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Many thanks for your supporting messages
We’ll never surrender
AASLD Investor event

Agenda

• Introductory remarks
  J-F. Mouney

• The NASH landscape in 2015
  V. Ratziu

• GOLDEN-505: Study Results

• Elafibranor Cardiometabolic
  B. Staels

• GENFIT: global NASH management
  S. Mégnien

• Launch of phase 3 in 2015

• Concluding remarks
  J-F. Mouney
GENFIT

A

BIOPHARMACEUTICAL

COMPANY
GENFIT: Innovative company in diagnostic & treatment of metabolic & inflammatory diseases

• Public company focused on metabolic diseases & associated complications, including liver related disorders
  › Elafibranor (GFT505) lead program Phase 2b completed, first-in-class PPAR α/δ candidate for NASH
  › Programs of companion tests / biomarkers associated with particularly NASH/NAFLD

• World-leading expert in nuclear receptor based drug discovery
  › Revenue-generating alliances with multiple major pharmaceutical companies

• Founded in 1999 (Lille, France – Cambridge, US) – 90 employees

• Since 2006, Euronext Paris - compartment B (GNFT)
GENFIT: Innovative company in diagnostic & treatment of metabolic & inflammatory diseases

Addressing the current need of non-invasive approaches to NASH diagnosis

March 2015
Positive results in NASH obtained

First-in-class orally available small molecule candidate

New treatment class autoimmune hepatitis

Fast-Track Designation

MARKETED DRUG
Oral administration, single pill, once daily

LAUNCH IN 2015

FIRST-IN-CLASS PPARα/δ DRUG CANDIDATE
NASH: Nonalcoholic steatohepatitis
Fibrosis & Cirrhosis

ELAFIBRANOR (GFT505)
THE NASH LANDSCAPE IN 2015
NASH: The liver manifestation of metabolic syndrome

Obesity prevalence

- 39% of adults aged 18 years and older were overweight.

Type 2 Diabetes prevalence

- 66% of obese or diabetic patients >50 years in the US have NASH with advanced fibrosis
- Prevalence of NASH in the adult US population (12%)

NASH: Urgent need to treat the increasing epidemic

### Incidence of Cardiovascular disease

<table>
<thead>
<tr>
<th>Author (Ref.)</th>
<th>Number of subjects</th>
<th>Diagnosis of NAFLD</th>
<th>Follow-up duration (years)</th>
<th>Proportion of deaths due to cardiovascular disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soderberg</td>
<td>118</td>
<td>Histology</td>
<td>24 (median)</td>
<td>30%</td>
</tr>
<tr>
<td>Ekstedt</td>
<td>129</td>
<td>Histology</td>
<td>13.7 ± 1.3 (mean)</td>
<td>16%</td>
</tr>
<tr>
<td>Adams</td>
<td>421</td>
<td>Imaging</td>
<td>7.6 ± 4.0 (mean)</td>
<td>25%</td>
</tr>
<tr>
<td>Dam-Larsen</td>
<td>170</td>
<td>Histology</td>
<td>20.4 (median)</td>
<td>38%</td>
</tr>
<tr>
<td>Rafiq</td>
<td>173</td>
<td>Histology</td>
<td>18.5 (median)</td>
<td>12.7%</td>
</tr>
</tbody>
</table>

### Waitlist registrations for liver transplantation

- Predicted to become the first indication for liver transplantation by 2020

Increase all-cause mortality, CV mortality leading cause

NASH, the severe form of NAFLD, leads to liver fibrosis, cirrhosis & HCC

- NASH is the underlying cause of progressive fibrosis resulting from necroinflammation, and leads to cirrhosis
- Estimated 20 millions US adult patients with NASH & advanced fibrosis

Resolution of NASH is a clinical objective to prevent progressive fibrosis, cirrhosis and related complications.

- Necroinflammation (ballooning & inflammation) is the driver leading to progressive liver fibrosis.
PHASE 2b
GOLDEN-505
BACKGROUND & STUDY DESIGN
Rationale for treating NASH with Elafibranor, a dual PPARα/δ agonist with multiple activities

**ELAFIBRANOR GOLDEN505 IN NASH**

- Improvement of glucose homeostasis and insulin sensitivity
- Phase 2a trials

- Favorable effects on plasma lipids
  - Decreases TG
  - Decreases LDL-C
  - Increases HDL-C

- Absence of safety concern

- Phase 2a trials

- Efficacy in NASH acting on:
  - Steatosis
  - Inflammation
  - Hepatocyte injury
  - Fibrosis

- Phase 2a trials

- Improvement of liver dysfunction markers

**Disease models**

- Phase 2a trials

- Anti-inflammatory properties

GOLDEN-505: The first international clinical trial with “Resolution of NASH without worsening of fibrosis” as the Primary endpoint

- **Inclusion criteria on liver biopsy**
  - Presence of NASH defined by at least “1” in each component (steatosis, inflammation, ballooning) = NAS score of 3 and above
  - High placebo effect with NAS=3 patients

- **Recruitment of 274 randomized treated patients, 237 patients with 2 biopsies**
  - 9 countries including USA + Europe
  - 3 arms: 80mg, 120mg, placebo
  - 56 centers

- **Primary endpoint**
  - “Resolution of NASH without worsening of fibrosis” after 52 weeks of treatment
  - Therapy Response according to Protocol definition:
    - NASH resolution = Score of 0 for at least one of the 3 components of NAS
    - Worsening of fibrosis = Progression to bridging fibrosis from F0, F1 and F2 - or progression to F4 for F3
    - “2012” consensus definition of resolution

- **Centralized reading**
  - Rigorous methodology
  - Centralized reading for both inclusion biopsy and end-of-treatment biopsy
  - Adequacy of sample checked by central pathologist
HISTOLOGICAL RESULTS

NEW "2015" DEFINITION OF RESOLUTION OF NASH
Newly recommended definition of resolution of NASH focuses on Necroinflammation

• **Resolution of NASH** is an appropriate approach to **stop fibrosis progression***

• The newly recommended definition of **Resolution of NASH** without worsening of fibrosis focuses on disease activity reflected by the extent of **ballooning** and **inflammation** (**necroinflammation**):
  - Absence of **ballooning** (score = 0)
  - Absent or mild **inflammation** (score 0-1)
  - Steatosis can be present (score 0-3)
  - Worsening of fibrosis = any progression ≥1 stage

*Sanyal, Gastro 2015
Elafibranor 120mg has significant effect vs placebo in both global and NAS≥4 populations

<table>
<thead>
<tr>
<th>N</th>
<th>NAS</th>
<th>Placebo</th>
<th>Elafibranor 120mg</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>274</td>
<td>All patients (ITT)</td>
<td>17%</td>
<td>21%</td>
<td>0.28</td>
</tr>
<tr>
<td>274</td>
<td>All patients (ITT)</td>
<td>12%</td>
<td>19%</td>
<td><strong>0.045</strong></td>
</tr>
</tbody>
</table>
Elafibranor 120mg has significant effect vs placebo in both global and NAS≥4 populations

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Placebo</th>
<th><strong>Elafibranor 120mg</strong></th>
<th>p-value</th>
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<tbody>
<tr>
<td>N</td>
<td>NAS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>274</td>
<td>All patients (FAS)</td>
<td>12%</td>
<td>19%</td>
<td>0.045</td>
</tr>
<tr>
<td>234</td>
<td>NAS≥4</td>
<td>9%</td>
<td>19%</td>
<td>0.013</td>
</tr>
<tr>
<td>204</td>
<td>NAS≥4 with fibrosis (any stage)</td>
<td>11%</td>
<td>20%</td>
<td>0.009</td>
</tr>
</tbody>
</table>
All histological beneficial effects of Elafibranor 120 mg increase with baseline severity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline severity, bNAS</th>
<th>Placebo (%)</th>
<th>Elafibranor 120mg (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NAS ≥2 point reduction</strong></td>
<td>Severe (6-8), N=90</td>
<td>23</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>Moderate (4-5), N=144</td>
<td>20</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Mild (3), N=40</td>
<td>31</td>
<td>14</td>
</tr>
<tr>
<td><strong>Hepatocyte ballooning</strong></td>
<td>Severe</td>
<td>16</td>
<td>40</td>
</tr>
<tr>
<td>(≥1 point improvement)</td>
<td>Moderate</td>
<td>29</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>38</td>
<td>21</td>
</tr>
<tr>
<td><strong>Lobular inflammation</strong></td>
<td>Severe</td>
<td>39</td>
<td>53</td>
</tr>
<tr>
<td>(≥1 point improvement)</td>
<td>Moderate</td>
<td>18</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>44</td>
<td>14</td>
</tr>
<tr>
<td><strong>Steatosis</strong></td>
<td>Severe</td>
<td>10</td>
<td>33</td>
</tr>
<tr>
<td>(≥1 point improvement)</td>
<td>Moderate</td>
<td>27</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>0</td>
<td>7</td>
</tr>
</tbody>
</table>

Descriptive statistics in the FAS population (N=274): missing end-of-treatment liver biopsy considered as non responders.
Elafibranor responders have significant histological responses

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

Elafibranor 120mg Responders vs Elafibranor 120mg Non-Responders

Elafibranor 120 mg Responders

Elafibranor 120 mg Non responders

NAS

Steatosis

Ballooning

Inflammation

Fibrosis

NASH components

###: p<0.001

#: p<0.05
LIVER ACTIVITY
MARKERS
AND SCORES
Elafibranor significantly lowers plasma marker-based NASH scores, indicating an antifibrotic effect.

Elafibranor 120mg vs placebo on composite scores

<table>
<thead>
<tr>
<th>Test</th>
<th>Effect Size (Absolute change)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIBROTEST</td>
<td>-0.05</td>
<td>***</td>
</tr>
<tr>
<td>STEATOTEST</td>
<td>-0.11</td>
<td>***</td>
</tr>
<tr>
<td>NAFLD-Fibrosis score</td>
<td>-0.25</td>
<td>**</td>
</tr>
</tbody>
</table>

** : p<0.01
*** : p<0.001
Elafibranor lowers liver markers

The effect size vs placebo was calculated and expressed as LSMean±Standard Error.

*** : p<0.001
Elafibranor has efficacy on inflammation markers, demonstrating beneficial impact on the liver.

Elafibranor 120mg vs placebo on inflammatory markers

The effect size vs placebo was calculated and expressed as LSMean±Standard Error.

** : p<0.01
*** : p<0.001
CARDIOMETABOLIC PROTECTION MARKERS
The NASH patient: A patient with high cardiovascular risk

- NASH is highly prevalent in obesity
- NASH is associated with atherogenic dyslipidemia and increased CV risk
- NASH is associated with type 2 Diabetes

Central role of the liver in NASH and its complications
New generation therapies should improve global risk management
Cardiovascular diseases is the leading cause of death in NASH patients

- Cardiovascular diseases: 38%
- Non-liver cancer: 19%
- Cirrhosis complications: 8%
- HCC: 1%
- Liver transplantation: <1%
- Infections: 8%
- Other: 18%
- Unknown: 8%
NASH patients should be managed for CVD risk

FDA/AASLD recommendation (Hepatology 2014)

• “NASH is associated with type II diabetes, increased cardiovascular risk and cancer-related mortality (74-76). For this reason, it seems important to monitor LDL- and HDL-cholesterol, triglycerides, and diabetes control (e.g. hemoglobin A 1C ) in phase 2b and 3 NASH trials.”

• “It is imperative that any drug developed for NASH be at least neutral from a cardiovascular risk perspective and ideally also reduce cardiovascular risks.”

Elafibranor has beneficial effects on top of standard treatments (statins, anti-diabetic drugs, etc.)
Elafibranor improves CV risk factors: TG, Cholesterol, LDL-C, Remnant-C, HDL-C

The effect size vs placebo was calculated and expressed as LSMean±Standard Error.

** : p<0.01
*** : p<0.001
Elafibranor improves glucose homeostasis / insulin sensitivity in type 2 diabetic NASH patients

Effect size vs placebo was calculated and expressed as LSMean±Standard Error.

# : p<0.05
## : p<0.01

Significant decrease in HbA1C vs placebo

HbA1C
Elafibranor improves NASH by acting on all cell types involved in NASH.

Elafibranor decreases Fibrosis

Elafibranor improves Vaso-reactivity

Elafibranor decreases Inflammation

Elafibranor increases Insulin Sensitivity

EC decreases Liver fat

PPRE decreases Inflammation Fibrosis

NFkB decreases Liver fat

AP-1 decreases Inflammation Fibrosis

FAO

Elafibranor

Parenchymal cells

KC

HSC

ET-1
HIGHLY FAVORABLE SAFETY OF ELAFIBRANOR
Elafibranor has very good safety and tolerance profiles, essential for a long-term treatment

- **Very good tolerance confirmed after one year of treatment**
  - **No** Death and no Major Cardiovascular Events (MACE)
  - **No** effect on body weight
  - **No** signal on cancer
  - **No** meaningful change in safety markers and hematology
  - **No** signal for pruritus

<table>
<thead>
<tr>
<th></th>
<th>Elafibranor 80mg N=93</th>
<th>Elafibranor 120mg N=89</th>
<th>Placebo N=92</th>
<th>Total N=274</th>
</tr>
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<tbody>
<tr>
<td>Nausea</td>
<td>13</td>
<td>9</td>
<td>9</td>
<td>31</td>
</tr>
<tr>
<td>Vomiting</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>Headache</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>21</td>
</tr>
<tr>
<td>Fatigue/asthenia</td>
<td>9</td>
<td>5</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>Creatinine increase</td>
<td>1</td>
<td>6</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>1</td>
<td>8</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td>Myalgia</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Rash</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Pruritus</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>
GENFIT focuses on global management of NASH patients

• **DIAGNOSIS**
  Development of non-invasive biomarkers
  Identification of patients who should receive therapeutic intervention

• **TREATMENT**
  Therapy to meet unmet need
  › Resolution of histological NASH
  › Safe and well tolerated for long term treatment
  › Beneficial cardioprotective profile for global management of NASH patients

Elafibranor enters phase 3
GENFIT’s new algorithms are more predictive than existing scores to identify NASH patients for treatment

- Identification of NASH patients with NAS≥4 with F2 or F3 fibrosis
- Important for the patient: non-invasive diagnosis for treatment
- Important for the physician: facilitated diagnosis of patients to treat
- Important for the NASH market: to meet full potential
- GENFIT is developing proprietary biomarker algorithms to identify, diagnose and follow-up patients
- Innovation with bioinformatics and miRNAs

FDA position on NASH diagnosis

“...there is an urgent unmet need to develop biomarkers that facilitate the diagnosis, identification of populations at risk, assessment of disease progression or regression, and/or response to treatment.” Page 1401 (Hepatology 2015;61:1392-1405)
Launch of Phase 3 in 2015

- **Study population: High-risk patients**
  - NASH with a NAS ≥4
  - Fibrosis stage F2 and F3
  - (F1 + cardiometabolic risk)

- **Subpart H (interim analysis)**
  - Histological **primary endpoint:**
    - NASH resolution, corresponding to ballooning=0, inflammation=0-1, without worsening of fibrosis (1 point increase)
    - Central reading for all biopsies (inclusion & follow-up)
  - Key secondary endpoint: improvement of histological fibrosis, to be considered as an additional labeling claim
  - Duration 18 months of treatment
  - All patients followed until the occurrence of a pre-defined number of progressions to cirrhosis and other liver related events

- **General design**
  - Approximately **900 patients** in interim analysis (total of 1,800 patients)
  - 2 arms: Elafibranor 120mg & Placebo
Launch of Phase 3 in 2015

**FIRST TREATMENT PERIOD**
- Placebo
- Elafibranor 120mg

**EXTENSION PERIOD**
- Placebo
- Elafibranor 120mg

**SUBPART H**
- 900 patients

**200 centers**

**TRIAL INITIATION**
- Q4 2015

**72-WEEK INTERIM ANALYSIS**
- PRIMARY ENDPOINT
- NASH RESOLUTION WITHOUT WORSENING OF FIBROSIS

**END OF STUDY**
- Occurrence of a pre-defined number of events including progression to cirrhosis (time-to-event)

**Up to 1800 patients**
Large unmet needs, large market size

THANK YOU FOR YOUR SUPPORT

PATIENTS
EXPERTS & INVESTIGATORS
INVESTORS

www.genfit.com