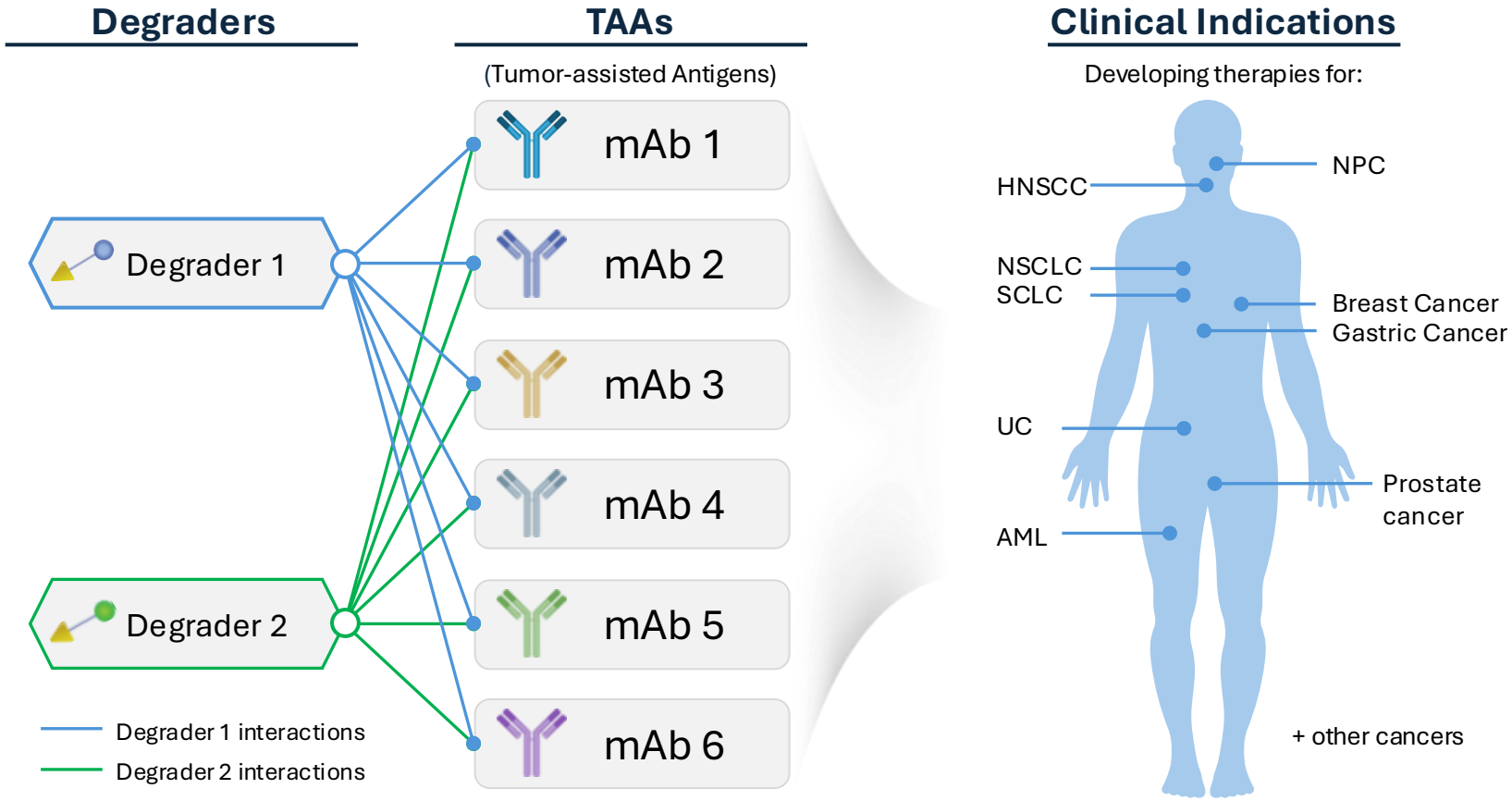
DACs represent the next generation of ADC therapies

Cullgen has developed >7,000 active degraders targeting >20 distinct proteins, serving as a valuable resource for payload selection

We have successfully generated multiple DACs and demonstrated their selectivity, efficacy and safety

Conjugating a diverse array of degraders to different antibodies offers the opportunity to selectively target different cancer types

Degraders May be Combined with Different mAbs to Create Novel DACs Targeting Specific Cancers



Gyre THERAPEUTICS

U.S. Management Team with Cross-Culture Operational Experience

Ying Luo, Ph.D.
Chief Executive Officer

Thomas Eastling
Chief Financial Officer

Weiguo Ye
Chief Operating Officer

Yue Xiong, Ph.D.
Chief Scientific Officer

Jialiang Wang, Ph.D.
Executive Vice President, General Manager

Joshua Bergmann, J.D.
General Counsel and Corporate Secretary

Ruoyu Chen
Chief Information Officer

Seth Goldblum, MBA
Senior Vice President - Corporate Development

Jing Liu, Ph.D.
Senior Vice President – Platform Chemistry

Mark Marino, M.D.
Senior Vice President – Clinical Development

Michael Plewe, Ph.D.
Senior Vice President - Medicinal Chemistry

Leslie Robinson, Ph.D., J.D.
Vice President - Intellectual Property and Licensing

Liang Zhao
VP Corporate Controller

Gyre THERAPEUTICS

Key Value Drivers

1. Robust and balanced therapeutic pipeline including assets from discovery to marketed products, with established manufacturing and commercialization operations
2. Utilization of highly efficient and cost-effective drug discovery and innovation capabilities in China to advance risk-mitigated products to the United States
3. Strong foundation in protein degrader development provides distinct advantage for the development of DACs as next generation ADC therapeutics
4. Accomplished management team in the United States and China with extensive international business operations experience

Thank You!

Gyre THERAPEUTICS