Full Year 2020 Financial Results

March 5, 2021

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Today's speakers

Frédéric Cren, MA/MBA, Chairman, CEO and Co-Founder

Pierre Broqua, Ph.D., CSO and Co-Founder

Michael Cooreman, MD, CMO

Jean Volatier, MA, CFO

Summary

- **Full year 2020 highlights**
- **Clinical pipeline update**
- **Financials**
- **Near-term catalysts**

Full Year 2020 Highlights

Full year 2020 highlights

Lanifibranor program in non-alcoholic steatohepatitis (NASH)

- **Launch of collaboration with Pr. Jérôme Boursier**, M.D., Ph.D, Professor of Medicine Angers University, **to develop biomarkers** to identify patients responding to lanifibranor with regards to NASH resolution and fibrosis improvement
- Confirmation by FDA that the **non-clinical toxicology package is complete and acceptable to support NDA filing** for the treatment of NASH and improvement of liver fibrosis
- **Positive topline results** from the NATIVE Phase IIb clinical trial in NASH
- **Decision by Pr. K Cusi to reduce the number of patients** in the trial evaluating lanifibranor in T2DM with NAFLD following higher than expected observed effects of lanifibranor in reducing steatosis during the NATIVE Phase IIb trial
- **Breakthrough Therapy** designation from FDA in NASH
- **Publication of new pre-clinical data on lanifibranor for the treatment of cirrhosis** in the Journal of Hepatology
- **Finalization of the design of the pivotal Phase III clinical trial with lanifibranor in NASH** following the end-of-phase II meeting with the FDA and the receipt of the Scientific Advice letter from the EMA
- Approval of a **new patent directed at the use of lanifibranor** for the treatment of several fibrotic diseases, including NASH, in China

Odiparcil program in mucopolysaccharidoses (MPS)

- **Publication of latest data on odiparcil's mechanism of action** in the leading peer-reviewed scientific journal PLOS ONE
- **Extension of the duration of the Phase I/II SAFE-KIDDS** trial in MPS VI children from 6 to 12 months following a scientific advice meeting with the EMA
- **Acceptance of the Investigational New Drug (IND)** application for odiparcil in MPS VI by the U.S. FDA
- **Fast Track designation** from FDA in MPS VI
- **Decision to focus our efforts on NASH** and to review all available options to optimize the development odiparcil. Suspension of all MPS-related R&D activities during such time

Full year 2020 highlights

Collaboration with AbbVie in auto-immune diseases

- Completion by AbbVie of the International Nonproprietary Names (INN) process for ABBV-157, now named **cedirogant**
- Confirmation by AbbVie of **clinical POC** in psoriasis with cedirogant in **Q2 2021**

Other

- **Opening of Inventiva's subsidiary in the U.S.** ahead of the initiation of the pivotal Phase III clinical trial with lanifibranor in NASH
- **Appointment of Dr. Michael Cooreman, M.D.,** as Inventiva's Chief Medical Officer based in the United States
- **Appointment of Dr. Arun J. Sanyal** to Inventiva's Scientific Advisory Board (SAB)

Financials

- **Capital increase of €15 million** subscribed by BVF Partners L.P., New Enterprise Associates (NEA), Novo Holdings A/S and Sofinnova Partners
- **Entry into a €10.0 million non-dilutive loan facility guaranteed by the French State** ("Prêt Garanti par l'Etat")
- **Successful €94.9 million IPO on the Nasdaq** in the United States
- Extension of **cash runway through Q4 2022**

Clinical pipeline update

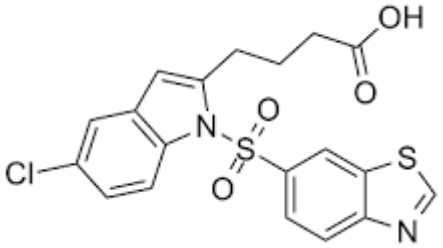
Lanifibranor

A new generation pan-PPAR agonist for the treatment of fibrotic conditions

Lanifibranor: the only pan-PPAR agonist in clinical development for the treatment of NASH and improvement in liver fibrosis

LANIFIBRANOR

Moderate and balanced pan-PPAR agonist activity



- **Differentiated chemical structure**
- **Once daily oral administration**

- Composition of matter patent granted in 55 countries and method of use patent granted in the US, China and in the EU: **limit of exclusivity in the US is 2035**
- **Breakthrough Therapy and FAST Track** designations granted by FDA

Compound	PPARα EC50 (nM)	PPARδ EC50 (nM)	PPARγ EC50 (nM)
Lanifibranor*	**1630**	**850**	**230**
Fenofibrate	*2400*	*-*	*-*
Pioglitazone	*-*	*-*	*263*
Rosiglitazone	*-*	*-*	*13*
*Elafibranor***	*10*	*100*	*-*
*Seladelpar^*	*-*	*2*	*-*

Results justifying a NASH development

- **Effects observed** on insulin-sensitivity, dyslipidemia, steatosis, ballooning, inflammation, hepatic fibrosis and cirrhosis in preclinical models
- **Phase IIa(^^) trial demonstrated pan-PPAR agonist activity,** supporting dose selection for NASH clinical trial

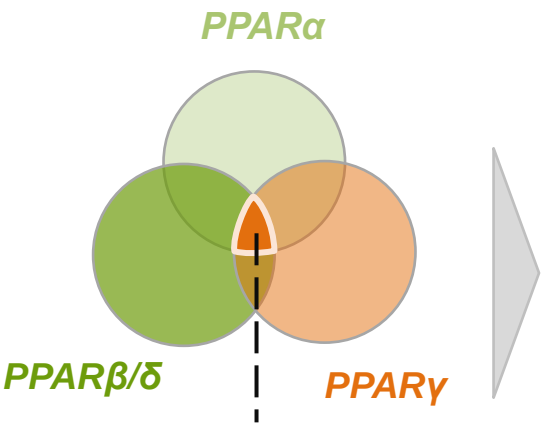
Favorable tolerability profile

- 24-months rodent and 12-month monkey studies leading to **PPAR class clinical hold lifted** by FDA
- Phase I trials with more than **200** healthy volunteers(^^^) and Phase IIa trial with **47** TD2M patients
- Approximately **250** patients treated for 24 or 48 weeks in Inventiva's completed Phase IIb clinical trials
- In connection with these trials, lanifibranor has undergone a total of **7 DSMB reviews without recommendations of protocol change**

(*) Company data; (**) Hanf R et al, Diabetes & Vascular Dis Res 2014; (^) Cymabay company presentation; (^^) Conducted by Abbott prior to our founding; (^^^) Including 125 healthy volunteers in the phase I conducted by Abbott prior to our founding

Lanifibranor's activation of the three PPAR isoforms designed to address the key features of NASH

LANIFIBRANOR



METABOLISM

PPARα PPARδ PPARγ

- ↑ Insulin sensitivity
- ↑ HDLc
- ↓ Triglycerides

STEATOSIS

PPARγ

- ↓ FA uptake
- ↑ FA catabolism
- ↓ Lipogenesis

INFLAMMATION AND BALLOONING

PPARα PPARδ PPARγ

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

FIBROSIS

PPARδ PPARγ

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

VASCULAR

PPARα PPARγ

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

Adverse events and toxicity previously seen in other single and dual PPAR agonists are not observed in lanifibranor

SAFETY

Organ	Isoforms activated	Reported PPAR side effects	lanifibranor effects
 HEART	*PPARγ*	▶ Fluid retention ▶ Cardiac hypertrophy	NOT OBSERVED
 SKELETAL MUSCLE	*PPARα*	▶ Myofiber degeneration	
 KIDNEY	*PPARα*	▶ > 50% increases in creatinine, degenerative changes in renal tubules	
 URINARY BLADDER	*PPARγ*	▶ Proliferative changes in bladder epithelium	

Adverse events and toxicity of single / dual PPAR agonists not observed in primate and rodent studies

FAVOURABLE TOLERABILITY PROFILE in a 12-month monkey study …

- ▶ No adverse clinical signs observed at any dose-level tested
- ▶ No effects on body and heart weight, no haemodilution or creatinine increase
- ▶ Electrocardiography and clinical pathology investigations did not reveal any undesirable effects

… and in two-year **CARCINOGENITY STUDIES** performed in rat and mice

- ▶ Rat: no observed neoplastic change or increase in tumor types commonly associated with single PPARγ and dual PPARα/γ agonists (liver, adipose, bladder, renal and skin)
- ▶ Mice: no observed neoplastic changes of human relevance

Confirmation by FDA that the non-clinical toxicology package is complete and acceptable to support NDA filing

Source: Company data

The overall development plan builds on the successful outcomes of the NATIVE Phase IIb trial

CLINICAL DEVELOPMENT

Phase I → **Phase IIa** → **Phase IIb (NATIVE)** → **F2-F3 Phase III** → **Registration**

Conducted by Abbott prior to Inventiva's founding

IIb DESIGN

- *24 weeks*
- *800 mg and 1200 mg once-daily lanifibranor*

IIb EFFICACY

- *First candidate to achieve statistically significant results on the two Phase III FDA and EMA primary endpoints*

IIb SAFETY

- *Favourable profile*

III OVERVIEW

- *72 week Part 1 + Part 2 extension period*
- *Inclusion criteria and patient profile in line with NATIVE Phase IIb*
- *Primary composite endpoint combining NASH resolution and fibrosis improvement*

Study in NAFLD patients with T2DM

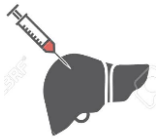
F4 potential development pathways

Combination therapies (outlook)

The Phase IIb NATIVE trial studied 800 mg and 1200 mg once-daily lanifibranor across 247 patients

PHASE IIb **DESIGN** **OVERVIEW**

Screening
▶ **Liver biopsy**



24-week treatment + 4-week follow-up
Double blind, randomized, placebo-controlled

Placebo

lanifibranor, 800 mg once daily

lanifibranor, 1200 mg once daily

End of treatment
▶ **Liver biopsy**

- Randomisation 1/1/1
- Stratification on type 2 diabetes mellitus (T2DM)

Patient population	# patients	Definition
Safety / Intention-to-Treat (ITT)	247	Patients randomized having received at least one dose of lanifibranor/placebo
Per Protocol (PP)	194	Patients with paired biopsies and without deviation impacting efficacy results

▶ **Main inclusion criteria**: patients with biopsy-proven NASH confirmed by central reader having Steatosis-Activity-Fibrosis (SAF) scores of 1-3 for steatosis, 3-4 for activity, and <4 for fibrosis

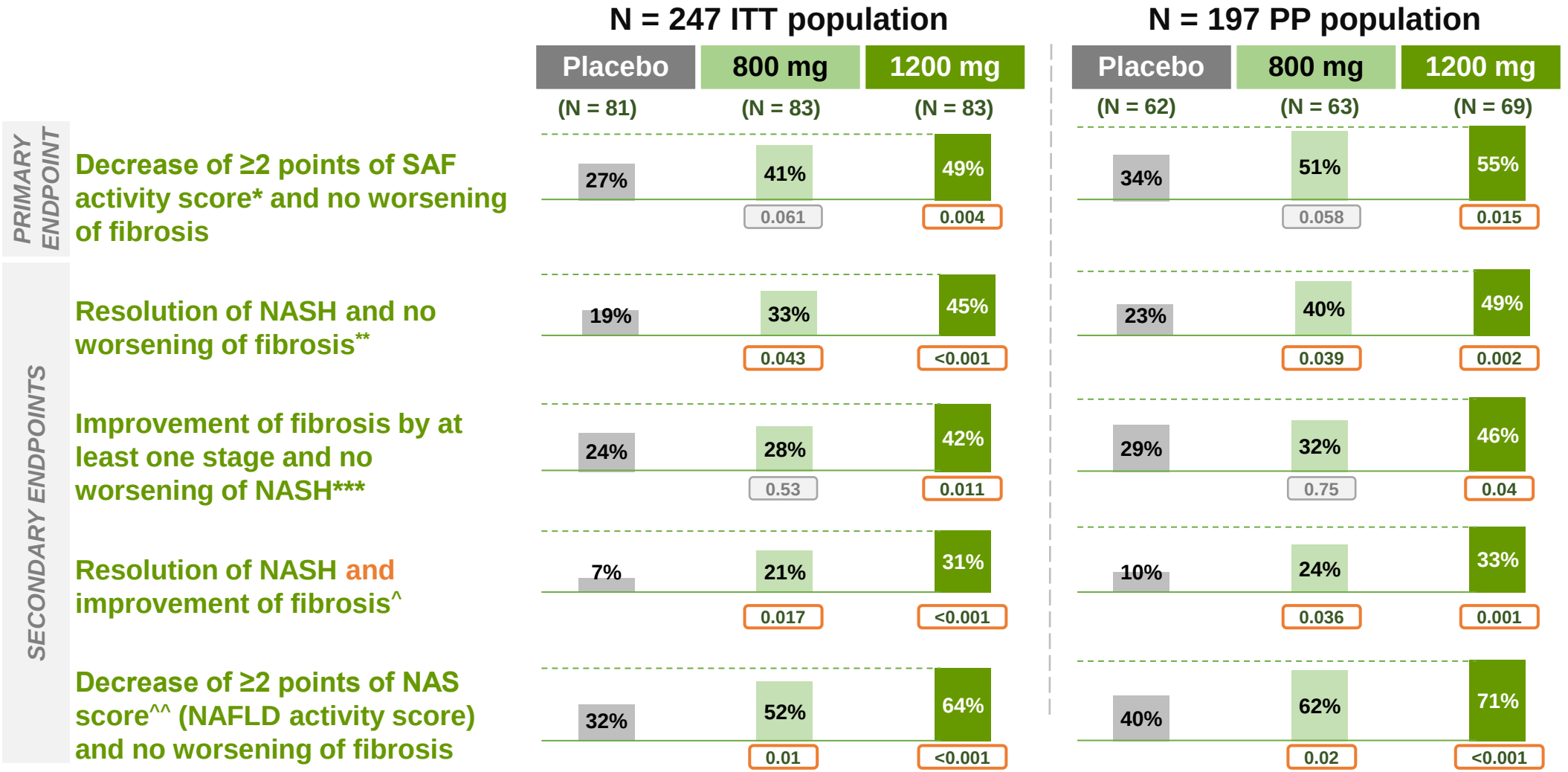
More information on: http://www.native-trial.com/

Lanifibranor is the first candidate to achieve statistically significant results on the two Phase III FDA and EMA primary endpoints

PHASE IIb | EFFICACY | KEY ENDPOINTS

xx Statistically significant xx Non-statistically significant

Key Phase IIb results by endpoint



** Response is defined as a decrease from baseline to week 24 of at least 2 points of the SAF Activity score (SAF-A) with no worsening of the NAS Fibrosis score (NAS-F). No worsening means that score remains stable or decreases ; ** Resolution of NASH and no worsening of fibrosis at week 24: NAS-I = 0 or 1 (NAS-Inflammation), NAS-B = 0 (NAS-Ballooning) and no worsening of NAS-F from baseline; *** Improvement of liver fibrosis ≥ 1 stage and no worsening of NASH at week 24; ^ Resolution of NASH and improvement of fibrosis at week 24: NAS-I = 0 or 1, NAS-B = 0 and an improvement of NAS-F ≥ 1 stage; ^^ NAS score is a commonly accepted, semi-quantitative evaluation of biopsy results that assesses the severity of steatosis, inflammation and ballooning in the liver.*

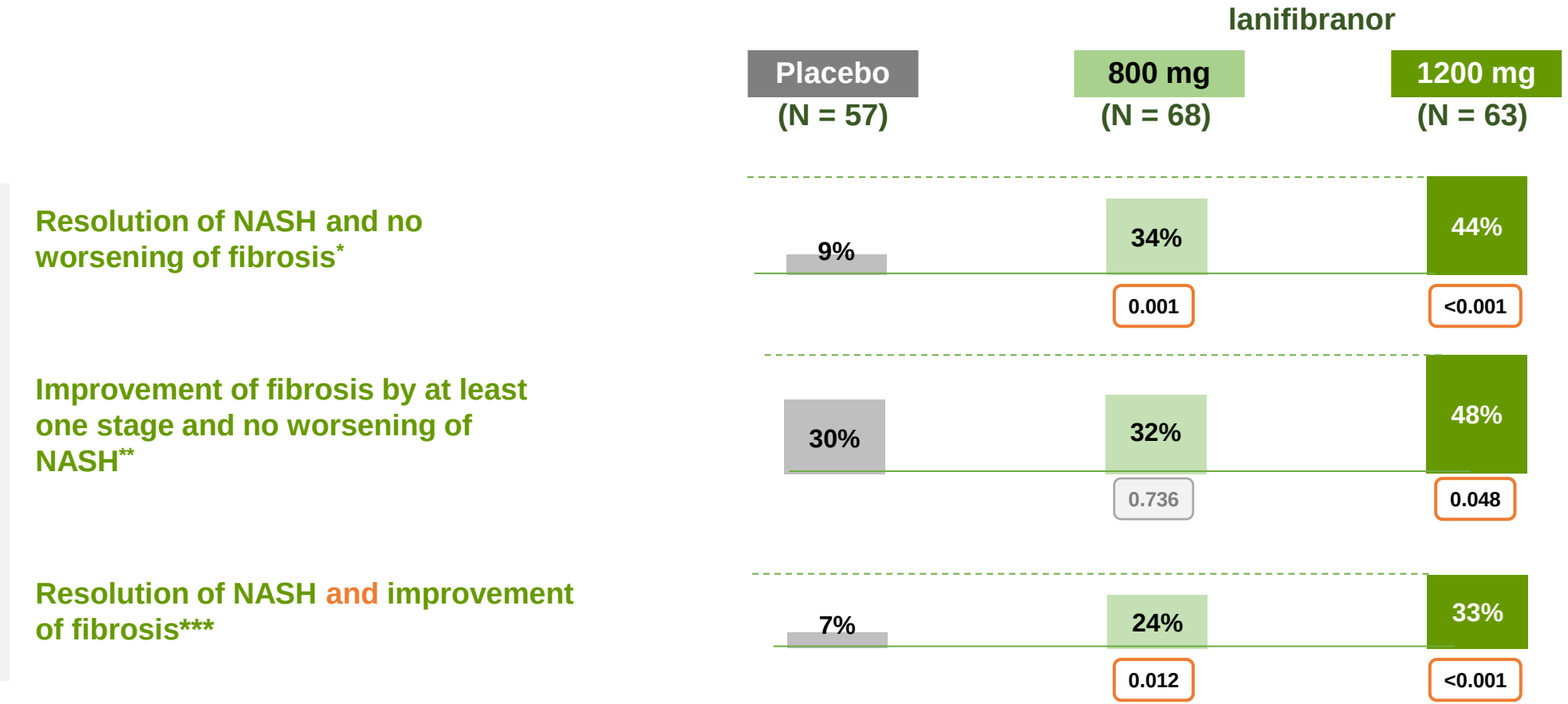
In F2-F3 patients, statistical significance was demonstrated for the main key histological secondary endpoints

PHASE IIb | EFFICACY | F2-F3 POPULATION

xx Statistically significant xx Non-statistically significant

Key secondary endpoints in FAS F2 F3 patients (N=188)

SECONDARY ENDPOINTS



- Similar results in the PP population
- Consistent response in diabetic and non-diabetic patients

** Resolution of NASH and no worsening of fibrosis at week 24: NAS-I = 0 or 1 (NAS-Inflammation), NAS-B = 0 (NAS-Ballooning) and no worsening of NAS-F from baseline; ** Improvement of liver fibrosis ≥ 1 stage and no worsening of NASH at week 24; *** Resolution of NASH and improvement of fibrosis at week 24: NAS-I = 0 or 1, NAS-B = 0 and an improvement of NAS-F ≥ 1 stage*

A significant decrease in circulating biomarkers was observed under lanifibranor treatment after 24-weeks

AASLD November 13-16, 2020 The Liver Meeting® Digital Experience

PHASE IIb | EFFICACY | OTHER

OTHER OUTCOME MEASURES

Median relative change (%)		Placebo	lanifibranor *(Two doses pooled)*	Pvalue
Fibrosis	Pro-C3	(4.1%)	(13.9%)	*p= 0.005**
	Pro-C3 >14 at baseline	(12.8%)	(20.5%)	*p= 0.017**
	Ratio TIMP-1/MMP-2	(4.6%)	(22.5%)	*p < 0.001**
Apoptosis	CK18-M30	0.5%	(41.1%)	*p < 0.001**
Inflammation	Ferritin	(9.1%)	(29.4%)	*p < 0.001**
	hs-CRP	13.0%	(35.5%)	*p < 0.001**

(1) Level where it is estimated that fibrogenesis is active and corresponding to F2/F3 patients
FAS (Full Analysis Set) population with available data at baseline and at week 24
** Statistically significant*

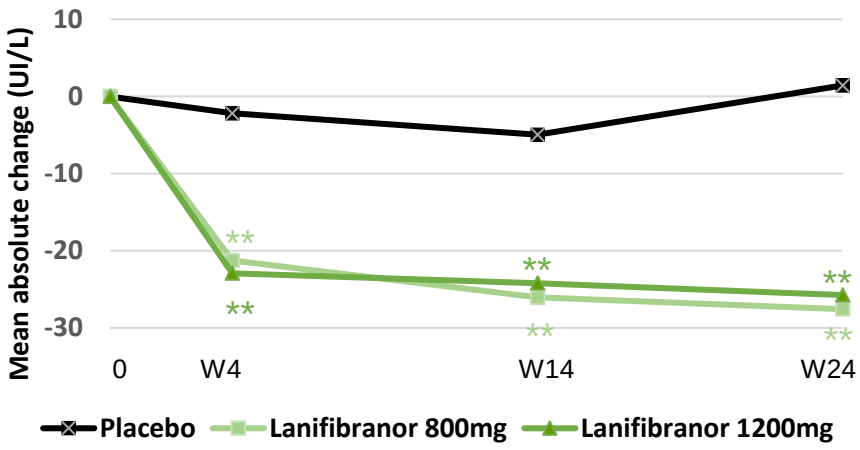
A statistically significant decrease in liver enzymes was observed

PHASE IIb | EFFICACY | OTHER

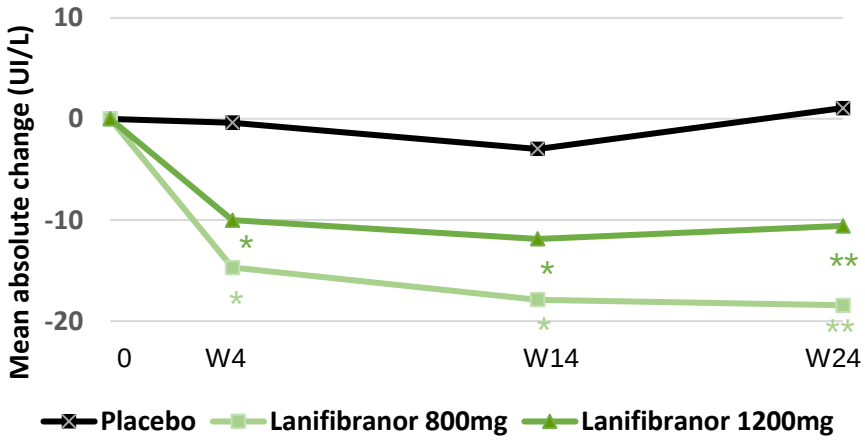
Other secondary endpoints in ITT (N = 247)

SECONDARY ENDPOINTS

Absolute change from baseline in ALT

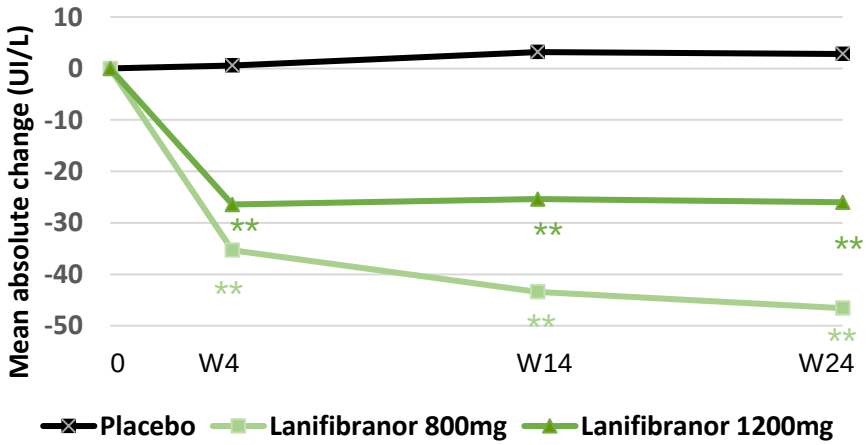


Absolute change from baseline in AST



* p<0.01 **p<0.001

Absolute change from baseline in GGT



A statistically significant decrease of ALT, AST and GGT in both lanifibranor dose groups observed after 4 weeks

A statistically significant change in HDL-cholesterol and triglycerides was seen, without a change in LDL-cholesterol

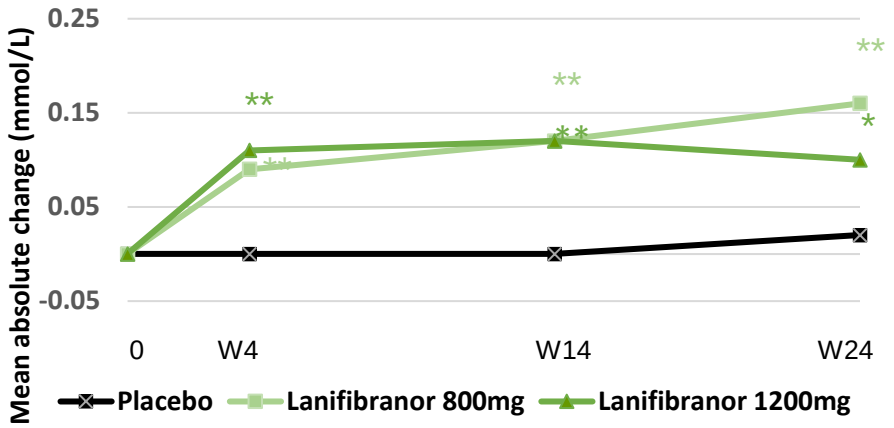
PHASE IIb | EFFICACY | OTHER

Other secondary endpoints in ITT (N = 247)

* p<0.01 **p<0.001

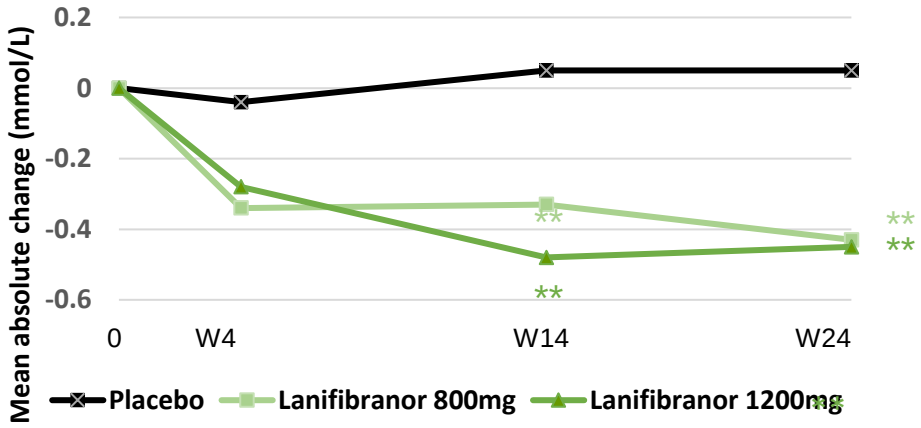
SECONDARY ENDPOINTS

Absolute change from baseline in HDL-C



Statistically significant change in HDL-cholesterol

Absolute change from baseline in triglycerides



Statistically significant change in triglycerides

- No change in LDL-cholesterol

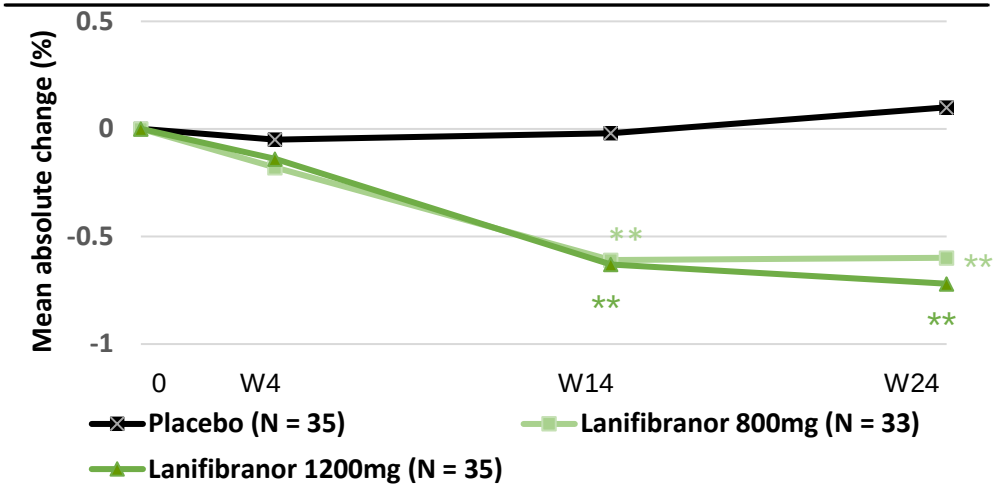
In type 2 diabetes (T2DM) patients with NASH, statistically significant reductions of fasting glucose and insulin, HbA1c were observed

PHASE IIb | EFFICACY | T2DM

Secondary endpoints in T2DM patients with NASH (N = 103)

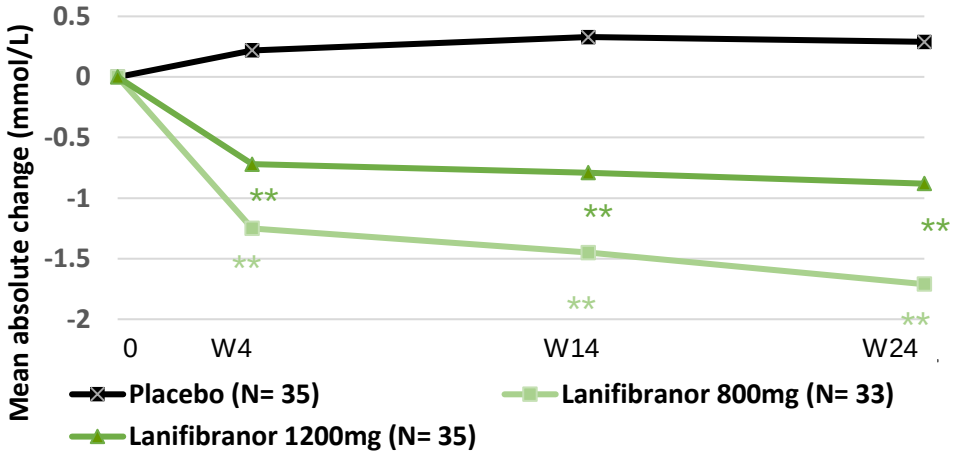
SECONDARY ENDPOINTS

Absolute change from baseline in HbA1c

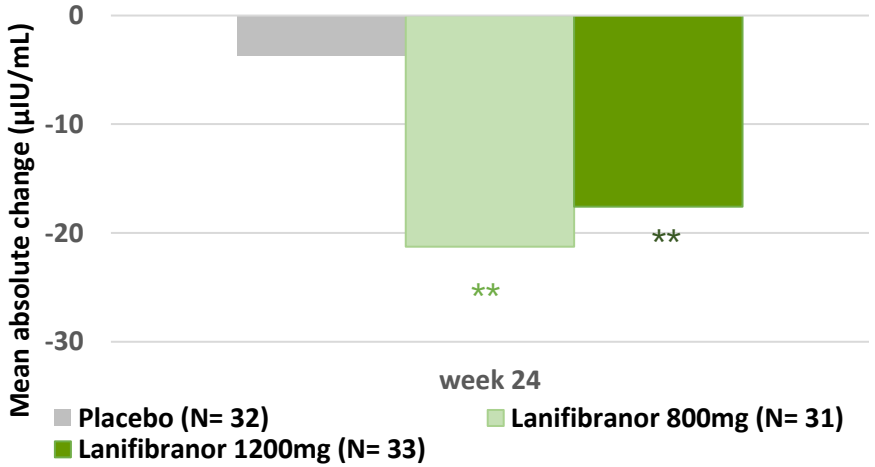


**p<0.001

Absolute change from baseline in fasting glucose



Absolute change from baseline in insulin at W24



Lanifibranor associated with improvements in insulin sensitivity and glycemic control in NASH patients

Lanifibranor has continued to show a favourable safety profile

PHASE IIb | **SAFETY** | **OVERALL**

N (%) patients reporting Adverse Event (AE)	Placebo (N = 81)	800 mg (N = 83)	1200 mg (N = 83)
▶ **Any Treatment-Emergent AE (TEAE)**	50 (61.7%)	59 (71.1%)	62 (74.7%)
Drug-related TEAE	19 (23.5%)	25 (30.1%)	23 (27.7%)
▶ **Any TEAE leading to drug withdrawal**	3 (3.7%)	4 (4.8%)	3 (3.6%)
Drug-related TEAE leading to drug withdrawal	2 (2.5%)	1 (1.2%)(1)	2 (2.4%)(2)
▶ **Any Serious TEAE**	3 (3.7%)	3 (3.6%)	7 (8.4%)
Drug-related Serious TEAE	2 (2.5%)(3)	-	-

Focus of next slide

(1) One patient with moderate diarrhea

(2) One patient with mild cardiac failure; one patient with mild diarrhea, abdominal pain, dizziness

(3) 2 SUSARs: one patient with mild cardiac failure; one patient with moderate urticaria

▶ Consistent with known insulin sensitizing pharmacology, a mean weight increase from baseline of 2.4 kg (2.6%) at the 800 mg/day dose and 2.7 kg (3.1%) at the 1200 mg/day dose was observed.

	Placebo (N = 81)	800 mg (N = 83)	1200 mg (N = 81)
▶ **Peripheral oedema**	2 (2.5%)	5 (6.0%)	7* (8.4%)
Drug-related peripheral oedema	-	2 (2.4%)	2 (2.4%)

* One AE of severe intensity

A limited number of serious TEAEs occurred

PHASE IIb | SAFETY | SERIOUS TEAE

Patients reporting treatment-emergent Serious AE (SAE); N (%)	Placebo (N = 81)	800 mg (N = 83)	1200 mg (N = 83)
Total	3 (3.7%)	3 (3.6%)	7 (8.4%)
Treatment-Emergent Serious AE linked to biopsy procedure			
Post-procedural haematoma/haemorrhage	-	1 (1.2%)	1 (1.2%)
Post-procedural pain	-	-	1 (1.2%)
Pneumobilia (post-procedural)	-	-	1 (1.2%)
Other Treatment-Emergent Serious AE			
Wrist fracture	1 (1.2%)	-	-
Angina unstable	-	-	1 (1.2%)
Cardiac failure	1 (1.2%)	-	-
Gastroenteritis	-	-	1 (1.2%)
Pyelonephritis	-	-	1 (1.2%)
Pancreatitis	-	1 (1.2%)	-
Undifferentiated connective tissue disease	-	1 (1.2%)	-
Urticaria	1 (1.2%)	-	-
Foot operation	-	-	1 (1.2%)

Peripheral oedemas were not flagged as a concern by study investigators

PHASE IIb | **SAFETY** | **PERIPHERAL OEDEMA**

#	Treatment group	Verbatim of AE Oedema peripheral	Intensity	Action taken or Corrective treatment	Relationship to treatment
1	**Placebo**	oedema in the lower leg and knees	Moderate	IMP interrupted + Meloxicam	Unlikely
2		bilateral lower extremity edema	Mild	No actions taken	Unlikely
3	**800 mg**	*bilateral lower extremity edema*	Mild	No actions taken	*Unrelated*
4		*edema in bilateral feet*			*Unlikely*
5		peripheral edema bilateral			Possible
6		right and left ankle swelling			Possible
7		*foot edema bilateral*	*Moderate*	*Bioflavonoids*	*Unrelated*
8	**1200 mg**	*leg edema - both legs*	*Mild*	*Torasemid*	*Unrelated*
9		*oedema in the 2 ankles*		*No actions taken*	*Unrelated*
10		*oedema lower leg, both sides*			*Unrelated*
11		*peripheral edema , both ankles*			*Unrelated*
12		bilateral edema leg			Probable
13		*bilateral postural extremities edema*	*Moderate*	*IMP stopped*	*Unrelated*
14		legs edema	Severe	IMP interrupted	Possible

Peripheral oedemas were not flagged as a concern by study investigators and were:

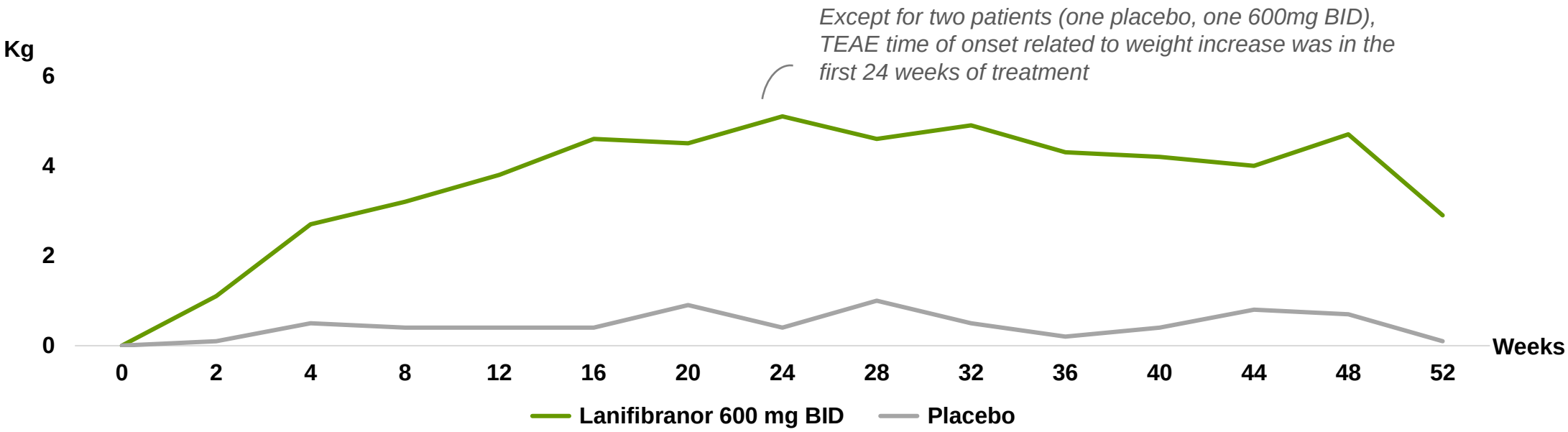
- Limited
- Transient
- Mostly mild
- Majority unrelated and not requiring treatment

Phase II results in NASH have demonstrated modest weight increase with no impact on efficacy

PHASE IIb | SAFETY | WEIGHT GAIN

- **CONSISTENT WITH KNOWN INSULIN-SENSITIZING PHARMACOLOGY**, a mean weight increase from baseline of 2.4 kg (2.6%) at the 800 mg/day dose and 2.7 kg (3.1%) at the 1200 mg/day dose was observed
- According to a six month study with pioglitazone in NASH patients* body weight gain is likely attributed to an **INCREASE IN ADIPOSE TISSUE** and **NOT WATER RETENTION**
- Based on a 52-week lanifibranor trial in systemic sclerosis (SSc) patient weight gain is expected **TO PLATEAU BY WEEK 24**

SSc lanifibranor study: weight (kg) relative change from baseline over 52 weeks (Observed cases under treatment – FAS population)



*Note: * Pioglitazone treatment increases whole body fat but not total body water in patients with non-alcoholiv steatohepatitis ; Balas, Belfort, Harrison et al. ; Journal of Hepatology 47 (2007) 565-570*

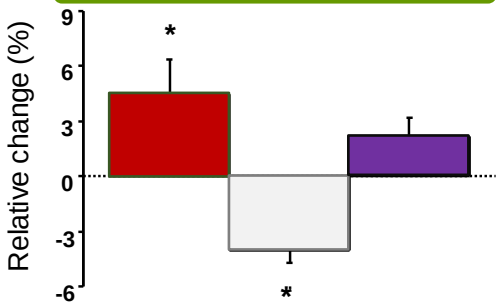
Weight gain is likely related to an increase in subcutaneous fat rather than water retention, while visceral and liver fat may be reduced

SAFETY

Weight gain driven by increase in subcutaneous fat

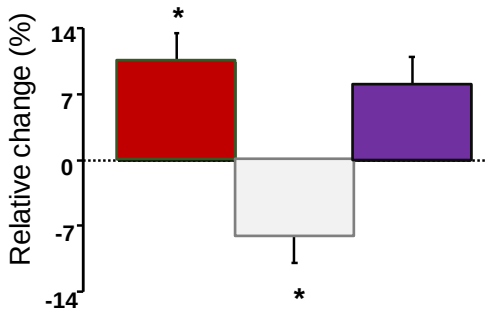
Bodyweight loss | Pioglitazone | Failed diet

Body weight



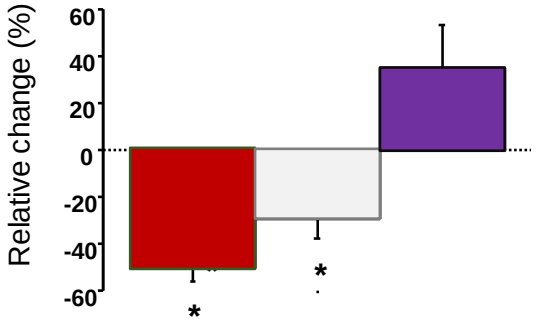
Patients on pioglitazone experienced a weight gain …

Subcutaneous fat



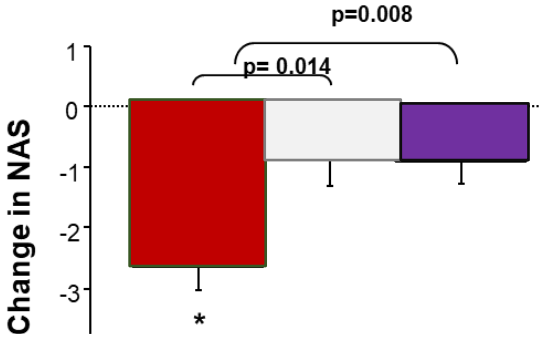
… driven by an increase in subcutaneous fat …

Liver fat



… while liver fat decreased …

Impact on NASH



… improving NASH

Experience with pioglitazone suggests weight gain is due to fat deposited in the subcutaneous tissue rather than visceral or liver fat or water retention; more harmful visceral and liver fat are reduced, improving NASH

*Note: *p < 0.01; ** p < 0.05*
Source: Gastaldelli/Cusi et al, 2020 (unpublished)

Pan-PPAR activation could lead to a favourable cardio-metabolic profile and a reduction in Cardio Vascular Disease (CVD)

SAFETY

Potential to improve cardio-metabolic profile and to reduce CVD

Pioglitazone | Failed diet

		Pioglitazone		Excess caloric intake	
INSULIN RESISTANCE	*Insulin levels (atherogenic)*	**IMPROVES**	↓	**WORSENS**	↑
	FFA levels (lipotoxicity)		↓		↑
	Liver fat and NASH		↓		↑
	Skeletal muscle fat		↓		↑
	Myocardial fat		↓		↑
DIABETES	*Risk of diabetes (non-DM)*	**IMPROVES**	↓	**WORSENS**	↑
	Hyperglycemia (T2DM)		↓		↑
CARDIOVASCULAR RISK	*CV events*	**IMPROVES**	↓	**WORSENS**	↑
	Myocardial function		↑		↓
	Atherogenic dyslipidemia		↓		↑
	Atherogenesis (many mechanisms)		↓		↑
	Endothelial dysfunction		↓		↑
	Subclinical inflammation		↓		↑

Multiple studies demonstrate no increased risk of heart failure for pioglitazone in patients without already pre-existing or suspected heart failure

Mazzone et al. (2006)	**Nissen et al. (2008)**	**Sanyal et al. (2010)**	**DeFronzo et al. (2011)**
Kernan et al. (2016)	**Cusi et al. (2016)**	**Vaccaro et al. (2017)**	**Strongman et al. (2018)**

Note: Mazzone et al. JAMA 2006;296, 2572-2581. Nissen et al. JAMA 2008;299, 1561-1573. Sanyal et al. N Engl J Med 2010;362, 1675-1685 (NASH) DeFronzo et al. N Engl J Med 2011;364, 1104-1115. Kernan et al. N Engl J Med 2016;374, 1321-1331 (IRIS study). Cusi et al. Ann Intern Med 2016;165, 305-315 (NASH). Vaccaro et al. Lancet Diabetes Endocrinol. 2017;5:887-897. Strongman et al. BMJ Open Diab Res Care 2018;6:e000481
Source: Cusi et al, 2020 (unpublished)

Lanifibranor has a differentiated potential to address all features of NASH in a safe and efficacious manner

EFFICACY AND SAFETY

Targeting all features of NASH…

- METABOLISM
- STEATOSIS
- VASCULAR
- INSULIN SENSITIVITY
- FIBROSIS
- INFLAMMATION AND BALLOONING

Tolerability profile similar or superior to other late stage competitors

Improved tolerability profile versus previously trialled single / dual PPARs

… ensures favourable comparison against competitors

- ✓ **NASH resolution with no worsening of fibrosis**
- ✓ **Fibrosis improvement with no worsening of NASH**
- ▶ **BREAKTHROUGH THERAPY** and **FAST TRACK** designations granted by the FDA
- ▶ **LANIFIBRANOR IMPROVES THE BIOMARKER RISK PROFILE** (HDL, TG) and could lead to a reduction in CVD
- ▶ **LANIFIBRANOR IS ASSOCIATED WITH IMPROVEMENTS IN INSULIN SENSITIVITY AND GLYCAEMIC CONTROL**
- ▶ PPAR class clinical hold **LIFTED** by FDA following 24-month rodent carcinogenicity studies and a 12-month monkey tox study
- ▶ Undergone seven DSMB reviews as part of Phase IIb trials **WITHOUT PROTOCOL CHANGE** recommendations

OTHER

PanNASH initiative

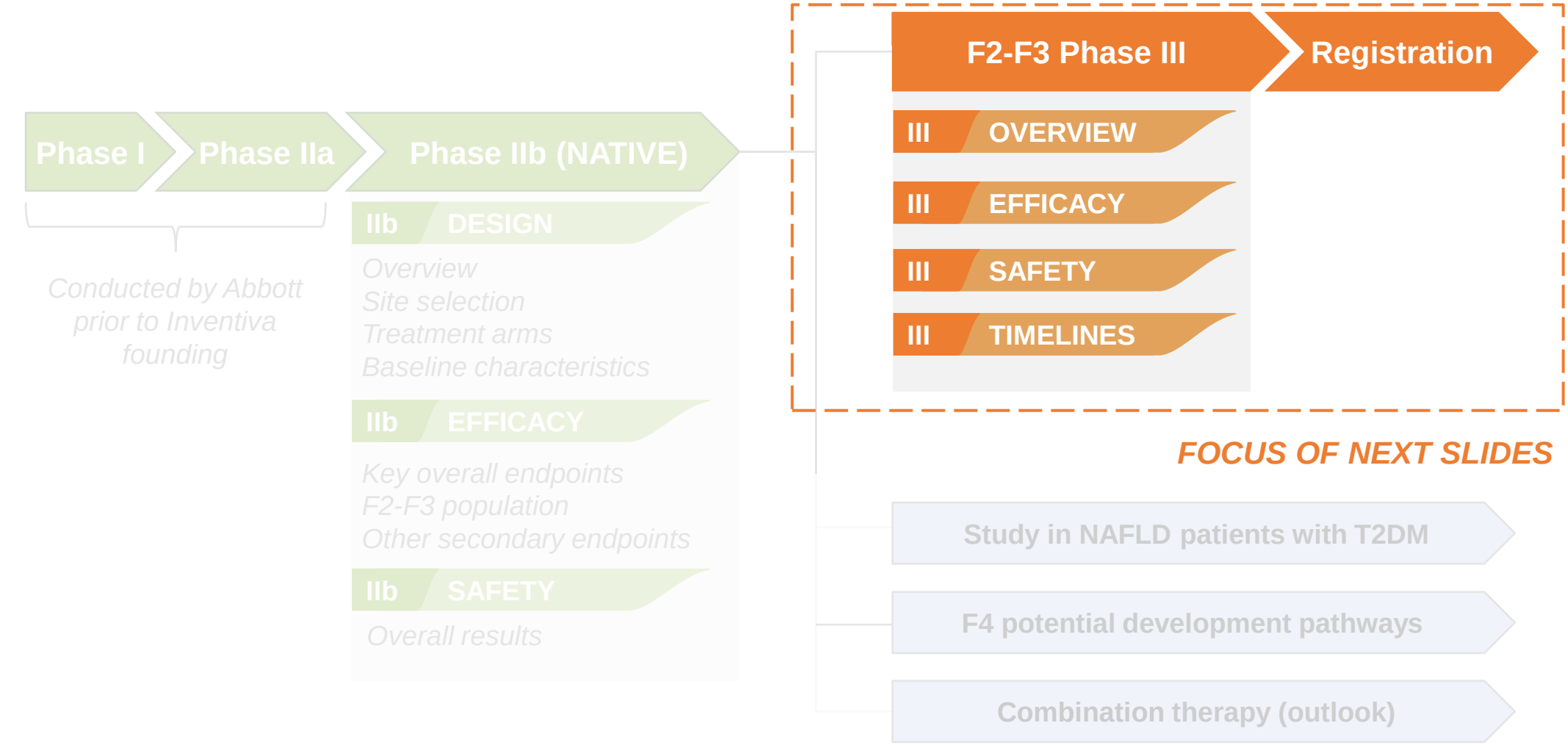
Supported by an experienced scientific and KOL network* that increases awareness and establishes best practices

*Note: * More information on: https://www.pannash.org/*

Lanifibranor's overall development plan builds on the successful outcomes of the NATIVE Phase IIb trial

OVERVIEW

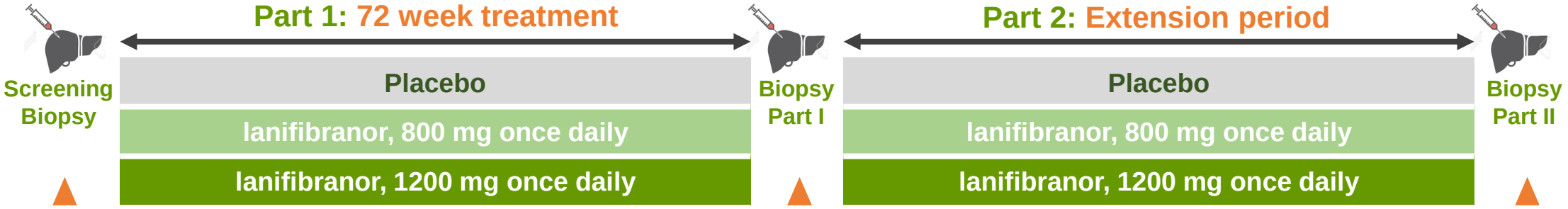
Lanifibranor clinical development overview



The F2-F3 Phase III inclusion criteria and patient profile are in line with the NATIVE Phase IIb trial

PHASE III OVERVIEW

A randomized, double-blind, placebo-controlled, multicenter, Phase III study evaluating long-term efficacy and safety of lanifibranor in adult patients with NASH and liver fibrosis



MAIN INCLUSION CRITERIA aligned with Phase IIb trial:

- Adults (≥18 years of age) diagnosed with NASH using SAF scoring (steatosis ≥1, activity ≥3 and fibrosis score of F2-F3)

RANDOMISATION AND STRATIFICATION

- Randomisation 1:1:1
- Stratification on T2DM and F2/F3 patients
- At least 30% of patients from the U.S.

STATISTICAL POWERING: 90% considered for sample size of c.900 patients

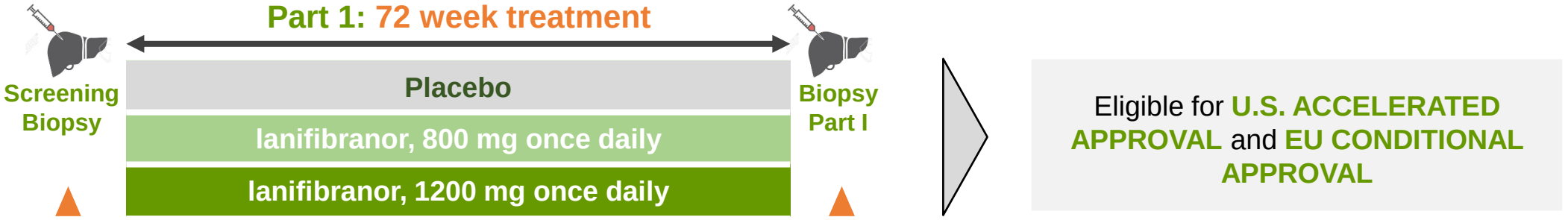
CENTRAL BIOPSY review done by two pathologists

*Note: * Patients with both resolution of NASH and improvement of fibrosis: Native phase IIb placebo 7%, 800mg dose: 24%, delta: **17%**. Nativ3 hypothesis: placebo 9%, 800mg dose: 21%, delta: **12%***

Part 1 of the Phase III may enable U.S. accelerated approval and EU conditional approval based on a 72-week histology analysis

PHASE III OVERVIEW

Part 1 F2-F3 Phase III



PRIMARY ENDPOINT at week 72 on c.900 patients

- Composite endpoint of patients having both NASH resolution and fibrosis improvement of at least one stage

KEY SECONDARY ENDPOINTS

- NASH resolution and no worsening of fibrosis
- Improvement of fibrosis and no worsening of NASH

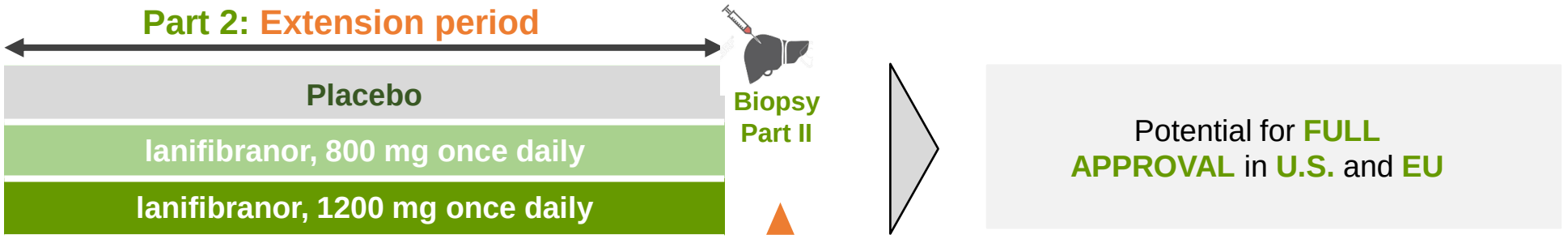
OTHER SECONDARY ENDPOINTS AND HIGH-LEVEL KEY EXPLORATORY ENDPOINTS *(non-exhaustive)*

- Glycaemic parameters at week 12 and week 24 in patients with T2DM not well controlled: proportion of patients with HbA1c back to normal
- Composite endpoint of diabetic patients having both NASH resolution and fibrosis improvement
- Improvement in renal function
- Reduction of cardiovascular risk (including major adverse cardiovascular events 'MACE'; non-fatal myocardial infarction, non-fatal stroke, cardiovascular death, hospitalisation for unstable angina)
- Quality of life (NASH-CLDQ) and PRO (PROMIS)

Part 2 of the Phase III study may allow for a broader “full approval” of lanifibranor in F2-F3 patients

PHASE III OVERVIEW

Part 2 F2-F3 Phase III



KEY ENDPOINTS IN PART 2 *(non-exhaustive)*

- Based on time to first clinical event on c.2,000 patients
  - histological progression to cirrhosis
  - all cause mortality
  - hepatic decompensation events
    - hepatic encephalopathy
    - variceal bleeding
    - new onset ascites requiring treatment
    - spontaneous bacterial peritonitis
  - MELD score ≥15
  - liver transplant

TRIAL END DATE

- Pre-defined number of clinical events

TIMING OF FINAL BIOPSY

- On per-patient basis, defined by:
  - suspicion of progression to cirrhosis (based on non-invasive testing), or
  - trial has ended

The composite endpoint combining NASH resolution and fibrosis improvement can help differentiate from key competitors

PHASE III **EFFICACY**

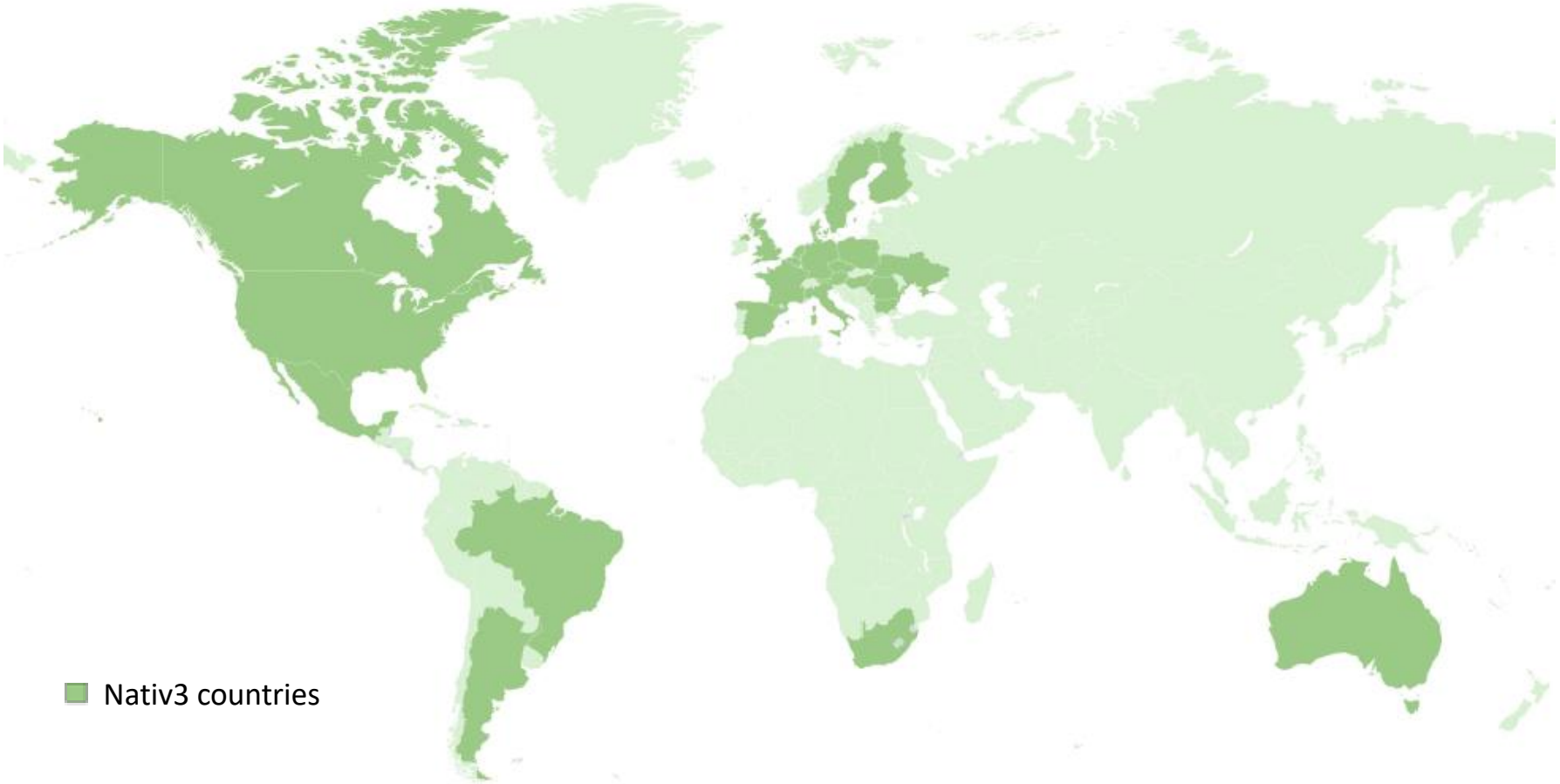
- The primary endpoint “resolution of NASH and improvement of fibrosis” addresses the major pathways of the disease: achieving both of these histological outcomes reflects a stronger impact on disease modification compared with improvement in either steatohepatitis or fibrosis alone
- If met, **a label for the treatment of NASH and the improvement in liver fibrosis** in adult non-cirrhotic NASH patients will be requested

Phase III study	lanifibranor (800 - 1200mg) At W72	Obeticholic acid (10 - 25mg) At W72	Resmetirom (80 - 100mg) At W52
Resolution of NASH and improvement of fibrosis	**Primary**	Secondary (not met)	/
Fibrosis improvement and no worsening of NASH	**Key secondary**	**Primary** (met)	Secondary
NASH resolution and no worsening of fibrosis	**Key secondary**	**Primary** (not met)	**Primary** *(with reduction of at least 2 pts of NAS)*
NASH resolution and fibrosis improvement in diabetic patients	**Secondary**	/	/

Note: / : information not available*

The Phase III patients will be randomised across approximately 300 sites worldwide

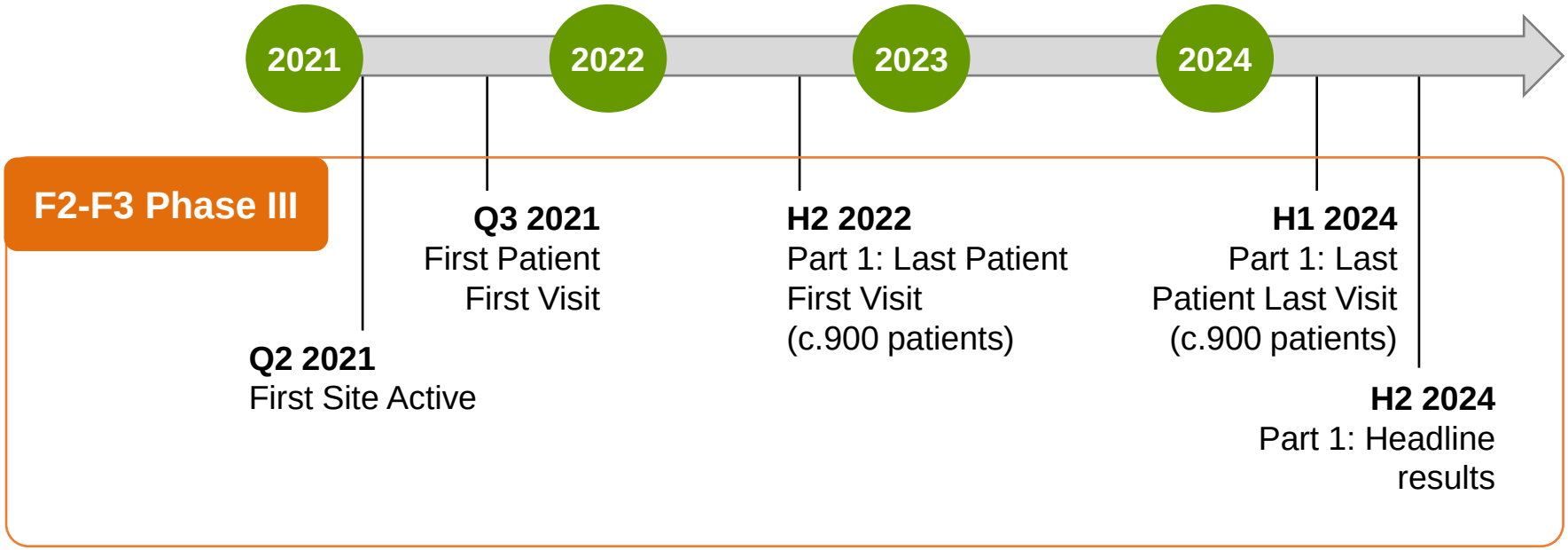
PHASE III | DESIGN | SITE SELECTION



25 countries worldwide for close to 300 sites expected to participate

Key milestones of the Phase III study in NASH (Part 1)

PHASE III **MILESTONES**



Lanifibranor's overall development plan builds on the successful outcomes of the NATIVE Phase IIb trial

OVERVIEW

Phase I → Phase IIa → Phase IIb (NATIVE) → F2-F3 Phase III → Registration

Conducted by Abbott prior to Inventiva founding

IIb DESIGN

Overview
Site selection
Treatment arms
Baseline characteristics

IIb EFFICACY

Key overall endpoints
F2-F3 population
Other secondary endpoints

IIb SAFETY

Overall results

III OVERVIEW

III EFFICACY

III SAFETY

III TIMELINES

Study in NAFLD patients with T2DM

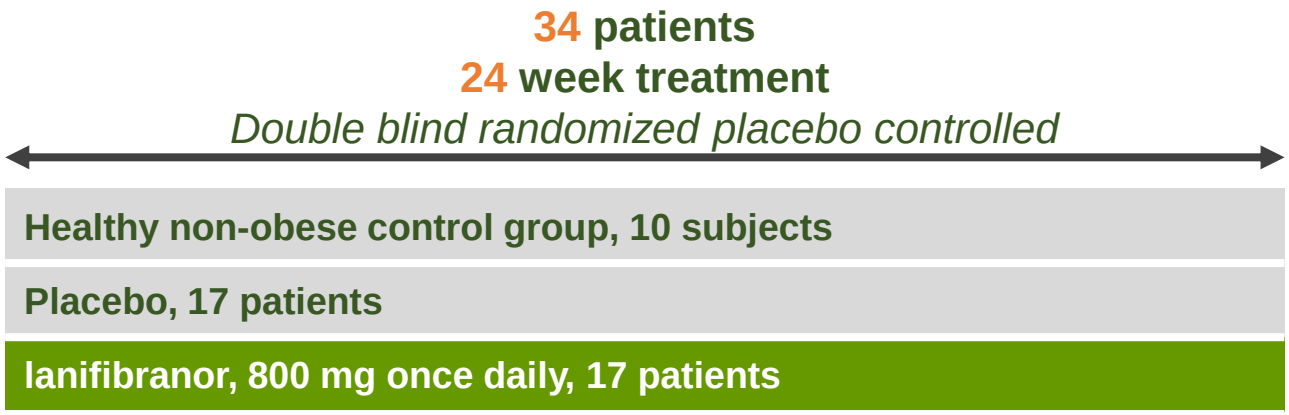
F4 potential development pathways

Combination therapies (outlook)

FOCUS OF NEXT SLIDES

Safety, efficacy and mechanism-of-action of lanifibranor in patients with T2DM and NAFLD: a profiling study (I)

- Collaboration with Dr. Ken Cusi, Professor of Medicine and Chief, Division of Endocrinology, Diabetes and Metabolism at the University of Florida, Gainesville, FL (sponsor)
  - **Clinicaltrials.gov identifier:** NCT03459079
- Objectives: Demonstration of beneficial therapeutic effects of lanifibranor on intrahepatic triglycerides (IHTG) and multiple additional markers of dysmetabolism in T2DM associated with NAFLD
- Study design:
  - 34 patients, randomized 1:1 to lanifibranor 800 mg/day or placebo
    - T2DM: 6.0 ≤ HbA1c ≥ 9.5
    - Hepatic steatosis ≥ 10% measured by Magnetic Resonance Spectroscopy (MRS)
    - Sample size is based on an assumed 50% reduction of IHTG
  - Control arm of 10 matched healthy subjects, for all metabolic and imaging investigations



A Phase II NASH – T2DM clinical profiling study to demonstrate efficacy of lanifibranor on biomarkers of metabolism relevant for NAFLD/NASH

Safety, efficacy and mechanism-of-action of lanifibranor in patients with T2DM and NAFLD: a profiling study (II)

Efficacy endpoints

- Primary efficacy endpoint: decrease of IHTG from baseline to end of therapy, measured by MRS
  - Responder: ≥ 30% reduction of hepatic fat from baseline
  - Proportion of patients having IHTG ≤ 5% at week 24 (NAFLD resolution)
- Secondary efficacy and exploratory endpoints include:
  - Insulin sensitivity, gluconeogenesis, de novo lipogenesis, glycemic control, lipid profile
  - Blood- and imaging markers of liver fibrosis
  - Noninvasive measurements of changes in liver fibrosis:
    - Ultrasound elastography (Fibroscan), Magnetic Resonance Elastography (MRE), and a novel T1 MRI protocol that allows accurate quantification of fibrosis
    - A panel of plasma markers of fibrosis

Status (March 2021)

- As everywhere, COVID-19 has had a large impact on enrollment during 2020; clinical research activities restarted toward the end of the year and are recovering
- Recruitment is supported through information sharing and networking with several other institutions
- Given COVID-19 impact, study results are expected in H1 2022 versus 2021 as previously planned

Evaluation of lanifibranor in F4 compensated cirrhotic patients

F4 compensated cirrhotic patients

In vivo	Thiocetamide (TAA)
In vitro	HSC biology
	Endothelial biology

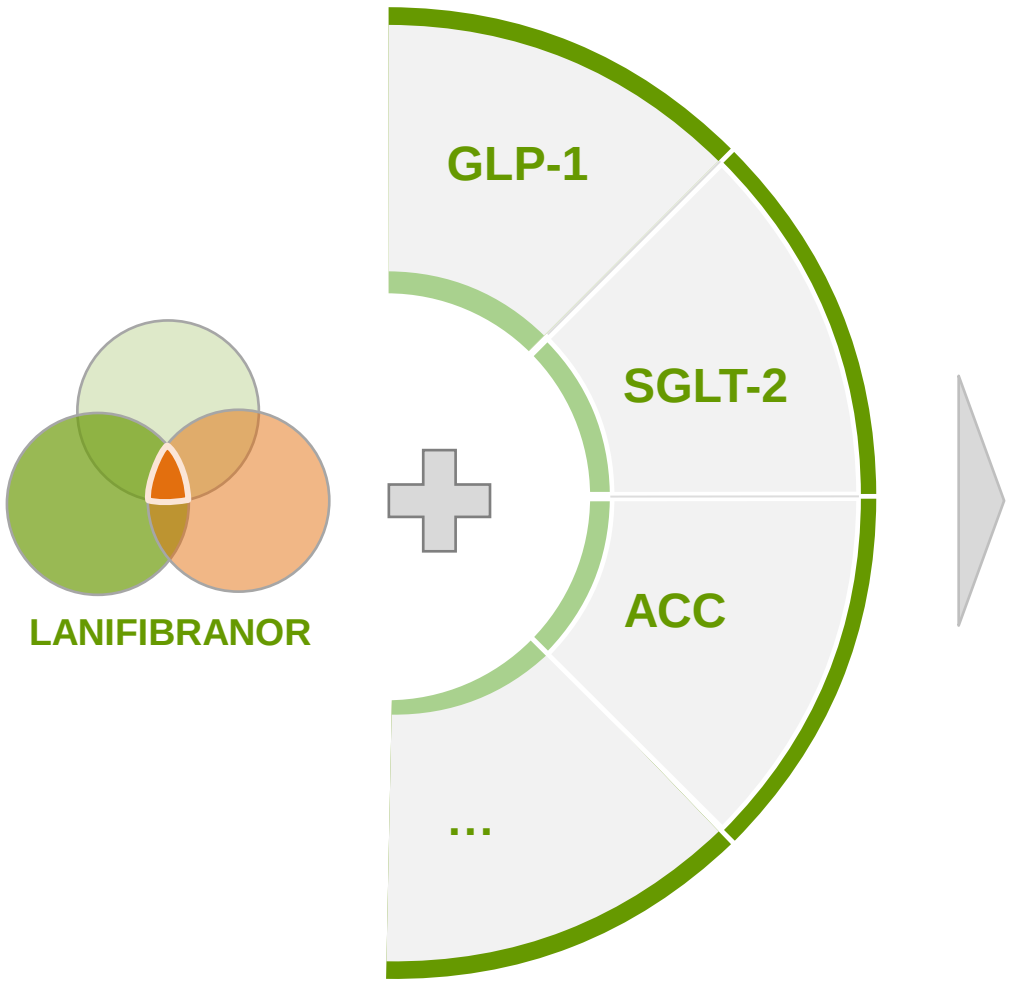
PROMISING PRECLINICAL DATA

- Improved portal hypertension through reduction in intrahepatic vascular resistance
- Rats exhibited significant improvement in liver sinusoidal endothelial phenotype and reduced number of ascites
- Promoted a significant improvement in hepatic stellate cell phenotype and significantly inhibited human primary HSC activation
- Lanifibranor-treated rats exhibited improved liver fibrosis
- Data has been published in the Journal of Hepatology (https://inventivapharma.com/wp-content/uploads/2020/12/Lefere-S-et-al-2020-JHEP.pdf)

Clinical options to evaluate lanifibranor in F4 compensated cirrhotic patients are currently being reviewed

Lanifibranor has the potential to be used in combination with other therapies to further strengthen its value proposition

Examples of potential combination therapies



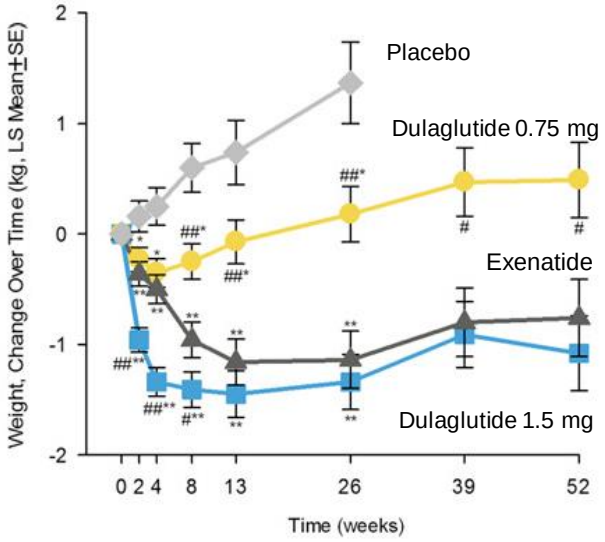
POTENTIAL BENEFITS OF COMBINATION USE

- **COMPLEMENTARY EFFECTS** on the multistep disease biology of NASH (dysmetabolism, insulin resistance, inflammation, fibrosis)
- Further **POTENTIATE THERAPEUTIC EFFICACY** with histological NASH resolution (disease activity) and fibrosis staging as primary efficacy endpoints
- Management of (metabolically 'healthy') **WEIGHT INCREASE** in association with lanifibranor
- Potential to generate new **IP PROTECTION**

Potential combination therapies could include GLP-1 agonists as well as SGLT-2 and ACC inhibitors

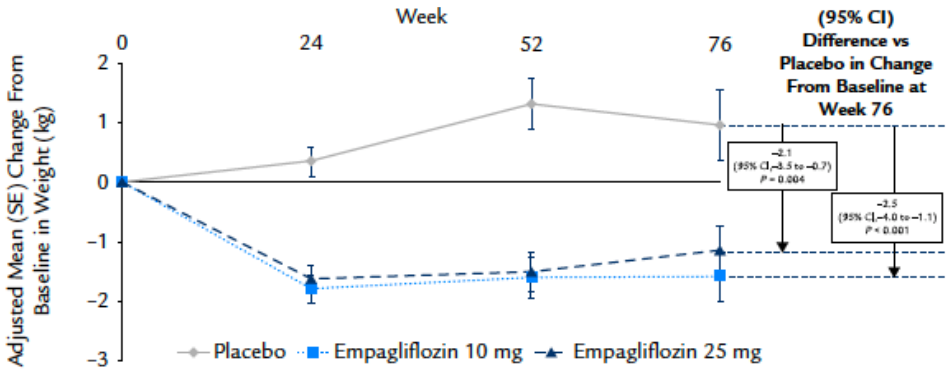
Examples of potential combination therapies

PPAR + GLP-1



- Clinical data suggests pioglitazone and GLP-1 RAs (exenatide or dulaglutide) leads to **WEIGHT LOSS** in T2DM patients

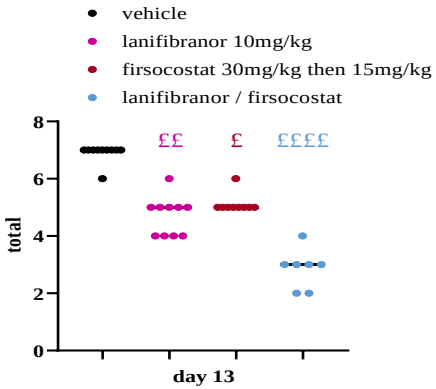
PPAR + SGLT2



- Clinical data suggests pioglitazone in association with SGLT-2 (empagliflozin) leads to **WEIGHT LOSS** in T2DM patients
- Potential for improvement in liver fat reduction

PPAR + ACC

Effect of lanifibranor and firsocostat association on NAS score



- Preclinical data suggest combination allows for greater efficacy in all parameters* including hepatic fat content vs. either monotherapy
- New **PATENT** covering combination filed

*Note: * Including steatosis, inflammation, fibrosis and the global NASH/fibrosis scoring*
Source: Wysham et al, Diabetes Care 2014;37:2159–2167; Kovacks et al, Clin Ther. 2015;37:1773–1788; internal data (unpublished)

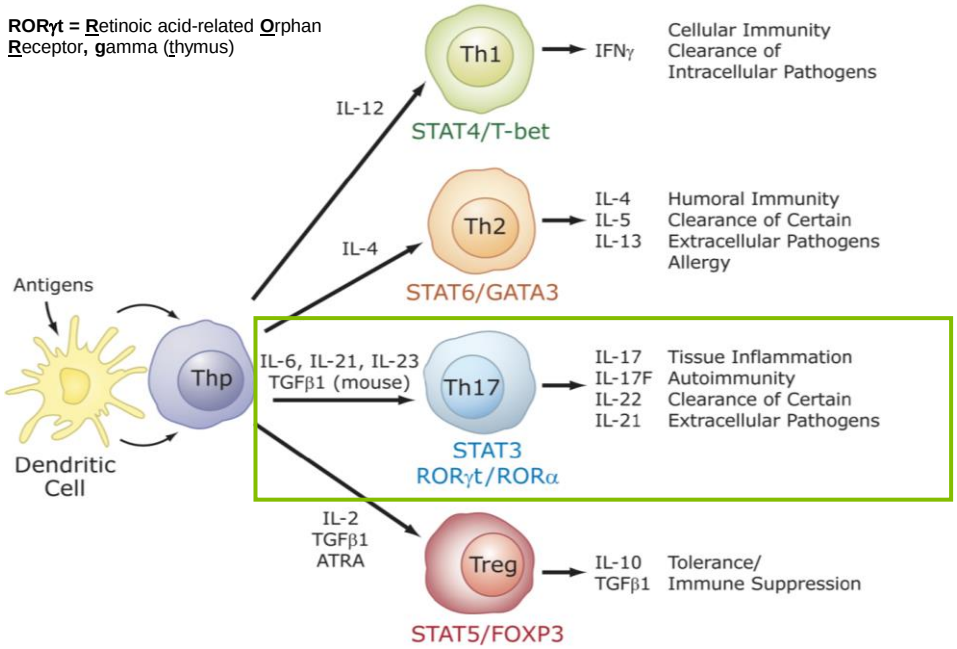
Collaboration with AbbVie in auto-immune diseases: cedirogant (ABBV-157)

abbvie

Cedirogant, a clinical compound co-discovered by Inventiva, with potential in several auto-immune diseases

abbvie

RORγ believed to be a master regulator of Th17 differentiation and IL-17 expression



IL-17 / 23 approach has been validated by several successful biologics

- Pharmacological inhibition of RORγ by small molecules has been observed **to suppress Th17 production, block cutaneous inflammation in animal models of psoriasis and inhibit TH17 signature gene expression** by cells isolated from psoriatic patient samples
- RORγ is therefore a **validated drug target** for the treatment of psoriasis and potentially other cutaneous inflammatory disorders

Cedirogant POC trial conducted by AbbVie

- **Single ascending dose and multiple ascending dose** trials in healthy volunteers **completed**
- **Second clinical trial initiated**: a randomized, double-blind, placebo-controlled, multiple-dose trial to evaluate the pharmacokinetics, safety and tolerability of cedirogant in 60 healthy volunteers and **patients with chronic plaque psoriasis** (clinicaltrials.gov identifier: NCT03922607)
- Results and clinical POC **expected Q2 2021**

Inventiva eligible for milestone payments and sales royalties
€3.5m milestone payment received in Q4 2019 for inclusion of first patient

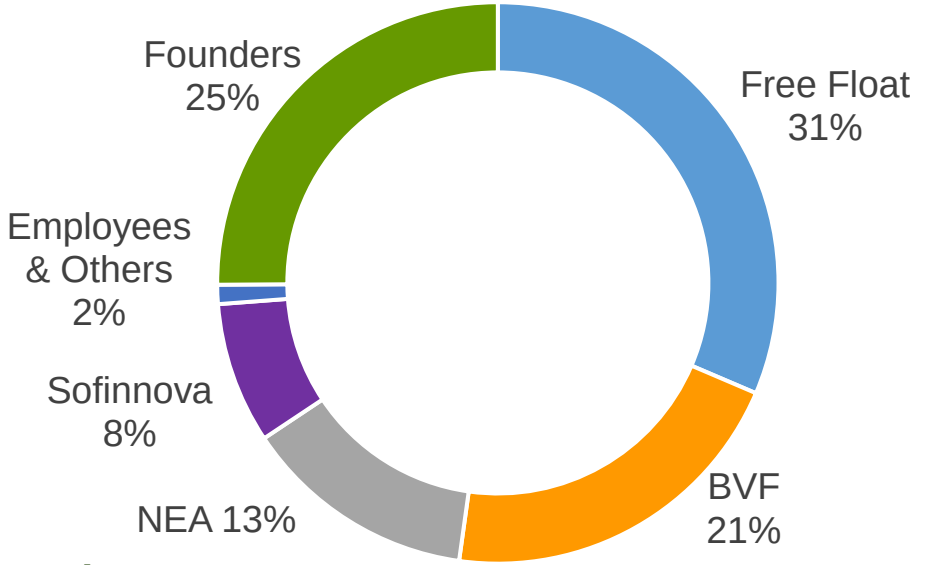
Financials

Key financials and shareholder base

Key financials

ISIN code	FR0013233012 / US46124U1079
Market	Euronext Paris / Nasdaq GM
Shares outstanding	38,630,261 (incl. 7,478,261 shares of the July 9 Nasdaq IPO)
Market cap (March 4, 2021)	Euronext Paris: €471m / Nasdaq Global Market: $558m
Cash position (as of December 31,2020)	€113m (vs €35.8m as of December 31, 2019) Current expected cash runway through Q4 2022
2020 revenues	€0.4m compared to €7.0 in 2019
R&D expenditures in 2020	€23.7m compared to €33.8m in 2019

Shareholder base(1)



Analyst coverage

Jefferies	L. Codrington / M. J. Yee
Stiffel	D. Archila
Guggenheim	E. Darout
HC Wainwright	E. Arce
Roth Capital	Z. Jallah
LifeSci Capital	P. Dolezal
Chardan	M. Morabito
KBC	L. Van Steenhuyse
Société Générale	D. Le Louët
Bryan Garnier	JJ. Lefur
Portzamparc	M. Kaabouni

Full year 2020: Key figures

Income Statement		
Key figures *(in thousands of euro, except share and per share amounts)*	**2020**	**2019**
Revenue	**372**	**6,998**
Other income	4,891	4,293
Research and development expenses	(23,717)	(33,791)
Marketing – business development expenses	(563)	(249)
General and administrative expenses	(8,499)	(6,088)
Other operating income (expenses)	(2,202)	(1,475)
Net operating loss	**(29,718)**	**(30,312)**
Net financial income (loss)	**(3,902)**	**93**
Income tax	-	-
Net loss for the period	**(33,619)**	**(30,218)**

Cash Position		
Key figures *(in thousands of euros)*	**December 31, 2020**	**December 31, 2019**
Cash & cash equivalents(1)	**113,022**	**35,840**

(1) The cash position includes cash and cash equivalents as well as short-term deposits which are included in the category "other current assets" in the IFRS statement of financial position as of December 31, 2020.

Highlights

- **No expected revenues in 2020 vs €3.5m and €2.6m for the collaborations with AbbVie and Boehringer Ingelheim in 20219**
- **29% decrease in R&D investment, €23.7m vs €33.8m in 2019**
  - Continued efforts dedicated to the development of lanifibranor (NASH) and odiparcil (MPS), offset by one full year of savings following the halt of the development in systemic sclerosis in 2019 and the Employment Safeguard plan introduced mid-2019
- **40% increase in R&D G&A, €8.5m vs €6.1m in 2019 due to 1st year of dual listing compliance costs starting 2020**
- **Cash position at €113.0m vs €35.8m as of 12.31.2019**
  - Net operating cash flow at - €30.6m vs - €28.4m in 2019 >> see above P&L analysis + 2 R&D French credits received in 2020 vs in 2019
  - Net financing cash flow at €111.7m vs €8.4m, due to the Nasdaq listing (€94.9m) in July, the French guaranteed loan in June (€10.0m) and the €15.0 capital increase in Q1

Financial Calendar

- **May 13, 2020: Publication of Q1 2021 financial results (revenues and cash)** (*after market closing*)

Near-term catalysts

Recent and anticipated key milestones

Lanifibranor

- ☑ Positive topline results of NATIVE Phase IIb trial in NASH
- ☑ Breakthrough Therapy Designation granted by FDA
- ☑ NATIVE Phase IIb meeting with FDA and EMA Scientific Advice
- ▶ Launch of phase III trial with lanifibranor in NASH **– H1 2021**

Odiparcil

- ▶ Strategy update on odiparcil development **– 2021**

Cedirogant

abbvie

- ▶ Results of cedirogant clinical POC trial (Phase I) in psoriasis **– Q2 2021**

Q&A

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