We structurally engineer fatty acids to generate a novel class of drugs with a unique ability to conquer metabolic diseases.

- Icosabutate
  - NASH Phase 2b
- SEFA-1024
  - Dyslipidemia (VHTG) Phase 1
- SEFA-6179
  - IFALD (orphan) Phase 1

- Phase 2b data in Q1-23
- Phase 1 data in Q2-22
- Phase 1 initiation in Q4-21

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Uniquely positioned as experts in next-gen Structurally Engineered Fatty Acids (SEFAs)

- Exclusive, global license of a library of pre-clinical and clinical SEFAs
- Excellent safety and efficacy data from Icosabutate Phase 1+2 lipid trials covering 300+ patients
- Phase 2b NASH trial of Icosabutate initiated in 3Q-19

Pronova Biopharma (acquired by BASF)
- Omacor/Lovaza: 1st generation omega-3 fatty acid with $1b+ peak sales
- Began development of next-generation SEFAs; 1st compound, Icosabutate, entered clinic in 2011
- Terminated clinical programs post-BASF acquisition

Validated by dedicated Lifesciences investors with deep experience in NASH and fatty acid drug development

Experienced team, including ex-Pronova scientists with vast experience in lipid engineering

SERIES A: €25M (Nov 2017)
SERIES B: USD 40M (Dec 2019)
SERIES C: USD 80M (Dec 2021)

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Our focus: develop a new class of drugs targeting metabolic and inflammatory disease using the SEFA technology

Naturally occurring fatty acids
Examples: Lovaza, Vascepa
- Pivotal regulators of multiple metabolic and inflammatory process
- Limited potential as oral drugs due to systemic distribution, incorporation into complex lipids, and use as energy
- Only 10-15% reaches liver
- Used as energy

Unmodified fatty acids

Structurally engineered fatty acids (SEFAs)
Example: Icosabutate
- Designed to facilitate direct hepatic uptake and minimize systemic distribution & accumulation in complex lipids
- Unavailable for β-oxidation
- Maximize ability to activate both intra- and extracellular fatty acid signaling pathways
- 100% liver-targeted
- Fully available

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The unique structure of SEFAs allows for cellular fatty acid pathway targeting not achievable with unmodified fatty acids

**EPA (unmodified fatty acids)**
- Natural fatty acids enter the liver via hepatic artery
- Stored in phospholipid
- Energy storage in lipid droplets
- CO₂
- Burned for energy in mitochondria

**SEFAs**
- Icosabutate enters liver via portal vein
- Stored in phospholipid
- Free Fatty Acid Receptor
- Oxygenated metabolites
- CYP2C8/2C9
- Rapid excretion

By avoiding storage and use as a source of energy, Icosabutate is maximally available for key signaling pathways

- Clear advantage for PK and for potency (PD)
- e.g., Vascepa = 4g/day vs. Icosabutate 600 mg/day
# SEFA pipeline development plan

<table>
<thead>
<tr>
<th>Product Candidate</th>
<th>Preclinical</th>
<th>IND/CTA-Enabling</th>
<th>Phase 1</th>
<th>Phase 2</th>
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<tr>
<td>SEFA-1024</td>
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<td>Phase 1 data in Q2-22</td>
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<tr>
<td>Very High Triglycerides (VHTG)</td>
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</table>
NASH: A large and growing market

High prevalence

3–12% of US population affected by NASH*

No therapies approved in the US and Europe

Blockbuster potential

Efficacious treatments can take share in a market anticipated to reach $27B globally in 2029**

2029 Total NASH Sales: $27.2 billion

- US: 94.3%
- UK: 1.6%
- Germany: 1.3%
- Japan: 1.2%
- France: 0.8%
- Italy: 0.6%
- Spain: 0.3%

* Mechanism of NAFLD development and therapeutic strategies; Friedman et al, Nature Medicine 2018
** Global data NASH report 2020

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Icosabutate targets three pillars of NASH: dysregulated metabolism, inflammation and fibrosis

Reduced oxidative stress

Reduces metabolic stress

Anti-inflammatory

Anti-fibrotic

FFAR1, FFAR4, PPAR-α
- Decreased oxidized lipotoxic lipids
- Improved glycemic control
- Increased fatty acid oxidation

FFAR4, arachidonic acid cascade
- Decreased hepatic macrophage numbers
- Decreased pro-inflammatory arachidonic acid metabolites
- Reduced pro-inflammatory gene expression

Anti-proliferative effect on stellate cells
- Reduced pro-fibrotic gene expression
- Reduced myofibroblast number
- Decreased collagen formation

Reduced oxidative stress
# Icosabutate is efficacious in all pre-clinical NASH models tested and outperforms competitors

<table>
<thead>
<tr>
<th>MODEL</th>
<th>Fibrosis</th>
<th>Inflammation</th>
<th>Steatosis</th>
<th>Ballooning/hypertrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td>CDAA (Schuppan)</td>
<td></td>
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<tr>
<td>vs GLP-1 agonist</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>– Equivalent</td>
<td>Not measured</td>
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<tr>
<td>vs EPA</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>Not measured</td>
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<tr>
<td>CDAA (Gubra)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>vs GLP-1 agonist</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>Not measured</td>
</tr>
<tr>
<td>vs obeticholic acid</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>Not measured</td>
</tr>
<tr>
<td>vs ACC inhibitor</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>– Equivalent</td>
<td>Not measured</td>
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<tr>
<td>ob/ob NASH (Gubra)</td>
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<tr>
<td>vs obeticholic acid</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>– Equivalent</td>
<td>✓ Superior</td>
</tr>
<tr>
<td>APOE3*L NASH (TNO)</td>
<td></td>
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</tr>
<tr>
<td>vs rosiglitazone</td>
<td>✓ Superior</td>
<td>– Equivalent</td>
<td>✓ No effect (macro)</td>
<td>✓ Superior</td>
</tr>
<tr>
<td>ob/ob NASH (Gubra)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>vs pioglitazone</td>
<td>✓ Superior</td>
<td>✓ Superior</td>
<td>Not measured</td>
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</tbody>
</table>

Comparison of Icosabutate (at human equivalent doses of 300-600mg/day) versus competitors in established pre-clinical NASH models

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Highlights from phase 1 and phase 2 clinical trials

- Multiple phase 1 and phase 2 clinical trials completed to date; in hypercholesterolemia, mixed dyslipidemia and VHTGs subjects
- Potent reduction in atherogenic lipids and apolipoproteins across diverse populations**
- Significant improvements in fasting insulin, HOMA-IR and hsCRP
- Marked reductions in ALT and GGT in patients with elevated levels of liver enzymes at baseline
- Well tolerated and excellent safety profile in > 280 patients

* Cardiology; Kastelein et al, 2015; Icosabutate, a structural engineered fatty acid, improves the CV risk profile in statin-treated patients with Hypertriglyceridemia
** Journal of clinical lipidology; Bays et al, 2016; Icosabutate for the treatment of very triglycerides: a placebo controlled, randomized, double blind, 12 week clinical trial

Phase 1b study in hypercholesterolemic subjects***

![Phase 1b study graph showing changes in lipid profile](image)
Phase 2b ICONA trial in NASH

- Target enrolment: 264 patients
- Three arms: 300mg, 600mg and placebo
  - Primary endpoint: histology after 1 year (resolution of NASH without worsening of fibrosis)
  - Secondary endpoints: histology components of NAS, fibrosis, imaging by MRI-PDFF and cT1, fibrosis biomarkers Pro-C3 and ELF panel and liver function tests
- Pre-specified Interim analysis after 90 patients complete 16 weeks treatment (announced in Q1-21)
- Conducted in the US (extended to 45-50 sites)
- Recruitment completed in H2-21
- Final data expected in Q1-23

Phase 2b ICONA trial in NASH

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Pre-specified hierarchical analysis provides strong support for the primary, secondary endpoints and shows comorbidity improvement

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline median</th>
<th>Reductions vs. placebo (LS means)</th>
<th>P value</th>
<th>Impact on primary endpoint and comorbidity</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT</td>
<td>56 U/L</td>
<td>-25 U/L</td>
<td>0.0001</td>
<td>Resolution of NASH</td>
</tr>
<tr>
<td>GGT</td>
<td>51 U/L</td>
<td>-29 U/L</td>
<td>0.0001</td>
<td>Resolution of NASH</td>
</tr>
<tr>
<td>Plasma TG</td>
<td>161 mg/dl</td>
<td>-34 mg/dl (-35%*)</td>
<td>0.02</td>
<td>CV comorbidity</td>
</tr>
<tr>
<td>Pro-C3</td>
<td>17.6 ng/dl</td>
<td>-4.58 ng/ml (-28%)</td>
<td>0.0006</td>
<td>Fibrosis</td>
</tr>
<tr>
<td>MRI-cT1</td>
<td>958 ms</td>
<td>-82 ms</td>
<td>NS</td>
<td>Inflammation/fibrosis</td>
</tr>
<tr>
<td>MRI-PDFF</td>
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</table>

* vs baseline

- The highly significant data from the hierarchical analysis provide **strong support for the ICONA primary histology endpoint**; resolution of NASH without worsening of fibrosis
- The lowering of plasma TG, in conjunction with reductions of ApoC3 and remnant cholesterol, **demonstrate improved lipoprotein metabolism and hence a reduction of CV risk**
- Additional parameters had **remarkable significant reductions** (e.g., hsCRP, HbA1c)
- MRI-PDFF is unchanged
NASH is a multi-factorial disease where Icosabutate has a multi-factorial MoA.

The typical NASH patient has metabolic syndrome with increased cardiovascular risk.

Chronic therapy for NASH patients requires high tolerability, a mild side effect profile and ease of use.

Given the complexity of NASH, combination therapy is anticipated for a large part of the NASH patient pool.

- Direct effect on the 3 pillars of NASH due to its pleiotropic targeting
- Unique anti-inflammatory MoA via activation FFAR4 and inhibition of arachidonic acid cascade
- Excellent efficacy in reducing atherogenic lipids and lipoproteins
- Improves glycemic control, decreases fasting plasma insulin
- Decreases hsCRP
- Excellent tolerability and safety profile shown in over 280 patients
- Once-daily oral drug (600mg)
- Complementary to most other NASH drugs
- Potential to be the backbone therapy for either monotherapy or in combination with other agents
## SEFA pipeline development plan

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</table>
## SEFA-1024 for the treatment of VHTGs

<table>
<thead>
<tr>
<th>VHTG (TG &gt; 500 mg/dl) is an attractive market with an unmet medical need</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Prevalence in the US is approximately 4 million patients, 3 million in Europe</td>
</tr>
<tr>
<td>• 2/3 of patients treated with statins, fibrates, niacin or high concentrate omega-3 do not get TG levels to treatment target*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>A cost-effective, oral, mono- and combination-therapy solution is required for the VHTG market</th>
</tr>
</thead>
<tbody>
<tr>
<td>• There is an unmet need for oral, cost-effective combination on top of a fibrate to reach treatment target and monotherapy for fibrate intolerant patients</td>
</tr>
<tr>
<td>• Injectable therapies in development (ApoC3, FGF21 and angPTL3) expected to treat VHTG patients in the most severe disease (&gt; 800 mg/dl)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Safety, tolerability and co-morbidities will drive differentiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Available VHTG oral therapies come with low tolerability (fibrates and nicotinic acid)</td>
</tr>
<tr>
<td>• Available VHTG oral therapies have no impact on glycemic control (approximately 30% of the VHTG patients are diabetics)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SEFA-1024 has an ideal drug profile for VHTGs based on pre-clinical data</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Once daily, oral and excellent safety and tolerability profile</td>
</tr>
<tr>
<td>• Triglyceride reduction of 40-50%, improvement of lipid profile</td>
</tr>
<tr>
<td>• Potent improvements in glycemic control</td>
</tr>
<tr>
<td>• Option for combination therapy on top of a fibrate</td>
</tr>
</tbody>
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* Source: Hypertriglyceridemia in statin-treated US adults: the National Health and Nutrition Examination Survey, 2019

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## SEFA-6179 for the treatment of IFALD

| IFALD is an orphan disease with a high unmet medical need | • Estimated number of patients in the US on total parenteral nutrition (TPN) for more than 3-6 months is 40,000  
• Most patients on TPN have signs of liver damage (elevated liver enzymes), around 20% will develop IFALD |
| --- | --- |
| No approved treatment available for adult IFALD patients | • Lipid amount/source is the main contributor to liver damage  
• Current best practice is reducing amount/source of intravenous lipids, not providing optimal nutrition regime  
• Off-label use of ursodiol and intravenous Omegaven (pediatric use) |
| The unmet need for an oral drug that prevents and treats IFALD and prevents TPN adjustment | • Treat IFALD-induced steatosis and inflammation  
• High availability in intestinal absorption compromised patients to enable low oral dose  
• Vulnerable patients can keep their optimal TPN regimen |
| SEFA-6179 has an ideal drug profile for IFALD based on pre-clinical data | • Oral dosing, excellent safety and tolerability shown in tox studies  
• High efficacy shown to reduce steatosis, cholestasis, inflammation and fibrosis  
• Normalization of liver enzymes |
Clinical development pipeline and key milestones in relation to corporate strategy

<table>
<thead>
<tr>
<th>YEAR</th>
<th>2019</th>
<th>2020</th>
<th>2021</th>
<th>2022</th>
<th>2023</th>
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<tbody>
<tr>
<td>Icosabutate NASH</td>
<td>ICONA Phase 2b</td>
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<td>Phase 3</td>
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<tr>
<td>SEFA-1024 VHT</td>
<td>Phase 1</td>
<td>DDI</td>
<td></td>
<td></td>
<td>Phase 2</td>
</tr>
<tr>
<td>SEFA-6179 IFALD (orphan)</td>
<td>Phase 1</td>
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<td>Phase 2</td>
</tr>
</tbody>
</table>

Interim analysis completed

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An experienced management team....

Rob de Ree  
CEO  
- > 25 years pharmaceutical and medtech experience  
- MSc Pharmacy  
- Byk Gulden, Medtronic, Crucell, BMEYE, Dezima Pharma, various board positions  
- Two transactions as CEO: BMEYE (2013; USD 40 mln) and Dezima Pharma (2015; USD 1.5 bln)  
- Operating Partner at BioGeneration Ventures

Stephen Harrison  
CMO  
- Consulting CMO  
- Medical degree from the University of Mississippi School of Medicine  
- Board certified in both Internal Medicine and Gastroenterology  
- Visiting Professor of Hepatology at the Radcliffe Department of Medicine, University of Oxford  
- Past Associate Editor for Hepatology and for Alimentary Pharmacology and Therapeutics  
- Internationally known for studies in NAFLD and NASH with over 200 peer reviewed publications in these fields. Focus is on drug development.  
- Serves as the Medical Director for Pinnacle Clinical Research and the president of Summit Clinical Research.

Hilde H. Steineger  
COO  
- > 25 years experience in life science in the interception of research/ business development /finance  
- Ph.D. in Medical Biochemistry (hepatic gene regulation of normal and structurally engineered fatty acids), M.Sc. in Molecular Biology  
- Nycomed Pharma, Nordea Securities, Neomed Management, Pronova BioPharma/BASF  
- Extensive board member experience for e.g in Strongbridge Biopharma plc, Nordic Nanovector AS, Algeta ASA  
- Inventor on multiple Icosabutate and SEFA patents

David Fraser  
CSO  
- Lead investigator in 4 public-sector clinical studies, 8 years Exploratory R&D at Pronova Biopharma  
- Ph.D. in Immunology/ Endocrinology, M.Sc. in Human Nutrition & Metabolism (hypotriglyceridemic effects of omega-3)  
- Nycomed Pharma, Sanofi-Aventis, Pronova BioPharma/BASF  
- Last position: Group Lead Exploratory Biology  
- Inventor on multiple Icosabutate and SEFA patents

Mats Blom  
CFO  
- 15 years experience from CFO roles in public and non-public specialty pharma/biotech companies  
- MBA from IESE, Barcelona  
- Zealond Pharma, Swedish Orphan, Anuto, Active Biotech, EY  
- As CFO responsible for two European and one US IPO plus several financing rounds, royalty transactions and license deals  
- Non-executive board member in Hansa Biopharma AB, Auris Medical Holding Ltd and Rare Thyroid Therapeutics AB

Stephen Rossi  
CDO  
- > 25 years experience in clinical development and medical affairs with a focus on metabolic, cholestatic and viral liver diseases  
- PharmD from University of California at San Francisco; Fellowship in Transplant Immunopharmacology  
- Roche Pharmaceuticals, Roche Molecular, Gilead Sciences, NGM Biopharmaceuticals, CymaBay Therapeutics, Pliant Therapeutics  
- Program lead for multiple novel clinical candidates in NASH and cholestatic liver disease
... supported by a world-renowned Scientific Advisory Board

Dr Detlef Schuppan
Advisor
- International expert in the fields of fibrosis and coeliac research.
- Director of the Institute of Translational Immunology and a professor of internal medicine, gastroenterology, and hepatology at the Medical Center of the Johannes Gutenberg University of Mainz in Germany
- From 2004 to 2010, he was lecturer, professor, and senior physician for gastroenterology and hepatology at the Beth Israel Deaconess Medical Center at Harvard Medical School.
- Professor of medicine and senior visiting scientist at Harvard Medical School.

Dr John Kastelein
Advisor
- Prof. of Medicine at the Department of Vascular Medicine at the Academic Medical Center, Amsterdam
- Published over 850 research papers in peer reviewed journals, including Nature Genetics, Lancet, New England Journal of Medicine, JAMA and Circulation and has a Hirsch index of 102 in March 2017.
- Co-founder of UniQure and Xenon Pharma
- Principal investigator and advisor for a wide range of dyslipidemia clinical trials

Dr Scott Friedman
Advisor
- Prof. of Medicine, liver diseases and professor in pharmacological sciences
- Dean for Therapeutic Discovery and Chief of the Division of Liver Diseases, at the Icahn School of Medicine at Mount Sinai.
- Pioneering research into the underlying causes of scarring, or fibrosis associated with chronic liver disease
- First scientist to isolate the hepatic stellate cell, which is responsible for hepatic fibrosis, scarring process that can lead to cirrhosis of the liver
- Authored over 300 scientific articles; and he has served as mentor to over 50 students, physicians, and postdoctoral trainees

Dr Stephen Harrison
Advisor
- Medical degree from the University of Mississippi School of Medicine
- Board certified in both Internal Medicine and Gastroenterology
- Visiting Professor of Hepatology at the Radcliffe Department of Medicine, University of Oxford
- Past Associate Editor for Hepatology and for Alimentary Pharmacology and Therapeutics
- Internationally known for studies in NAFLD and NASH with over 200 peer reviewed publications in these fields. Focus is on drug development.
- Serves as the Medical Director for Pinnacle Clinical Research and the president of Summit Clinical Research.

Dr Arun Sanyal
Advisor
- Prof. of Medicine, Physiology and Molecular Pathology at Virginia Commonwealth University School of Medicine in Richmond, Virginia
- > 25 years of experience as a hepatologist
- Has served as the secretary and president of the AASLD
- Founding member of Hepatology board of the American Board of Internal Medicine
- Member of the council of the NIH
- Considered as one the greatest NASH specialists in the diagnosis and treatment of NASH
- Published over 300 papers in leading medical journals and periodicals throughout his career

Dr Sander van Deventer
Advisor
- Prof. Translational Gastroenterology at Leiden University Medical Center
- Authored over 400 peer reviewed publications
- Developed Remicade, first to infuse reconstituted HDL in humans
- Co-founder, former CEO and current CSO of UniQure
- Founding CEO and scientific advisory board member at Dezima Pharma
- Operating Partner at Forbion Capital Partners

Dr Stephen Harrison
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- Medical degree from the University of Mississippi School of Medicine
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Conclusions

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<thead>
<tr>
<th>SEFAs are novel structurally modified fatty acids with a unique therapeutic potential</th>
<th>NorthSea Therapeutics optimizes structural engineering of fatty acids to achieve therapeutic effects not possible with unmodified fatty acids. Developing a broad pipeline including three products in the clinical by Q4-2021</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead compound Icosabutate has a unique approach for targeting NASH. The phase 2b ICONA study is ongoing. Excellent interim readout data of the first 90 patients treated for 16 weeks</td>
<td>Icosabutate leverage an extensive pre-clinical, two phase 2 studies and a strong CMC package. The lead compound has excellent safety data from over 300 patients. Phase 2b ICONA interim results demonstrate potent anti-inflammatory, anti-fibrotic and hepatic antioxidant properties of Icosabutate in NASH patients in conjunction with an improvement in cardio-metabolic risk profile</td>
</tr>
<tr>
<td>The company completed a $80 mln Series C round in December 2021 to develop an attractive early and late-stage clinical pipeline</td>
<td>In addition to the lead program in NASH two additional clinical programs are initiated: SEFA-1024 will be developed for dyslipidemia and entered phase 1 in H2-2020, SEFA-6179 will be developed for IFALD and entered phase 1 in Q4-2021.</td>
</tr>
<tr>
<td>The experienced team and world-renowned SAB are a solid basis to execute the plans</td>
<td>The organization is further developed with a clinical team in the US and will further developed in key areas as BD and regulatory and is supported by the key experts in the different clinical areas.</td>
</tr>
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