Advancing Lanifibranor: Potential Best-in-Disease Oral Therapy for MASH

A DIFFERENTIATED ORAL ANTIFIBROTIC CANDIDATE DESIGNED TO ADDRESS PROGRESSIVE FIBROSIS IN F2/F3 MASH

J.P. Morgan Healthcare Conference 2026

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Please refer to the Universal Registration Document for the year ended December 31, 2024, filed with the Autorité des Marchés Financiers on April 15, 2025, and the Annual Report on Form 20-F for the year ended December 31, 2024, filed with the Securities and Exchange Commission (the “SEC”) on April 15, 2025, for other risks and uncertainties affecting Inventiva, including those described under the caption “Risk Factors”, and in future filings with the SEC. Other risks and uncertainties of which Inventiva is not currently aware may also affect its forward-looking statements and may cause actual results and the timing of events to differ materially from those anticipated. All information in this presentation is as of the date of the release. Except as required by law, Inventiva has no intention and is under no obligation to update or review the forward-looking statements referred to above. Consequently, Inventiva accepts no liability for any consequences arising from the use of any of the above statements. This presentation includes information and statements of third parties. Inventiva does not make any representation with respect to the accuracy or otherwise with respect to such third party information.

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Addressing Remaining Unmet Medical Needs in MASH

Advancing a differentiated oral antifibrotic as a potential new treatment option for patients with MASH

Structured to deliver topline results expected in H2 2026 and prepare for potential launch

Clinical momentum, financial backing and strong leadership

Moving Towards Approval & Launch of Lanifibranor

- Phase 3 trial fully enrolled, patient population mirrors successful Phase 2b
- Topline data expected in second half of 2026
- Regulatory filings expected in 2027, potential commercial launch in 2028 if approved

Robust Financial Position

- Successful follow-on financing in November 2025
- $172M raised from top-tier investors (existing and new)(1)
- Cash runway until the middle of Q3 2027 assuming full exercise of 3rd tranche(2)

Extensive Medical, Regulatory and Commercial Experience

- **Andrew Obenshain, MBA,** CEO (formerly BlueBird Bio)
- **Jason Campagna, MD, PhD,** Chief Medical Officer, Head of R&D
- **Martine Zimmermann, PharmD,** EVP, Regulatory Affairs and Quality
- **Nazira Amra, MD, MBA** Chief Commercial Strategy Officer

Poised to Potentially Capture Significant Value in a Multi-Billion Dollar MASH Market

(1) Gross proceeds. (2) The third tranche of up to €116 million would consist of potential proceeds from the exercise of warrants for ordinary shares, at the discretion of investors, following the potential publication of positive results in NATiV3 by no later than June 15, 2027. This estimate is based on the Company's current business plan and excludes any potential milestones payable to or by the Company, any additional expenditures related to other product candidates or resulting from the potential in licensing or acquisition of additional product candidates or technologies, or any associated development the Company may pursue. The Company may have based this estimate on assumptions that are incorrect, and the Company may end up using its resources sooner than anticipated.

Addressing a growing urgency for MASH treatments

Medical and scientific understanding of the disease has undergone significant recent changes

MASH is a chronic, progressive disease and the **no. 1 cause of liver-related death**.

MASH is a cardiometabolic disease:
Not just a liver disease; part of a “cardiometabolic continuum”

Systemic risk in patients: Highly associated with comorbidities; over 60% of patients with MASH and advanced fibrosis have T2D, increasing their risk of major adverse liver outcomes

Cirrhosis (F4) now understood as a dynamic process: Cirrhosis isn’t just scar tissue; but a combination of structural fibrosis and altered hepatic physiology

MASH is not a single-pathway disease.

Durable impact requires liver-targeted activity across metabolic, inflammatory and fibrotic drivers.

Clin Mol Hepatol. 2025 Feb;31(Suppl):S32-S50
Journal of Diabetes and its Complications, Volume 39, Issue 10, 2025,

Underdiagnosed and undertreated, with rapidly expanding commercial validation

Early uptake signals demand, not disease resolution — creating a potential multi-billion-dollar opportunity for lanifibranor

Clinical need and market are expanding: Strong and growing demand for more potent liver directed oral therapies such as lanifibranor that lead to improvement in fibrosis

~ 10%

Only ~1.9 million diagnosed with MASH in U.S in 2025, representing ~ 10% of the patients with MASH.
+25% compared to the 2024 estimate.

~ 40%

~910k patients have clinically actionable F2/F3 disease, 374k of them are under treater care (~40%).
+20% of patients under treated care compared to the 2024 estimate.

$15B

MASH market expected to exceed $15 billion by 2035.

The only other oral drug approved has already reached approximately $1 billion in sales since its launch in April 2024.

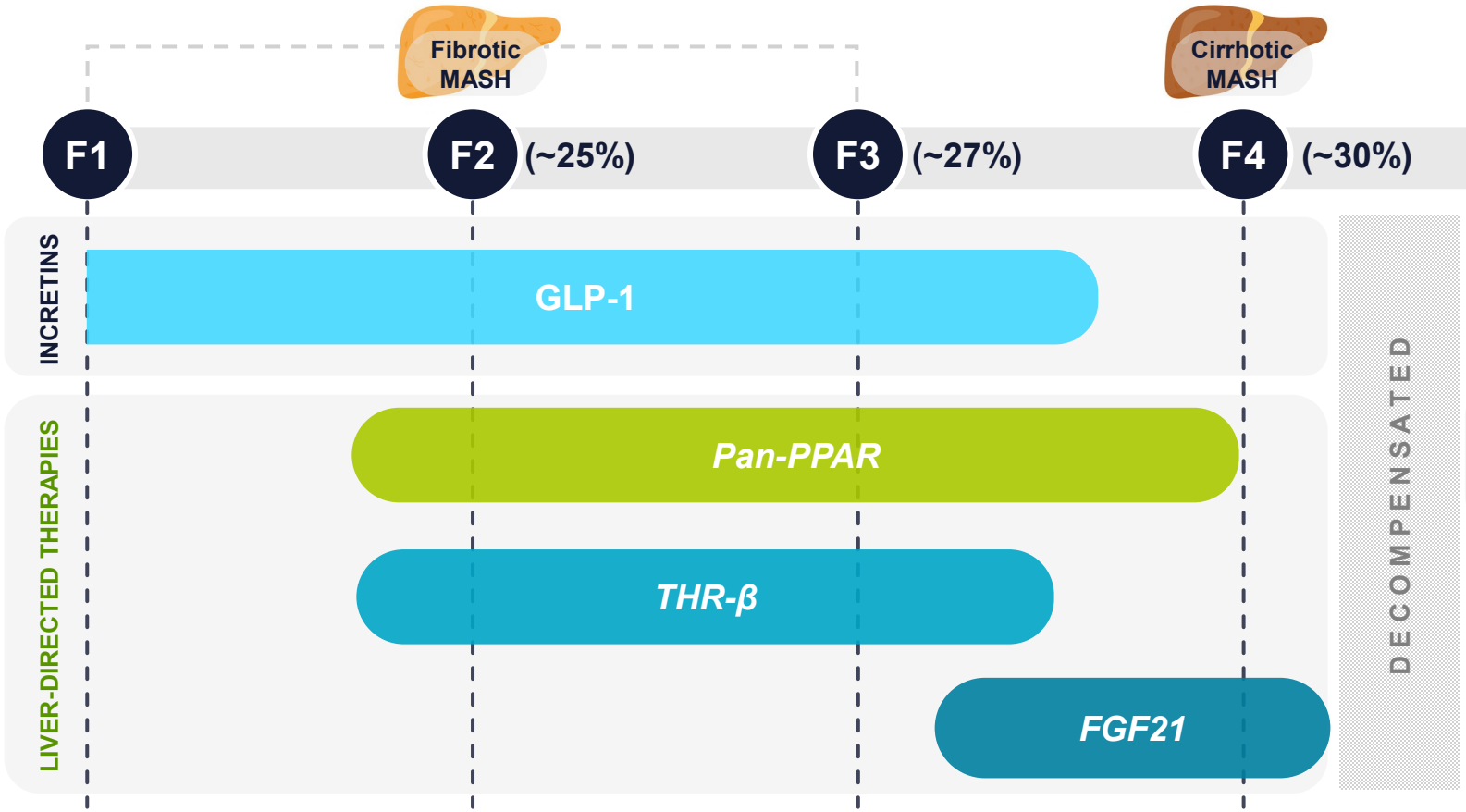
Inventiva is well-positioned to potentially **bring the best-in-disease oral drug to patients** with progressive fibrotic MASH.

Estes. 2018; Lui et al 2022, Prevalence taken from 2018 and conservative growth adjustments applied; 2) Analysis conducted by Forian using CHRONOS™
Based on based on recent industry market research forecasts.
Madrigal Pharmaceuticals Third-Quarter 2025 Financial Results

Different mechanisms address different stages of fibrotic MASH

Lanifibranor is designed to play a key role in several high-unmet-need MASH patient segments

Priority patient segment at time of anticipated lanifibranor launch, if approved, based on KOL feedback



GLP-1s are modestly effective on fibrosis, but key backbone therapy for patients with obesity and T2D with cardiometabolic benefits, MASH specific Rx primarily anticipated in early stage

Pan-PPAR agonists have a strong antifibrotic effect and insulin sensitivity profile, and manageable adverse events

THR-β agonists have modest efficacy, best suited for early-stage patients

FGF21s most likely used in advanced patients due to GI side effects, bone density reduction, and injectable administration

Source: KOL Interviews; Inventiva Analysis based on Phase 3 and Phase 2b results and safety profiles, as well as route of administration.

Balanced pan-PPAR Designed to Address the Biology of MASH

A new chemical entity engineered to deliver coordinated metabolic, anti-inflammatory and antifibrotic effects

Rational pan-PPAR design informed by decades of biology

Lanifibranor: Low-potency, balanced PPAR-α/δ/γ activation designed to avoid receptor dominance

First-in-class new chemical entity.
Distinct from fibrates and thiazolidinediones (TZDs).

Balanced and low potency across all three isoforms.
Engineered to mirror the safe potency range of pioglitazone and avoid the high γ-potency of rosiglitazone (~ 20x higher).

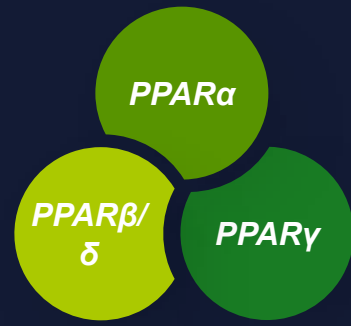
Balanced PPAR isoforms engagement.
Avoid dominance of a single receptor and delivers coordinated metabolic, inflammatory, and antifibrotic activity.

Differentiated co-activator fingerprint.
Enable metabolic, anti-inflammatory, and antifibrotic benefits.

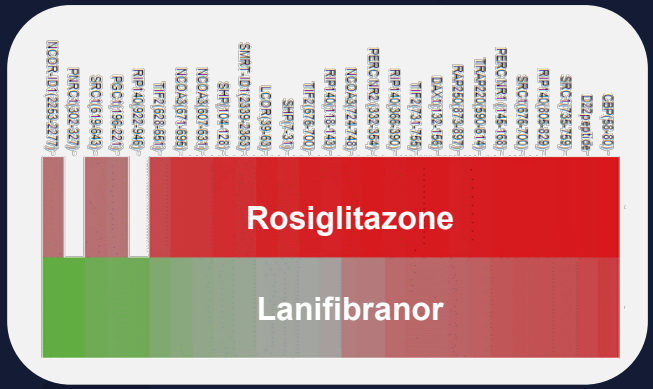
- ***Lanifibranor has been designed at the physiologic sweet spot for broad and best-in-class efficacy, clean and durable safety profile.***
- ***Engineered to maximize therapeutic benefit without transcriptional overload or high-potency risks.***

Master Regulators of Nutrient and Energy Metabolism

PPARs (peroxisome proliferator-activated receptors) enable coordinated modulation of the metabolic, inflammatory, and fibrotic pathways at the core of MASH.



Cofactor Recruitment Comparison



Striking the PPAR Balance

LIVER

Steatosis (abnormal fat buildup)

PPARγ

- ↓ Fatty acid uptake
- ↑ Fatty acid breakdown (catabolism)
- ↓ Fat creation (lipogenesis)

Inflammation and ballooning

PPARγ, PPARα, PPARδ

- ↓ NFkB-dependent gene activation
- ↓ Inflammasome
- ↓ Ballooning

Fibrosis

PPARγ, PPARα

- ↓ Stellate cell proliferation and activation
- ↓ Collagen and fibronectin production

Vascular

PPARγ, PPARδ

- ↓ Portal pressure
- ↓ LSEC capillarization
- ↓ Intrahepatic vascular resistance

WHOLE BODY

Metabolism

PPARγ, PPARα, PPARδ

- ↑ Insulin sensitivity
- ↑ HDL-C
- ↓ Triglycerides

Clinical Milestones

Phase 2b NATIVE trial showed compelling data;

Phase 3 underway, with topline results expected in H2 2026

NATIVE Phase 2b trial results support antifibrotic activity with broader metabolic impact

Histologic improvement observed alongside favorable cardiometabolic markers at 24 weeks

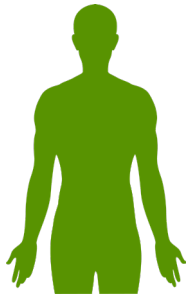
Within 24 weeks, lanifibranor improved fibrosis and key metabolic markers.

18%

effect size on **improvement in fibrosis** with no worsening of MASH versus placebo.

24%

effect size on the dual endpoint of **fibrosis improvement and MASH resolution.**



Improved cardiometabolic, glycemic, and metabolic markers.

Phase 2b NATIVE trial design

Once-daily oral dosing; two doses, 800mg and 1200mg

Screening
Liver biopsy

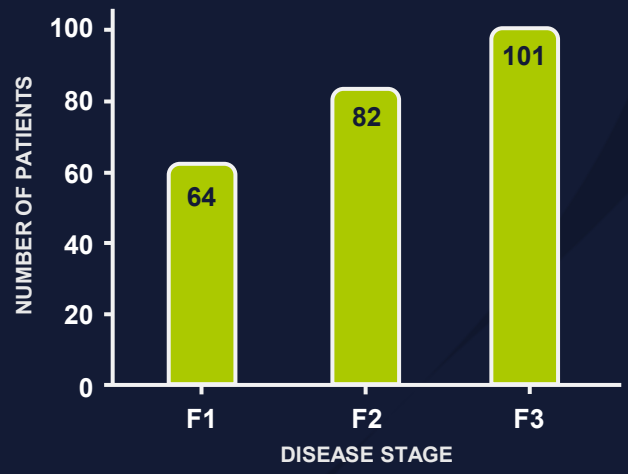
24-week treatment + 4-week follow-up
Double blind, randomized, placebo-controlled ITT: 247 patients

End of treatment
Liver biopsy

- **Placebo**
- **lanifibranor, 800 mg once daily**
- **lanifibranor, 1200 mg once daily**

Main Inclusion Criteria:

Patients with biopsy-proven MASH confirmed to have Steatosis-Activity-Fibrosis (SAF) scores of 1-3 for steatosis, 3-4 for activity, and <4 for fibrosis.



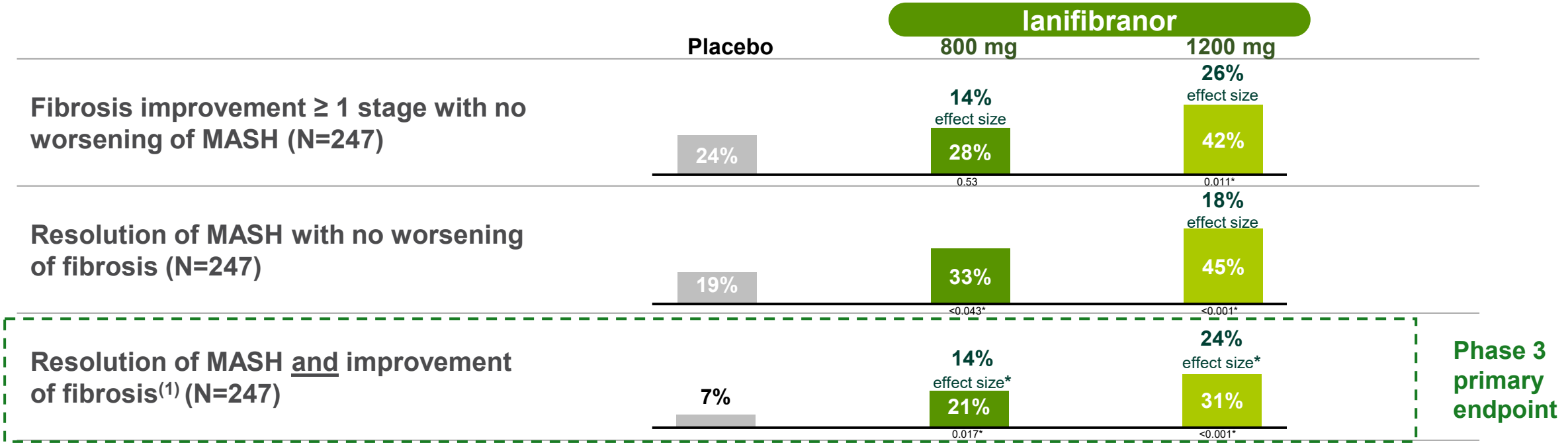
1:1:1 randomization

Stratification on type 2 diabetes

A Randomized, Controlled Trial of the Pan-PPAR Agonist Lanifibranor in NASH, N Engl J Med 2021;385:1547-1558

Statistically significant results on key Phase 3 FDA and EMA primary endpoints

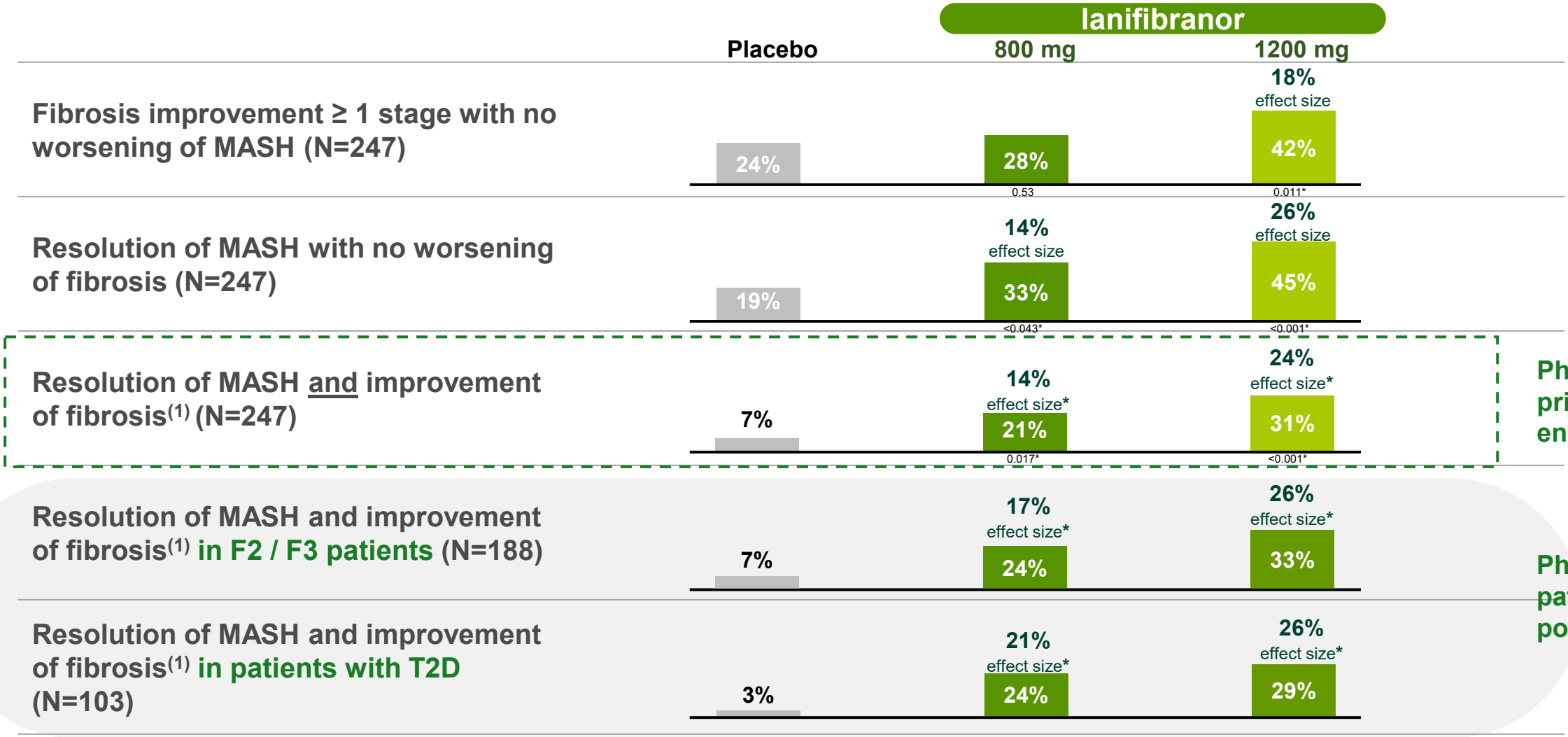
Significant fibrosis improvement versus placebo at only 24 weeks



(1)) Resolution of MASH and improvement of fibrosis at week 24: NAS-I = 0 or 1, NAS-B = 0 and an improvement of NAS-F ≥ 1 stage

Statistically significant results on key Phase 3 FDA and EMA primary endpoints

Significant fibrosis improvement versus placebo at only 24 weeks with greater effect size in patients with T2D



(1)) Resolution of MASH and improvement of fibrosis at week 24: NAS-I = 0 or 1, NAS-B = 0 and an improvement of NAS-F ≥ 1 stage

Biomarkers support compelling histological findings

Statistically significant, rapid, and sustained biomarker responses at 24 weeks

Intra-hepatic Benefits

Liver Health

- Statistically significant decrease in liver enzymes (ALT, AST, GGT), with rapid and sustained improvement

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Extra-hepatic Benefits

Cardiometabolic Health

- Lipids: Improvements in HDL cholesterol and TGs without a change in LDL cholesterol
- Glycemic Control: Significant reductions in HbA1c +
- Significant improvements in hepatic and muscular insulin sensitivity
- Decrease in diastolic blood pressure
- Increase in adiponectin levels, with a relative beneficial increase of ~2.8x and ~3.5x for each dose group

NATIVE safety and tolerability profile

Investigator-reported most-frequent adverse events[(1)]

	Placebo (N = 81)	800 mg (N = 83)	1200 mg (N = 83)
Diarrhea	1 (1%)	8 (10%)	10 (12%)
Fatigue§	8 (10%)	3 (4%)	11 (13%)
Nausea	3 (4%)	8 (10%)	7 (8%)
Weight gain	-	8 (10%)	7 (8%)
Peripheral edema**	2 (2.5%)	5 (6%)	7* (8%)
Drug-related peripheral edema	-	2 (2%)	2 (2%)
Headache	4 (5%)	4 (5%)	7 (8%)
Abdominal pain¶¶	4 (5%)	4 (5%)	5 (6%)
Dizziness	3 (4%)	2 (2%)	6 (7%)
Anemia‡	-	1 (1%)	6 (7%)
Constipation	6 (7%)	3 (4%)	5 (6%)
Increase in aminotransferase levelΦ	1 (1%)	5 (6%)	3 (4%)

¶ Adverse events are the ones reported by investigators and include those that occured in more than 5% of patients in either lanifibranor group.
§ Fatigue included asthenia.
¶¶ Abdominal pain included upper and lower abdominal pain.
** Peripheral edema (bilateral ankle edema): usually mild, in most cases no treatment was required, a few patients received diuretics. 4 cases were considered study drug related by the investigator (2 at 800 and 1200 mg each). One case of severe intensity, which resolved by stopping treatment (lanifibranor 1200mg) for 12 days, without reoccurrence when the study treatment was resumed. All were female patients.
‡ Anemia included iron deficiency anemia and decrease hemoglobin level.
Φ Increase in aminotransferase level included increased in ALT, AST, or abnormal liver function test result.

Approximately 30% of treated patients experienced >5% body-weight increase→ **Less than 10% of cases reported as adverse events by investigators.**

Weight gain occurred alongside favorable changes in:

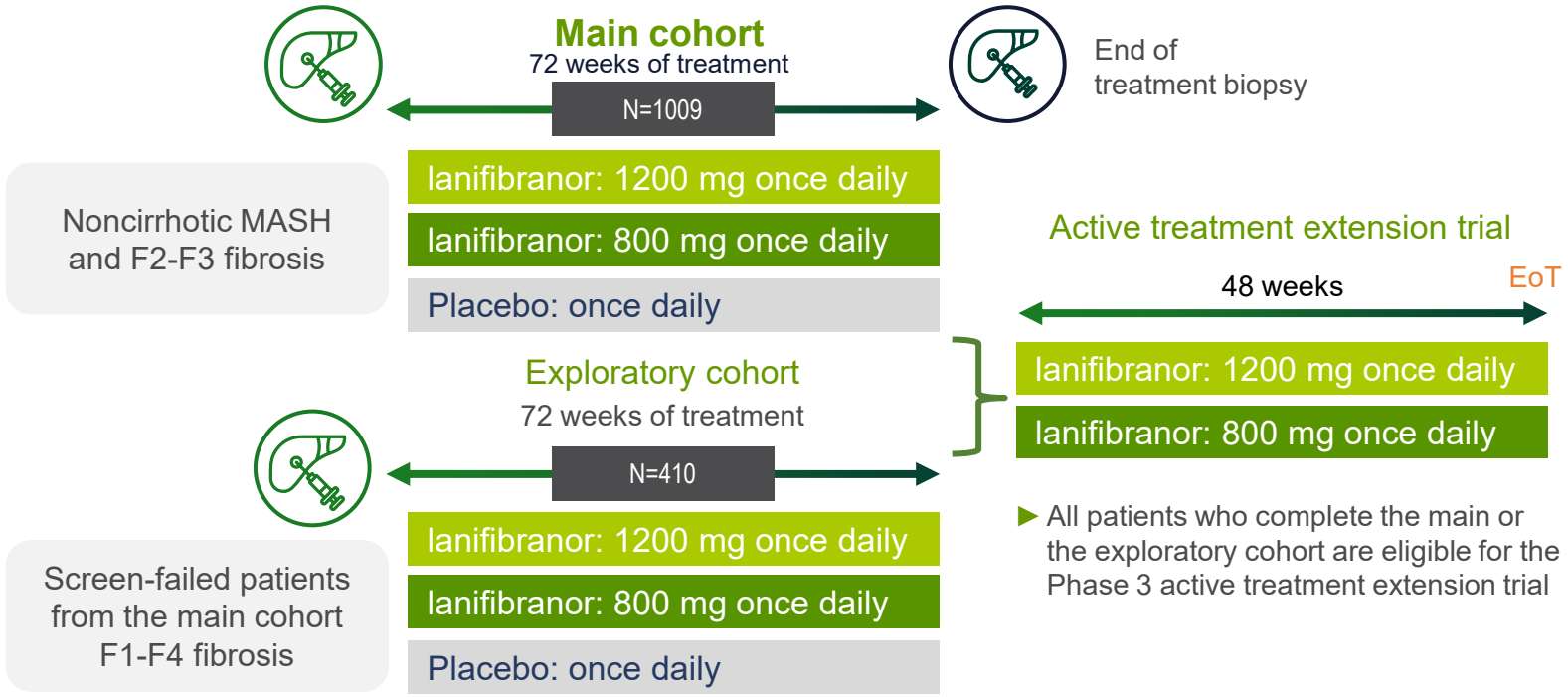
- ✓ adiponectin
- ✓ lipids
- ✓ liver enzymes
- ✓ liver histology

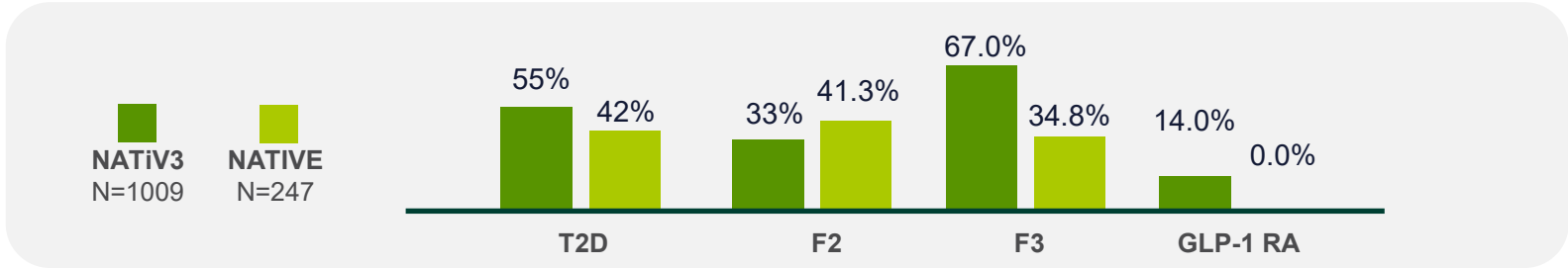
Pattern consistent with improved insulin sensitivity rather than adverse metabolic effect.

Lanifibranor was purposely designed to achieve low-potency, and balanced PPAR-α/δ/γ activation to avoid receptor dominance and associated safety concerns.

(1) A Randomized, Controlled Trial of the Pan-PPAR Agonist Lanifibranor in NASH, N Engl J Med 2021;385:1547-1558

Phase 3 trial fully recruited, Designed to be de-risked





EoT, end of treatment; GLP-1, glucagon-like peptide-1; GLP-1-RA, glucagon-like peptide-1 receptor agonist; MASH, metabolic dysfunction-associated steatohepatitis.
* As of October 2025.

Phase 3 NATiV3 Clinical Trial

Primary Endpoint
Composite end point of patients having both MASH resolution and 1 stage of fibrosis improvement.

Key Secondary Endpoints
MASH resolution and no worsening of fibrosis, fibrosis improvement, and no worsening of MASH.

GLP-1 Inclusion
Patients under a stable dose of GLP-1-RA for at least 3 months prior to screening can be included.

Statistical Powering
- 90% considered for sample size calculations.
- Stratification by fibrosis stage and diabetic status.
- NATiV3 fully recruited with 1,009 patients in the main cohort and 410 in the exploratory cohort.

Next Steps

An accelerated timeline for potential market approval, product launch, and commercial partnerships

Plan to confirm clinical benefit in compensated cirrhosis

Planned Regulatory Filing

Accelerated Approval (FDA) and Conditional Approval (EMA) filing on Phase 3 histology.

Confirmatory Trial

Starting outcomes study in patients with compensated cirrhosis MASH to confirm clinical benefits:

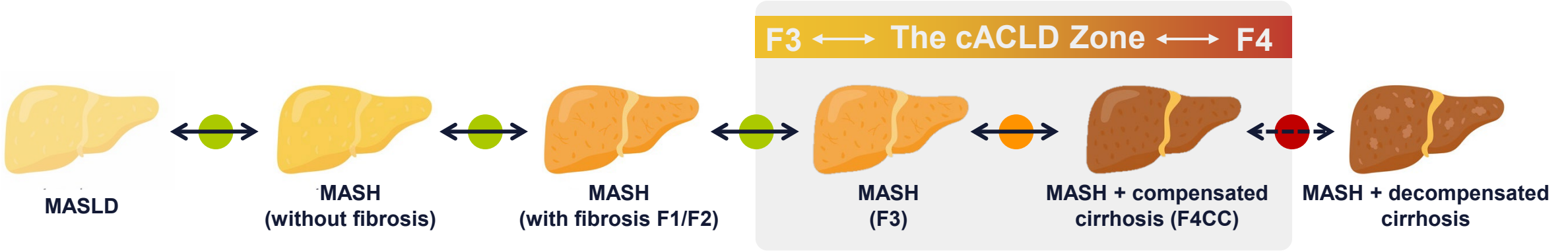
- Prevents or reduces associated clinical outcomes: risk of decompensation, HCC, liver transplant

Preclinical and early clinical signals suggest lanifibranor may provide a disease-modifying oral option for patients with compensated cirrhotic MASH, a high unmet need.

Lanifibranor targets the core pathophysiology underlying both MASH and cACLD

Physiology, not anatomy, drives active progression; reversal is possible

Many patients are asymptomatic until later stage, then need critical intervention.
MASH is a major driver of cACLD (compensated advanced chronic liver disease),
in which patients have **advanced fibrosis or cirrhosis but no outward signs of liver failure**.

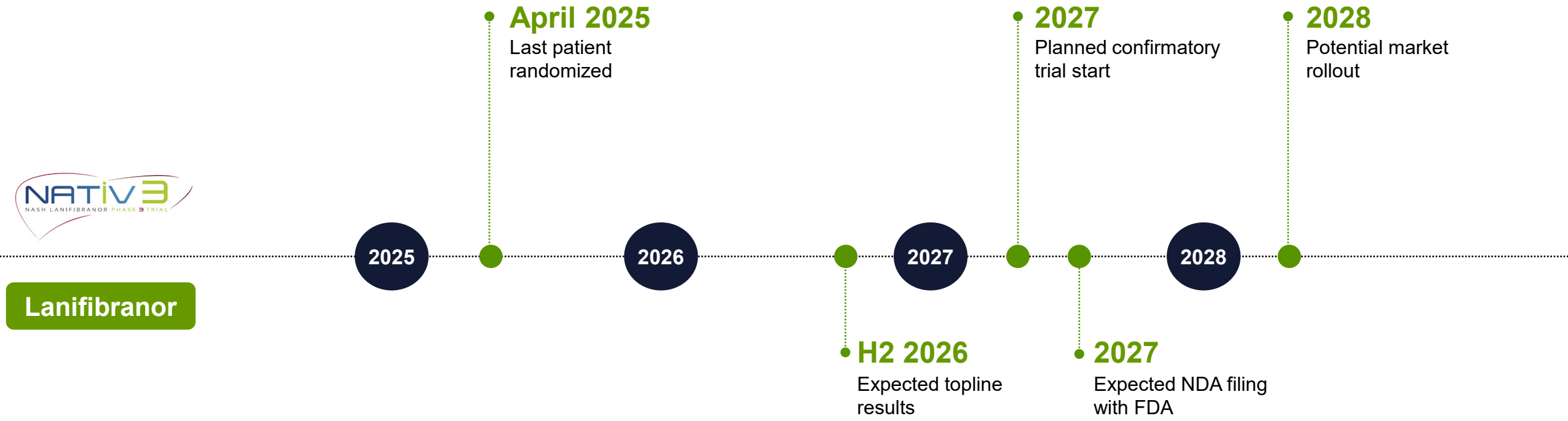


Lanifibranor engages the core drivers of F4 progression:

- PPAR-γ inactivates hepatic stellate cells; restores quiescence
- PPAR-α/δ normalize mitochondrial function and lipid flux; reduce lipotoxic stress
- Adipose–liver axis: ↑ adiponectin → antifibrotic, anti-inflammatory, endothelial benefits
- Broad reduction of systemic inflammation across macrophage and vascular beds
- Portal pressure biology: preclinical models show improved sinusoidal tone and vascular remodeling.

Topline data expected in H2 2026, NDA filing expected in 2027

Lanifibranor could be the second oral liver-directed agent for the treatment of MASH



Cash runway projected beyond Phase 3 readout

- **Q3 2025 –** Cash balance of €122.3M(1)
- **Q4 2025 –** Follow-on Raise of €149M(2)
- **Q4 2026 and Q1 2027 –** Repayment deadline of €65M EIB debt
- **Q3 2027 –** Assuming exercise of 3rd Tranche of Structure Financing of €116M

Cash runway until the middle of Q3 2027 assuming fully exercised T3 upon positive Phase 3 readout(3)

(1) Consisting of Cash and cash equivalents of €97.6 million, and €24.7 million in short-term deposits.
(2) Gross proceeds
(3) The third tranche of up to €116 million would consist of potential proceeds from the exercise of warrants for ordinary shares, at the discretion of investors, following the potential publication of positive results in NATiV3 by no later than June 15, 2027. This estimate is based on the Company's current business plan and excludes any potential milestones payable to or by the Company, any additional expenditures related to other product candidates or resulting from the potential in licensing or acquisition of additional product candidates or technologies, or any associated development the Company may pursue. The Company may have based this estimate on assumptions that are incorrect, and the Company may end up using its resources sooner than anticipated.

Shares dilution

Current Fully Diluted Share	**193M ordinary included in a total of 313M fully diluted**
Post T3 Fully Diluted Share (+77M)	**390M shares fully diluted**

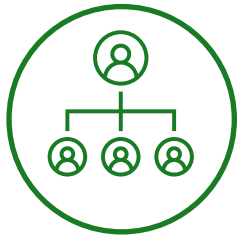
Royalty certificates

Commences upon launch	**2% capped at €92M, 15-year term**
	3%, 14-year term

Strategic partnerships

CTTQ Greater China	**$265 million in additional potential milestones. Low-single-digit royalty rate.**
Hepalys Japan, South Korea	**$231 million in additional potential milestones. Mid-double-digit to low-20s tiered royalty rate.**

Pioneering the Future of MASH



Inventiva has an experienced management team and strong financial position

Novel investigational pan-PPAR agonist engages the core drivers of fibrosis progression, with once-daily oral dosing

Phase 2b showed 24% effect on the dual endpoints of fibrosis regression and MASH resolution in just 24 weeks

Potential expansion into later-stage patient populations with high unmet medical need

Targeting multi-billion in potential peak sales by 2035 as MASH market expands to >$15 billion