Gyre THERAPEUTICS

cullgen

A Fully-Integrated Biopharmaceutical Company Featuring a Robust Pipeline of Degraders and DACs

Forward-looking Statements

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Gyre Therapeutics: At-A-Glance

1ST

to receive **IPF[1] treatment approval** (pirfenidone) in **China** (2011):

Pioneering fibrosis treatment with a track record of success

#1

IPF market share in China for **10 consecutive years[2]**

(~50% IPF market share, 90% + share in pirfenidone in 2024)

~ 600

dedicated global employees:

~ 400 commercial team across **China** and the **U.S.**
~ 70 focused on **R&D**

150,000 +

IPF patients treated with **pirfenidone**

3,000+

hospitals and pharmacies covered in **China** across **870+ cities**

EBITDA positive

since 2017[3], while revenue grew at **~32%** compounded annual growth rate **(CAGR)[3]** during the same period

2023 Revenue	$113.5M
2024 Revenue	$105.8M

2 state-of-the-art, **GMP compliant manufacturing** facilities built for growth in China, currently running at **40%** and **18%** capacity

1. IPF = Idiopathic Pulmonary Fibrosis.
2. Per IQVIA CHPA.
3. Financial data inclusive of pro forma data prior to GNI Group and Catalyst Biosciences business combination for comparison purposes only.

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Cullgen: At-A-Glance

A Targeted Protein Degrader Company: Founded in 2018

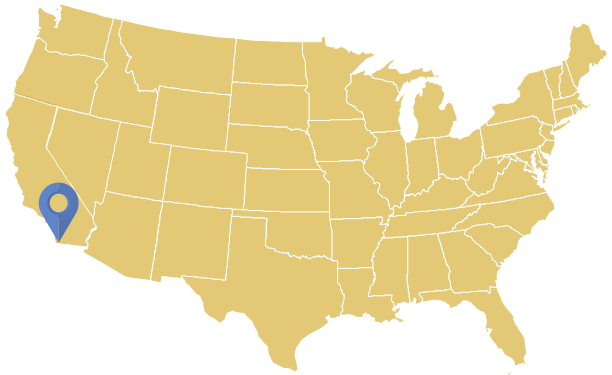
>120 employees worldwide
~50% with advanced degrees (PhD, MD, JD, MBA, MS)

8 Announced therapeutic programs:
- **1** forthcoming Phase 2 clinical trial
- **2** currently in Phase 1 clinical trials
- **2** IND-enabling studies
- **2** DACs in lead optimization
- **1** degrader in discovery

Programs cover multiple therapeutic areas including cancers and pain / inflammatory diseases

A Powerful Combination

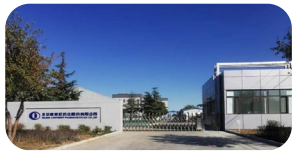
Combined entity intends to **leverage established and cost-efficient China operations** for accelerated discovery, early validation, and development of next generation therapeutics based on degraders and DACs



- Global innovation
- Late-stage clinical trials
- Access world's largest healthcare market
- Governance and compliance

- Accelerated development
- Initial validation / risk mitigation
- Cost efficiency
- Early commercialization

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



Beijing, China
Manufacturing, Clinical Development and Commercialization

Shanghai, China
Drug Discovery, Clinical Development

Post-Combination Gyre Therapeutics: At-A-Glance

Expected Date of Close Early Q2 2026

Company Name	Gyre Therapeutics, Inc. (Nasdaq: GYRE)	
Company Headquarters	San Diego, CA, with subsidiaries in Beijing and Shanghai	
Post-Merger Leadership	• Ying Luo – President & CEO • Yue Xiong – CSO	• Ping Zhang – Executive Chairman • Thomas Eastling – CFO
Therapeutic Assets	**10 announced therapeutic programs:** **1** Marketed **1** pre-NDA **2** Phase 2 **3** Phase 1 **3** IND-enabling studies + line extensions	
Therapeutic Areas Addressed	• Inflammation / Pain • Cancer	
WW Employees	**~740 Total:** ~170 R&D ~85 Manufacturing ~370 Sales & Marketing ~115 G&A	

Key Value Drivers

1. Robust and balanced therapeutic pipeline including assets from discovery to development, with established manufacturing and commercialization operations
2. Utilization of highly efficient and cost-effective drug discovery 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 with extensive international business operations experience

Broad Product Portfolio from Discovery to Commercialization

Robust Portfolio: Addressing Inflammatory Diseases, Pain and Cancer

Discovery / Lead Optimization	IND Enabling	Phase 1a	Phase 1b/2	Pre-NDA	Marketed
DAC DAC Blood Cancers	**CG923308** Degrader Solid Cancers	**CG009301** Degrader Leukemia and MYC+ cancers	**CG001419** Degrader Acute and Chronic Pain (IND filed)	**Hydronidone (F351)** CHB-associated Liver Fibrosis	**ETUARY® (Pirfenidone)** Idiopathic Pulmonary Fibrosis (IPF)
DAC DAC Solid Cancers	**CG620953** Degrader Inflammatory Diseases	**CG001419** Degrader Solid Tumors	**F573** Acute Liver Failure (ALF)		
Protein Degrader Degrader Fibrotic diseases	**F528** Chronic Obstructive Pulmonary Disease (COPD)	**F230** Pulmonary Arterial Hypertension (PAH)			

Key:

Inflammation / Fibrosis / Pain

Cancer

Degrader DAC

Table above does not include line extensions for ETUARY and F351

Therapeutic Pipeline: Significant Focus on DACs & Degraders

Program and Description	Indication	Discovery	IND Enabling	Phase 1a	Phase 1b/2	Phase 3
TGF-β Inhibitor **F351**	CHB – associated liver fibrosis					
Degrader | TRK **CG001419**	Acute and chronic pain					
Degrader | TRK **CG001419**	Solid tumors					
Degrader | GSPT1 **CG009301**	Leukemia and MYC+ cancers					
Degrader | CDK2 **CG923308**	Breast cancer and multiple solid tumors					
Degrader | TYK2 / JAK1 **CG620953**	Inflammatory diseases					
Erythromycin derivative inhibitor **F528**	COPD					
DAC | Epigenetic Factor **Undisclosed Target**	Prostate, lung & bladder cancers					
DAC | Translation Factor **Undisclosed Target**	Blood cancers & multiple solid tumors					
Degrader | cAMP Regulator **Undisclosed Target**	Fibrotic diseases					

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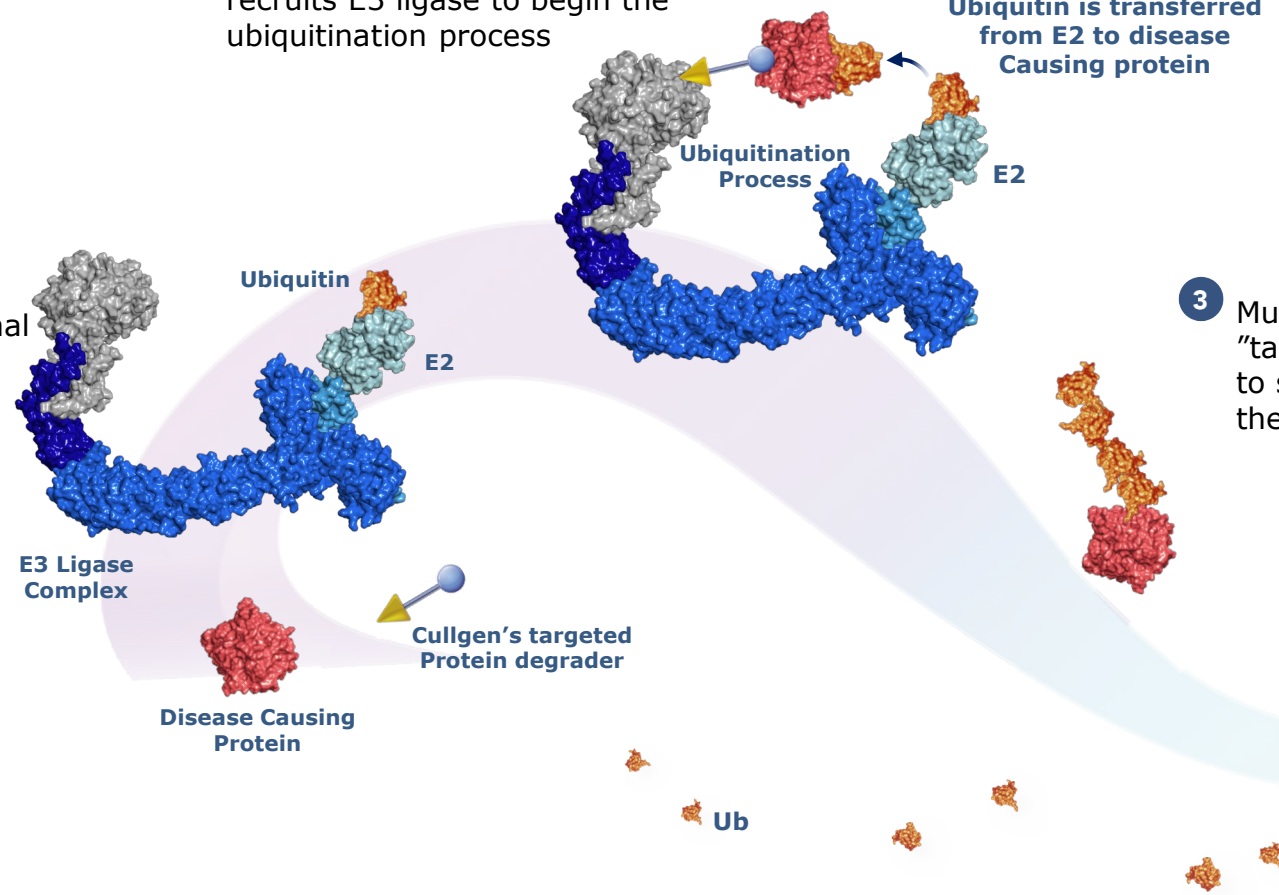
Inflammation / Fibrosis / Pain Cancer Degrader DAC

TPD and DAC Platform Technology to Revolutionize Drug Discovery

Hijacking the Ubiquitin Proteasome System to Target Undruggable Disease-causing Proteins

1 Targeted protein degraders are bi-functional molecules that bind to disease causing proteins and specific E3 ligases inside cells

2 Targeted protein degrader binds to disease causing protein and recruits E3 ligase to begin the ubiquitination process

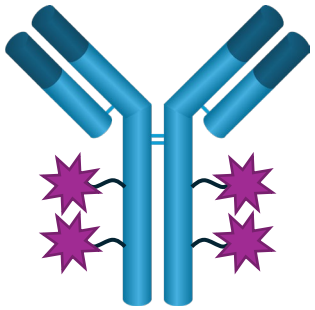


3 Multiple ubiquitin molecules "tag" disease causing protein to signal it for degradation by the UPS

4 Disease causing protein is degraded by the proteasome

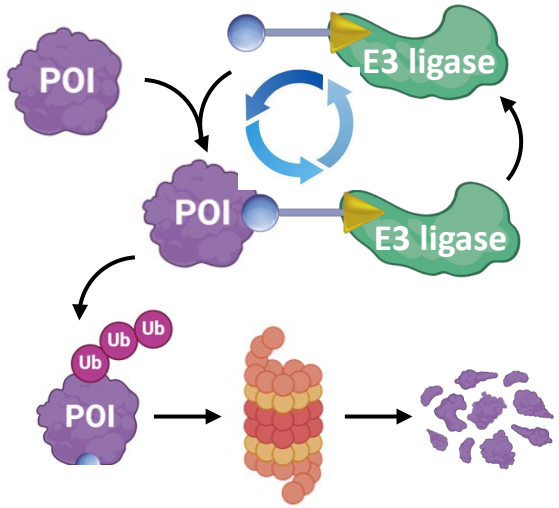
Degrader-Antibody Conjugates (DACs) Combine the Advantages of ADCs and TPD and Represent the Next-Generation of ADCs

ADC
(Antibody drug conjugate)



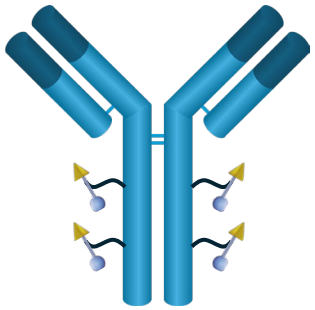
+

TPD
(Targeted protein degradation)



→

DAC
(Degrader antibody conjugate)



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.

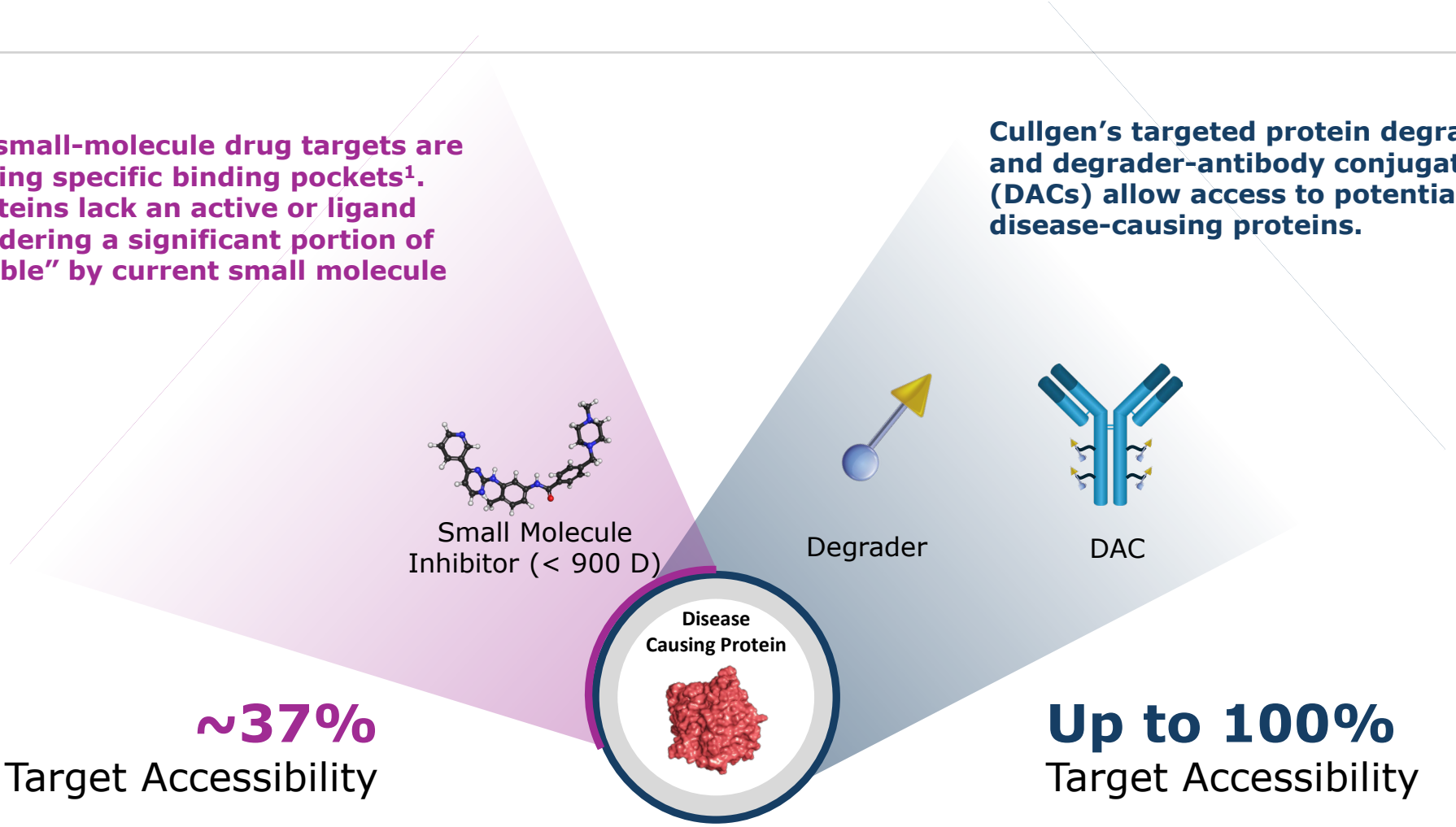
IMPROVED SAFETY

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

Targeted Protein Degraders and DACs Expand Druggable Disease Space

The majority of small-molecule drug targets are proteins containing specific binding pockets[1]. Most human proteins lack an active or ligand binding site, rendering a significant portion of them "undruggable" by current small molecule pharmacology.

Cullgen's targeted protein degraders and degrader-antibody conjugates (DACs) allow access to potentially all disease-causing proteins.



~37%
Target Accessibility

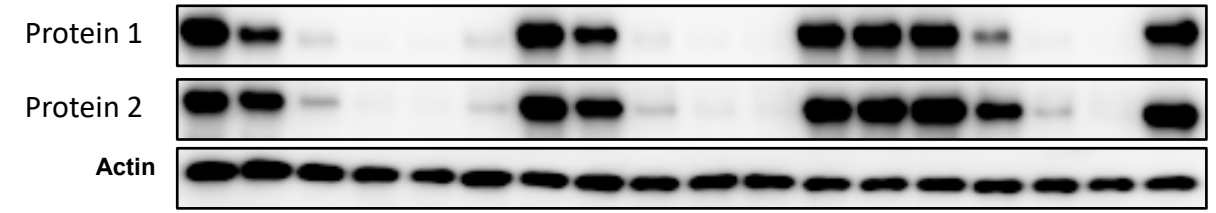
Up to 100%
Target Accessibility

1. Santos et al. (2017) *Nat Rev Drug Discov* PMID: 27910877

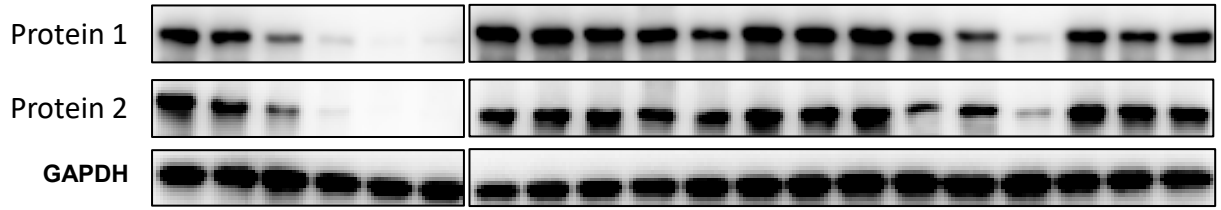
Prostate Cancer DAC Demonstrates Potent and Tumor-Associated Antigen (TAA)-dependent Target Degradation and Cell Killing

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

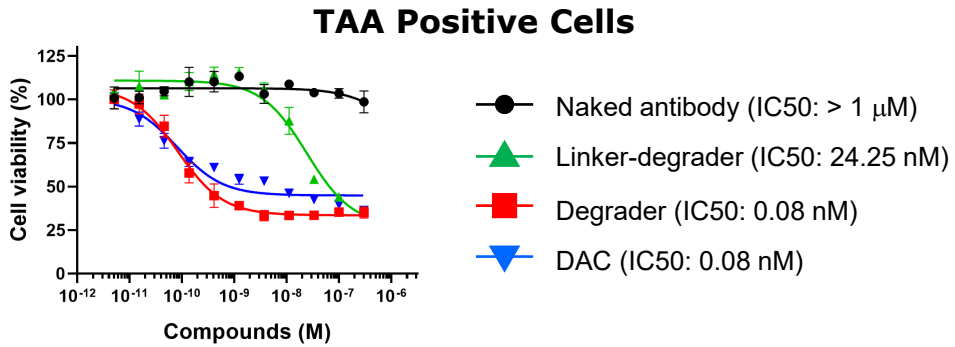
Cell Line	TAA Positive Cell Line																	
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

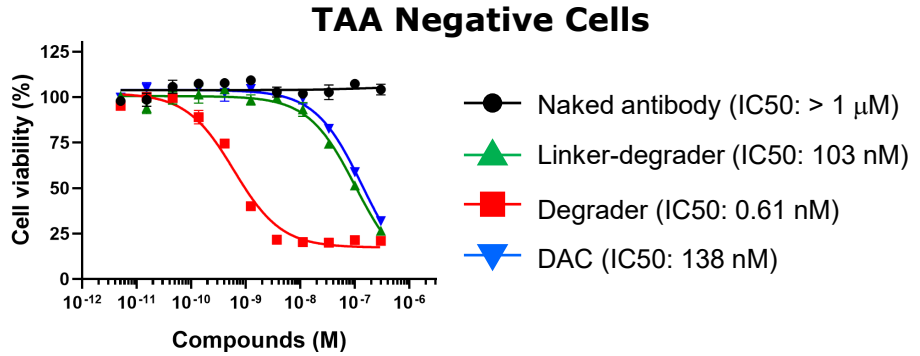


Cell Line	TAA Negative Cell Line																		
Compounds (nM)	DMSO	Degrader					DAC					DMSO	Linker-degrader					mAb		DMSO
		0.01	0.1	1	10	100	0.01	0.1	1	10	100		0.01	0.1	1	10	100	10	100	
Lane	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20



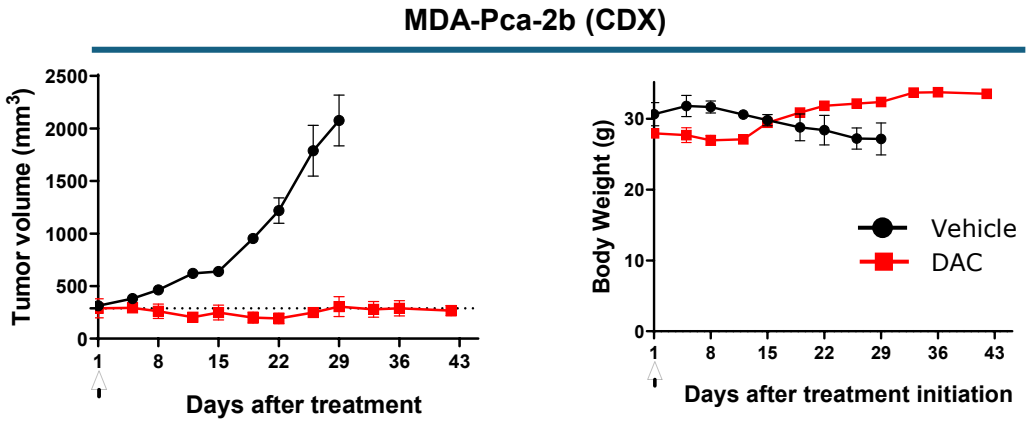
B. Cullgen prostate cancer DAC kills cancer cells in a TAA-dependent manner



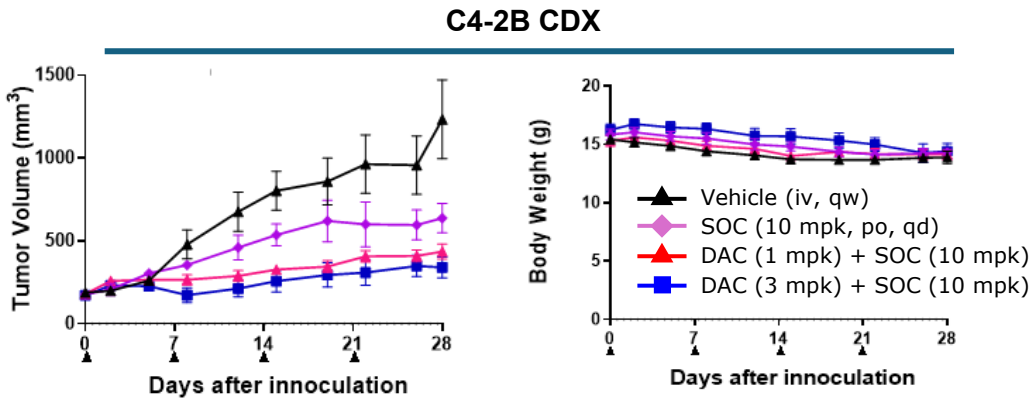


Cullgen's Prostate Cancer DAC Demonstrates Durable Tumor Growth Inhibition and Overcomes Resistance to Current Therapy

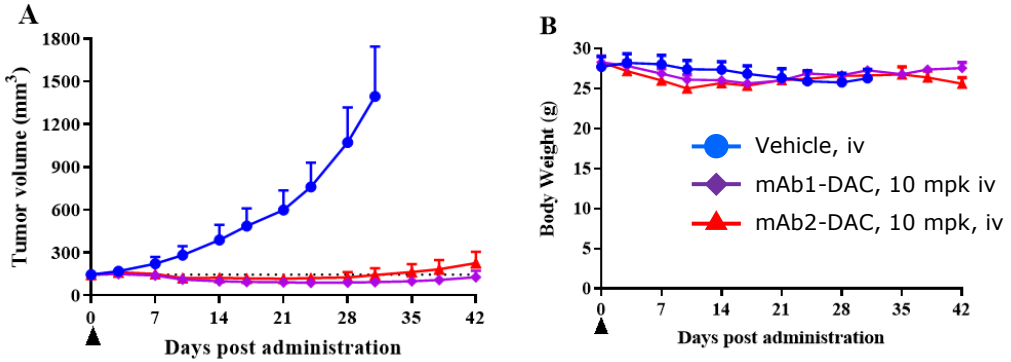
A. DAC demonstrates durable tumor growth inhibition *in vivo*



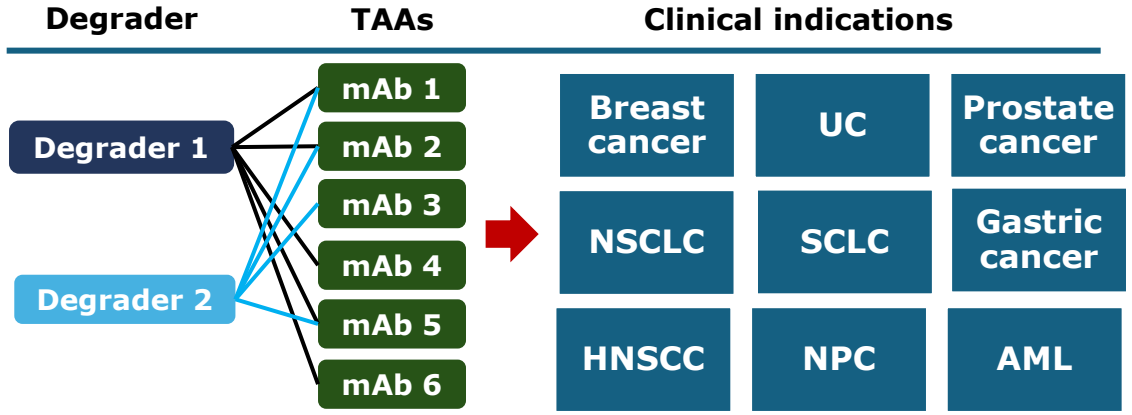
B. DAC synergizes with the standard-of-care (SOC) agent



C. DACs overcome therapy-resistance in CPRC PDX



D. Targeting different cancer indications by DAC



Leading Inflammation/Fibrosis Therapeutic Programs

Expanding F351's Global Market Potential
Tyk2 for Rheumatoid Diseases

F351 Overview

Product:	F351 - Hydronidone
Primary Indication	Liver fibrosis - Chronic Hepatitis B (CHB) / Metabolic dysfunction-Associated SteatoHepatitis (MASH)
Summary	A structural analogue of pirfenidone, chemically modified to reduce metabolism liabilities. Anti-fibrotic with TGF-β1 targeting mechanism.
Mechanism of Action	Inhibits HSC (hepatic stellate cell) activation via Smad7-mediated TGF-β degradation; inhibits p38γ kinase; reduces fibrosis-related gene expression.
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 anticipated to be filed with NMPA in 1H 2026.
Regulatory	Breakthrough Therapy designation from NMPA (March 2021) for hepatitis B-induced liver fibrosis by NMPA and CDE. U.S. IND for MASH filed, with anticipated Phase 2 start in 2026.
Opportunity	China has the largest burden of hepatitis B world-wide, with an estimated 79 – 86 million cases of chronic HBV infections[1].

1. https://pmc.ncbi.nlm.nih.gov/articles/PMC11806133/

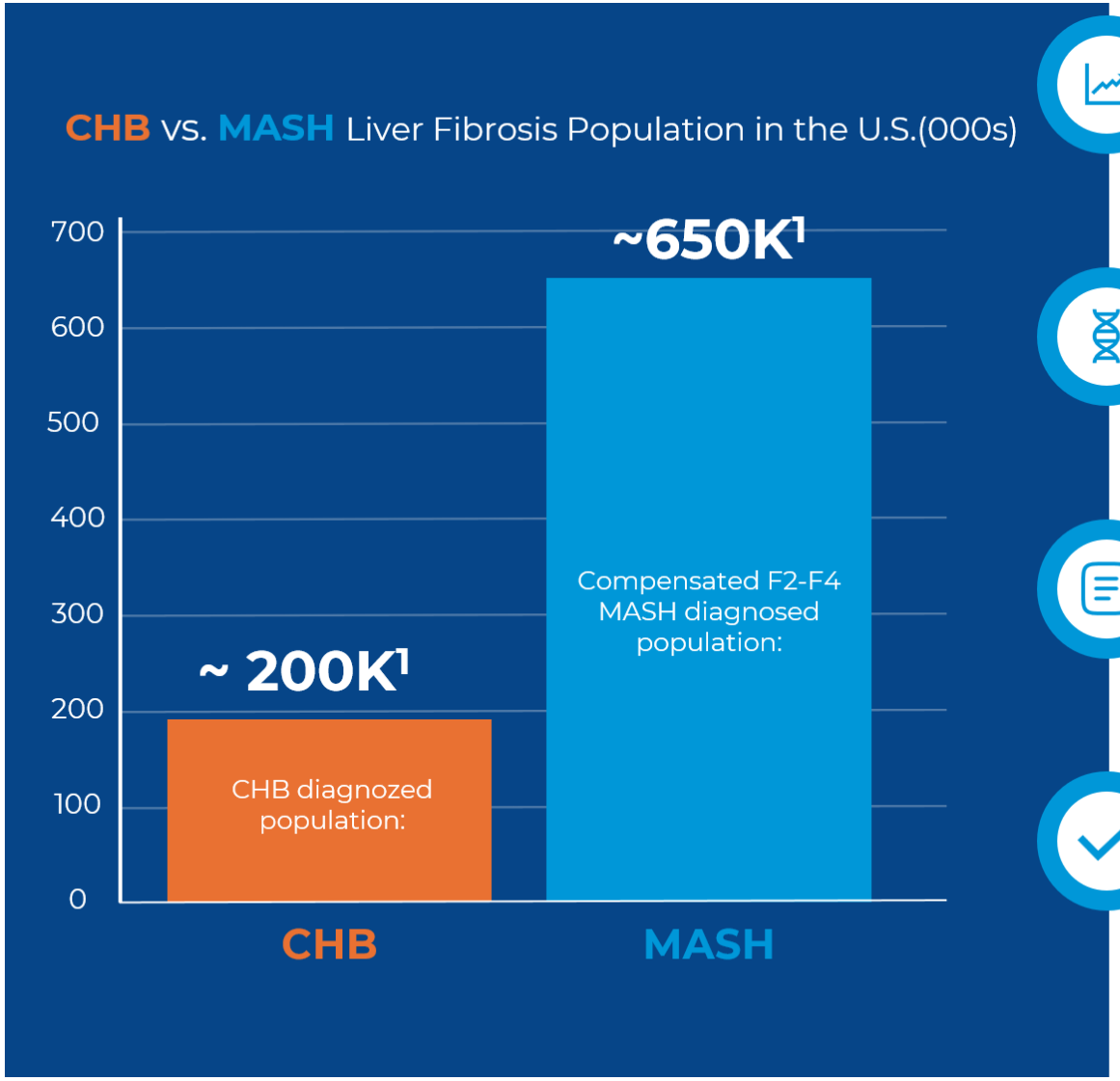
F351 Phase 3 Results Demonstrate New Global Potential in Liver Fibrosis and Cirrhosis

NDA expected to be filed with NMPA in 1H 2026

Primary Endpoint Met with High Statistical Significance	Key Secondary Endpoint Reduction in Liver Inflammation	Favorable Safety Profile
≥1-stage fibrosis regression at Week 52: • Hydronidone: 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	**≥1-grade inflammation improvement without fibrosis progression at Week 52:** • Hydronidone: 49.57% (n=123) vs. • Placebo: 34.82% (n=124) • **Delta: 14.75%** • **P = 0.0246** • Reinforces anti-inflammatory activity	**Serious Adverse Events** • Hydronidrone: 4.88% (6/123) vs • Placebo: 6.45% (8/124) **No discontinuations, dose interruption or dose reduction due to adverse events**

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

Exploring F351's Potential in CHB-related and/or MASH-related Compensated Liver Fibrosis and Cirrhosis in the U.S.



Market Opportunity

In the U.S., the MASH fibrosis market is approximately **7.2 times larger** than the CHB fibrosis market.

Clinical Rationale

Hydronidone modulates **TGF-β / p38γ / Smad7** signaling pathway — directly targeting fibrosis progression and **offering a differentiated approach from metabolic agents**.

Regulatory Pathway

Hydronidone's CHB data **helps to reduce risks in MASH development** and potentially supports *accelerated regulatory review and fast track*.

Competitive Differentiation

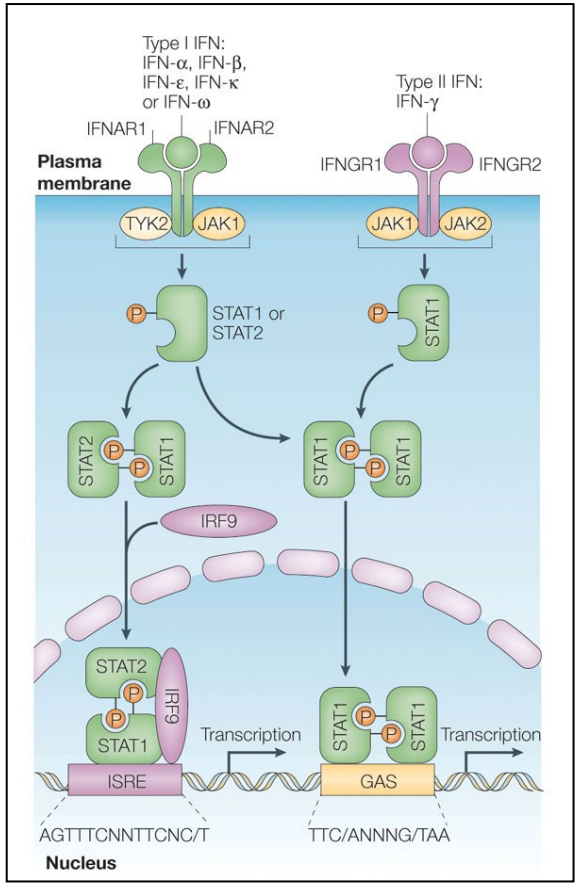
Hydronidone's unique anti-fibrotic approach positions it as a **complementary therapy** — not a competitor — to metabolic agents like THR-β, GLP-1s, and FGF21.

1. Based on analysis of third-party epidemiological research, published academic studies, and internal modeling.

Note: Market projections based on epidemiological research report prepared by L.E.K. Consulting for Gyre on 12-18-2025

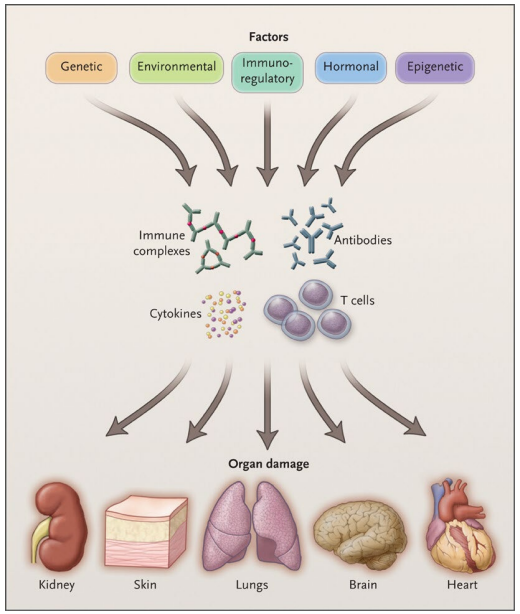
Dual Targeting of TYK2 and JAK1 for Autoimmune Diseases, Focus on Systemic Lupus Erythematosus and Rheumatoid Arthritis

A. TYK2/JAK - STAT signaling



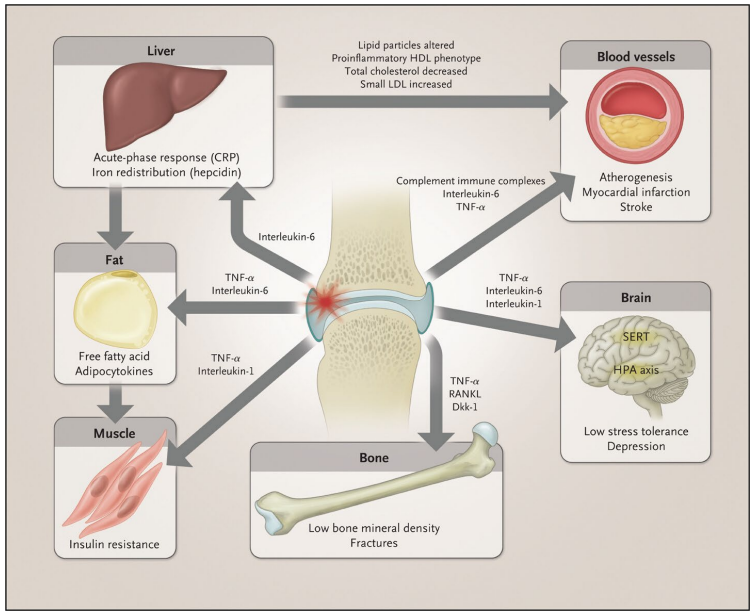
Platanias, LC. (2005) *Nat Rev Immunol* PMID:15864272

B. SLE Mechanism



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

C. RA Mechanism



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

Significant Opportunity

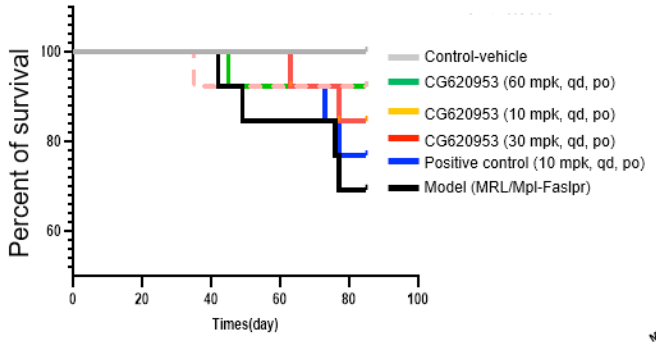
- 125,000,000 psoriasis patients worldwide[1]
- 18,000,000 rheumatoid arthritis patients worldwide[2]
- ~204,000 lupus patients in the US in 2018[3]

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

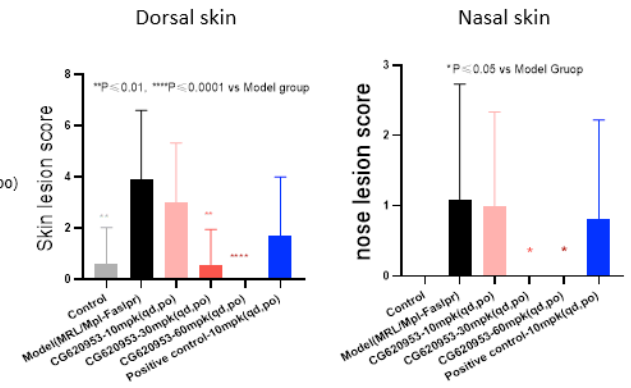
TYK2-JAK1 Dual Degrader Demonstrates Superior Efficacy in Preclinical Models for Lupus And Rheumatoid Arthritis

CG620953 demonstrates superior efficacy vs Deucravacitinib

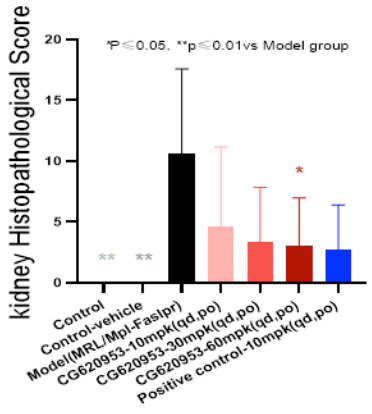
Survival Rate



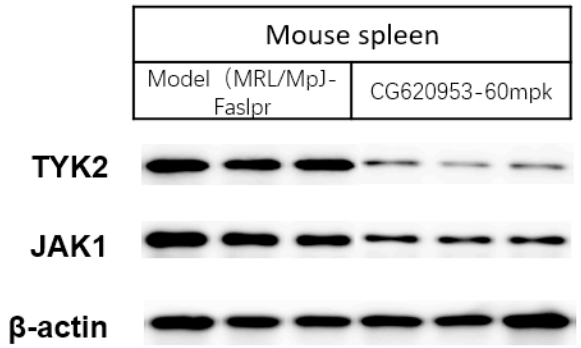
Skin lesion



Kidney Histopathological Score

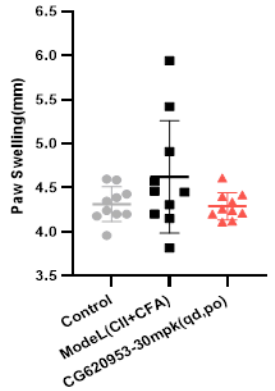


TYK2/JAK1 degradation in immune organ

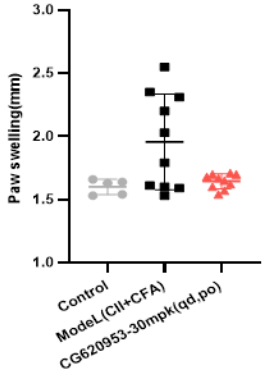


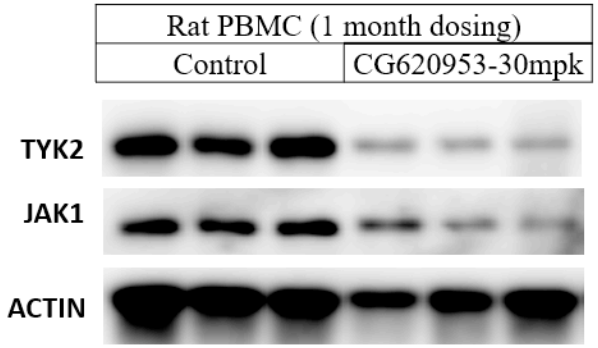
CG620953 reverses CII-Induced joint and paw swelling

CII-induced RA in SD Rat Prophylactic



CII-induced RA in DBA1 mice Therapeutic

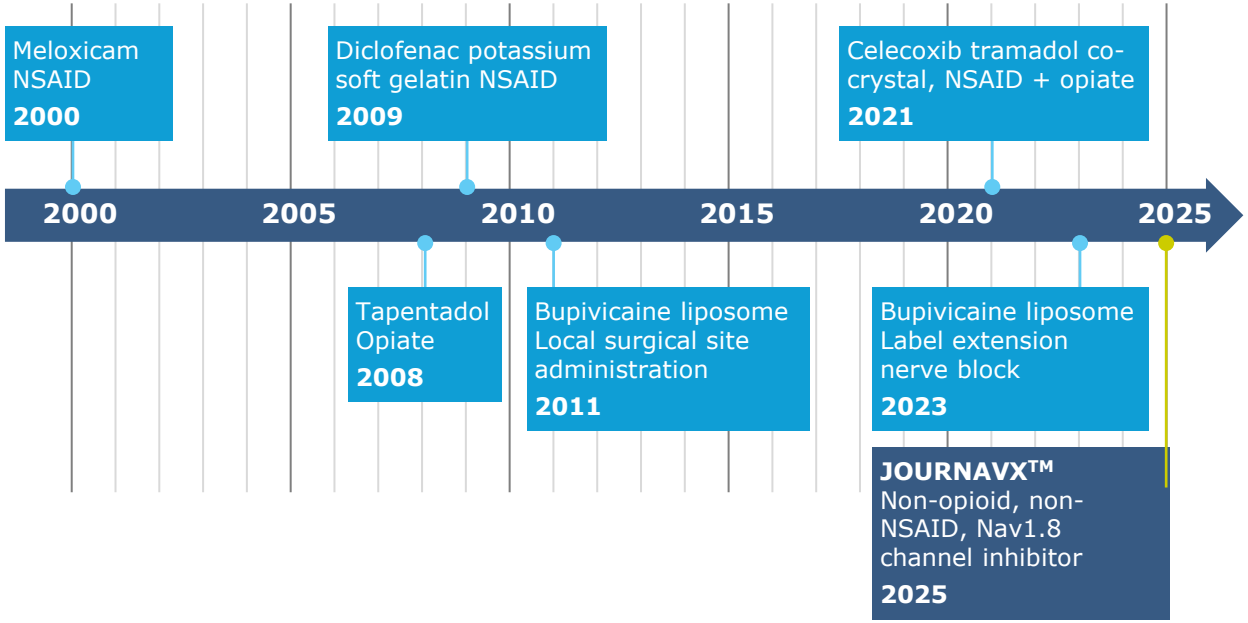




Pain Therapeutic Program
CG001419 for Acute and Chronic Pain

Opioid Crisis Created an Urgent Unmet Medical Need and Significant Market for Pain Management

A. Only one non-NSAID non-opiate analgesic has been approved in the last 25 years for acute pain

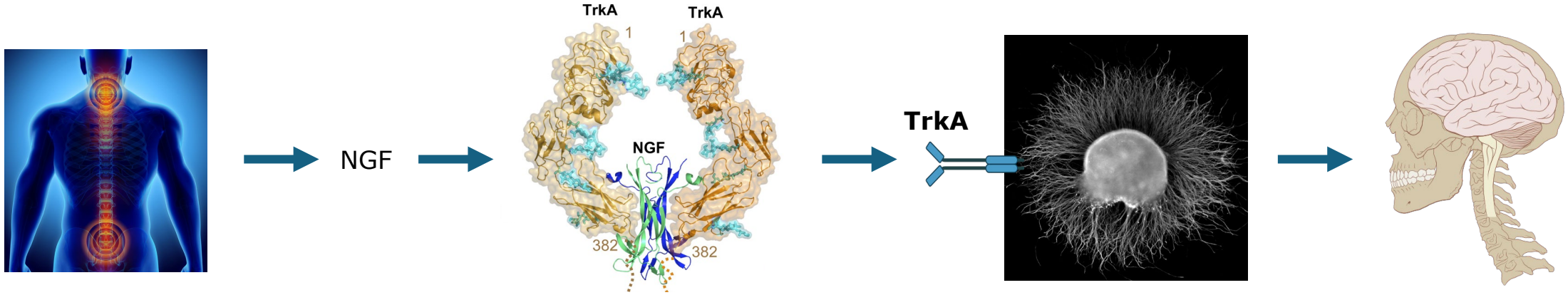


B. Chronic and Acute Pain Management is a multi-billion-dollar market

Source: https://www.precedenceresearch.com/pain-management-drugs-market

The Role for TRKA in Nociception and Analgesia Has Been Validated in Humans

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

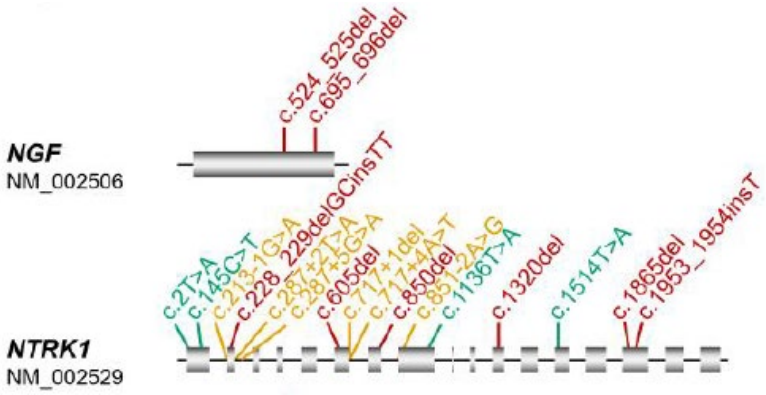


B. TRKA mutations cause congenital insensitivity to pain and anhidrosis (CIPA)

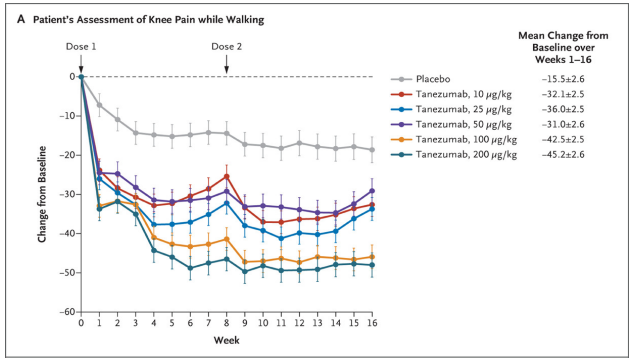
Sequencing of a cohort 78 CIPA patients

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



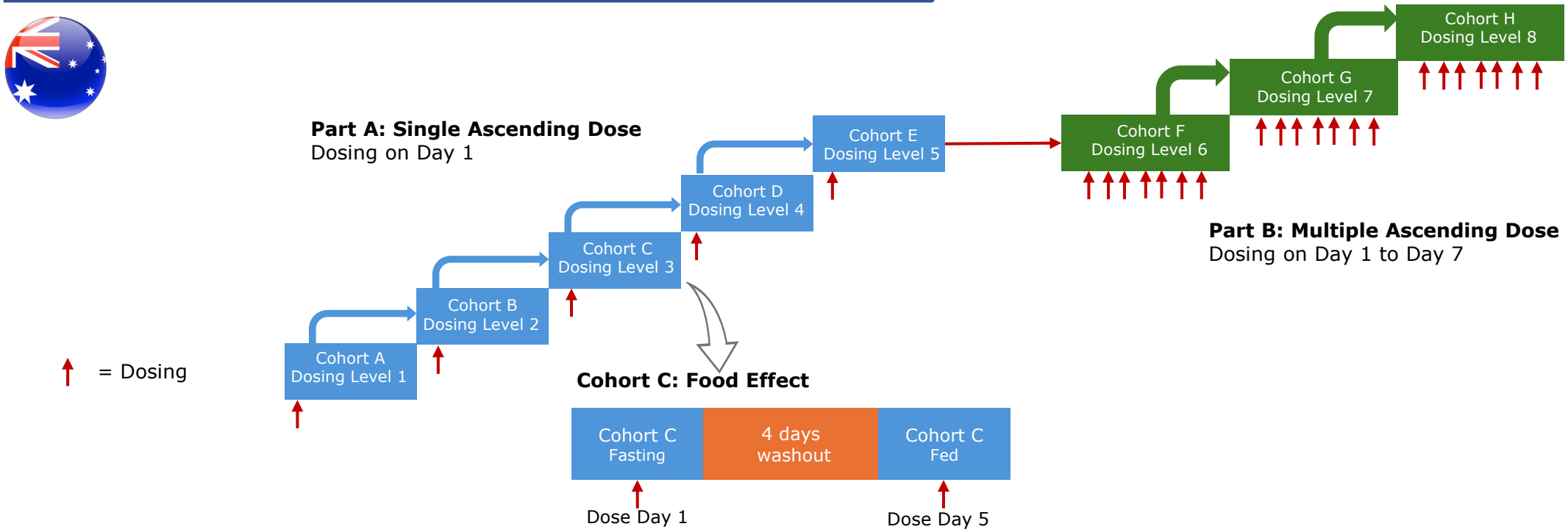
C. Blocking NGF reduces osteoarthritis pain



Lane et al. (2010) *NEJM* PMID: 20942668

Clinical Development of Cullgen's CG001419 for Acute Post-Operative Pain

Phase 1a Dose Escalation, IND Enabling



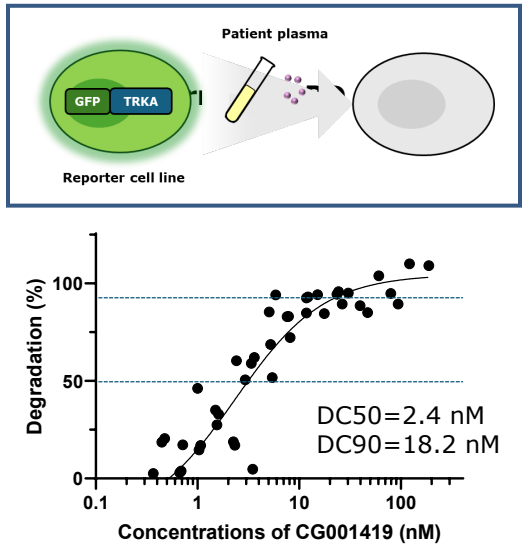
Key Findings from Phase 1 Study

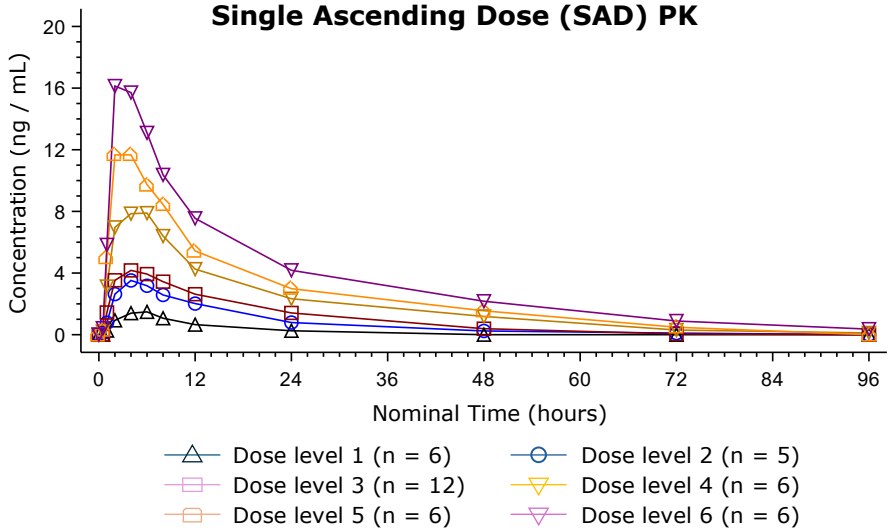
- All dose regimens were well tolerated
- Plasma levels are consistent with DC50 and DC90 levels from preclinical studies
- Results provide sufficient guidance for appropriate dosing levels to evaluate in the phase 2 bunionectomy study

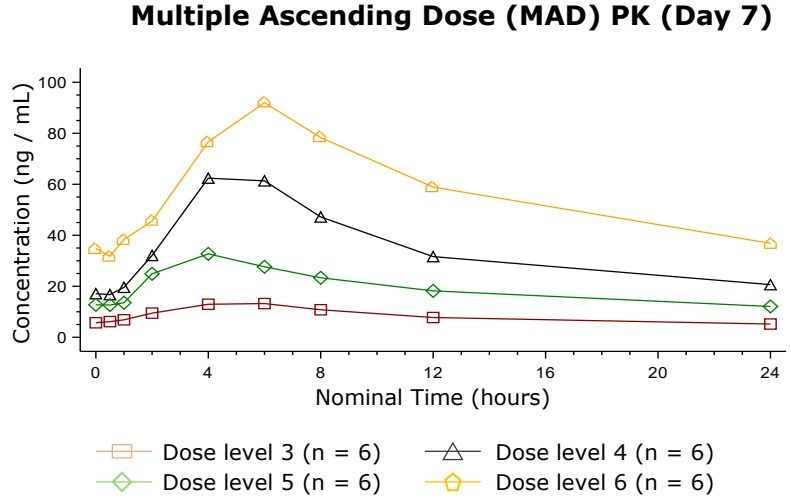
Summary of Phase 1 PD, PK and Safety Study of CG001419

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







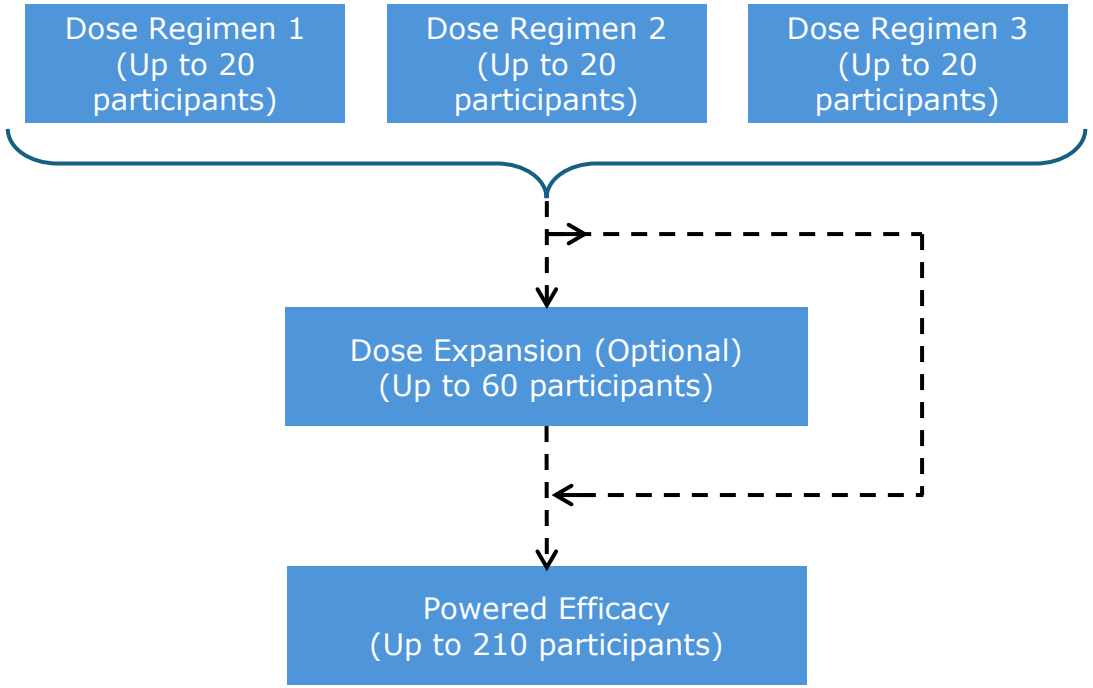
Clinical Development of Cullgen's CG001419 for Acute Post-Operative Pain

Phase 1b/2a Acute Pain Bunionectomy Study*

Part A: Dose regimen evaluation to select dose for Part C

Part B (Optional): Dose expansion to inform power calculations and size for Part C

Part C: Powered efficacy portion of study



Primary Endpoint:
SPI48 (time-weighted sum of pain-intensity over 48 hours since dosing will begin pre-op) in comparison of CG001419 with placebo

Secondary Endpoints:
Reduction in NPRS score at rest at 48 hours: Patients with ≥ 30% reduction, patients with ≥ 50% reduction and patients with ≥ 70% reduction

PK parameter estimates of CG001419 and its metabolites

Safety and tolerability based upon AEs, changes from baseline in clinically significant laboratory endpoints, vital signs and ECGs

Additional Endpoints:
Percentage of subjects using rescue medication, and total rescue medication usage, 0-48 hours after the first dose of study drug

Percentage of subjects using opiates, and total opiate equivalents (OEs) used, 14 days post-discharge

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* Subject to regulatory alignment

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

	Opioids	NSAIDs	Cebranopadol	Journavx (Suzetrigine, VX-548)	VX-993	LTG-001	STC-004	CG001419
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

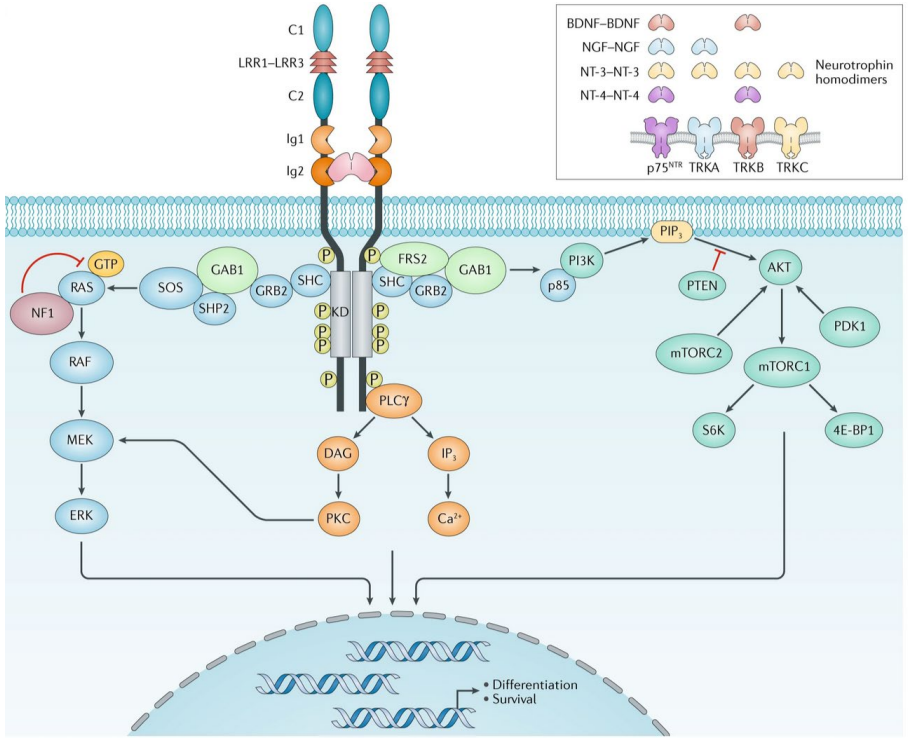
Cancer Programs

CG001419: for pan-TRK Cancers

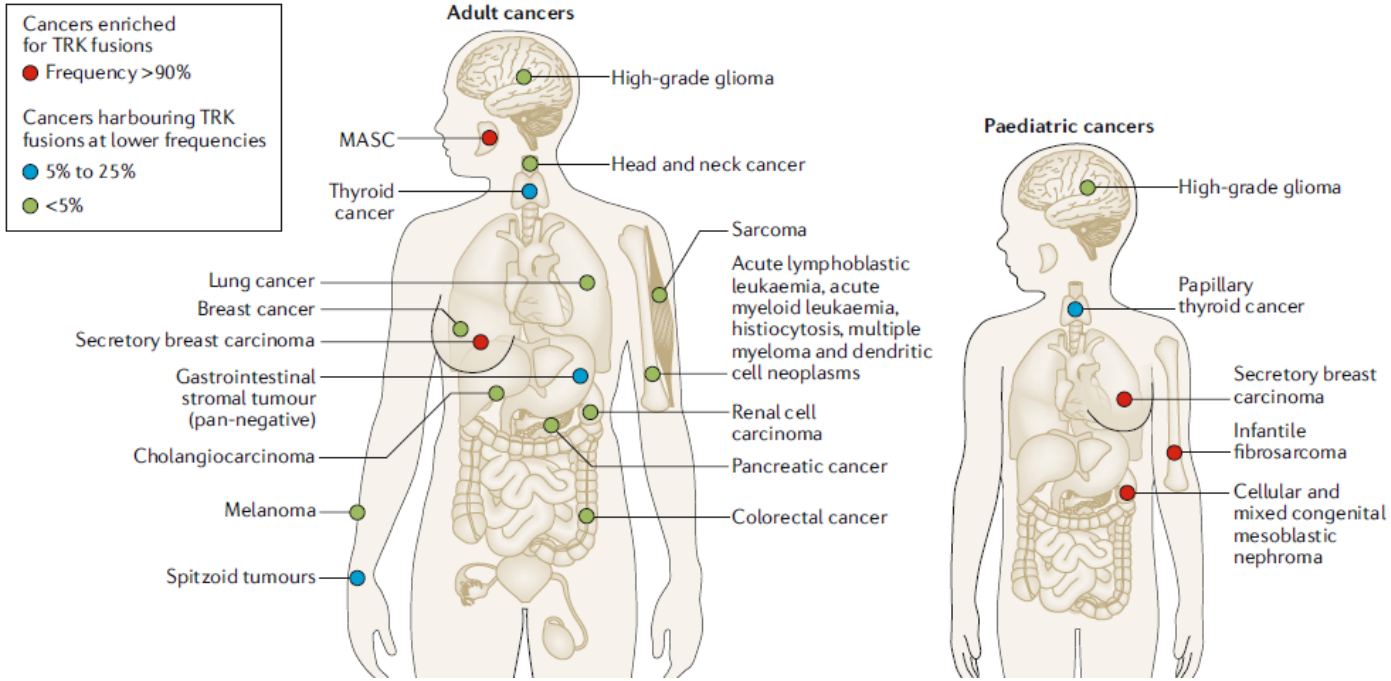
CG009301: GSPT1 Degrader for AML and MYC+ Cancers

Tropomyosin Receptor Kinases (TRKs) Are Receptors for Neurotrophins Including NGF

A. TRK signaling pathways

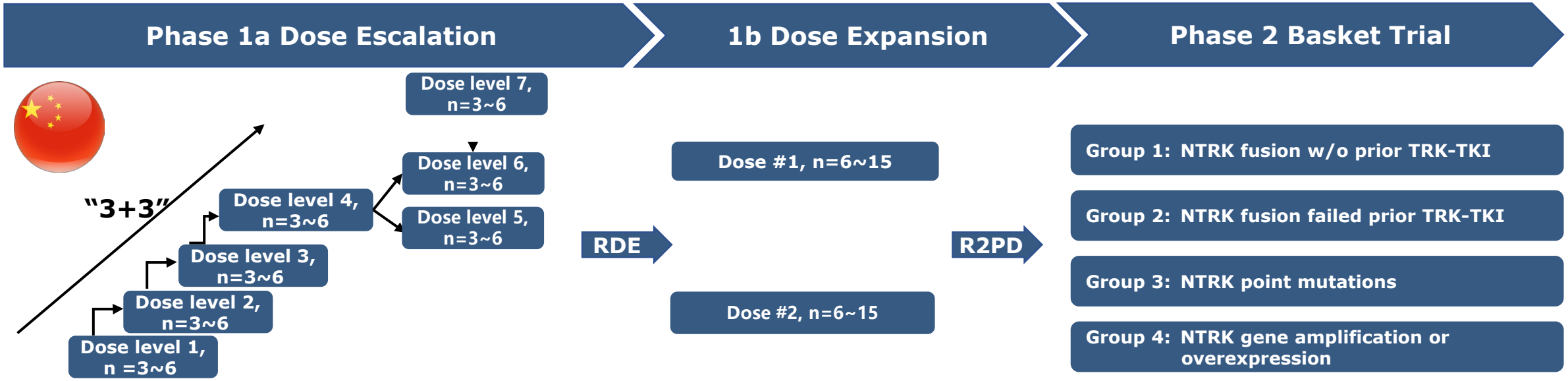


B. Activation of TRK in multiple solid tumors



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

Clinical Development of CG001419 for Cancer

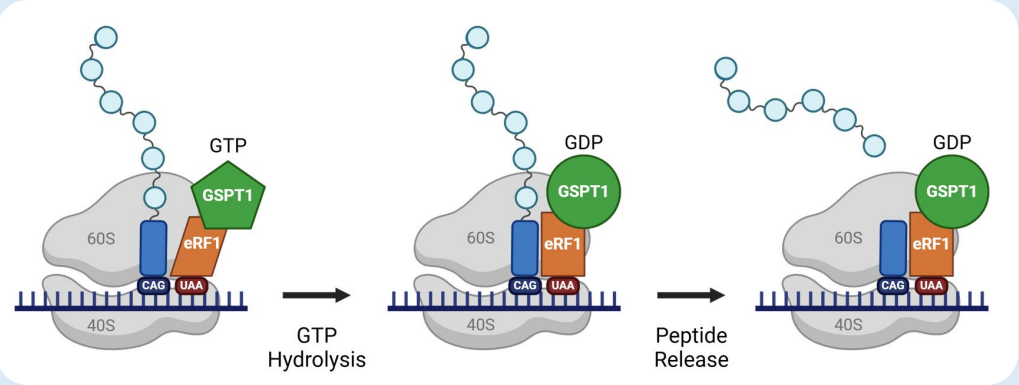


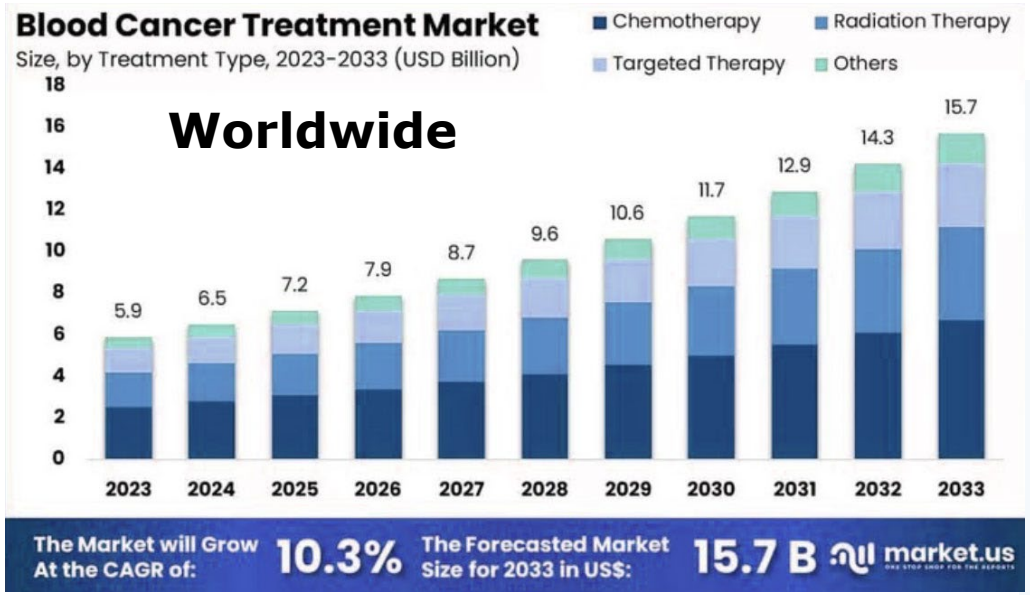
Future Data Points

- Data from first 18 patients demonstrated no observed DLTs, treatment-related SAEs or grade ≥3 treatment related AEs.
- Enrollment to dose expansion portions is anticipated to begin Q1 2026.

CG009301 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.



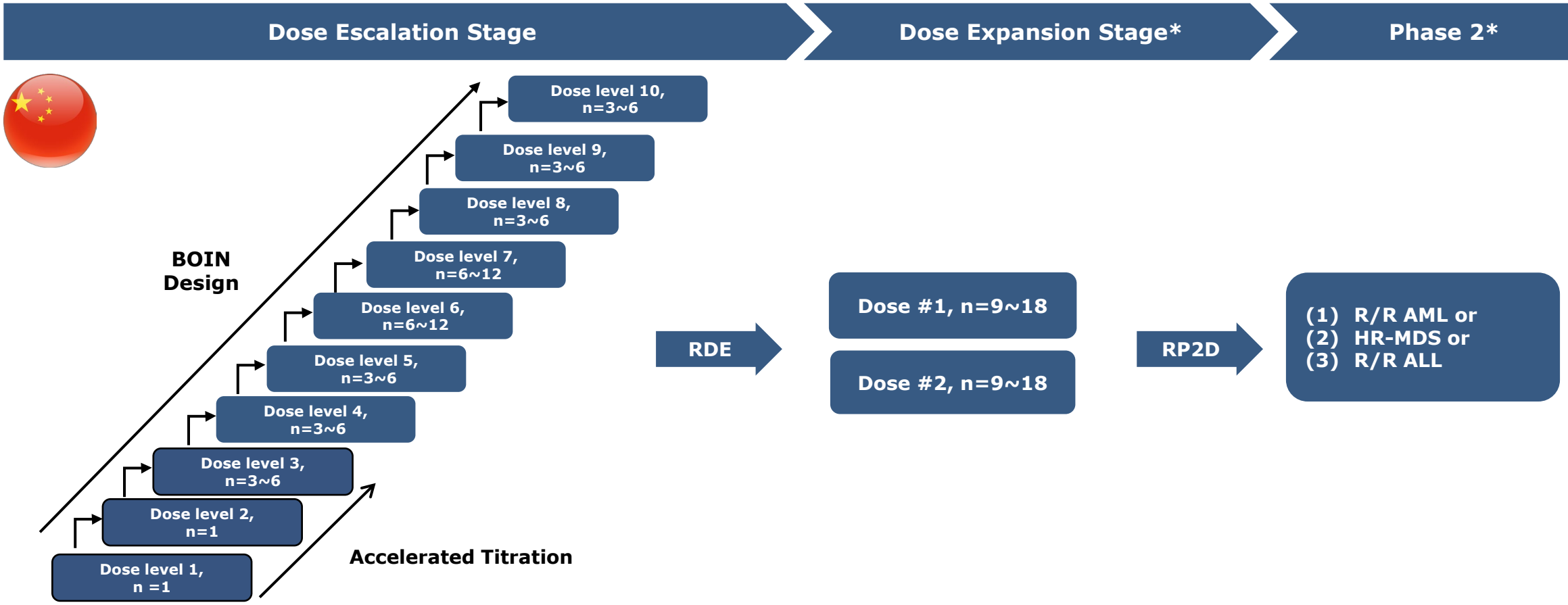


US Patient Population

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 all (2022) *Clin Lymphoa Myelom Leuk*, PMID: 34544674

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



- Study commenced in April 2025 and dose escalation stage currently underway.
- Anticipate enrollment of approximately 30 – 45 patients.

* Subject to regulatory alignment

Anticipated Upcoming Catalysts

2026

- IND submission of CG001419 for a Phase II trial for the acute pain in the US — **COMPLETED**
- Initiation of Phase II trial of CG001419 in the US — **Q2-2026**
- NDA submission of Hydronidone (F351) for fibrosis to NMPA — **1H-2026**
- Completion of the Phase I trial of F230 for PAH in China — **Q4-2026**

2027

- IND submission of Phase I trial of CG620953 for inflammatory diseases in China — **Q1-2027**
- IND submission of Phase I trial of CG923308 for cancer in the U.S. and China — **Q1-2027**
- IND submission of Phase I trial of F528 COPD in the U.S. and China — **Q1-2027**
- Completion of the Phase I trial of CG009301 for AML in China — **2H-2027**
- Completion of the Phase I trial of CG001419 for cancer in China — **Q4-2027**

The milestones set forth above are subject to adjustment with respect to F351/MASH and other clinical development milestones.

Investment Summary: Gyre's Acquisition of Cullgen

1. Robust and balanced therapeutic pipeline including assets from discovery to development, with established manufacturing and commercialization operations
2. Utilization of highly efficient and cost-effective drug discovery 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 with extensive international business operations experience

Future Board of Combined Company

Gordon Carmichael, PHD
Professor of Genetics and Genome Sciences at the University of Connecticut Health Center. Published 110 papers. on kinase signaling in oncogenesis, transcriptional and post-transcriptional gene regulation, long noncoding RNAs, stem cell biology, innate immunity, RNA modifications

David Epstein, PhD
Founder of PairX and Black Diamond (NASDAQ BDTX). Vice Dean of Duke-NUS Medical School at Singapore. CSO of OSI Pharmaceuticals. Developed Izervay in Archemix

Ying Luo, PhD, CEO
>30 years of biotech experience. PhD from U. Connecticut. Management at Aviron, Clontech, and Rigel. Founded Shanghai Genomics and led GNI IPO (TSE 2160). >40 research articles and >20 patents.

Rodney Nussbaum, CPA
Managing director of Atago Advisory. Former Senior Partner of E&Y Japan and Asia Pacific. Former partner of Arthur Anderson.

Renate Parry, PhD
25 years of research experience in global pharma. Developed 3 novel drugs for oncology and fibrosis into clinical development

Dan Weng, MD, MA
CEO of Medelis. Former CEO of EPS International. Held executive positions at MedPace, ICON, PharmaNet and Quintiles

Ping Zhang, MBA
Executive Chairman
20 years experience in healthcare investment with senior postings in Japan and China. Managing director of String Capital. Executive director of GNI Group Ltd.

Future Leadership Team of Combined Company

Ying Luo, PhD
President and CEO

>30 years of biotech experience. PhD from U. CT. President of GNI Group. Founded Shanghai Genomics and led GNI IPO (TSE 2160). Responsible for 6 IND approvals and 1 class 1 drug approval (Etuary) by China FDA. Author of >37 research articles and >20 patents.

Ping Zhang, MBA
Executive Chairman

20 years experience in healthcare investment with senior postings in Japan and China. Managing director of String Capital. Executive director of GNI Group Ltd.

Yue Xiong, PhD
CSO

William R. Kenan Distinguished Professor, UNC Chapel Hill. Pew Scholar. AACR Gertrude B. Elion Cancer Research Award. >220 papers. Discovery of Cyclin D, CKD4, p21, and ROC1/2.

Thomas Eastling
CFO

>25 years experience in global health care, financial services and investment banking, with senior postings in New York, London, Tokyo and China. Previously CFO of GNI Group Ltd.

AVIRON · GNI · rigel · String Capital · CSH Cold Spring Harbor Laboratory · THE UNIVERSITY of NORTH CAROLINA at CHAPEL HILL · SMBC日興証券

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Thank You