GENFIT
TOWARDS BETTER MEDICINE

Corporate Presentation

June 2021

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Update on Implementation of Corporate Roadmap

Lead program in PBC Therapeutics

- Phase 3 ELATIVE™[1] evaluating drug-candidate elfibranor[2]: patient enrollment **started in 3Q20**
- Topline data readout **expected in 1Q23**
- Next corporate **update in Sept 2021**

▶ PBC Day, February 21

Pipeline

- **R&D rationalized**: focus on **ACLF** and **Cholestatic diseases**, leveraging the potential of GENFIT's internal assets in therapeutic areas where highest probability of success for GENFIT: **moving into clinical stage in 2021**

▶ Pipeline Update, May 21

Lead program in NASH Diagnostics

- NASHnext™, powered by NIS4® technology: large scale **commercial launch by Labcorp in April 2021**
- Next corporate **update in Sept 2021**
- NIS4® technology available for use in clinical research by Covance

Finances

- 1Q21 cash position: €108.9M
- On track to achieve **~€45M operational cashburn in 2022**
- Convertible debt maturity pushed back to **Oct 2025**
- Residual convertible debt down to nominal amount of €57.2M as of April 13, 2021

Note: PBC: Primary Biliary Cholangitis; ACLF: Acute-on-chronic-Liver-Failure; NASH: Non-alcoholic steatohepatitis
1. CT.Gov identifier: CT04526665; 2. Elafibranor is an investigational compound that has not been reviewed and has not received approval by any regulatory authority. 3. *Prêt Garanti par l'Etat* (Bank loan guaranteed by the French government)

Therapeutics:
Cholestatic diseases

Cholestatic Diseases: Multiple Opportunities, Beyond Lead Program in PBC

High unmet medical need in cholestatic diseases

PBC

- Standard of Care (SoC): still unmet medical need calling for new differentiated therapies[1]

PSC

- Rare chronic cholestatic disorder characterized by disrupted bile-acid homeostasis that may result in cirrhosis, liver failure, cholangiocarcinoma - no approved treatment[2]
- Significant burden of disease (high prevalence of pruritus, liver transplant, etc.)[2]

Other rare pediatric cholestatic diseases

- Several indications in need of treatment options: Progressive familial intrahepatic cholestasis (PFIC) / Biliary atresia / Alagille syndrome / Pediatric PSC

GENFIT's rationale

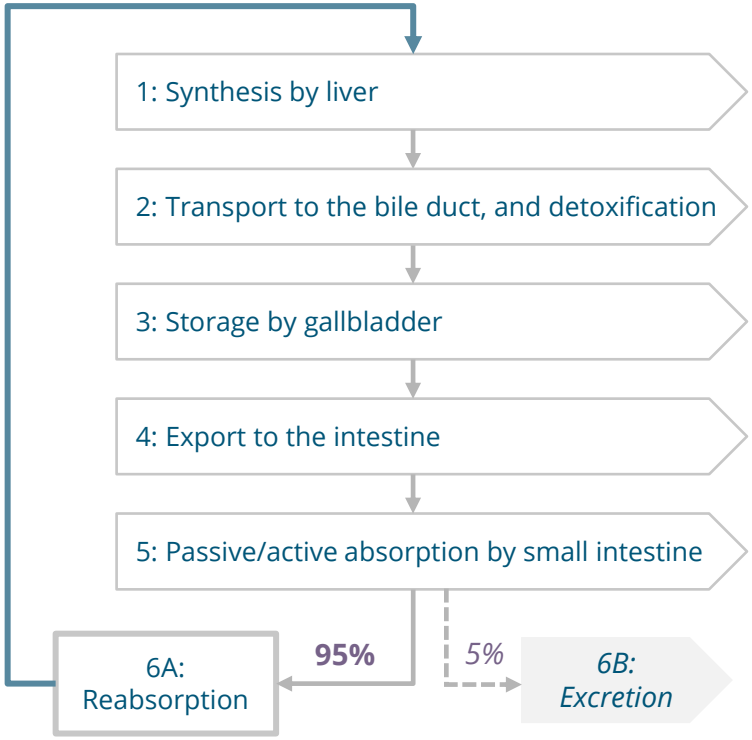
- **In-house expertise (discovery to late stage) in severe liver diseases**
- **Strong scientific rationale to support the effects of elafibranor on cholestasis[3]**
- **Late stage clinical development in PBC, clinical development initiated in PSC, research program initiated on potential candidates for other rare cholestatic pediatric diseases**
- **New clinical evidence expected in cholestatic diseases, with PSC data in 2022 (elafibranor)**

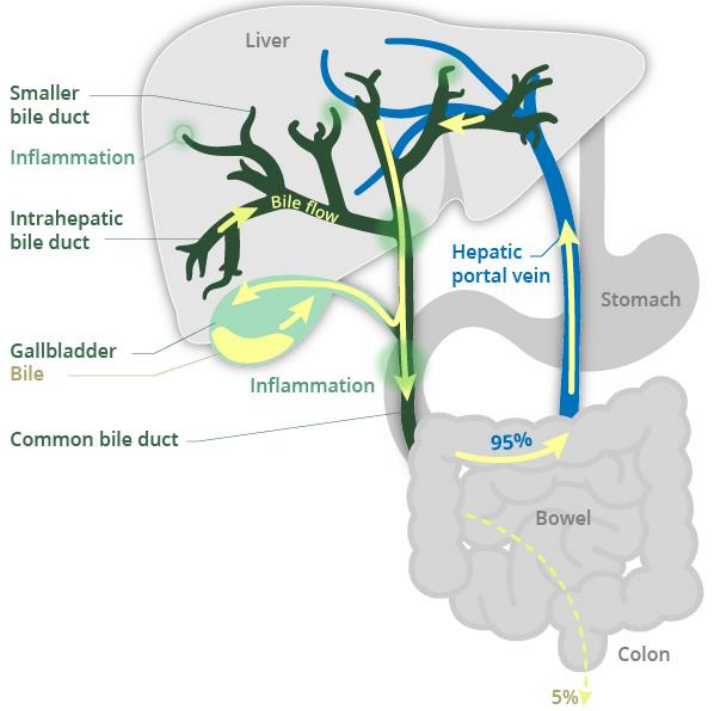
Market opportunity

- **PBC and PSC orphan indications**: enhanced market exclusivity and clinical research subsidies[4]
- **Eligible population in PBC**: ~90,000 patients US+EU for second line treatment, representing a market potential of $1bn by 2025[5]
- **Eligible population in PSC**: ~50,000 patients US+EU, with a market potential of ~$700M[5]
- **Other rare pediatric cholestatic diseases: ~**100,000[6] patients with pediatric cholestatic liver disease in top 25 countries

References:1. Onofrio et al. Gastroenterol. Hepatol. 2019; Vol.15, Issue 3:145-154; 2. Karlsen, et al. J. of Hepatol. 2017; 3. Schattenberg et al. J. of Hepatol. 2021;Vol. 74, Issue 6:1344-1354.4. Orphan Drug Act of 1983, US Food and Drug Administration. 4 January 1983, PUBLIC LAW 97-414; 5. Iqvia Commercial Opportunity Presentation, 2020 - Resarch on File, November 2019; 6. Pediatric CLD: Data on file, excluding China & India.

Cholestatic Diseases: Characterized by Inflammation and Fibrosis of the Bile Ducts





Enterohepatic cycle (bile acid cycle) and potential dysfunction

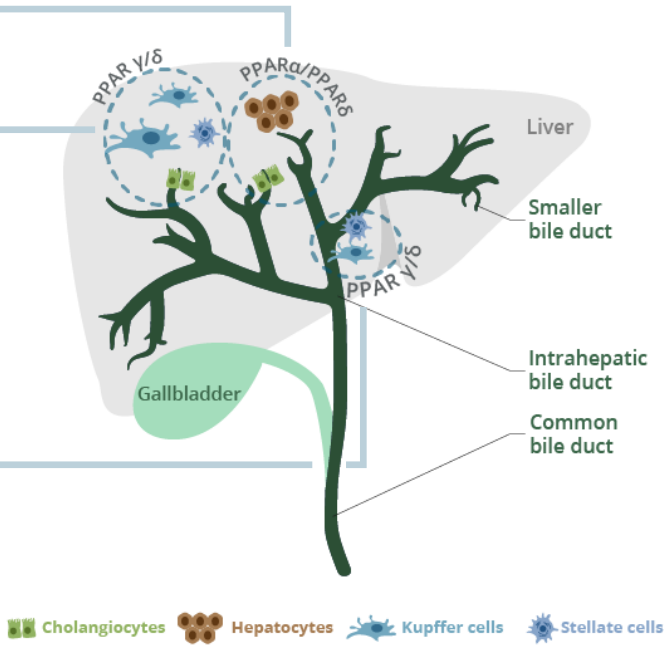
PPARs Mechanism of Action in Cholestatic Diseases

PPARs activation

↑	Increase Bile Acid Detoxification **CYP3A4 | SULT2A1 | UGT2B4**	Intracellular bile acid metabolism
↓	Decrease Bile Acid Synthesis **CYP7A1 | CEP27A1 | CYP8B1**	
↑	Increase Bile Acid Output **BSEP | MRP2**	Effects on apical membrane transporters
↓	Decrease Bile Toxicity **MDR2/3 | ABCG5/G8**	
↓	Inhibition of Inflammatory Pathways **AP1 | NFκb | BCL6**	

PPARα/PPARδ

PPARγ/δ



PPARs engage complementary pathways to drive potential activity in cholestatic diseases

PPAR: Peroxisome proliferator-activated receptor. References:1. Lens et al. Liver International 2014, 34:197-203; 2. Zang et al. Drug Design, Development and Therapy 2015;9:2757-2766; 3. Levy et al. Aliment. Pharmacol. Ther. 2011; 33:235-242; 4. Yin et al. Drig Design, Devleopment and Therapy 2015;9:5407-5419; 5. Hosonuma et al. Am. J. Gastroenterol. 2015;110:423-431; 6. Tanaka et al. J. Gastroenterol. 2015; 50:675-6826; 7. Grigorian et al. Clin. Res. Hepatol. Gastroenterol. 2015;39:296-306; 8. Cheung et al. Aliment.Pharmacol. Ther. 2016;43:283-293; 9. Honda et al. Hepatology 2013;57:1931-1941; 10. Adapted from Corpechot Curr Hepatology Rep 2019:18, 107–114.

Literature on PPARs in Cholestatic Diseases

PPAR Proof of Concept in Cholestatic Disease

Gastroenterology 2021;160:734–743

CLINICAL—LIVER

Fibrates for Itch (FITCH) in Fibrosing Cholangiopathies: A Double-Blind, Randomized, Placebo-Controlled Trial

De Vries et al. Gastroenterology 2021:160:734-743

↳ **Double-blind, randomized, placebo-controlled study**

Patients with cholestatic disease (PBC, PSC or SSC) w/ moderate to severe pruritus

Treatment (n=38) vs placebo (n=36) for 21 days

Results

- Pruritus (≥50% reduction in moderate/severe pruritus assessed by VAS) 45% response rate vs 11% for placebo (p=0.003)
- Reduced serum ALP 35% vs 6% for placebo (p=0.03)
- Can be considered as a new treatment option for cholestasis-associated pruritus given anti-pruritic and anticholestatic properties and a favorable safety profile

Conclusion

- Superior to placebo in improving moderate to severe pruritus in patients with PSC and PBC

Reduced Levels of ALP May be Associated with Improved Outcomes in PSC

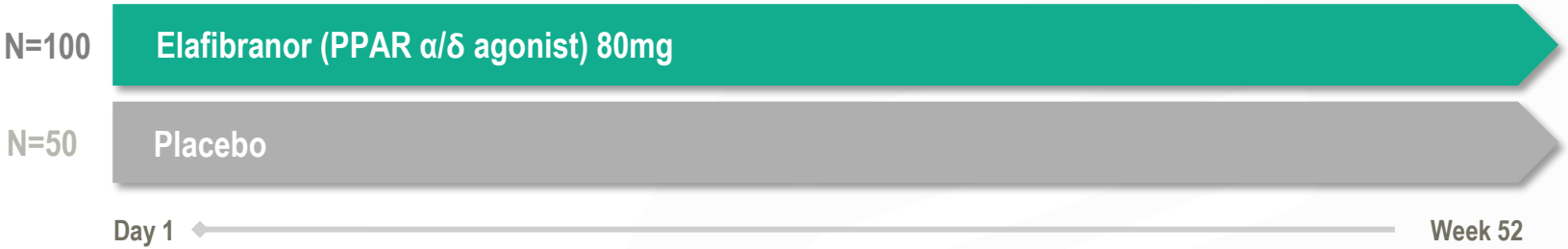
- Stanich PP , Bjornsson E , Gossard AA et al. Alkaline phosphatase normalization is associated with better prognosis in primary sclerosing cholangitis. Dig. Liver Dis. 2011 ; 43 : 309 – 13.
- Al Mamari S , Djordjevic J , Halliday JS et al. Improvement of serum alkaline phosphatase to <1.5 upper limit of normal predicts better outcome and reduced risk of cholangiocarcinoma in primary sclerosing cholangitis. J. Hepatol. 2013 ; 58 : 329 – 34.
- Lindstrom L , Hultcrantz R , Boberg KM et al. Association between reduced levels of alkaline phosphatase and survival times of patients with primary sclerosing cholangitis. Clin Gastroenterol Hepatol 2013 ; 11 : 841 – 6.

PBC = primary biliary cholangitis; PSC = primary sclerosing cholangitis; SSC = secondary sclerosing cholangitis.

Elafibranor as a Potential Treatment for PBC (1/4)

ELATIVE™ – a Pivotal Phase 3 Study in Patients with PBC

Randomized 2:1, double blind, placebo-controlled, global study[1]



Primary Endpoint

*Response to treatment defined as **Alkaline phosphatase (ALP) < 1.67 x Upper Limit Normal (ULN) and Total Bilirubin (TB) ≤ ULN and ALP decrease ≥ 15 percent***

Key Secondary Endpoints

- *Response to treatment based on ALP normalization (At week 52)*
- *Change in pruritus from baseline (Over 52 weeks of treatment) based on PBC Worst Itch Numeric Rating Scale (NRS) score**

SEPTEMBER 2020
Beginning of enrollment

1Q 2023
Anticipated Data readout

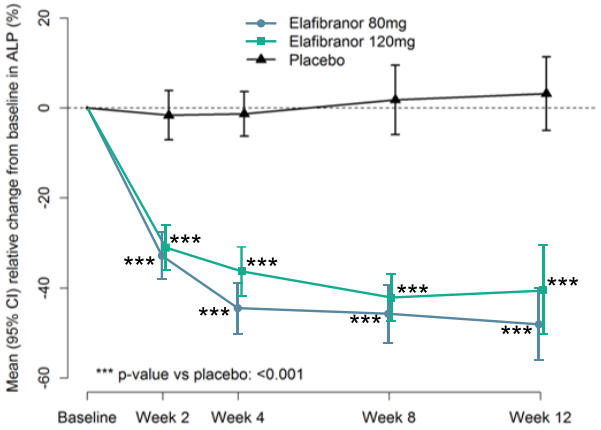
Note: *Measuring itch over the past 24 hours from 0 (no itch) to 10 (worst itch imaginable)
References: 1. NCT04526665

Elafibranor as a Potential Treatment for PBC (2/4)

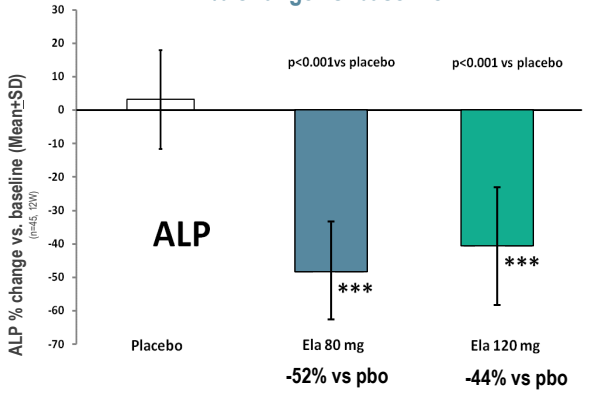
Elafibranor Phase 2a PBC Study

Statistically significant treatment effects in both 80mg and 120mg doses on the primary end-point (confirmed in mITT* set) of serum alkaline phosphatase (ALP) change from baseline

Change from baseline in ALP at week 12[1]



ALP % Change vs. baseline[1]



*Non-parametric randomization ANCOVA with baseline as covariate
*** P-value vs. placebo: <0.001

Elafibranor awarded Breakthrough Therapy designation by the FDA and Orphan Drug Designation by the FDA & EMA for PBC[2]

JOURNAL OF HEPATOLOGY
The Home of Liver Research

A randomized placebo-controlled trial of elafibranor in patients with primary biliary cholangitis and incomplete response to UDCA[1]

Jörn Schattenberg *et. al.* | Accepted for publication in Journal of Hepatology Jan. 7, 2021

Note:* mITT (All subjects w/ available baseline value and at least one post baseline value under treatment for ALP)=Placebo (N=15), Elafibranor 80mg (N=15), Elafibranor 120mg (N=14). Per Protocol Set = Placebo (N=14), Elafibranor 80mg (N=14), Elafibranor 120mg (N=13). ITT (intend to treat) = Placebo (N=15), Elafibranor 80mg (N=14), Elafibranor 120mg (N=15)

References:1. Schattenberg et al. J. of Hepatol. 2021;Vol. 74, Issue 6:1344-1354; 2. GENFIT Corporate Press Release June 29, 2019 "GENFIT Announces FDA Grant of Breakthrough Therapy Designation to Elafibranor for the Treatment of PBC.".

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Elafibranor as a Potential Treatment for PBC (3/4)

Elafibranor is a Competitive 2L Candidate for PBC

	Elafibranor*[2] Phase 2a Week 12 Data NCT03124108 EudraCT2016-003817-80		Ocaliva™[3], Phase 3 POISE Month 12 Data NCT01473524	
	80mg (N=15)	Placebo (N=14)	10mg (N=73)	Placebo (N=72)
Composite endpoint *% responders, ALP<1.67 x ULN; Bili<ULN and ALP reduction >15%*	**67%** (p=0.001)	6.7%	**47%** (p<0.001)	10%
Alkaline phosphatase *(% change vs baseline)*	**-48%** (p<0.001)	3%	**~-36%**** (p<0.001)	~-4%**

► Access to the replay of GENFIT PBC Day, February 2021 for more information

Note: Indirect Comparison of Selected Biochemical Endpoint[1]. Both studies were add-on investigational therapy to UDCA or monotherapy in patients unable to tolerate UDCA. 2L: Second-line. ***Elafibranor –*** mITT: All subjects w/ available baseline value and at least one post baseline value under treatment for ALP. **These are estimations-based figures as reported data is based on actual change from Baseline n ALP (U/L) .Elafibranor is an investigational compound and has not been approved by any regulatory authority in any indication. Obeticholic acid is registered in US and EU under the trade name OCALIVA®, please refer to the approved PI and SmPC. **References** : 1. Data from referenced clinical trials; 2. Schattenberg et al. J. of Hepatol. 2021;Vol. 74, Issue 6:1344-1354; 3. Nevens, et al. NEJM 2016;375(7):631-43.

Elafibranor as a Potential Treatment for PBC (4/4)

Phase 2a Efficacy and Safety Data Support ELATIVE™ Phase 3 PBC Trial

Elafibranor demonstrated beneficial effects on clinically proven markers of PBC[1]

Cholestatic Markers

- ↓ **GGT**
  - - 39%

  (elafibranor 80mg, p=0.001)
- ↓ **5'-nucleotidase**

Inflammation Markers

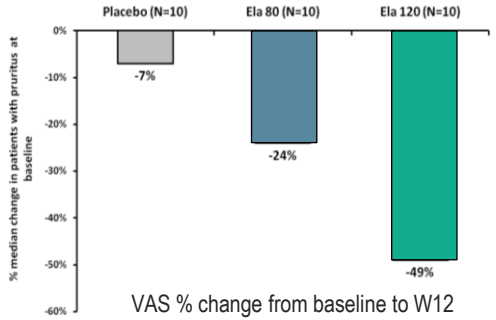
- ↓ **IgM**
- ↓ **hsCRP**

Bile Acid Precursors

- ↓ **C4**

Generally safe and well tolerated

Pruritus trend[1]



VAS % change from baseline to W12

- 24% (elafibranor 80mg)
- 7% (PBO)

Summary of Adverse, Treatment-Emergent Adverse, and Serious Adverse Events

Elafibranor[1] (Phase 2a – 12W study)	80mg (N=15), N (%) [#AEs]	120mg (N=15), N (%) [#AEs]	Placebo (N=15), N (%) [#AEs]
Patients with at any AE	13 (86.7) [46]	13 (86.7) [51]	12 (80.0) [28]
Patients with at any TEAE	12 (80.0) [41]	13 (86.7) [46]	12 (80.0) [25]
Patients with any treatment-related TEAE	2 (13.3) [6]	5 (33.3) [5]	1 (6.7) [1]
Patients with any serious TEAE	0 [0]	2 (13.3) [3]	0 [0]
Patients with any severe TEAE	2 (13.3) [3]	2 (13.3) [5]	2 (13.3) [2]
Patients with any serious treatment related TEAE	0 [0]	1 (6.7) [1]	0 [0]
Patients with any TEAE leading to study drug discontinuation	0 [0]	1 (6.7) [2]	0 [0]

Successful Phase 2a trial in PBC demonstrating efficacy and safety of elafibranor 80mg[1,2]

Note:GGT= gamma-glutamyl transferase, **IgM**= immunoglobulin M, **hsCRP**= High sensitivity C-reactive protein, **C4**= serum 7α-hydroxy-4-cholesten-3-one, **VAS** = Visual Analog Score,
References: 1. Schattenberg et al. J. of Hepatol. 2021;Vol. 74, Issue 6:1344-1354 ; 2. GENFIT Corporate Press Release June 29, 2019 "GENFIT Announces FDA Grant of Breakthrough Therapy Designation to Elafibranor for the Treatment of PBC".

Therapeutics:
Acute on chronic liver failure

ACLF: a New Growth Opportunity for GENFIT

High unmet medical need in ACLF

- **Life threatening disease** with high mortality at 30-90 days[1]
- **Standard of Care (SoC):** no approved drugs[2]
- **Medical need**: a therapy that helps patients to survive ACLF without liver transplantation

Market opportunity

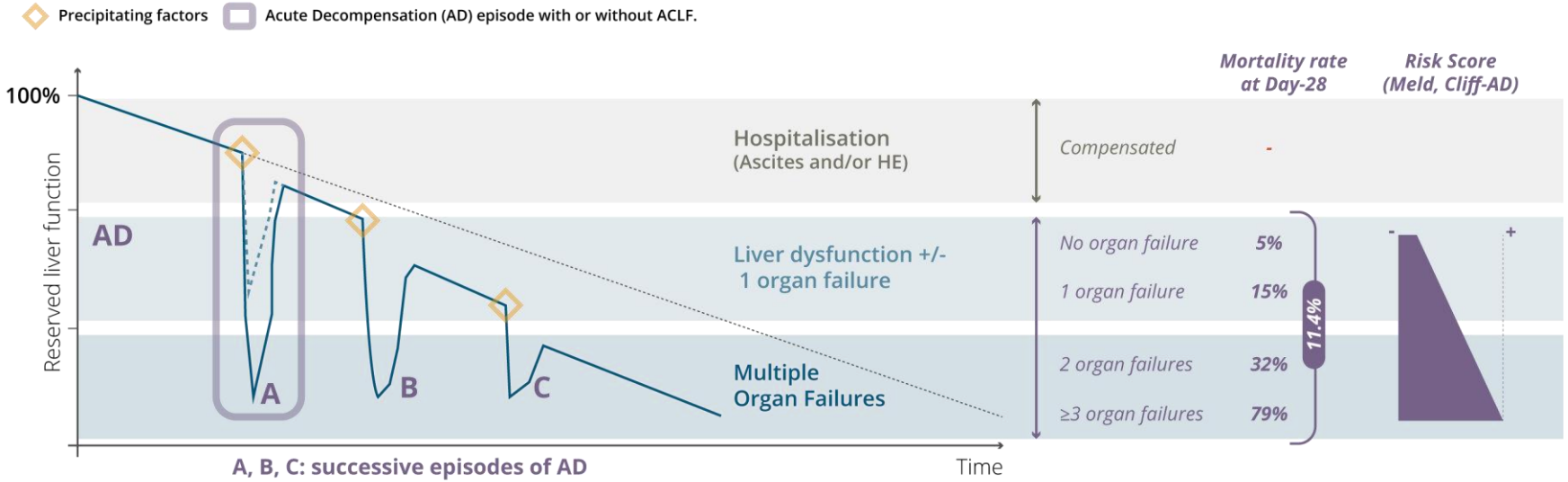
- **Significant cost to the U.S. healthcare system**: in-hospital costs 3.5 times higher for an ACLF patient than for a decompensated cirrhosis patient without ACLF[3]
- **Hospital stays**: 16 days average for ACLF patient (vs 7 days for cirrhotic patient)[4]
- **Nb of patients**: 180,000 in the U.S. only (10-30% prevalence in cirrhotic patients hospitalized in the U.S.)[5]
- **Market opportunity:** up to ~$4bn in the U.S. and ~$2bn in EU[6]
- **Orphan condition:** enhanced market exclusivity and clinical research subsidies[7]

GENFIT's rationale

- **In-house expertise (discovery to late stage) in severe liver diseases**
- **Strong scientific rationale based upon MOA for elafibranor and GFT1575 as well as supportive preclinical data for NTZ**[8]
- **Initiation of clinical program for NTZ in ACLF, on-going preclinical program for GFT1575 and elafibranor**
- **First clinical evidence expected in 2022 (NTZ)**

References:1. Jalan R et al. World Gastroenterol. Org. Working Party. Gastroenterology 2014;147:4-10; 2. Gustot, Discussion Part 2015; 3. Allen et al. Hepatology, 2016;64:2165-2172; 4. Kamath, Acute on Chronic Liver Failure. 2017; 5. Hernaez et al. J. of Hepatol. 2019; 6. Derived from assumptions taken from Delveinsight, ACLF Market Insight, Epidemiology and Market Forecast -2030 Report published in Oct. 2020; 7. Orphan Drug Act of 1983, US Food and Drug Administration. 4 January 1983, PUBLIC LAW 97-414; 8. Shou et al. Inflammation Vol. 42:1336–1349(2019), data on file.

Natural History of AD and ACLF in Cirrhotic Patients



- ACLF Definition : **acute deterioration of preexisting chronic liver disease**, usually related to a **precipitating event** and associated with **increased mortality at three months due to multi-system organ failure.**
- Precipitating factors **: Infection (bacterial, fungal or viral)** / Reactivation of hepatitis B (or C) or superimposed viral hepatitis (e.g hepatitis E in India) / Alcohol / Drug Induced Liver Injury (DILI, herbal medicine) / Gastrointestinal bleeding / Portal vein thrombosis / Surgery / Ischemia / Flare of autoimmune hepatitis or Wilson disease.
- ACLF patient with known chronic liver disease experiencing sudden **multiple organ failures affecting: liver, kidney, coagulation, brain and lung functions**

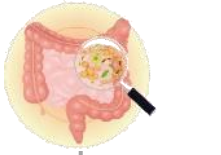
References:1. Kim TY, Kim DJ. Clin Mol Hepatol. 2013 Dec;19(4):349-59; 2. Moreau et al. Gastroenterology.

NTZ and PPARs May Address Multiple Relevant Pathways in ACLF

NTZ effects
PPARs effects

Gut microbiota dysbiosis
(>90% anaerobic Gram negative)
Bacterial overgrowth
Decreased bacterial diversity

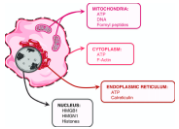
Impaired intestinal barrier and increased bacteria translocation:
Bacterial products (PAMPs: LPS, peptidoglycan….)
Non-pathogen and pathogen bacteria



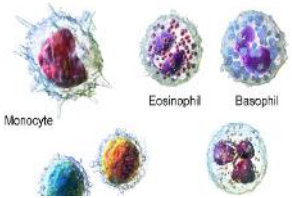
PAMPs
(LPS, peptidoglycan…)

Cirrhotic liver

Dying cell



DAMPs
(HMGB1, mitDNA,ATP…)



Overactivation of innate immune system by PAMPs and DAMPs

Cytokine storm:
- Increased TNFa, IL6, IL1b, IL8
- Increased ROS NOS

Exacerbated Metabolic stress
- Increased aerobic glycolysis
- FFA accumulation and lipotoxicity
- Mitochondrial dysfunction
- Decreased beta-oxidation
- Decreased OXPHOS

Multiple Organ Failures (MOFs)
➔ ACLF ➔ Death

Organ injury and acute loss of function
Oxidative stress, Apoptosis, Necrosis

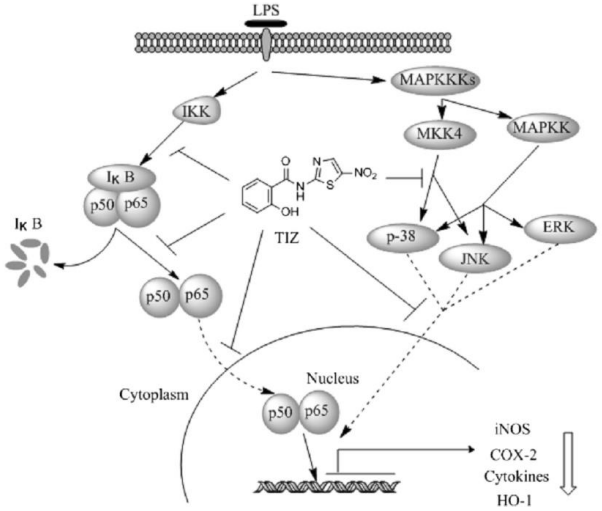
PAMP: pathogen-associated molecular patterns; DAMP: damage-associated molecular patterns

References:1. Arroyo V et al. J. Hepatol, 2021 Mar;74(3):670-685; 2. Trebicka J et al. Nat Rev Gastroenterol Hepatol 2021 Mar;18(3):167-180.

Highlights from the Literature on NTZ and PPARs in ACLF

NTZ

- **Anti-infectious** properties may act on intestinal microbiota dysbiosis/overgrowth and improve intestinal barrier.
- Direct dose-dependent **anti-inflammatory** effects on immune cells (macrophages and PMN).



References:1. Shou et al. Inflammation Vol. 42:1336–1349(2019); 2. Dubreuil et al. Antimicrob Agents Chemother 1996 Vol.40:2266–2270; 3. Hecht et al. Antimicrob Agents Chemother 2007 Vol.51:2716–2719; 4. Hoffman et al. Antimicrob Agents Chemother 2007 Vol.51:868–876; 5. MacVay et al. Antimicrob Agents Chemother 2000 Vol.44: 2254-2258; 6. Megraud et al. Antimicrob.Agents Chemother. 1998 Vol. 42:2836-2840; 7. Musher et al. Clinical Infectious Diseases 2006 Vol.43:421-427; 8. Pankuch et al. Antimicrob Agents Chemother. 2006 Vol.50(3):1112-7.

PPARs

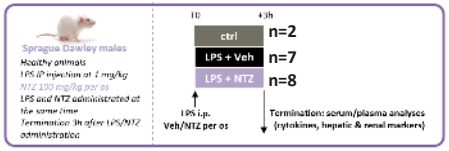
- In ACLF, an over-activation of the innate immune-system induces a **critical energetic adaptation** i) increased aerobic glycolysis ii) profound decrease in FFA beta-oxidation[1,2]
- In the liver and other peripheral organs, an over-metabolic adaptation leads to:
  - Energy deprivation
  - FFA acid accumulation and lipotoxicity
  - Mitochondrial dysfunction and ER stress
  - Oxidative stress
  - Hepatocyte necrosis and apoptosis…[1,2]
- **PPARs are master switches of energetic adaptation**, and their activation directly **modulates inflammatory response** of immune cells. [3]
- In animal models, over-activation of the immune system, some selective agonists (PPARα, PPARγ and PPARδ) have shown certain **favorable effects on MOF's and mortality.** [4]

Notes: MOF=Multiple organ failures

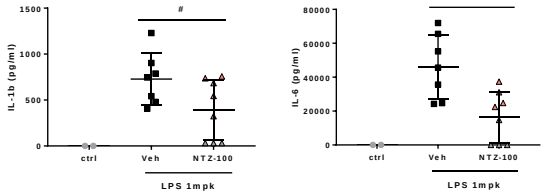
References:1. Zaccherini G. JHEP Reports 2021 Vol.3:100176; 2. Moreau et al. J. of Hepatol. 2020 Vol.72(4):688-701; 3. Standage et al. Critical Care Medicine 2016 Vol. 44(6):594-603; 4. Paumelle et al. J. of Hepatol. 2019 Vol.70(5):963-973.

Evidence Generated by GNFT – NTZ in ACLF

In vivo model #1



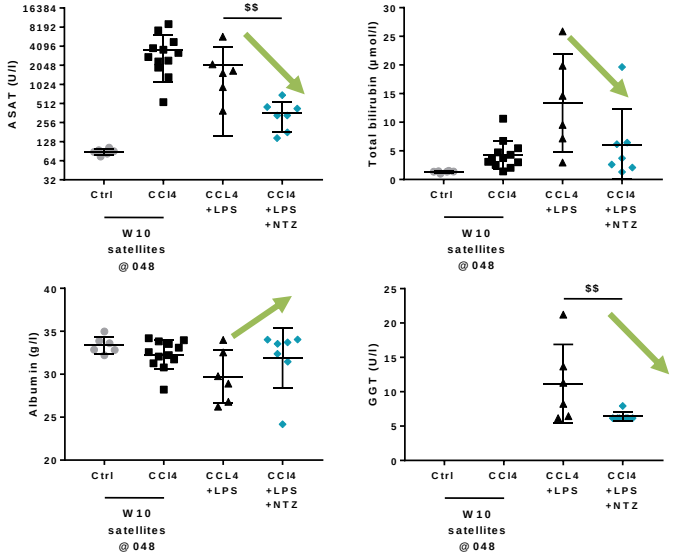
Activity on inflammatory markers



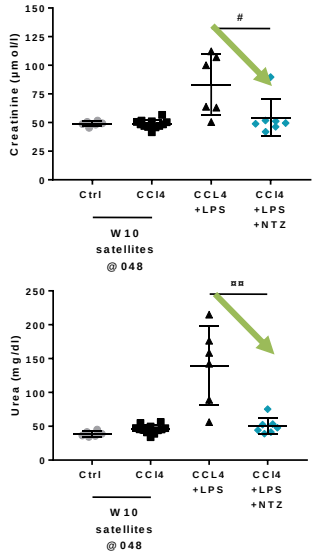
Activity on liver enzymes

In vivo model #2 (ACLF)

Activity on liver failure markers



Activity on kidneyfailure markers



Reference: GENFIT data on file. In this experiment, 40% mortality was observed in each group. liver and kidney failure markers were measured on the remaining 60% survivors.

Diagnostics: NASH

NIS4® Technology to Diagnose Millions of Patients with Active NASH and Fibrosis

High unmet medical need in NASH diagnostics

- 6.7M patients have NASH and significant fibrosis (F≥2) in the US[1†], only 900,000 are diagnosed
- Poor disease awareness among patients with NAFLD due to nonspecific symptoms[2,3]
- Liver biopsy, the reference standard for NASH, poses risks for patients and has technical limitations[4]
- Patients who have NASH and Significant Fibrosis (F≥2), also referred to as at-risk NASH, are at increased risk of developing cirrhosis and/or complications of severe liver disease [5-7]
- There are no non-invasive diagnostic tests specifically developed to identify at-risk NASH

GENFIT's rationale

To identify patients with at-risk NASH, GENFIT has invested in a strategic R&D program:

- **Statistical analysis of >100 circulating blood-based biomarkers[5]**
- **Comparison of results against liver biopsy results[5]**
- **Testing and validation in 3 independent cohorts with suspected NAFLD[5]**
- **Designed for utilization and commercialization in clinical research and clinical management settings**

Market opportunity

- Prevalence of NASH among patients with diabetes: 25 to 30%[8]
  - Diabetes patients in U.S.: 34M (13% = prevalence of diabetes in U.S. population) 2010 US Census: 308,745,538 U.S. population (74,181,467 U.S. population under age 18 + 234,564,071 U.S. adults)[9]
- Prevalence of NASH among patients with obesity (BMI>30): 25 to 30%[8]
  - Obese patients in U.S.: 94M- (39.8% = prevalence of obesity in U.S.)[10]

Go To Market

- NASHnext™ clinical diagnostic launched by Labcorp in April 2021.

Test is powered by GENFIT's NIS4® Technology

Note: †Case numbers for the specified populations are approximated as follows: Active NASH and fibrosis, i.e. at-risk NASH is F2 + F3 + F4, NASH with cirrhosis is F4, and ESLD is decompensated cirrhosis.
References: 1. Estes et al. Hepatology. 2018;67(1):123-133; 2. Chalasani et al. Hepatology. 2018;67(1):328-357; 3. Bugianesi et al. J. of Hepatol. 2005;42(5):784-785; 4. Cleveland et al. Clin Liver Dis (Hoboken). 2018;11(4):98-104; 5. Harrison et al. Lancet Gastroenterol Hepatol. 2020; 5(11):970-985; 6. Angulo et al. Gastroenterology. 2015;149(2):389-397.e10; 7. Sanyal et al. Presented at: The Liver Meeting 2019 (abstr 1190); 8Anstee, Q. M. & Day, C. P. Nat. Rev. Gastroenterol. Hepatol. 10, 645–655 (2013); 9. National Diabetes Statistics Report 2020; 10. National Center for Health Statistics.

NIS4®: A Proprietary and Differentiated Technology for the Diagnosis of At-Risk NASH

Currently Available Diagnostic Method	NASH Activity	Fibrosis	Standard Ordering HCP	Method	Designed for NASH
NIS4®,1	✓	✓	**Any healthcare provider**	**Non-invasive**	✓
BIOPSY	✓	✓	**Hepatologist/GI**	Invasive	-
ULTRASOUND	✓ Steatosis Only	-	**Any healthcare provider**	**Non-invasive**	-
FibroScan®	✓ Steatosis Only	✓	**Hepatologist or GI**	**Non-invasive**	-
NFS	-	✓	**Any healthcare provider**	**Non-invasive**	-
FIB-4	-	✓	**Any healthcare provider**	**Non-invasive**	-
APRI	-	✓	**Any healthcare provider**	**Non-invasive**	-
ELF™	-	✓	**Hepatologist/GI**	**Non-invasive**	-

Note: At-risk NASH as defined by NASH Activity Score (NAS)≥4 and Fibrosis stage (F)≥ 2.
NFS (NAFLD fibrosis score), FIB-4 Index (Fibrosis-4), APRI (AST / platelet ratio index), FibroScan® is a registered trademark of EchoSens™, Paris. ELF™ (Enhanced Liver Fibrosis) is a trademark of Siemens Healthineers and is not commercially available in the U.S. but used widely outside of the U.S.

References: 1. Harrison et al. Lancet Gastroenterol Hepatol. 2020; 5(11):970-985

NIS4® Technology: An Innovative Approach Built Upon miRNA Science

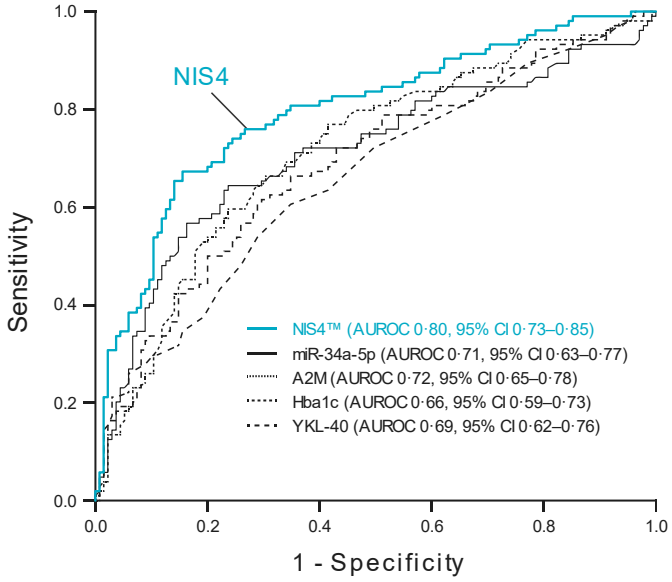
NIS4® is the only non-invasive, blood-based technology specifically designed to assess both NASH activity and liver fibrosis among patients with metabolic risk factors

NIS4® assigns a single score that ranges from 0.00 to 1.00 based on blood/serum levels of 4 biomarkers:

- **miR-34a-5p**
- **Alpha2-macroglobulin (A2M),**
- **YKL-40,**
- **Hemoglobin A1c (HbA1c),**

- NIS4® significantly outperformed other blood biomarker-based NASH or fibrosis diagnostics, including FIB-4, NFS, APRI, and ELF for the detection of at-risk NASH
- NIS4® performance data have been generated against liver biopsy in more than 900 patients across the NASH/NAFLD spectrum

Comparison of NIS4 vs individual biomarker components to identify patients with at-risk NASH within the discovery cohort (n=239)*



Note: At-risk NASH (NAS≥4,F≥2), NAS, non-alcoholic fatty liver disease activity score; AUROC, area under the receiver operating characteristics curve.
References: Harrison et al. Lancet Gastroenterol Hepatol. 2020; 5(11):970-985

NIS4® Technology Published in *The Lancet Gastroenterology & Hepatology*

THE LANCET
Gastroenterology & Hepatology

A blood-based biomarker panel (NIS4) for non-invasive diagnosis of non-alcoholic steatohepatitis and liver fibrosis: a prospective derivation and global validation study[1]

Stephen A. Harrison, Vlad Ratziu *et. al.* | Accepted for publication in *The Lancet Gastroenterology & Hepatology* Aug. 5, 2020

NIS4® Technology

- High diagnostic performance with low misclassification rates to rule in and rule out at-risk NASH
- Consistent test performance vs. other tests – more consistent results irrespective of BMI, gender, presence or absence of diabetes, dyslipidemia, hypertension, or aminotransferase levels
- Provides a definitive diagnosis of at-risk NASH for over 72% of patients with high accuracy

Tests powered by NIS4® technology can be adapted to different clinical trial or clinical goals

- Potential to reduce unnecessary liver biopsies in patients with lower risk of disease progression
- May improve referral pathways amongst multiple patient sub-populations to liver specialty care
- May enable earlier identification of higher risk patients and allow for focused patient management to mitigate disease progression

GENFIT
TOWARDS BETTER MEDICINE

Note: *There is currently no NIS4-based test approved as an IVD. NIS4® Technology has been licensed to Labcorp, which has used it to develop and launch its own NIS4-based for use in the commercial setting.

References: **1.** Harrison et al. Lancet Gastroenterol Hepatol. 2020; 5(11):970-985

The Progression and Future of NIS4® Technology

A test powered by clinically validated NIS4® technology is the simple solution to identify at-risk NASH patients

Licensing of NIS4® Technology for research use in clinical trials
Signed with Labcorp-Covance
1Q19

Commercial availability of NIS4® Technology in clinical research (Covance)
Utilization in clinical trials
4Q19

NIS4® Technology Pivotal Publication in *The Lancet G&H*[3]
NIS4® derivation and validation
3Q20

Licensing of NIS4® Technology for Commercialization of a NASH Diagnostic Test
Signed with Labcorp
3Q20

Launch of NASHnext™ based on NIS4® Technology
By Labcorp
2Q21

A development and commercialization plan for an untapped market

Large scale commercial launch of NASHnext™ by Labcorp: a non-invasive, blood-based, molecular LDT for the identification of patients with at-risk NASH powered by GENFIT's NIS4® Technology:

Target Populations with Suspected NASH

- Diabetes patients in U.S.: 34M[1]
- Obese (BMI≥30) patients in U.S.: 94M[2]

Upcoming projected development milestones

- Future Submission to FDA for IVD approval
- Future Submission to EU Notified Body for CE mark

LDT: Laboratory Developed Test ; **IVD:** In vitro Diagnostic ; **CE mark:** (*Conformité Européenne)* mandatory conformity marking for certain products sold within the European Economic Area (EEA) since 1985.
References: 1. National Diabetes Statistics Report 2020 (13% = prevalence of diabetes in U.S. population) ; +2010 US Census: (308,745,538 U.S. population – 74,181,467 U.S. population under age 18 = 234,564,071 U.S. adults); 2. National Center for Health Statistics (39.8% = prevalence of obesity in U.S.); 3. Harrison et al. Lancet Gastroenterol. Hepatol. 2020; 5(11):970-985.

NASH Diagnostics: Accelerating Technology

A platform of fit-for-purpose NASH diagnostic solutions

Commercial

NIS4® Technology

- NIS4® Research*
- NIS4® LDT*
- NIS4® IVD

Today, addressing the unmet need of identifying people with at-risk NASH

NASHNext™
Powered by NIS4® Technology

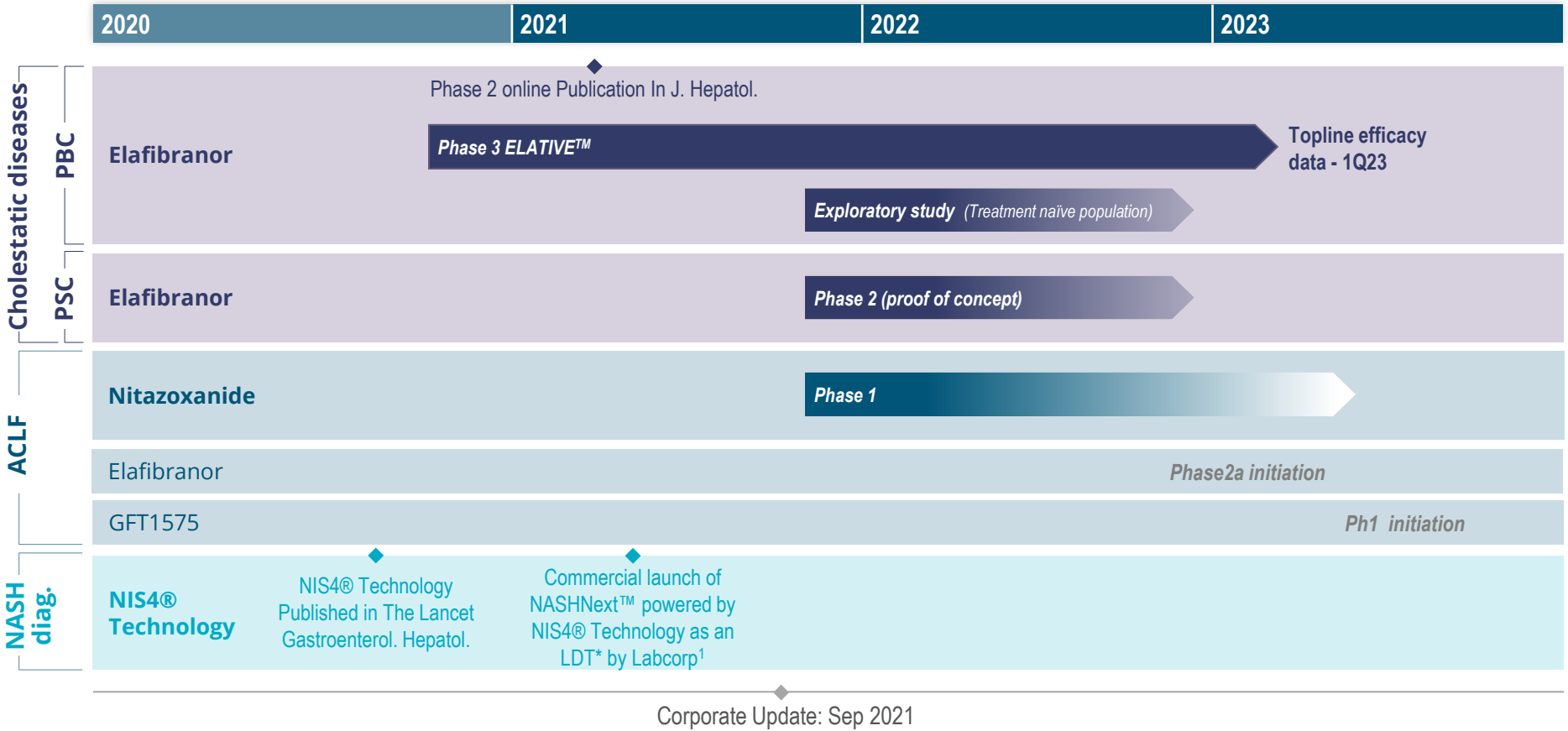
labcorp
Learn more

R&D

- **Disease Progression** — NAFL/NASH Monitoring
- **Partnership** — Targeted Populations
- **Artificial Intelligence** — Automated Biopsy Reading

Note: *NIS4®Technology has been licensed to Labcorp, which has used it to develop and launch its own NIS4® Technology-based research tool and NIS4® Technology-based LDT for use in the commercial setting. There is currently no NIS4® Technology-based test approved as an IVD.

Development Roadmap



Upcoming milestones, data announcements and launch dates are anticipated and subject to change. Elafibranor is an investigational compound and has not been approved by any regulatory authority in any indication.
LDT: Laboratory developed test. 1. Exclusive licensing to Labcorp for development and commercialization of NIS4® Technology to power a next-generation NASH diagnostic test

GENFIT: A Pioneer in the Diagnosis and Treatment of Liver Disorders

Leader in PPAR research • Pioneer in NASH and PBC • Proven team with global scientific, regulatory, and commercialization expertise

Milestones

Cholestatic diseases Therapeutics

- 3Q20 ELATIVE™ Clinical study beginning of enrolment
- 1Q21 Publication of Phase 2 study evaluating elafibranor in PBC in J. of Hepatol.
- 2Q21 Creation of a new franchise on cholestatic diseases - Pre-clinical R&D programs progress to clinical phase

ACLF Therapeutics

- 2Q21 Creation of the new franchise - Pre-clinical R&D programs progress to clinical phase

NASH Diagnostics

- 1Q19 Licensing agreement with Labcorp-Covance for use of NIS4® technology in clinical research
- 2Q19 Partnership with Terns Pharmaceuticals for commercialization of elafibranor in Greater China (PBC)
- 3Q20 Exclusive licensing agreement with Labcorp for NIS4® Technology development and commercialization of a NASH diagnostic test
- 2Q21 Commercial launch of NASHnext™ powered by NIS4® technology as an LDT by Labcorp

Financials

- 1Q21 Renegotiation of convertible debt & corporate reorganization: Convertible debt maturity pushed back to Oct 2025
- 1Q21 Cash position: €108.9M
- 2Q21 Residual convertible debt down to nominal amount of €57.2M as of April 13, 2021

Next step September 2021: Corporate update