UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of report (Date of earliest event reported): **September 10, 2025**

Gyre Therapeutics, Inc.

(Exact name of registrant as specified in its charter)

Delaware	**000-51173**	**56-2020050**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

12770 High Bluff Drive **Suite 150** **San Diego, CA**	**92130**
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: **(858) 567-7770**
N/A
(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock	GYRE	The Nasdaq Capital Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01 Other Events.

On September 10, 2025, Gyre Therapeutics, Inc. (the "Company") made available an updated corporate presentation on the Company's website. A copy of the corporate presentation is filed herewith as Exhibit 99.1 and incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits

(d) *Exhibits*.

EXHIBIT INDEX

Exhibit No.	Description
99.1	Corporate Presentation, dated September 2025
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

GYRE THERAPEUTICS, INC.

Date: **September 10, 2025**

By: /s/ Ping Zhang
Name: Ping Zhang
Title: Executive Chairman and Interim Chief Executive Officer

Exhibit 99.1

Gyre THERAPEUTICS

Developing **Anti-Fibrotic Therapeutics** for Chronic Organ Diseases

H.C. Wainwright Global Investment Conference
September 2025

Forward Looking Statements

This presentation contains "forward-looking statements" within the meaning of the federal securities laws 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 statements concerning: 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 or ongoing preclinical studies and clinical trials; Gyre's research and development efforts; timing of expected clinical readouts, including timing of the filing of an NDA with the PRC's NMPA for Hydronidone for the treatment of CHB-related liver fibrosis, submission of U.S. IND filing for Hydronidone for advanced fibrosis and trial initiation pending regulatory review, timing of completion of Gyre's Phase 2 clinical trial in the PRC of F573 for ALF/ACLF, IND submission of F528 in COPD, the expectations regarding commercial revenues from the sales of Etorel and Contiva maleate tablets, management's plans and objectives 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," "design," "estimate," "expect," "forecast," "intend," "may," "might," "plan," "potential," "predict," "objective," "should," "strategy," "will," "would," or the negative of these terms, and similar expressions intended to identify forward-looking statements. 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, 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, and competition from other therapies or products, as well as those described in "Risk Factors" and "Management's Discussion and Analysis of Financial Condition" in Gyre's Annual Report on Form 10-K for the year ended December 31, 2024 filed on March 17, 2025 with the Securities and Exchange Commission (the "SEC") and elsewhere in such filing and in Gyre's other 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 expected consequences or affect Gyre or its business in the ways 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 made in this presentation 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. Gyre obtained the data used throughout this presentation from its own internal estimates and research, as well as from research, surveys and studies conducted by third parties. Internal estimates are derived from publicly available information and Gyre's own internal research and experience, and are based on assumptions made by management based on such data and its knowledge, which it believes to be reasonable. In addition, while Gyre believes the data included in this presentation is reliable and based on reasonable assumptions, Gyre has not independently verified any third-party information, and all such data involve risks and uncertainties and are subject to change based on various factors.

This presentation concerns a discussion of investigational drugs that are under preclinical and/or clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration. They are currently limited by Federal law to investigational use, and no representations are made as to their safety or effectiveness for the purposes for which they are being investigated.

Gyre Therapeutics (Nasdaq: GYRE): 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.

Innovative fibrosis focused development pipeline with sentinel indications in liver and lung

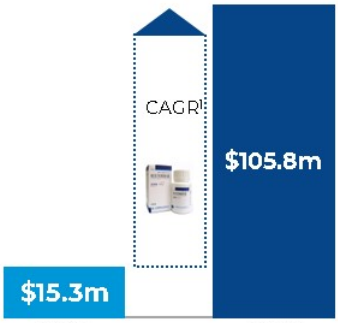
Gyre THERAPEUTICS

CANDIDATE	INDICATION	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKETED	RIGHTS	TRIAL
F351 (hydronidone)	Advanced Liver Fibrosis	IND preparation in progress ★					Global	🇺🇸
	Chronic Hepatitis B (CHB) Liver Fibrosis							First 🇨🇳
F573	Acute Liver Failure / Acute-on-Chronic Liver Failure							
F230	Pulmonary Arterial Hypertension (PAH)							
F528	Chronic Obstructive Pulmonary Disease (COPD)							
ETUARY (pirfenidone)	Idiopathic Pulmonary Fibrosis (IPF)						China	First 🇨🇳
	Radiation-induced lung injury (RILI) with or without immune-related pneumonitis (CIP)	4Q 2025: Start Phase 2/3 Trial		(Adaptive Approach)				
	Pneumoconiosis (PD)	4Q 2025: Complete enrollment						
	Diabetic Kidney Disease (DKD)							

Strategic moves to strengthen our pirfenidone franchise and prepare for potential future Hydronidone launch

Gyre THERAPEUTICS

ETUARY® (pirfenidone)[1]



Etorel - Launched in 2025

Contiva – Launched in 2025

Expanding Our IPF Market:

Gyre's **ETUARY** has **dominated China's IPF market.** Through the strategic acquisition of a generic Etorel (nintedanib), we further strengthen our leadership by offering physicians a full spectrum of IPF treatments.

Securing a Foothold in Liver Disease

We acquired rights to a generic **Contiva (avatrombopag)** as a strategic entry point into the liver physician network—paving the way for the future **launch of Hydronidone.**

ETUARY was approved in 2011 before the Reference Listed Drug (RLD) requirement. Without RLD, generic competitors can not conduct the required bioequivalence (BE) studies. This creates a market exclusivity beyond patent protection.

Financial data inclusive of proforma data prior to GNI Group and Catalyst Science merger for comparison purposes only.

Note: 2017 sales number is audited (China) and 2024 is audited (U.S. GAAP). See Note 2 above for further clarification.

Phase 3 CHB-associated liver fibrosis - recently announced positive topline results

1

Primary Endpoint Met with High Statistical Significance

≥1-stage fibrosis regression at **Week 52**: Hydronidone: **52.85%** vs. Placebo: **29.84%** **(P = 0.0002;** ITT[1] analysis with central blinded pathology review)

2

Key Secondary Endpoint Achieved

≥1-grade inflammation improvement without fibrosis progression at Week 52: Hydronidone: **49.57%** vs. Placebo: **34.82%** (P = 0.0246)

3

Favorable Safety & Tolerability Profile

- Serious Adverse Events: **4.88%** (6/123, Hydronidone) vs. **6.45%** (8/124, Placebo)
- **No discontinuations** due to adverse events

4

Clinical and Regulatory Pathways

- Breakthrough Therapy Designation (China NMPA[2], 2021), potentially **first-in-class approval**
- **New Drug Application** (NDA) to **NMPA** expected in **Q3 2025,** with accelerated approval to be sought
- **U.S. IND** filing for **advanced fibrosis** expected in **2025**; trial initiation planned pending regulatory review

ITT = Intent-To-Treat. 2. NMPA = National Medical Products Administration of China

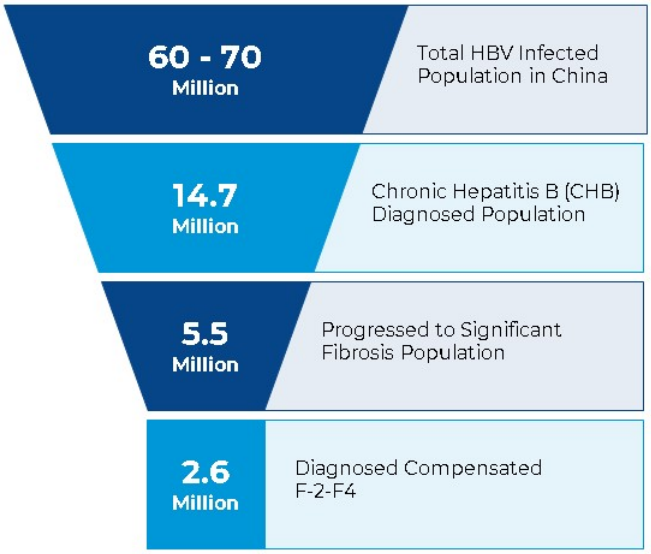
Phase 3 Safety Profile

Safety Event	Hydronidone (N=123)	Placebo (N=124)
Any TEAE	98 (79.67%)	103 (83.06%)
Grade 1 AES	27.64%	33.06%
Grade 2 AES	43.90%	43.55%
Grade ≥3 AES	8.13%	6.45%
Drug-related AEs (ADRs)	32.52%	33.87%
Grade ≥3 ADRs	1.63%	1.61%
Discontinuation due to AE	0	0
Temporary interruption due to AE	0	0.81%
Dose reduction due to AE	0	0
Any SAE	6 (4.88%)	8 (6.45%)
Due to Investigational Drug:		
Possibly unrelated	2	3
Unrelated	4	5
Death	0	0

All SAEs were assessed to be **unrelated to Hydronidone**

No discontinuations due to SAEs across either treatment arm.

Hydronidone targets CHB fibrosis -- a high-need and untapped market in China



The market for **CHB-associated liver fibrosis** is significantly **unmet.**

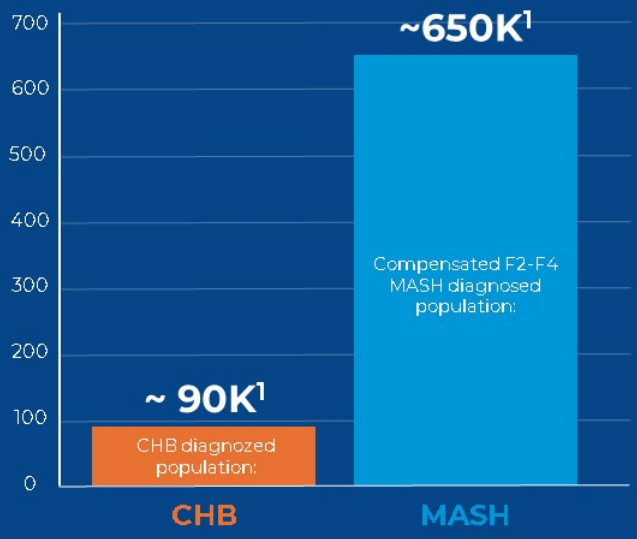
Current standard of treatment, e.g. entecavir, tenofovir, focuses on only **reducing liver inflamation**.

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.

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

Expanding Hydronidone's potential: from CHB fibrosis in China to MASH 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

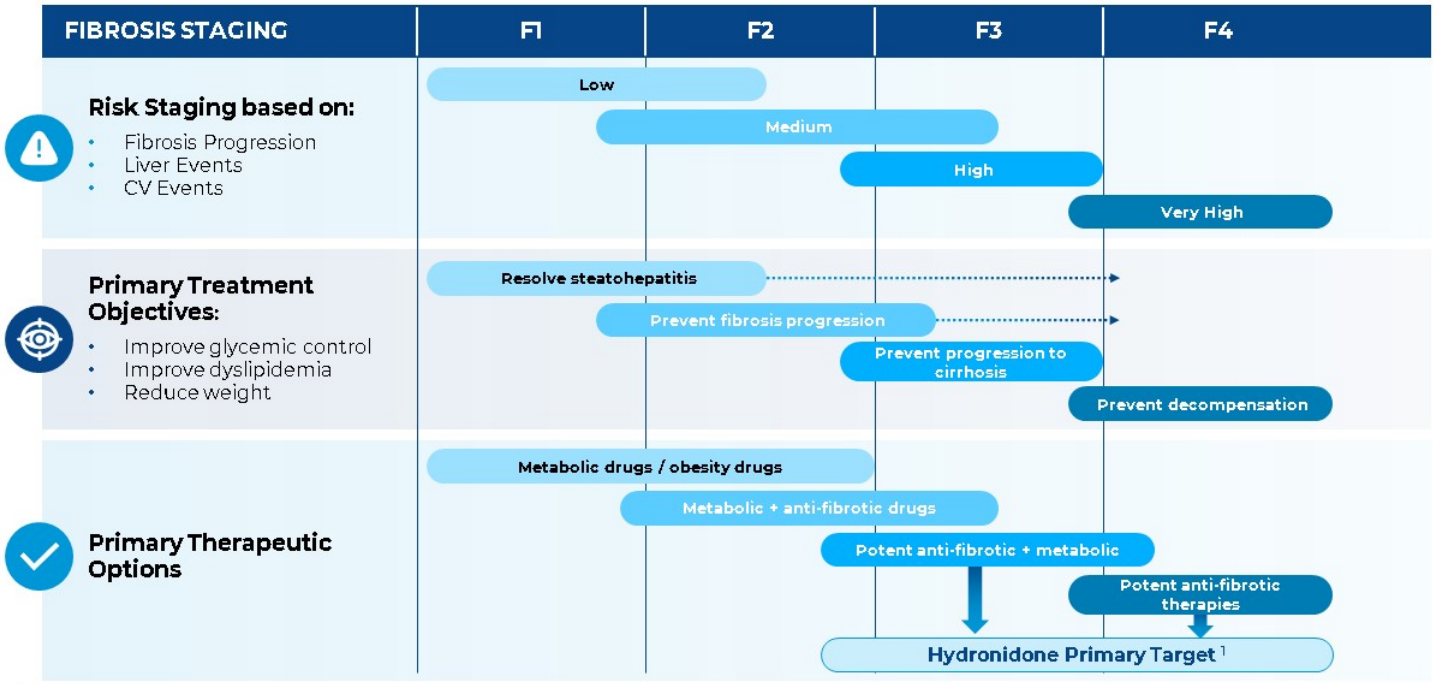
Hydronidone's CHB data **helps to reduce risks in MASH development** and potentially supports *accelerated regulatory review and fast track*.

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.

Hydronidone Targets Fibrosis Specifically for Advanced MASH



1. We estimate ~650K compensated F2–F4 MASH patients in the U.S., based on market data and internal modeling.

What makes Gyre different ?

Strong Pipeline

Our lead asset, F351 (Hydronidone), has the potential to become a **first-in-class therapy** for CHB-related liver fibrosis, addressing a significant unmet medical need in China.

Robust pipeline spanning various clinical stages focused on treating organ diseases.

Efficient R&D Strategy

China-first validation strategy leveraging faster patient enrollment and cost efficiency, followed by expansion into the U.S. helps mitigate clinical and regulatory risks.

Hydronidone U.S. IND filing for advanced fibrosis expected in 2025, trial initiation planned pending regulatory review.

Proven Commercial Execution

Maintaining market leadership since the commercialization of first-in-class pirfenidone in 2014, with extensive and effective nationwide commercial coverage in China across more than 3,000 hospitals and pharmacies.

Fully Integrated Platform

Comprehensive in-house capabilities covering discovery, clinical development, regulatory affairs, manufacturing, and commercialization.

Two GMP-compliant manufacturing facilities are strategically located to support robust expansion.

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.

Gyre THERAPEUTICS

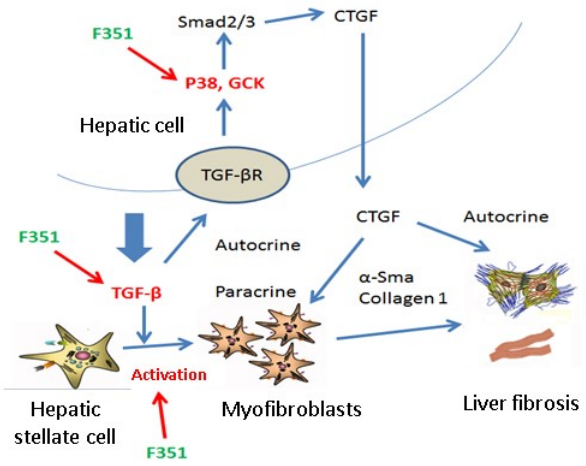
Thank you

Contact:

David Zhang
David.Zhang@Gyretx.com

Comparison of Hydronidone and Pirfenidone metabolism

Liver Injury → TGF-β ↑ triggers multiple fibrosis pathways:
1. → p38γ → HSC Activation → α-SMA ↑ → ECM Accumulation → Fibrosis
2. → Smad2/3 (phosphorylation) → Fibrosis
3. ⊣ Smad7 (inhibitory) → Upregulation of TGF-beta signaling → Activation of both p38gamma and SMAD2/3 cascades



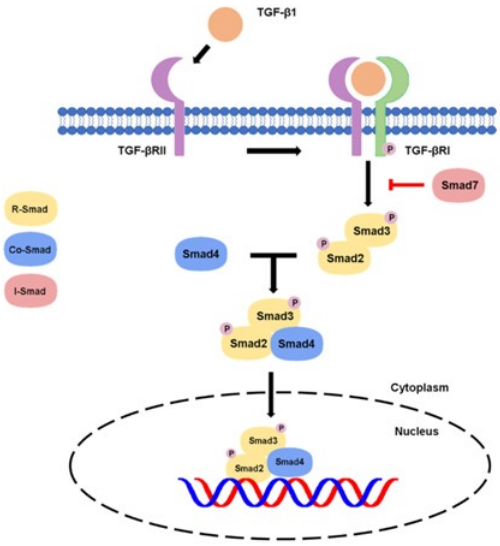
As a key profibrotic cytokine, TGF-β drives hepatic stellate cell (HSC) activation, promotes extracellular matrix (ECM) deposition, and triggers fibrogenesis.

The p38γ isoform plays a pivotal role in TGF-β-stimulated collagen production. Hydronidone attenuates fibrosis, at least in part, by targeting the p38 MAPK transduction pathway.

During hepatic injury, TGF-β upregulation triggers hepatic stellate cell (HSC) activation and differentiation into myofibroblasts. This phenotypic transformation is characterized by cytoskeletal remodeling, including α-smooth muscle actin (α-SMA) expression, which serves as a specific marker for myofibroblasts and the onset of fibrogenesis.

Extensive preclinical and clinical studies indicate that activated myofibroblasts with elevated α-smooth muscle actin (α-SMA) expression serve as the dominant producers of fibrillar collagen and key ECM proteins, thereby driving hepatic fibrogenesis.

TGF-β plays important role in liver fibrosis by activating HSCs

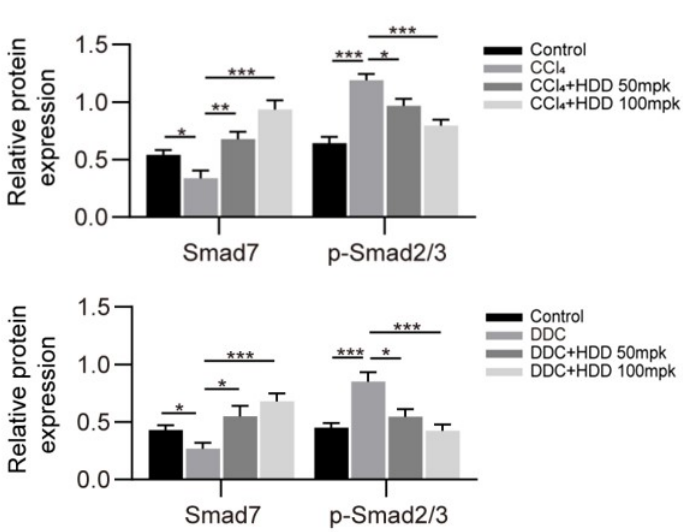


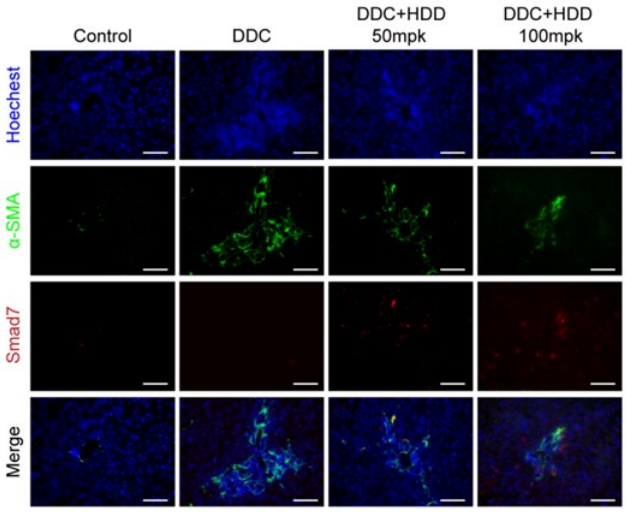
- Smad7 is a negative regulator of TGF-β signaling.
- Smad7 knockdown can promote HSC activation and liver fibrosis.
- Smad7 overexpression can prevent liver fibrosis.
- **Hydronidone is believed to effectively target this pathway.**

Inhibiting HSC activation is believed to be one of the most effective therapeutic strategies to fight liver fibrosis

1. Xu, Xianjun et al. "Hydronidone ameliorates liver fibrosis by inhibiting activation of hepatic stellate cells via Smad7-mediated degradation of TGFβRI." *Liver international : official journal of the International Association for the Study of the Liver* vol. 43,11 (2023): 2523-2537. doi:10.1111/liv.15715

Animal studies demonstrated Hydronidone upregulated the expression of Smad7 and inhibited phosphorylation of Smad2/3



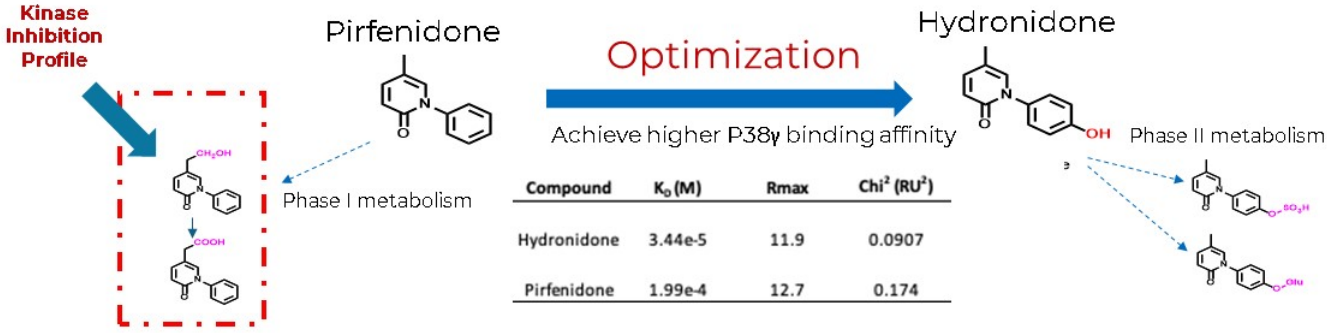


Smad7 is a known negative regulator of liver fibrosis, suggesting clinical potential in a recognized cascade

Note: Statistical significance: *p < .05, **p < .01, *p < .001 (one-way ANOVA with post hoc tests). Data from Xu et al., Liver International (2023), 43(11): 2523–2537. doi:10.1111/liv.15715.

Hydronidone vs. Pirfenidone: Mechanistic and safety advantages

- The introduction of a hydroxyl group shifts its metabolic profile from Pirfenidone's dominant Phase I oxidation to preferential Phase II conjugation (M3/M4 metabolites). Phase II metabolism, known as "detoxification metabolism," can prevent the formation of active metabolites and covalent binding to proteins, suggesting a mechanistic basis for hydronidone's improved hepatic safety profile compared with Pirfenidone.



Compound	K_D (M)	Rmax	Chi² (RU²)
Hydronidone	3.44e-5	11.9	0.0907
Pirfenidone	1.99e-4	12.7	0.174

- In vitro kinase assay shows that both hydronidone and Pirfenidone effectively inhibit p38γ activity, with hydronidone exhibiting a higher inhibition potency than Pirfenidone.
- These findings indicate that hydronidone exhibits stronger inhibition of the p38γ pathway, potentially contributing to its enhanced antifibrotic activity.

Hydronidone shaping up to be Pirfenidone 2.0

Feature	Hydronidone	Pirfenidone
Mechanism of Action	Tri-pathway mechanism: inhibits p38γ, upregulates Smad7, and suppresses TGF-β/Smad2/3 signaling	Broadly downregulates TGF-β levels, with less defined pathway specificity
Metabolism	Undergoes Phase II metabolism, known for safer detoxification and fewer reactive byproducts	Primarily metabolized through Phase I oxidation (CYP1A2), which can generate reactive metabolites
Liver Safety	Designed to reduce hepatotoxicity; favorable liver safety profile in trials	Observed increases in liver enzymes in some patients; rare hepatic events documented
Fibrosis Efficacy (in humans)	Shown to reverse fibrosis in 55% of patients with CHB (270 mg group)[1]	Exploratory clinical data in liver fibrosis; not approved for fibrotic liver disease

1. Cai et al. Clin Gastroenterol Hepatol. 2023;21(7):1893–1901. doi:10.1016/j.cgh.2022.05.056

Hydronidone is Purpose-Built on Pirfenidone's Foundation - with Enhanced Potency and Safety

Pirfenidone → [Structural Analog + Hydroxyl Group] → Hydronidone

↓ ↓

Modest Liver Activity ↑ Hepatotoxicity

Enhanced Smad7 Upregulation + Phase II Metabolism [1] → ↓ *Hepatotoxicity* + ↑ Anti-fibrotic Potency

Attribute	Pirfenidone	Hydronidone	Benefit
Structure	Parent compound	Analog with –OH group	↑ Smad7
MoA	TGF-β	TGF-β + p38γ + Smad7	↑ Potency
Metabolism	Phase I (oxidation)	Phase II (conjugation)	↓ Toxicity
Hepatic Safety	Known liver risk	Improved	↑ Tolerability
MASH Evidence	Some benefit (PROMETEO, model)[2]	Strong effect in a validated preclinical model	↑ Rationale

Hydronidone enhances pirfenidone's anti-fibrotic effect by also inhibiting p38γ and upregulating Smad7, improving hepatic safety and supporting its expansion into metabolic liver diseases like MASH.

1. Phase II metabolism is associated with improved hepatic safety due to faster detoxification. 2. González-Huezo M, et al. Real-life proof-of-concept trial of prolonged-release pirfenidone in advanced liver fibrosis (PROMETEO study). Hepatol Int. 2021;15(2):377–388.