The HDL Company
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An experienced management

Jean-Louis DASSEUX, PhD, MBA

Founder and CEO
- More than 25 years of experience in the pharmaceutical industry (Pfizer, Esperion Therapeutics, Fournier Laboratories)
- A leading world expert in lipid metabolism, atherosclerosis and cardiovascular diseases
- Inventor of more than 60 patent families relating to HDL and the treatment of cardiovascular diseases

Cyrille TUPIN, CPA

CFO
- Audit Director at Sygnatures, the largest private auditing and consulting company in Toulouse, France
- More than 7 years at PWC working on high-profile business transactions
Value proposition: why invest in CERENIS?

CER-001: major potential in the treatment of patients post-ACS

1. A therapy targeting the 2/3 of patients who are poorly served with available medical treatments
2. Advanced and promising clinical developments currently in Phase II (CARAT)
3. Compelling to big pharma (e.g., OMTHERA $443 m; Esperion $1.3 bn; KOS $3.7 bn)¹
4. A manufacturing process validated on an industrial level with proven clinical safety and tolerability

In the short term: CER-001, a drug for treating orphan diseases

1. A potential of value creation in the short term, currently in Phase III (TANGO)
2. A major unmet medical need
3. Application for marketing approval before 2018

CER-209: major potential in the treatment of patients with atherosclerosis and NAFLD/NASH

1. A significant unmet medical need
2. CER-209, a highly specific P2Y13 receptor agonist promoting lipid elimination

A WELL-CAPITALIZED (€33 MILLION), LISTED COMPANY WITH SUBSTANTIAL POTENTIAL IN HDL THERAPY

¹ Press releases,
OMTHERA: http://www.astrazeneca.com/Newsroom/Press-releases/Article/20130528-omthera
Esperion: http://www.bloomberg.com/apps/news?pid=newsarchive&sid=apU2qYxCmKO4&refer=us
HDL therapy: one of the most promising treatments for removing cholesterol

Fundamental role of HDL in removing cholesterol

- At each LDL level, it is the HDL level that determines the cardiovascular risk
- An HDL therapy that increases the number of HDL particles is one of the best approaches for treating atherosclerosis
- No HDL medical treatment that can treat or eliminate atherosclerosis is yet available

A major epidemiological study on HDL

![Graph showing incidence of cardiovascular events (per 1,000) over 10 years vs. HDL level (mg/dl)]

CERENIS IS THE COMPANY THAT OFFERS ONE OF THE MOST COMPREHENSIVE INNOVATIVE HDL SOLUTIONS FOR TREATING ATHEROSCLEROSIS

1. PROCAM: 7,152 men aged 35 to 65, 406 coronary events over 10 years
Cardiovascular disease: Still an unmet medical need after more than 30 years of existing therapy

Leading cause of death in the world

- 1 out of 3 deaths worldwide (source: WHO)
- The disease category with the greatest health expenditure:
  - $107 bn in the United States, in 2010
  - $110 bn in Europe, in 2009

A primary cause: atherosclerosis

- Atherosclerosis: accumulation of cholesterol plaque in the arteries

Only 1/3 of cardiovascular patents receive benefit from the best current treatments

ONLY ONE REAL SOLUTION: ELIMINATE CHOLESTEROL PLAQUE WITH CERENIS
### 10 years of R&D to achieve one of the world’s most advanced HDL solutions

**Product** | **Indications** | **Preclinical** | **Clinical** |
---|---|---|---|
CER-001 (HDL mimetic) | Post-Acute Coronary Syndrome (ACS) | POC² Preclinical development | Phase I 1st Phase II 2nd Phase II |
| | HDL genetic defects (FPHA¹) | Preclinical development |  |

CER-209 (P2Y13 receptor agonist) | NAFLD/NASH/Atherosclerosis | POC² Preclinical development | Phase I Phase II Phase III |

**Financing to date**

<table>
<thead>
<tr>
<th>Year</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>2005</td>
<td>€25 m</td>
</tr>
<tr>
<td>2006</td>
<td>€42 m</td>
</tr>
<tr>
<td>2010</td>
<td>€50 m</td>
</tr>
<tr>
<td>2015</td>
<td>€53.4 m</td>
</tr>
</tbody>
</table>

**Investors**

Cerenis Therapeutics

**3 TARGETED INDICATIONS:** ACS, FPHA AND NAFLD/NASH/ATHEROSCLEROSIS

1. Familial Primary Hypoalphalipoproteinemia
2. Proof of Concept
**LDL Approach: reduces bad cholesterol**

No direct action on atherosclerotic plaque

**AVAILABLE DRUGS:**
- **Statins:** inhibit cholesterol synthesis
- **Resins and Inhibitors:** limit intestinal absorption of cholesterol
- **Fibrates:** reduce the level of triglycerides containing LDL cholesterol

Indirect long-term effect with no direct action on plaque:
only 1/3 of patients get benefit

**HDL Approach: reduces plaque**

Reduces atherosclerotic plaque

**NO DRUGS YET AVAILABLE:**
- **CER-001:** Cerenis HDL mimetic candidate that reduces atherosclerotic plaque

**Rapid direct effect:**
reduction in atherosclerotic plaque

**LDL Drugs have a limited efficacy on plaque reduction**
Tremendous market opportunity for HDL-focused therapies

Cardiovascular disease

2 main indications

Acute Coronary Syndrome
2.8 million patients (US + EU)

- 1/3 of patients receive benefit
- 2/3 of patients do not receive benefit

- Stent
- LDL therapies

HDL genetic defect (FPHA)
100,000 – 150,000 patients (US + EU)

- No existing HDL treatment

No existing HDL treatment

NO HDL DRUG IS CURRENTLY AVAILABLE FOR ALMOST 3 MILLION PATIENTS
12% of patients relapse during the 12 months following an ACS, 2/3 of them during the first 2 months

19-26% of patients over age 45 die during the 12 months that follow a cardiovascular event

ACS hospitalization costs: $20,000 - $60,000 per patient per event

HDL THERAPY IS THE ONLY SOLUTION ADDRESSING THE CRITICAL 2-MONTH POST-ACS PERIOD

2. PLATO clinical study, AstraZeneca
3. Source: AHS
Phase I showed:

- Mobilization of HDL cholesterol
  - Increase in HDL cholesterol: +700% for 45 mg/kg dose
  - Mobilization observed beginning with the 2 mg/kg dose
  - No patient safety issues

Concentration of HDL cholesterol following the infusion of CER-001

A PROVEN SAFETY PROFILE AT ALL DOSES
Compelling clinical results: proof of efficacy in Phase II

CHI SQUARE: Phase II post-ACS study

Effects on plaque

- Significant regression in the volume of atherosclerosis plaque, substantially better than existing treatments
- Rapid action in just 2 months vs. at least 2 years for other treatments

Change in the percentage atherosclerosis volume (PAV)

- **CER-001**
- **ASTEROID**
- **REVERSAL**
- **ACTIVATE**
- **CAMELOT**
- **A-Plus**
- **LDL (mg/dl)**

Change in PAV (%)

- Regression
- Progression

An independent analysis (SAHMRI) showed:

- A significant reduction in atherosclerotic plaque compared with the placebo

**CER-001 IS THE MOST EFFICIENT OF ALL TREATMENTS**

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3 Nissen S and al. JAMA 2006;295 (13):1556-1565
5 Nissen S and al. JAMA 2004; 291:1071–1080
Conclusions of CHI-SQUARE, the 1st Phase II study:

- Cholesterol mobilization by CER-001 at every dose level
- Demonstrated patient safety profile
- Primary endpoint (reduction in plaque at 12 mg/kg dose vs. placebo) not achieved
- Reduction in the total volume of atherosclerosis vs. baseline was statistically significant at 3 mg/kg

An independent analysis (SAHMRI) confirmed the optimal dose\(^2\):

Change in the percentage atherosclerosis volume (PAV)

*Patients with PAV ≥30 at baseline*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Placebo (n=69)</th>
<th>3 mg/kg (n=58)</th>
<th>6 mg/kg (n=78)</th>
<th>12 mg/kg (n=66)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAV</td>
<td>-0.259</td>
<td>-0.963</td>
<td>-0.619</td>
<td>+0.177</td>
</tr>
<tr>
<td>P value</td>
<td>0.038 (^1)</td>
<td>0.287</td>
<td>0.587</td>
<td></td>
</tr>
</tbody>
</table>

- Too high a concentration of HDL induces a down-regulation of ABCA1 transporter, which is necessary for cholesterol efflux. The 12 mg/kg dose caused such a down-regulation whereas 3 mg/kg did not resulting in the highest efficacy
- The optimal dose enabling a maximization of the plaque regression vs. placebo: 3 mg/kg
- Next study: number of infusions

THE OPTIMAL DOSE HAS BEEN IDENTIFIED
THE OPTIMAL NUMBER OF INFUSIONS STILL NEEDS TO BE DETERMINED

1. Statistically significant result
2. Greater regression of coronary atherosclerosis with the pre-beta high-density lipoprotein mimetic CER-001 in patients with more extensive plaque burden

American Heart Association sessions 2015, S. Nicholls et al.
Validation of the optimal dose

The publication highlights CER-001 preclinical positive results:

- CER-001 mimics native HDL
- Ability of CER-001 to inhibit the formation of atherosclerotic plaque with better efficacy at lower doses

Dose-response mechanism follows a U-shaped curve

- At high dose see strong down-regulation of the ABCA1 transporter, the cellular gatekeeper for eliminating excess tissue cholesterol
- Confirmation of the optimal 3mg/kg dose of the Phase II CARAT clinical trial in the post-ACS indication

Decrease percentage of an atherosclerotic plaque within carotids

CONFIRMATION OF OPTIMAL DESIGN FOR CARAT AND TANGO STUDIES

1 PLOS ONE: Tardy et al. September 3, 2015, DOI: 10.1371/journal.pone.0137584
CER-001 targets plaque: LOCATION clinical study

The LOCATION study provides the first evidence of

- CER-001’s ability to:
  - Penetrate atherosclerotic plaques
  - Preferentially target atherosclerotic plaques

- CER-001’s capacity to increase cholesterol efflux

Increased cholesterol efflux capacity is a predictive marker of a reduction in cardiovascular-related morbidity and mortality:

Association between cardiovascular risk and cholesterol efflux capacity*


* Source: 17th SYMPOSIUM INTERNATIONAL DE L’ATHEROSCLEROSE (IAS), 23 au 26 mai 2015 à Amsterdam, Erik Stroes et al., Academic Medical Center of Amsterdam, The Netherlands
Dr. Stephen Nicholls

I’m particularly enthusiastic about collaborating with Cerenis Therapeutics for the future Phase II CARAT clinical study of CER-001. On the basis of our convincing analyses of the Phase II CHI-SQUARE study highlighting the efficacy of the optimal 3mg/kg dose, I’m highly confident regarding the potential success of this important clinical step to establish CER-001 as the market benchmark in HDL mimetic.”
The CARAT study should show:

- A significant reduction in the percentage atherosclerosis volume vs. placebo
- The superior efficacy of an increase in the number of doses
- Enrollment completed in August 2016

**Study led by the South Australian Health and Medical Research Institute Limited (SAHMRI)**

- 292 subjects, randomized
- Treatment period: 9 weeks
- Observation period: 2 weeks
- 10 infusions of CER-001
- Placebo: n=146
- 3mg/kg: n=146
- IVUS visit: Follow-up

**IDENTIFICATION OF THE OPTIMAL TREATMENT AND ENROLLMENT OF PATIENTS WITH SUBSTANTIAL ATHEROSCLEROSIS PLAQUE**
The key advantage of HDL therapy for FPHA

FPHA: a rare syndrome of severe HDL deficiency

- Caused by mutations in the genes responsible for HDL synthesis/maturation
- Characterized by accelerated atherosclerosis

CER-001 treatment

- CERENIS’ solution restores the blood’s ability to mobilize cholesterol into HDL to facilitate its elimination
- Two Orphan Drug designations obtained
  - HDL deficiency (no apoA-I synthesis)
  - Tangier disease (absence of ABCA1)

Mobilization of HDL cholesterol in the blood

CERENIS: A THERAPEUTIC SOLUTION TO MEET THE UNMET FPHA MEDICAL NEED
Confirmed universal proof of concept amongst those deficient in HDL

The Phase II SAMBA study:

- Evaluated the efficacy of CER-001
- 7 FPHA patients in an open-label, single-arm, active-treatment study for a one-month treatment of 9 doses
- Assessed the reconstitution of the endogenous reverse lipid transport pathway
- Showed reduction of the vascular wall thickness
  - Behaves like a natural HDL
  - Eliminates cholesterol
  - Reduces plaque

**Efficacy on carotid atherosclerosis**

<table>
<thead>
<tr>
<th>8mg/kg</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ 20%</td>
<td></td>
</tr>
</tbody>
</table>

**Average thickness of the carotid wall (%)**

- 1 Month
- 6 Months

IN THE BODY, CER-001 RESULTED IN A STATISTICALLY SIGNIFICANT REDUCTION OF PLAQUE IN THE CAROTID ARTERY VESSEL WALL
The TANGO study should show:

- A reduction in coronary plaque in the carotid and aorta
- Enrollment began in December 2015
# A safe and industrializable natural mimetic solution

<table>
<thead>
<tr>
<th></th>
<th>CERENIS</th>
<th>The Medicines Company</th>
<th>CSL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Product specificity</strong></td>
<td>Only mimetic with the biological properties of natural HDL</td>
<td>Mutant protein produced in an <em>E. coli</em> bacteria</td>
<td>Protein extracted from plasma</td>
</tr>
<tr>
<td><strong>Composition of the nanoparticle</strong></td>
<td>Natural HDL mimetic</td>
<td>Mutant form</td>
<td>Multiple forms of A-I apolipoprotein</td>
</tr>
<tr>
<td><strong>Purity</strong></td>
<td>✓ ✓ ✓</td>
<td>✓</td>
<td>✕</td>
</tr>
<tr>
<td><strong>Mobilization of cholesterol / Efficacy</strong></td>
<td>✓ ✓ ✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Side effects/Toxicity</strong></td>
<td>✓ ✓ ✓</td>
<td>✕</td>
<td>✕</td>
</tr>
<tr>
<td><strong>Intellectual property</strong></td>
<td>✓ ✓ ✓</td>
<td>✕</td>
<td>✕</td>
</tr>
<tr>
<td><strong>Composition</strong></td>
<td>✓ ✓ ✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Manufacturing process</strong></td>
<td>✓ ✓ ✓</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

**Competitive advantage of CER-001**

- Homogenous particle population
- Lower required dosage
- No identified toxicity
- Protection of the active principle blocking any reproduction of the nanoparticle
- Only charged-complex natural HDL mimetic
- Only 3 purification steps
CER-209, a potential breakthrough treatment for atherosclerosis and nonalcoholic steatohepatitis (NASH)

HDL therapy enables to address atherosclerosis and NAFLD/NASH

- Atherosclerosis is frequently observed in patients with NASH, thus presenting high cardiovascular risk, in addition to steatohepatitis and liver inflammation
- Current treatments based on lipid-lowering drugs attempt to reduce LDL cholesterol but they often increase liver enzymes, thereby limiting the benefits for treating NASH patients
- Other treatments currently under development for NASH, such as targeting the nuclear receptor PPAR as well as FXR agents, may face problems associated with their multiple effects

CER-209 increases HDL elimination by the liver...

- A new mechanism of action that involves the last steps of the RLT pathway
- Agonist activity of CER-209 on the liver P2Y13 receptors facilitates elimination of mature HDL particles loaded with lipids such as cholesterol, through better HDL liver recognition and increased bile secretion

...by stimulating the activity of HDL receptors

CER-209, THE UNIQUE FIRST-IN-CLASS THERAPEUTIC SOLUTION TO ADDRESS BOTH NASH AND ATHEROSCLEROSIS
Experimental positive results for CER-209 presented at the 25\textsuperscript{th} Conference of the APASL (\textit{ASIAN PACIFIC ASSOCIATION FOR THE STUDY OF THE LIVER})

CER-209, an agonist of the P2Y13, decreases both atherosclerosis and liver steatosis

**Plaque regression after treatment with CER-209**

Regression of liver steatosis after high-cholesterol diet and treatment with CER-209

*C P2Y13 receptor agonist CER-209 decreases both atherosclerosis and liver steatosis in vivo: Rudi Baron, Marine Goffinet, Nadia Boubekeur, Claudine Tardy, Guy Cholez, Daniela C. Oniciu, Narendra D. Lalwani, Jean-Louis H. Dasseux and Ronald Barbaras*

CER-209 HAS A STRONG POTENTIAL FOR THE TREATMENT OF NASH AND NAFLD

CLINICAL DEVELOPMENT STRATEGY SHOULD BE FINALIZED IN 2016
A well-protected platform and technology

- 9 patent families protecting the products, indications and manufacturing / diagnostic methods

<table>
<thead>
<tr>
<th>PRODUCT</th>
<th>INDICATION</th>
<th>MANUFACTURING/DIAGNOSTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family 1: Formulation of CER-001 and its use</td>
<td>Family 4: Treatment of dyslipidemias</td>
<td>Family 2: Manufacturing methods for reconstituted HDL particles and highly-homogenous resulting populations of HDL particles</td>
</tr>
<tr>
<td>Family 6: HDL mimetic peptide including CER-522</td>
<td></td>
<td>Family 3: Companion diagnostics and dosage of CER-001</td>
</tr>
<tr>
<td>Family 7: P2Y13 receptor agonists (CER-209)</td>
<td></td>
<td>Family 5: Synthetic sphingomyelin synthesis / production methods</td>
</tr>
<tr>
<td>Family 8: PPAR agonists (CER-002)</td>
<td></td>
<td>Family 9: Carrier particles for administering drugs</td>
</tr>
</tbody>
</table>

NO COMPETITOR CAN REPRODUCE THE CHARGED NANOPARTICLE, EVEN PARTIALLY
### Products due to enter a new development phase

<table>
<thead>
<tr>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CER-001</strong></td>
<td></td>
<td>Post-ACS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recombinant HDL</td>
<td>FPXA: Orphan disease ApoA-I and ABCA1 deficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>CER-209</strong></td>
<td>Stimulation of HDL receptors</td>
<td>Dyslipidemia with low HDL</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Non-alcoholic steatohepatitis (NASH/NAFLD/atherosclerosis)</td>
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</table>

### Products in the portfolio

<table>
<thead>
<tr>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CER-522</strong> (back-up)</td>
<td>Peptide HDL mimetic</td>
<td>Aortic valve stenosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Specific PPAR delta agonist</td>
<td>Dyslipidemia with low HDL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non-alcoholic steatohepatitis (NASH)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systemic lupus erythematosus (SLE)</td>
<td></td>
<td></td>
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</tbody>
</table>

### Future growth drivers

A structured and diversified portfolio
Solid near- and medium-term news flow

Published News Flow

- 1st patient enrolled, CARAT study
- LOCATION study
- Board appointments
- Poster at the IAS
- PLOS ONE Publication
- Two poster presentations on CER-209 at the APASL
- Last patient enrolled, CARAT
- Poster presentation on CER-001 safety at the ESC

- 1st patient enrolled, FPHA study
- FPHA development, TANGO
- Application filed for orphan diseases
- Last patient visit, FPHA study
- FPHA results
- FPHA EU market approval
- CARAT results
- Last IVUS visit CARAT
- Poster presentation on CER-001 safety at the ESC

Timeline to be defined

WEALTH CREATION PERSPECTIVE IN BOTH THE NEAR AND MEDIUM TERM
### Consolidated accounts (IFRS)

#### BALANCE SHEET

<table>
<thead>
<tr>
<th>ASSETS</th>
<th>31/12/2015</th>
<th>30/06/2016</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total non-current assets</td>
<td>446</td>
<td>340</td>
</tr>
<tr>
<td>Total current assets</td>
<td>45,661</td>
<td>37,152</td>
</tr>
<tr>
<td>Total assets</td>
<td>46,107</td>
<td>37,492</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LIABILITIES</th>
<th>31/12/2015</th>
<th>30/06/2016</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total shareholders’ equity</td>
<td>33,198</td>
<td>22,359</td>
</tr>
<tr>
<td>Total non-current liabilities</td>
<td>7,120</td>
<td>7,082</td>
</tr>
<tr>
<td>Total current liabilities</td>
<td>5,790</td>
<td>8,051</td>
</tr>
<tr>
<td>Total liabilities</td>
<td>46,107</td>
<td>37,492</td>
</tr>
</tbody>
</table>

#### INCOME STATEMENT

<table>
<thead>
<tr>
<th>Operational income</th>
<th>31/06/2015</th>
<th>30/06/2016</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marketing and Admin. costs</td>
<td>(1,064)</td>
<td>(3,828)</td>
</tr>
<tr>
<td>R&amp;D costs</td>
<td>(5,239)</td>
<td>(10,213)</td>
</tr>
<tr>
<td>Operating profit / loss</td>
<td>(6,303)</td>
<td>(14,041)</td>
</tr>
<tr>
<td>Financial profit / loss</td>
<td>(760)</td>
<td>(626)</td>
</tr>
<tr>
<td>Net profit / loss</td>
<td>(7,062)</td>
<td>(14,662)</td>
</tr>
</tbody>
</table>

- Gross cash position of:
  - €7.8 m on December 31, 2014
  - €43.0 m on December 31, 2015
  - €37.2 m on June 30, 2016

- Of which €6.6 m is linked to Bpifrance (OSEO) advance payment

- Of which €6.8 m is trade payables

- Enrollment of clinical studies: CARAT, TANGO and LOCATION

- Affected by non-cash elements:
  - IFRS treatment of the BPI repayable advances

* Unaudited
<table>
<thead>
<tr>
<th>Cash Flow Table</th>
<th>30/06/2015</th>
<th>30/06/2016</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>€ thousands</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cash flow from operations</td>
<td>(6,079)</td>
<td>(11,018)</td>
</tr>
<tr>
<td>Cash flow from investments</td>
<td>(25)</td>
<td>(2)</td>
</tr>
<tr>
<td>Cash flow from financing</td>
<td>48,924</td>
<td>940</td>
</tr>
<tr>
<td><strong>Change in cash position</strong></td>
<td>42,820</td>
<td>10,079</td>
</tr>
<tr>
<td>Cash position at start of period</td>
<td>7,843</td>
<td>42,951</td>
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<tr>
<td>Currency effect</td>
<td>(3)</td>
<td>0</td>
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<tr>
<td><strong>Cash position at end of period</strong></td>
<td>50,660</td>
<td>32,872</td>
</tr>
</tbody>
</table>

*Unaudited

- March 2015 IPO
- Cash position as of June 30, 2016
Value proposition: why invest in CERENIS?

CER-001: major potential in the treatment of patients post-ACS

1. A therapy targeting the 2/3 of patients who are poorly served with available medical treatments
2. Advanced and promising clinical developments currently in Phase II (CARAT)
3. Compelling to big pharma (e.g., OMTHERA $443 m; Esperion $1.3 bn; KOS $3.7 bn)\(^1\)
4. A manufacturing process validated on an industrial level with proven clinical safety and tolerability

In the short term: CER-001, a drug for treating orphan diseases

1. A potential of value creation in the short term, currently in Phase III (TANGO)
2. A major unmet medical need
3. Application for marketing approval before 2018

CER-209: major potential in the treatment of patients with atherosclerosis and NAFLD/NASH

1. A significant unmet medical need
2. CER-209, a highly specific P2Y13 receptor agonist promoting lipid elimination

A WELL-CAPITALIZED (€33 MILLION), LISTED COMPANY WITH SUBSTANTIAL POTENTIAL IN HDL THERAPY

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