Developing breakthrough therapies in fibrosis, oncology and orphan diseases

Corporate Presentation
April 2017

IVA
LISTED
EURONEXT

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A clinical stage biopharma with a focus on fibrosis

State of the art R&D capabilities	▶ Fournier/Solvay and Abbott spin-off in 2012 ▶ State of the art owned 12,000 m² R&D facility and a library of ~240,000 compounds ▶ Solid portfolio of patents: 13 families approved
Leading technology in gene-modulation	▶ Expert in gene-modulation (nuclear receptors, transcription factors, epigenetic targets) ▶ Large fibrosis expertise ▶ Promising and innovative preclinical pipeline in oncology
Three innovative clinical programs	▶ **IVA337 NASH:** phase IIb enrolling since February 2017. Results expected mid-2018 • NASH market potential: $35-40bn ▶ **IVA337 Diffuse Cutaneous Systemic Sclerosis:** phase IIb enrolling since December 2015. Results expected second half of 2018 • SSc market potential: > €1.8bn ▶ **IVA336 MPS VI:** biomarker study ongoing and POC Phase I/II study initiated with first patient enrolment expected in 2017. Results expected mid-2018.
Two R&D collaborations	▶ **AbbVie:** ABBV-553 RORγ program phase I initiated in 2016. Inventiva eligible to research funding, milestone payments and royalties ▶ **BI:** collaboration in Idiopathic Pulmonary Fibrosis (IPF). Inventiva eligible to research funding, and up to 170M€ in milestones excluding royalties on sales
Strong financial position	▶ ~24,8M€ cash position at December 31, 2016 (before IPO proceeds) ▶ Successful IPO on Euronext Paris in February 2017: 48,5M€ raised ▶ 9,4M€ turnover in 2016 (+94%)

Fibrosis: one process leading to many diseases with high unmet needs

inventiva Boehringer Ingelheim

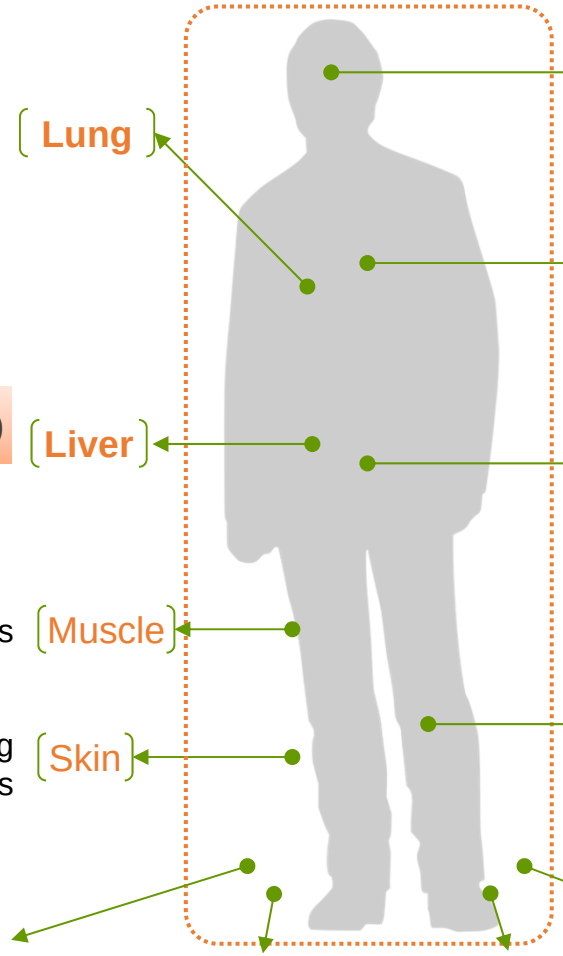
Idiopathic Plumonary Fibrosis (IPF)

Interstitial lung diseases
Asthma
Pulmonary arterial Hypertension

inventiva **IVA337**

Non-Alcoholic Steatohepatitis (NASH)

Cholestatic liver diseases
Cirrhosis



Age-Related Macular Degeneration (AMD)
Diabetic retinopathy
Proliferative Vitreoretinopathy (PVR)
Glaucoma

Cardiac Scarring Post-Myocardial Infarction
Cardiomyopathy induced by diabetes and hypertension

Transplant Nephropathy
Nephropathy induced by diabetes and hypertension
Lupus Nephritis
Focal Segmental Glomerulosclerosis
Alport's syndrome

inventiva **IVA337**

Systemic Sclerosis (SSc)

Nephrogenic Systemic Fibrosis

Fibrosis: ~45% of all deaths in the developed world (1)

Source: (1) The Journal of Clinical Investigation; Common and unique mechanisms regulate fibrosis in various fibroproliferative diseases; March 2007.

Rich pipeline approaching major inflection points

Program	Indication	Program Stage	Commercial Rights
IVA337	Nonalcoholic steatohepatitis (NASH)	Phase IIb	inventiva
IVA337	Systemic Sclerosis (SSc)	Phase IIb	inventiva
IVA336	Mucopolysaccharidosis type VI (MPS VI)	Phase I/II	inventiva
ABBV-553	Moderate to severe psoriasis	Phase I	abbvie Sales royalties for Inventiva
YAP/TEAD	Malignant Mesothelioma, Lung Cancer	Discovery	inventiva
NSD2	Multiple Myeloma	Discovery	inventiva
EPICURE institutCurie	Immuno-oncology	Research	inventiva
Undisclosed target	Idiopathic Pulmonary Fibrosis (IPF)	Discovery	Boehringer Ingelheim Sales royalties for Inventiva

IVA337 NASH and SSc

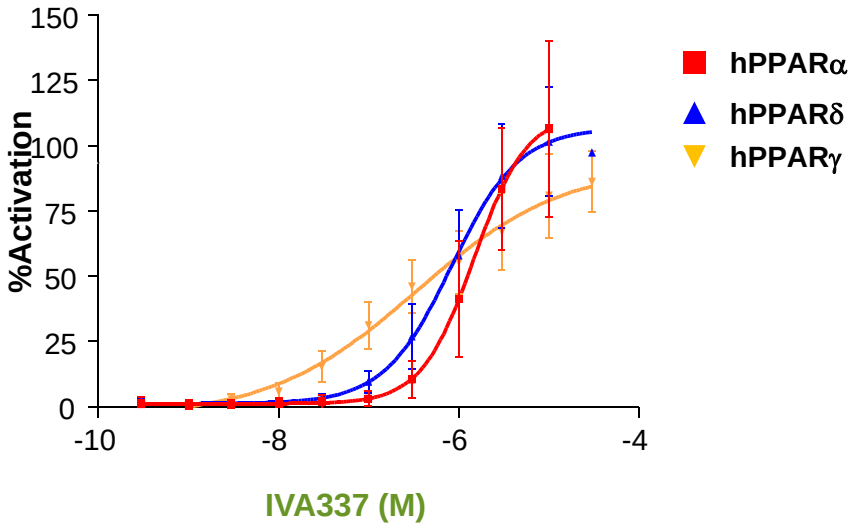
A new generation pan-PPAR agonist for a safe and efficacious treatment of fibrotic conditions

IVA337: a next generation pan-PPAR agonist for a safe and efficacious treatment of fibrotic conditions

Activity	▶ Unprecedented chemical structure with moderate and balanced Pan-PPAR agonist activity (PPARα, PPARγ and PPARδ) ▶ Oral administration ▶ Efficacy demonstrated on insulin resistance, dyslipidemia, steatosis, ballooning, inflammation and liver fibrosis. Anti-fibrotic activity also demonstrated in skin, kidney, lung ▶ 100 volunteers treated in Phase I trials and 56 patients treated in phase IIa study ▶ Phase IIa demonstrated Pan-PPAR agonist activity supporting dose selection for NASH and systemic sclerosis (SSc) ▶ Phase IIb SSc FPI December 2015 ▶ Phase IIb NASH FPI February 2017
IP	▶ Composition of matter patent granted in 59 countries: LOE August 2031 including 5-year extension ▶ Use patent filed in 2015 (LOE when granted: 2035) ▶ OSD granted in SSc in the US and EU
Safety	▶ Good safety profile different from other PPAR compounds demonstrated in 6-month rodent and monkey studies ▶ 52 weeks toxicity studies in primates completed (HR expected first-half of 2017) and carcinogenicity studies ongoing (HR mid-2018) ▶ Safety profile in phase I and phase IIa T2DM studies similar to placebo

IVA337: a next generation panPPAR with moderate and well balanced activity on the 3 PPAR isoforms

IVA337 DRCs and EC50s for hPPARs (nM)



Compound	PPARα EC50 (nM)	PPARδ EC50 (nM)	PPARγ EC50 (nM)
IVA337[1]	1630	850	230
Fenofibrate	2400	-	-
Pioglitazone	-	-	263
Rosiglitazone	-	-	13
Elafibranor[2]	10	100	-

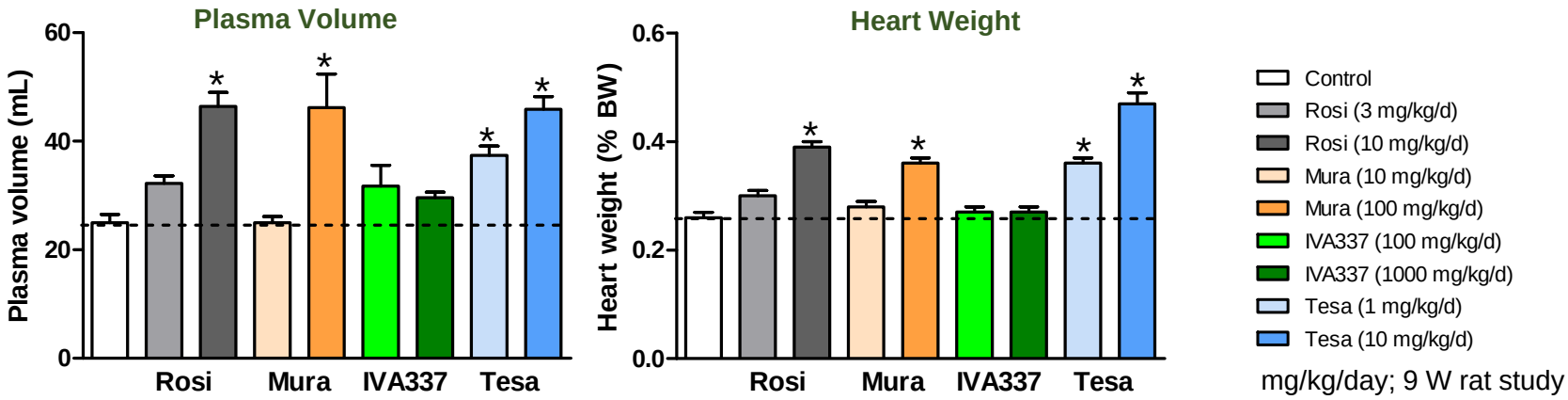
IVA337 presents a similar profile for the 3 rodent PPAR isoforms

(1) Company data (2) Hanf R et al, Diabetes & Vascular Dis Res 2014

A good safety profile differing from previously developed PPARs

Organ	Molecule	Reported PPAR liabilities	IVA337 effects	No Observed Adverse Effect Level (NOAEL)
Heart	Glitazone	Fluid retention Cardiac hypertrophy	Not observed	1000 mg/kg in rodents and primates 26w study
Skeletal muscle	Fibrate	Myofiber degeneration	Not observed	
Kidney	Fibrate	> 50% increases in creatinine, Degenerative changes in renal tubules	Not observed	
Urinary bladder	Glitazone	Proliferative changes in bladder epithelium	Not observed	

Contrasting with other PPARγ agonists , IVA337 does not produce plasma volume expansion



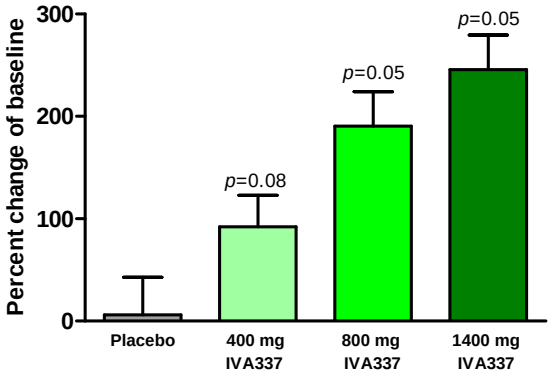
mg/kg/day; 9 W rat study

Phase I and Phase IIa clinical studies confirmed IVA337 safety and efficacy on key metabolic markers

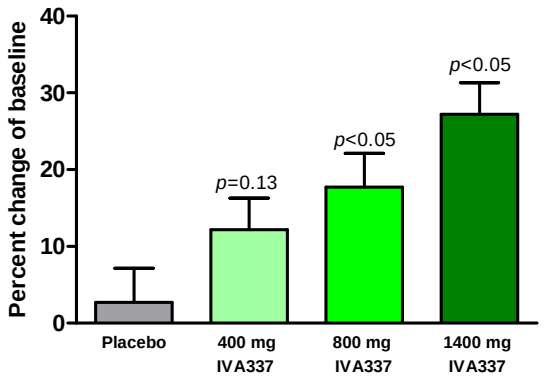
IVA337 strongly improves metabolic markers in type II diabetic patients

- Insulin resistance (HOMA-IR, adiponectin)
- Dyslipidemia (increase in HDL-C, reduction of TG)

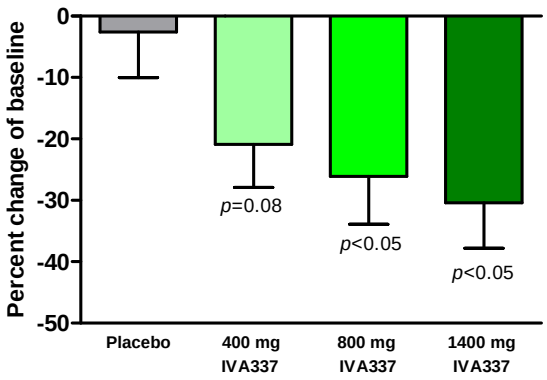
Adiponectin (PPARγ)



HDL Cholesterol (PPARα)



Triglycerides (PPARα/δ)



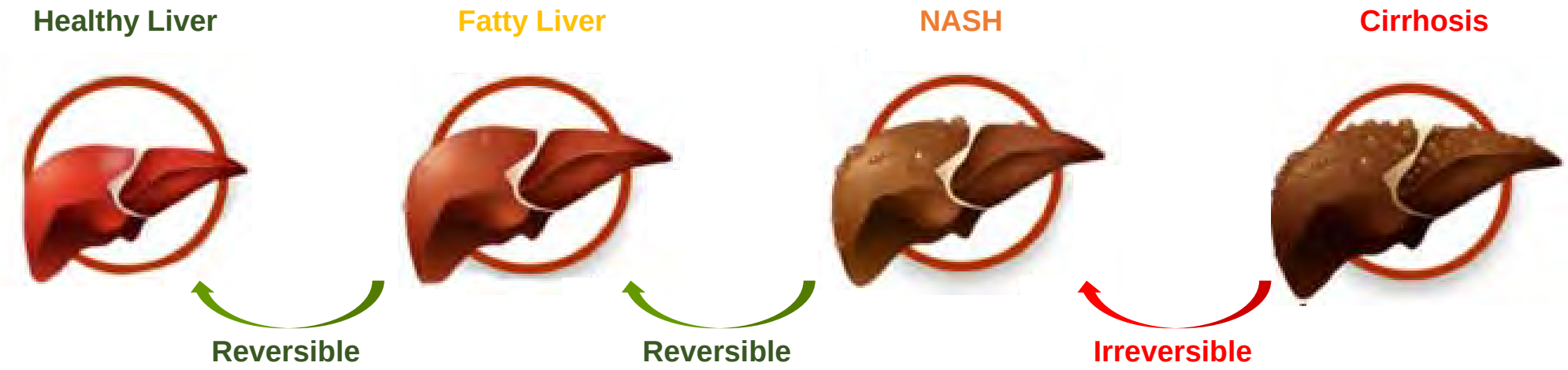
Clinical findings underline the excellent tolerability of IVA337

- Good overall tolerance and no major safety findings
- No increases of creatinine, LFTs, or CPK
- No changes in blood pressure
- No signal of fluid overload or hemodilution
- No clinically relevant weight gain

*Source: Company data and * Ohashi, Endocr Metab Immune Disord Drug Targets. 2015.*

NASH : overview

A severe disease with no approved treatment



A market estimated at 35-40B$

NAFLD (US)

~ 80MM adults

>30%
of adult population

NASH (US)

>30MM adults

>10%
of adult population

NASH with Fibrosis (US)

>14MM adults

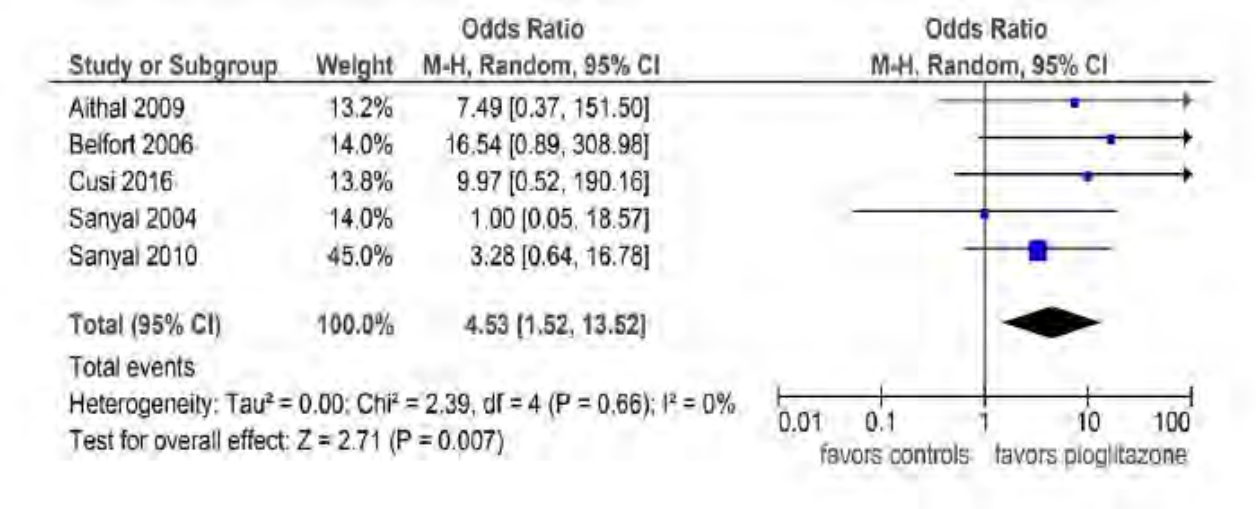
>6.7%
of adult population
with NASH & fibrosis

Sources: NASH Market, Allied Market Research 2016 ; Deutsche Bank Markets Research; Intercept website.; Epidemiology and natural history of non-alcoholic steatohepatitis.Clinical Liver Disease 2009 Nov;13(4):511-31.

panPPAR clinical rationale in NASH

- **PPARγ activation by pioglitazone significantly improves steatosis, ballooning and inflammation as well as metabolic markers in NASH patients after 6 months or 18 months of treatment:**

Pioglitazone	6 mo (Belfort et al, 2006)	18 mo (Cusi et al, 2016)
Steatosis (% patients improved)	65% vs 38%, p < 0.001	71% vs 26%, p < 0.001
Inflammation (% patients improved)	65% vs 29%, p < 0.001	49% vs 22%, p < 0.001
Ballooning (% patients improved)	54% vs 24%, p < 0.001	51% vs 24%, p < 0.001
NASH resolution (% patients)	NA	51% vs 19%, p < 0.001
Fibrosis (mean change in score)	NS	- 0.5, p = 0.039



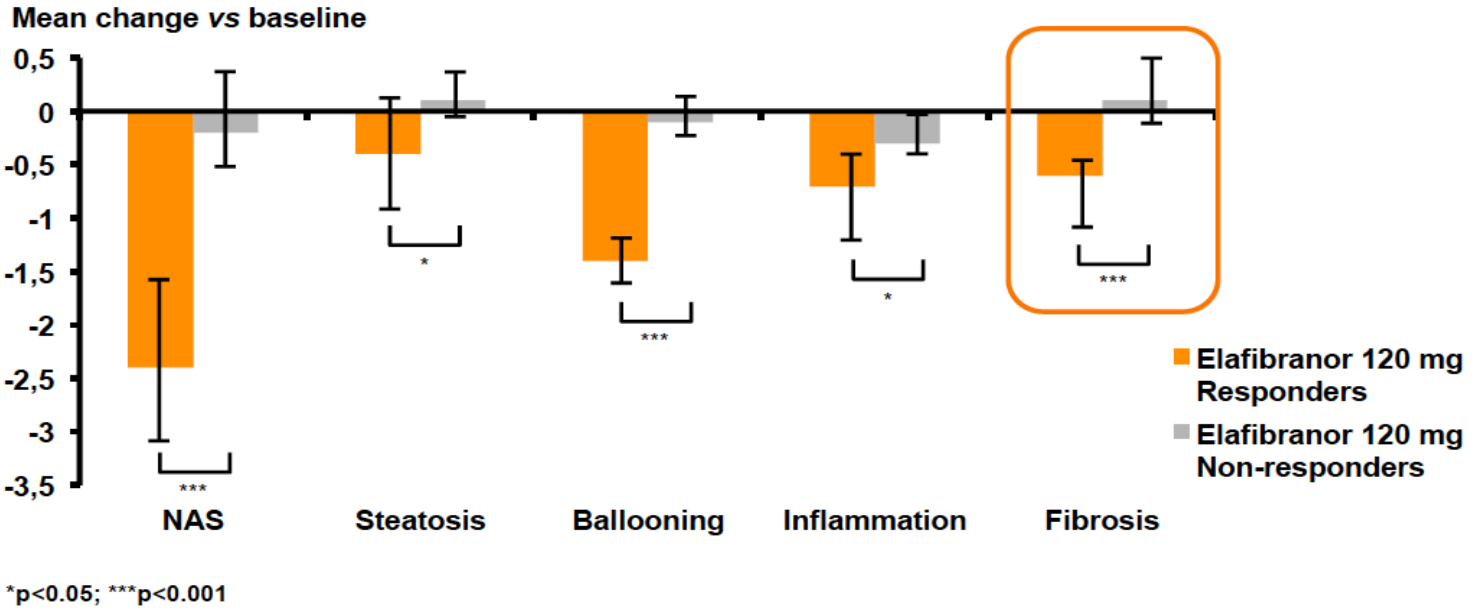
- **Pioglitazone improves advanced fibrosis (stage F3-F4) as indicated by an increase in the number of NASH patients whose fibrosis stage changed from F3-F4 to F0-F2 at the end of treatment**

Corey KE and Malhi H, Hepatology 2016

panPPAR clinical rationale in NASH

- **PPARα/δ activation by elafibranor leads to significant improvement of ballooning and inflammation as well as metabolic markers in NASH patients after 12 months of treatment:**
  - **NASH resolution in ITT: 19% vs 12%, p = 0.045**
  - **Steatosis in patients with bNAS>4 (% patients improved): 35% vs 18%, NS**
  - **Inflammation in patients with bNAS>4 (% patients improved): 55% vs 33%, p < 0.05**
  - **Ballooning in patients with bNAS>4 (% patients improved): 45% vs 23%, p = 0.02**

- **Patients who resolved NASH showed significant reduction in liver fibrosis while non-responders did not show any change from baseline**

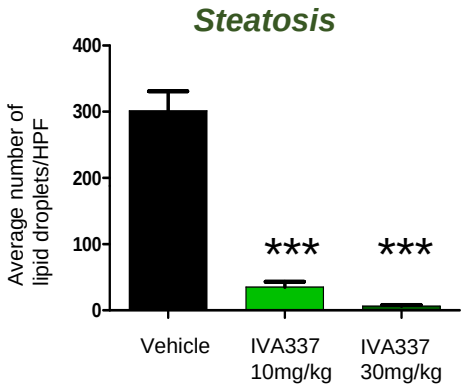


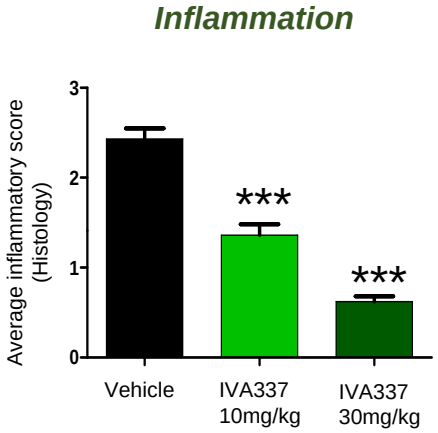
*p<0.05; ***p<0.001

Ratziu V, *et al.* Gastroenterology 2016

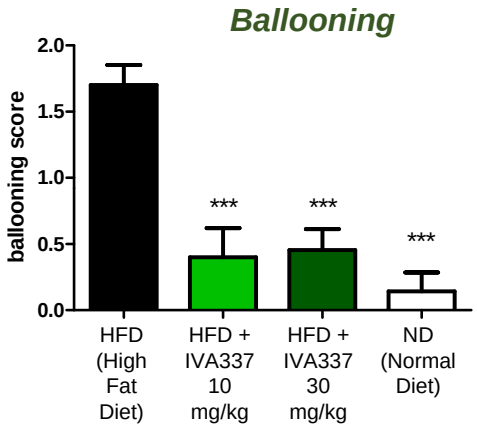
IVA337 strongly reduces steatosis, inflammation, ballooning and fibrosis in preclinical models

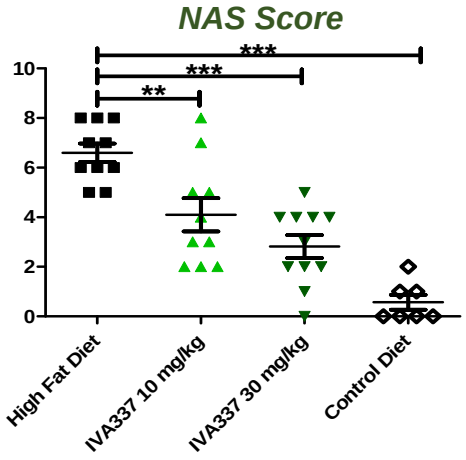
IVA337 inhibits steatosis and inflammation in the MCD model



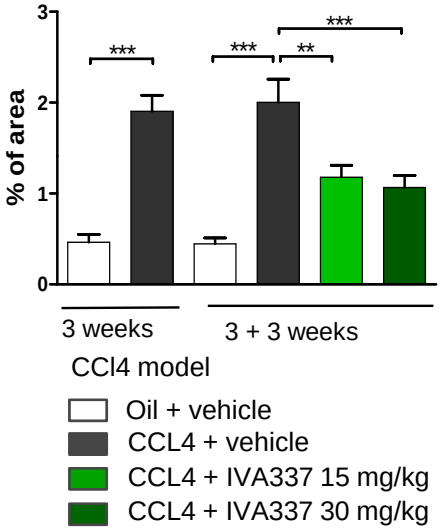


IVA337 strongly reduces ballooning and the NAS score in the foz/foz model





IVA337 reverses established liver fibrosis



IVA337 positively impacts all NASH-relevant liver lesions

NATIVE Phase IIb in NASH

Trial design

European study: 12 countries, 41 sites

- Principal investigator: Pr Francque (Universitair Ziekenhuis, Antwerpen)
- Other: Pr Ratziu (Hôpital Pitié-Salpêtrière, Paris), Pr Anstee (Newcastle University), Pr Bedossa (Hôpital Beaujon, Paris), Pr Bugianesi (Ospedale S Giovanni Battista, Turin)

Clinicaltrials.gov identifier:

- NCT03008070

Inclusion criteria

- Liver biopsy
- Moderate to severe patients with a SAF activity score of 3 or 4
- SAF Steatosis score ≥ 1
- SAF Fibrosis score: < 4

225 patients
24 week treatment
Double blind randomized placebo controlled

First patient

- February 2017

Placebo, 75 patients

IVA337, 800 mg, 75 patients

IVA337, 1200 mg, 75 patients

End of treatment

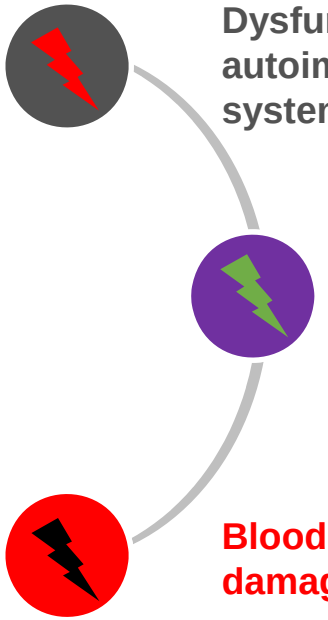
- Liver biopsy

Primary endpoint

- Decrease from baseline ≥ 2 points of the SAF activity score combining hepatocellular inflammatory and ballooning

Systemic sclerosis overview

A severe disease with no approved treatment [1]



Dysfunction of the autoimmune system

The immune system produces cytokines leading to Inflammation

Overproduction of collagen

Overproduction of collagen leading to skin and vital organs fibrosis and failure (lung, kidney, heart)

Blood vessel damage

Reduction in blood flow to tissues causing damage

Patients: More than 170,000 patients diagnosed and a market potential > €1.8bn [2]

USA	Europe Top 5	Japan
~102,000 patients [2]	~67,000 patients [2]	~4,800 patients [2]

Sources: (1) Eular SSc Trials and Research Group, EUSTAR, SSc Research Foundation, Canadian SSc research group ; (2) Venture Valuation.

IVA337 addresses all the relevant clinical features of systemic sclerosis

Data generated in several relevant preclinical models demonstrate that IVA337 positively impacts the most relevant clinical features of SSc

Skin	**IVA337 reduces skin fibrosis**
Lung	**IVA337 reduces vasculopathy and inflammatory driven lung fibrosis** **IVA337 restores lung functional capacity**
Heart	**IVA337 reduces right ventricular systolic pressure and right ventricular hypertrophy**
Kidney	**IVA337 reduces kidney fibrosis**

IVA337 FASST phase IIB study will have as primary endpoint the reduction of skin fibrosis, measured by the Modified Rodnan Skin Score

Source: Company data

FASST Phase IIb in SSc

Trial design

European study: 8 countries; 50+ sites

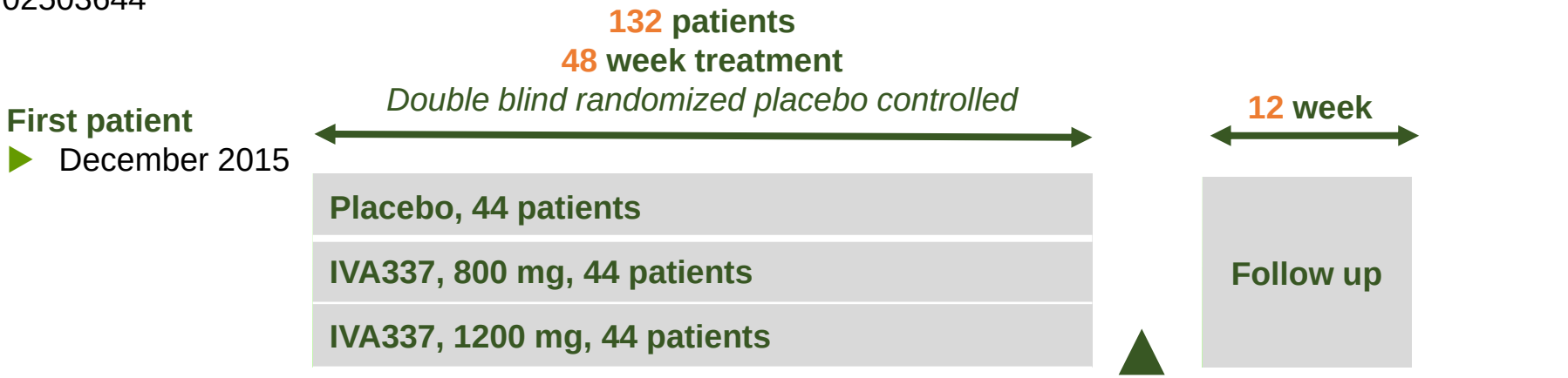
- Principal investigators:
  Pr Allanore (Hôpital Cochin, Paris) and Pr Denton (University College of London)
- Other: Pr Matucci (Florence University, Italy), Pr Distler (University of Erlangen, Germany), Pr Distler (Universitaet Zurich, Switzerland)
- 100th patient randomized in April 2017

Clinicaltrials.gov identifier:

- NCT02503644

Inclusion criteria

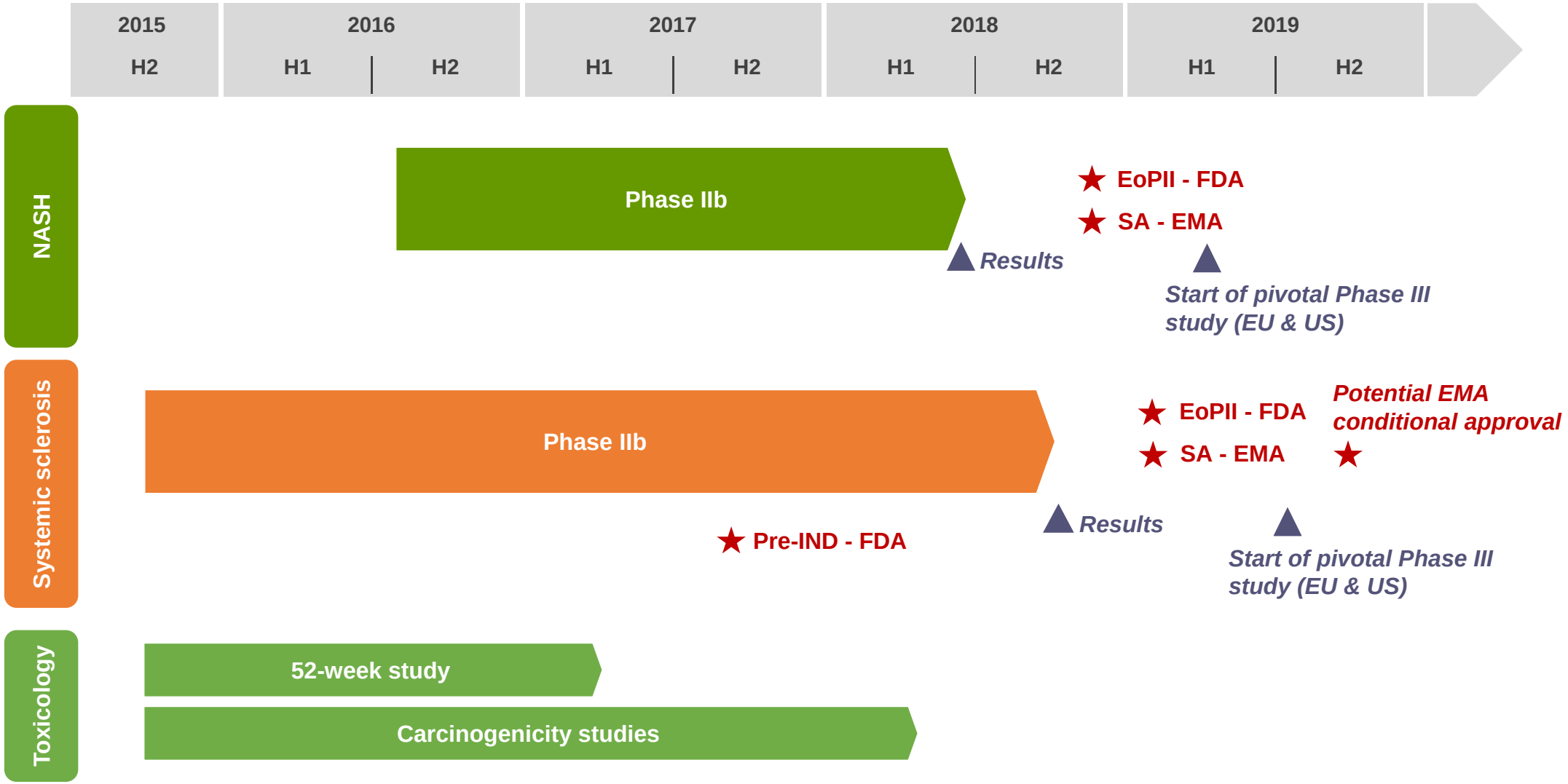
- MRSS (Modified Rodnan Skin Score) between 10 and 25
- SSc diagnosed from less than 3 years



Primary endpoint

- Mean change of the MRSS from baseline to 48 weeks

IVA337 a phase III ready program in NASH and SSc by end-2018



IVA336 MPS I, II, VI

The first oral therapy for MPS I, II and VI patients

MPS are devastating diseases with high unmet medical needs

MPS diseases are inherited lysosomal storage diseases

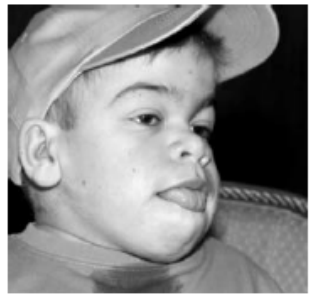
Autosomal recessive disorder characterized by accumulation of glycosaminoglycan(s) (GAG) due to lack of an enzyme

Seven distinct clinical types based on the enzyme affected

IVA336 could be the first substrate reduction therapy for 3 forms of MPS:

- MPS I: 1/100,000 live births[1] ~2,000 patients
- MPS II: 1/100,000 live births[1] ~2,000 patients
- MPS VI: 1/225,000 live births[1];~1,100 patients, increased frequency in Turkish and Portuguese subpopulations)[2]

Kathleen (MPS I)



Scotty (MPS II)

Karima (MPS VI)

The three targeted MPS have devastating clinical consequences

Consequences	MPS I	MPS II	MPS VI
Mental retardation	☑	☑	
Coarse facies, short stature	☑	☑	☑
Dysostosis multiplex	☑	☑	☑
Joint stiffness	☑	☑	☑
Spinal cord compression	☑	☑	☑
Organomegaly	☑	☑	☑
Poor vision (corneal clouding)	☑	☑(1)	☑
Hearing loss	☑		
Cardiac/respiratory disease	☑	☑	☑
		Pebbled skin Diarrhoea	Odontoid hypoplasia Kyphoscoliosis, genu valgum

(1) Retinal degeneration with no corneal clouding

Sources: (1) MPs society; (2) Valayannopoulos V, Nicely H, Harmatz P, Turbeville S; Mucopolysaccharidosis VI. Orphanet J Rare Dis. 2010 Apr 12;5:5.

Enzyme replacement therapy (ERT) are commercially successful, but with limited therapeutic efficacy

Enzyme Replacement Therapies

Recombinant human enzymes, administered once a week as an intravenous infusion over 4 hours

Product	Company	MPS	Estimated yearly cost	2014 sales
ALDURAZYME (LARONIDASE)	genzyme	MPS I	$ 298K	$ 192M
elaprase (idursulfase)	Shire	MPS II	$ 522K	$ 593M
Naglazyme (GALSULFASE - rch)	BIOMARIN	MPS VI	$ 485K	$ 334M

Source: LifeSci Capital equity research, Analysis of Orphan Drug Market, February 4, 2016, National MPS Society, presse, sites Internet de la Société : taux de change : 1$ = 1,12 €

ERT have not been able to resolve the symptoms occurring in certain regions of the ophthalmological system, joints, cartilages, cardiac valves, … due to poor penetration of the enzyme(1)

Sources: (1) H. Noh, J. I. Lee; Current and potential therapeutic strategies for muvopolysaccharidoses; Journal of Clinical Pharmacy

IVA336, the first oral therapy in MPS I, II and VI

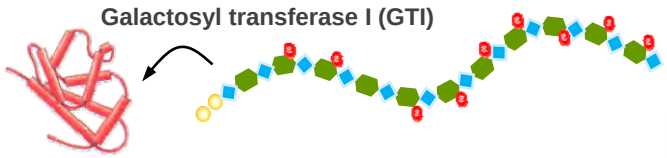
Activity	▶ Mechanism of action via modulation of GAG synthesis which accumulation triggers MPS ▶ Oral administration ▶ IVA336 reduction of GAG intracellular accumulation demonstrated in in vitro and in vivo relevant models ▶ IVA336 widely distributed in tissues that are poorly treated by enzyme replacement therapy ▶ IVA336 has the potential to replace current ERT treatments, especially in MPS VI patients ▶ 1,809 subjects treated in 32 phase I and II clinical trials for up to 16 weeks ▶ US biomarker study ongoing, and POC Phase I/II study initiated with first patient enrolment expected in 2017
IP	▶ Use patent filed in 2013 and granted in EU (Nov. 2015) and the US (Feb. 2017) ▶ LOE 2039 including 5-year extension
Safety	▶ Good safety profile ▶ Very low toxicity in vivo ▶ Well tolerated and safe in multiple phase I and phase II clinical studies allowing to start a POC study in MPS VI patients

MPS VI selected as first indication to demonstrate IVA336 efficacy

IVA336 original mechanism of action could provide additive benefit to enzyme replacement therapies (ERT) in MPS I, II and VI patients

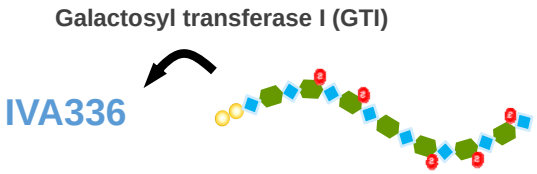
IVA336 diverts endogenous protein-bound GAG synthesis to soluble IVA336-bound CS and DS synthesis

Synthesis of proteoglycans (HS, CS, DS)



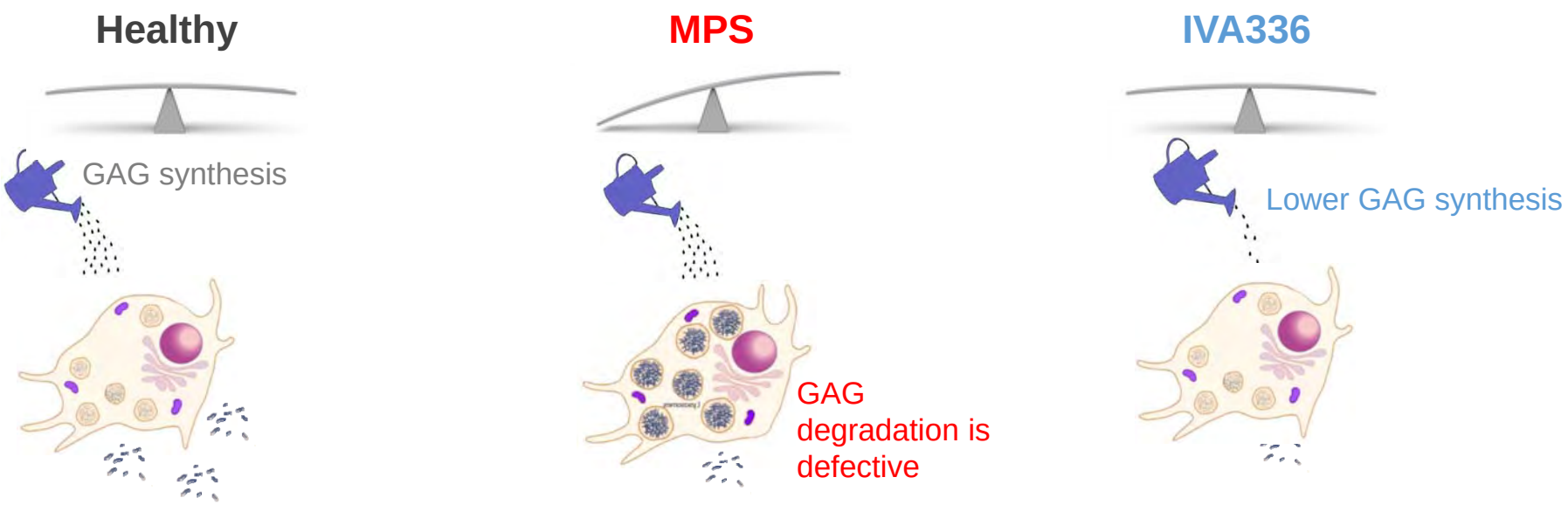
GTI activity

Substrate	Vm/Km
D-xylose	0.8
IVA336	1857



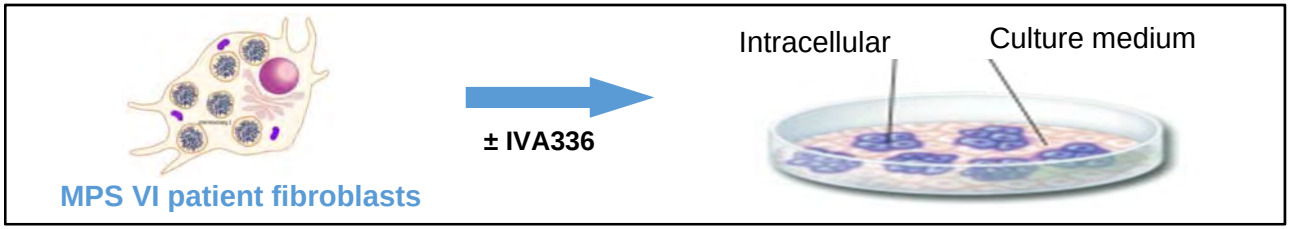
Synthesis of soluble GAGs (DS and CS)

IVA336 could restore GAG balance in MPS I, MPS II and MPS VI where CS and/or DS accumulate



Sources: H. Noh, J. I. Lee; Current and potential therapeutic strategies for muvopolysaccharidoses; Journal of Clinical Pharmacy

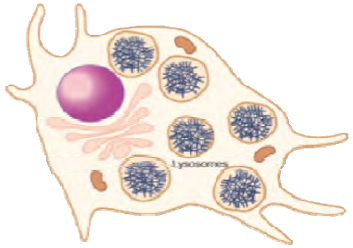
IVA336 triggers the synthesis and excretion of soluble CS/DS from MPS VI cells



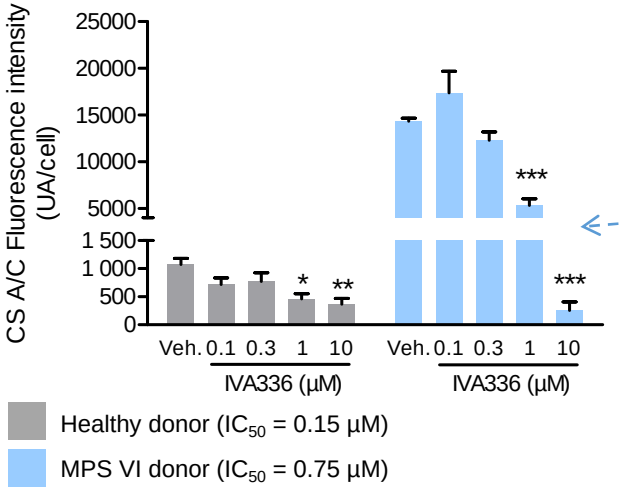
IVA336 decreases intracellular CS levels

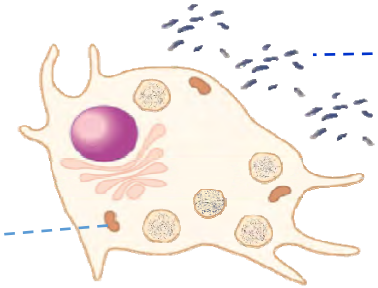
IVA336 increases GAG levels excreted

MPS VI patient cells

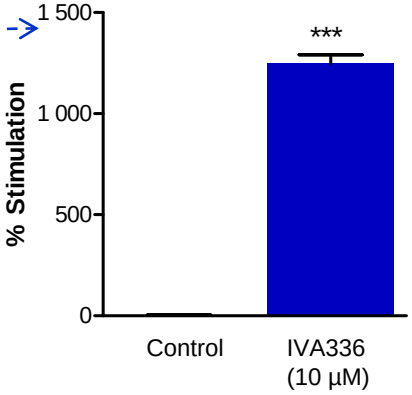


Intracellular CS/DS





Extracellular CS/DS



IVA336 decreases the accumulation of CS/DS in cells from MPS VI patients

Source: Company data

IVA336 decreases organ/tissue GAG accumulation and restores mobility *in vivo* in MPS VI mice

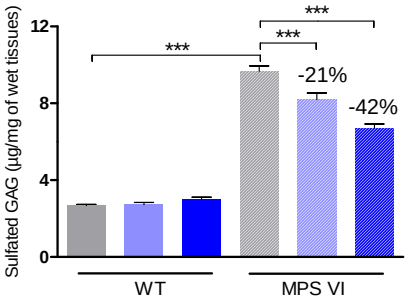
Wild-type and Arsb mutant mice
Treatment start when animals are one month old

6 months

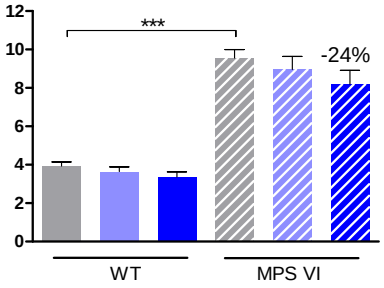
± IVA336
Given in food

- Sulfated GAGs in organs
- Mobility test
- Laboratory analysis: Alkaline phosphatase (ALP)

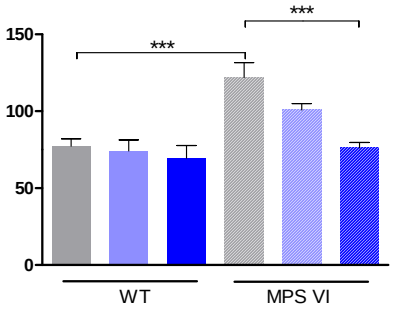
Liver GAG levels



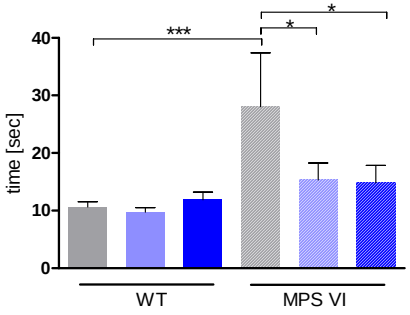
Spleen GAG levels



Serum ALP



Mobility in Pole-test



Wild-type mice (WT)
- Chow diet
- IVA336: 1.5 g/kg in food
- IVA336: 4.5 g/kg in food

Arsb mutant mice (MPS VI)
- Chow diet
- IVA336: 1.5 g/kg in food
- IVA336: 4.5 g/kg in food

IVA336 the first substrate reduction therapy approach for MPS VI patients

Source: Company data

IVA336 has the potential to positively differentiate versus current therapies in MPS VI

	IVA336 (inventiva)	Naglazyme (BIOMARIN)
Effect on mobility		
Effect on eyes, cartilage, bones, heart valves, spinal cord compression		No distribution
Safety		
Dose regimen		Weekly infusion

IVA336's iMProveS phase I/II study in MPS VI patients

Trial design

European study: 3 sites in UK, Turkey, Portugal

- Principal investigators: Pr Harmatz (Children's Hospital Oakland, USA); Pr Ezgü (Pediatric Disorders, Ankara, Turkey); Pr Hendriksz (Manchester , UK)

Inclusion criteria

- MPS VI patients (≥ 16 year-old) and treated with ERT for ≥1 year

First patient
- H2 2017

18 patients *double blind* + ***6 patients*** *open label*
24 week treatment

- **Placebo + ERT, 6 patients**
- **IVA336, 250 mg bid + ERT, 6 patients**
- **IVA336, 500 mg bid + ERT, 6 patients**
- **IVA336, 500 mg bid, 6 patients**

End of treatment

4 week

Follow up

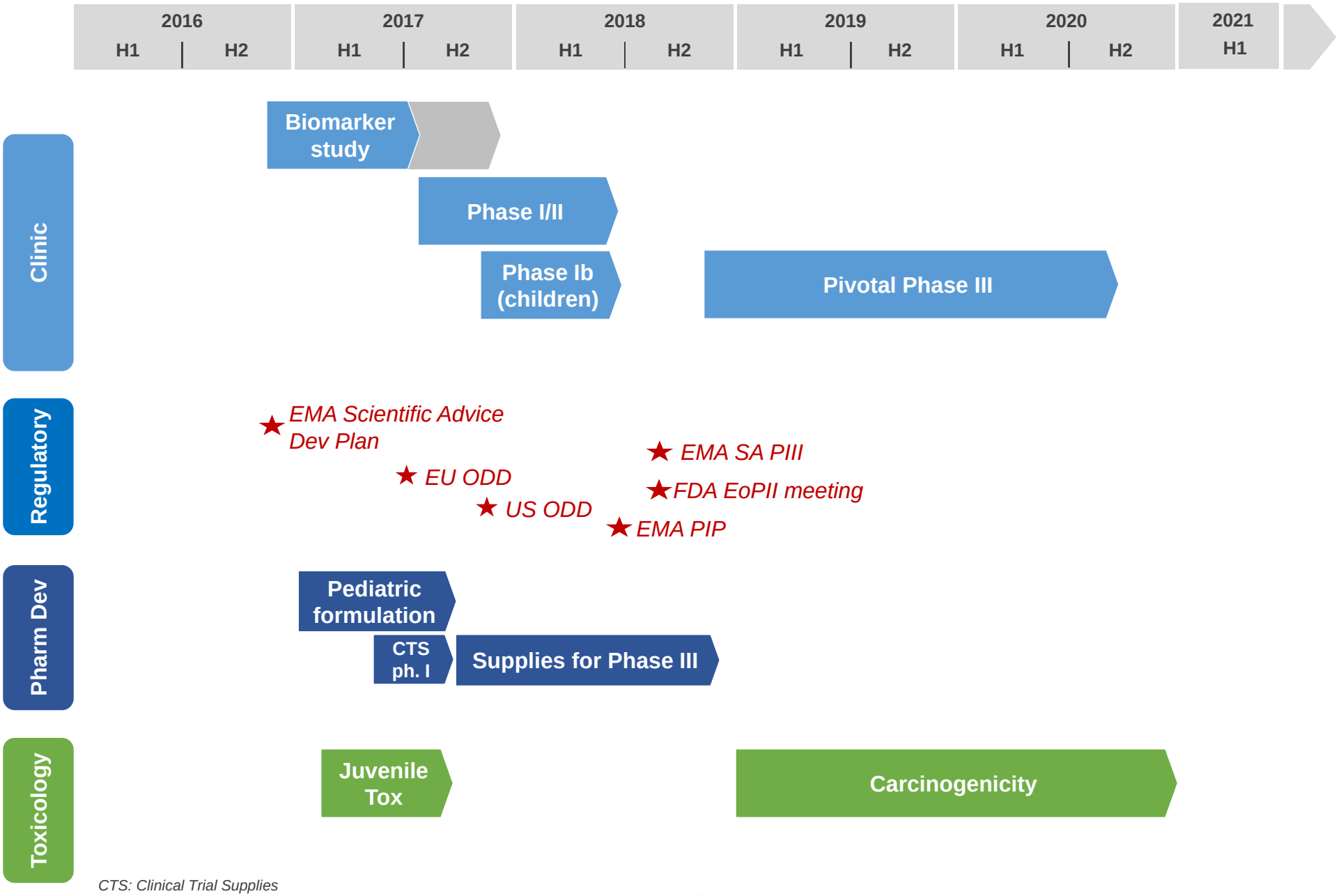
Safety

- Clinical and biological assessments (standard tests)

Efficacy

- Leukocyte and urinary GAG content, 6 minutes walk test,…

IVA336 a phase III-ready program in MPS VI by end-2018



CTS: Clinical Trial Supplies

Two partnerships with leading pharma companies

abbvie

Boehringer Ingelheim

A successful collaboration in place with AbbVie for an expanding market

ROR-γ drug discovery collaboration

- **RORγ program (ABBV-553) initiated Phase I in 2016**
  - Inventiva and AbbVie RORγ small molecule inhibitors suppress the production of all Th17 (IL17A, IL17F and IL22) inflammatory cytokines and is orally active in several models of psoriasis
  - Program well positioned to be best in class and first in class
- **RORγ program addresses large markets currently dominated by biologics**
  - Psoriasis, rheumatoid arthritis, ankylosing spondylitis, ulcerative colitis.
- **RORγ could prove to be superior to biologics**
  - Greater efficacy compared to biologics only block IL17A
  - Orally available vs injections
  - Potentially safer due to shorter half-life
- **Inventiva is eligible to multiple milestones payments and sales royalties**

Fibrosis target validation

- **Inventiva's fibrosis team validating new targets**
  - Focus on liver fibrosis
  - Access to in vitro and in vivo models in a fully integrated collaborative research program with one global project team
- **Eligible to fee for service payments**

Sources: (1) Datamonitor Psoriasis Forecast 2014

Inventiva's collaboration with BI leverages the company's fibrosis expertise

Collaboration to discover anti-fibrotic drugs

- **Multi-year R&D collaboration and licensing partnership**
  - Apply Inventiva's technology and expertise in validating an undisclosed target as an innovative approach for the treatment of IPF and potentially other fibrotic diseases.
  - The target addresses a central hypothesis for the pathogenesis of IPF: distinct mechanism of action to the approved therapeutics Nintedanib (commercialized by BI) and Pirfenidone (commercialized by Roche) with potential added benefit to the patients
- Target validation carried out by Inventiva and the subsequent search for a drug candidate will be jointly conducted by Inventiva and BI teams, with the latter to take full responsibility of clinical development and commercialization.
- Inventiva is also eligible to up to **€170 million** in milestones and to royalties

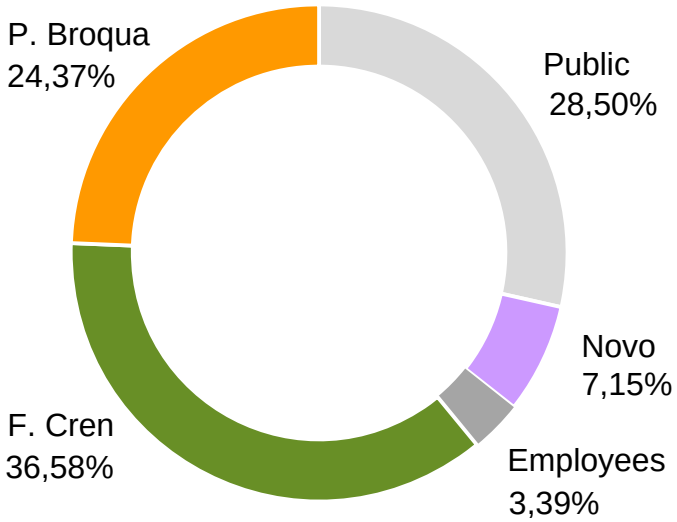
Financials

Strong cash position and shareholder base

Key financials

- Cash balance at 31/03/2017: €68,0M
- February 2017 IPO proceeds: €48,5M
- 2016 sales: €9,4M
- 2016 R&D expenditures: €22,1M

Shareholder base



- Key public investors include:
  - BVF Partners L.P: ~11%
  - Arbevel: ~4.5%
  - Perceptive: ~3%
  - …
- Listed on Euronext Paris: IVA
- Market cap (7/04/2017): €114M

Key achievements reached in 2016 and dense news flow in 2017/2018

	2016	2017	2018
IVA337	☑ Initiation of NASH phase IIb ☑ Enrollment of SSc phase IIb	▶ Last patient enrolled in phase IIb NASH ▶ Last patient enrolled in phase IIb SSc	▶ Results Phase IIb NASH ▶ Results Phase IIb SSc
IVA336	☑ Efficacy in ARSB mutant mice achieved ☑ Biomarker study launched ☑ MPS patent granted in EU	☑ MPS patent granted in US ▶ Biomarker study results ▶ First patient enrolment for Phase I/II in MPS VI ▶ Orphan status designation	▶ Results Phase I/II in MPS VI ▶ Start pivotal Phase III in MPS VI
Collaborations	☑ Oral RORγ Pre-CC candidate delivered ☑ Entry in Phase I of ABBV-553 ☑ Signature of BI collaboration	▶ Inventiva eligible to receive multiple milestones from AbbVie and BI	
Discovery	☑ 2,3M€ of non-dilutive funding received for Yap-Tead	▶ Yap-Tead: Vivo POC ▶ NSD2: Vitro POC	

Appendix

Management team

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

- Wide expertise within the areas of research, development, marketing, strategy and commercial operations.
- Held senior positions at Abbott, Fournier, Solvay Pharma and The Boston Consulting Group.
- Former member of both Fournier and Solvay Pharma Executive Committees.

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

- Has successfully managed numerous research programs leading to the discovery, development and commercialization of innovative compounds, including IVA337 and Ferring's GnRH antagonist Degarelix/ Firmagon®.
- Held several senior research positions at Fournier, Solvay Pharma and Abbott.

Jean Volatier, MA, CFO

- Started his career with PwC in Paris and Philadelphia.
- Former Head of controlling at URGO & Financial Director International Operations of Fournier.
- Held various positions as CFO with Soufflet and Naos groups.

Jean-Louis Abitbol, MD, MSC

- Former R&D and Global Medical Affairs director at HRA Pharma, responsible for achieving the European OTC Switch of EllaOne® and the registration of Ketoconazole and mutual recognition of Metopirone in Cushing syndrome.
- CMO for Trophos: led the clinical development of Olesoxime.
- Several positions of increasing responsibility in pharmaceutical companies in France and the USA (Pierre Fabre Médicament, Jouveinal/Parke-Davis/Pfizer and CERNEP-Synthélabo).

Board of Directors
Other than Frédéric Cren and Pierre Broqua

Karen Aiach, Director

- Founder and CEO of Lysogene, a clinical stage biotech company focused in CNS diseases. Karen has served as a patient representative and member of the Paediatric Committee of the European Medicines Agency (EMA). She is a founding and executive member of the International Rare Diseases Research Consortium (IRDiRC).

Chris Buyse, Director

- More than 30 years' expertise in international finance and financial management. CFO of the Belgian company CropDesign, where he coordinated the acquisition by BASF and CFO and Director of ThromboGenics, a biotechnology company listed on the NYSE Euronext Brussels. He currently holds a Director position in several private and public companies.

Philippe Goupit, Director

- Until recently Vice President Corporate Licences at Sanofi covering M&A and licensing activities. Philippe also served for some years as the Head of Investor Relations at Sanofi. Philippe is a member of MedDay's Board of Directors.

Jean-Louis Junien, Director

- Large expertise in the discovery and development of drugs as Vice President R&D Jouveinal-Warnert Lambert, Director of the Ferring Research Institutes in Southampton (UK) and La Jolla (United States), Global CSO for Ferring Pharmaceuticals, CSO of Laboratoires Fournier.

Chris Newton, Director

- Founding member and CSO of Argenta Discovery and Board Member and CSO of BioFocus. SVP Galapagos Services, managing the services business of Galapagos, after the acquisition of BioFocus by Galapagos. Previously Chris occupied several senior positions within Rhone-Poulenc/Aventis R&D organization.

Annick Schwebig, Director

- Annick was the founder and CEO of Actelion Pharmaceuticals France and held senior positions in the pharmaceutical industry as Vice President Medical Affairs France and Vice President Research and Development Europe at BMS. Annick is a member of Cellectis' Board of Directors.