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MADX: ORY A publicly traded company in the Madrid Stock Exchange

A clinical stage biopharmaceutical company developing innovative therapies in oncology and neurodegeneration leading the field of Epigenetics

A competitive EPIGENETIC Platform with a first program that validates scientifically and clinically the platform

- Two therapeutic programs in clinical development with multiple indication opportunities
- Additional assets in preclinical development to be progressed quickly

Signed global strategic partnership with ROCHE valued at 500M USD

Strong IP portfolio with technology developed in-house

Raised €27m in the last 12 months. Cash runway till 1H2018
Epigenetics – the study of heritable changes in genome function that occur without a change in DNA sequence

These changes mainly occur due to variations in the structure of chromatin that silence or activate whole regions of the chromosome and all the genes that reside in this region

These variations are caused by post-translational modifications on histones, the proteins that serve as scaffold for the DNA to conform the chromatin

Lysine methylation and demethylation is one of the key epigenetic modifications of the Histone tails
LSD1 is an enzyme that demethylates histones: specifically mono and dimethylated H3K4 and H3K9. LSD1 belongs to the family of FAD-dependent amine oxidases, which include known CNS drug targets, such as MAO-A and MAO-B. The general MAO inhibitor tranylcypromine is a chemical starting point to design LSD1 inhibitors.
<table>
<thead>
<tr>
<th>INDICATION</th>
<th>TARGET</th>
<th>MOLECULE</th>
<th>DISCOVERY</th>
<th>H2L</th>
<th>LEAD OPTIMIZATION</th>
<th>PRECLINICAL</th>
<th>PHASE I-IIA</th>
<th>PHASE IIIB</th>
<th>PHASE III</th>
<th>PARTNER</th>
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<tr>
<td>CANCER</td>
<td>LSD1</td>
<td>ORY-1001</td>
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<td>LSD1-MAOB</td>
<td>ORY-2001</td>
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<td>LSD1</td>
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</tbody>
</table>
ORY-1001: ONCOLOGY PROGRAM

- LSD1 is a key effector of the differentiation block in MLL leukemia
- MLL Leukemic stem cells are addicted to LSD1 activity
- ORY-1001 a highly potent and selective LSD1 inhibitor with orphan drug status granted by the European Medicines Agency (EMA)
- Currently in Phase I/IIA
  - Completed Part 1 of the study (Phase I) in acute leukemia
  - Extension Arm (Phase II-A) ongoing
- Potential for additional indications in solid tumors
**Phase I Highlights: ORY-1001 Leukemia**

Licensed to ROCHE in 2014

**Trial Design**

- Refractory & Relapsed Acute Leukemia
- Multi-Center (5)
- Multiple Ascending Dose (8 Cohorts)

**Primary Endpoint**

Evaluate Safety (hematological and non-hematological toxicities) and Tolerability

**Secondary Endpoints**

- Characterize PK
- Assess Responses (CR/Cri/PR), particularly for rMLL gene
- Evaluate surrogate PD markers for target engagement

**Preliminary Results**

- Excellent safety profile
- Demonstrated impact on pharmaceutical target
- PD clear readings several biomarkers
- Good PK
- Established maximum recommended dose

After the MRD, a 14 patients Expansion arm (Phase II-A), which included patients with target mutations (MLL and others), has been culminated to evaluate preliminary signs of efficacy

- ✔️ $23m received in 2014-15
- ✔️ +$500m in future contingent milestones
- ✔️ Tiered royalties up to double digit
- ✔️ Clinical development and all related investments beyond the ongoing Phase I/IIA trial are the responsibility of ROCHE

**Expected Report**

Preliminary Data in ASH 2016

$23m received in 2014-15

+ $500m in future contingent milestones

Tiered royalties up to double digit

Clinical development and all related investments beyond the ongoing Phase I/IIA trial are the responsibility of ROCHE
A number of scientific reports point out the potential of LSD1 inhibition as a target in a number of solid tumors.

Non oncological diseases as SCD and others may also be a CDP option.

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**Acute Myeloid Leukemia**

- 12% of all Blood Cancers
- 18,860 new cases in US in 2014

**Global Mk Potential of $932 million in 2024,**

CAGR of 10.5%

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**Small Cell Lung Cancer**

- 15% of all Lung Cancers
- 32,420 new cases in US in 2014

**Global Mk Potential of $684 million in 2017**

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**Sickle Cell Disease**

- SCD Epidemiology
- US/EU Prevalence ~150K

**US Mk Potential of $200 million in 2017,**

(Market to grow at 17% CGAR till 2019)

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**NOTE:** ROCHE is the sole responsible for the further Clinic Development Plan for ORY-1001. The indications and markets mentioned above are only presented on its likelihood based on the development of competitors or published scientific reports.

1. ACS, Cancer Facts & Figures 2014
2. [www.hematology.org](http://www.hematology.org)
3. [www.lungcancer.org](http://www.lungcancer.org)
4. Global Data 2015
5. Decision Resources 2015
Identical twins (monozygotic)
Same DNA with GBA risk mutation
Disconcordant for symptoms of Parkinson's
Up to 20 years difference in onset
Patient derived iPSCs: difference in MAO-B levels

Luca Lovrečić, et al., 2013 The Role of Epigenetics in Neurodegenerative Diseases
LSD1 in the CNS.
ORY-2001 inhibits both LSD1 and MAO-B by irreversible binding to the FAD cofactor

- LSD1 is a key component of different transcriptional complexes interacting with different transcription factors and very often with HDAC1 and HDAC2.
- In the brain, one of these TFs is REST. The LSD1-REST-CoREST-HDAC1/2 repressor complex is involved mainly in controlling developmental programs and modulating neuronal morphology in the CNS. Different to what happens in HDACs, it has been proven that it is possible to develop extremely selective LSD1 inhibitors with excellent pharmacological properties.
- LSD1 is known to be an important regulator in the maintenance of pluripotency and in specification of neuronal commitment of pluripotent or multipotent cells.
- In C. elegans, Drosophila, and mammalian cells, LSD1 suppression has been reported to significantly enhance the removal of misfolded proteins with a critical role on neurodegeneration like SOD1, TDP-43, FUS, and polyglutamine-containing proteins, indicating a general improvement in protein quality control.
Pharmacological Properties

- A selective dual LSD1-MAO-B inhibitor
- Optimal ADMET and PK profiles
- Crosses efficiently the BBB
- Once daily oral bioavailable
- Good pharmaceutical properties
- Selectivity against MAO-A demonstrated in-vitro and in-vivo
- High therapeutic window in animals: a safe drug for chronic settings
- Target engagement demonstrated in vivo

Exclusively owned by Oryzon

Preclinical Proof of Concept

Achieved in different animal models of:

- Alzheimer’s Disease
- Huntington’s Disease
- Multiple Sclerosis

Other additional indications being explored preclinically

Clinical development → In Phase I:

LPO expected in Dec2016

- Alzheimer’s Disease is lead indication
- Potential for additional indications: MS, HD and others

Biomarkers identified
The senescence accelerated mouse (SAMP8) as a model for oxidative stress and Alzheimer’s disease

John E. Morley, Harvey James Armbrecht, Susan A. Farr, Vijaya B. Kumar

The senescence accelerated mouse (SAMP8) is a spontaneous animal model of oxidative stress and amyloid deposition. It develops early memory and motor deficits and these symptoms are accompanied by the deposition of amyloid-β protein in the brain. SAMP8 mice show increased oxidative stress in the brain and in the liver, and this is associated with decreased antioxidant capacity. These results indicate that the SAMP8 mouse model may be a useful tool for studying the pathogenesis of Alzheimer’s disease.

Harboring the behavioral and histopathological signatures of Alzheimer’s disease (AD), senescent SAMP8 mice are currently considered a robust model for studying AD. However, the underlying mechanisms, triggered pathways and genes in SAMP8 mice linked to AD remain unclear. In this study, we provide a biological interpretation of the molecular underpinnings of SAMP8 mice. Our results were derived from differentially expressed genes in the hippocampus and cerebral cortex of SAMP8 mice compared to age-matched SAMRI mice at 2, 6, and 12 months of age using cDNA microarray analysis. On the basis of PPI, MetaCore and co-expression network, we constructed a distinct genetic sub-network in the brains of SAMP8 mice. Next, we verified that the regulation of synaptic transmission and apoptosis were disrupted in the brains of SAMP8 mice. We found abnormal gene expression of RAFl, MAPF, PEG32, CDKN2A, CAMK2A, TRIB2, ACEI, ADRAPI, MCM17 and TUB1, which may have induced the dysfunction of biological processes in the brains of SAMP8 mice. Specifically, we found microRNA, including miR-20a, miR-12, miR-34a, miR-165, miR-19a, miR-22, miR-26a, miR-101, miR-10b, and miR-125b, that might regulate the expression of genes in the sub-network. Taken together, these results provide new insights into the biological and genetic mechanisms of SAMP8 mice and add important new dimensions to our understanding of the neuro-pathogenesis in SAMP8 mice from a systems perspective.

Keywords: Alzheimer’s disease, senescent accelerated mouse prone 8, molecular network, hippocampus, cerebral cortex, differentially expressed genes, synaptic transmission, apoptosis

Table 1
Comparison of Alzheimer’s disease, SAMP8 mouse and transgenic models.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Alzheimer’s disease</th>
<th>SAMP8</th>
<th>Transgenic models</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overproduction of amyloid-β</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Amyloid plaques</td>
<td>Yes, Late*</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Phosphorylated tau</td>
<td>Increased</td>
<td>Increased</td>
<td>In some models</td>
</tr>
<tr>
<td>Cerebral amyloid angiopathy</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Neuron loss</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Synaptic dysfunction</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Dendritic spine loss</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Glossalt</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Cholinergic deficit</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Learning and memory impaired</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Circadian rhythm disturbances</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Oxidative damage</td>
<td>Yes</td>
<td>Yes</td>
<td>4 months</td>
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<tr>
<td></td>
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<td></td>
<td>8 months</td>
</tr>
</tbody>
</table>

* = uncertain.

Occur at 16 to 18 months.
PoC studies in SAMP8 mice

- The SAMP8 mouse is an excellent model to examine the pathophysiology of early defects seen in Alzheimer’s disease. They develop accelerated aging and senescence and show deficits in learning and memory as well as other similarities to pathology of AD.
- ORY-2001 cognitive effect tested by NORT in five different studies.
- Study 1 (below) After 2 and 4 month of oral treatment, ORY-2001 provides a robust protective effect in the medium and long-term memory of female mice, compared to age-matched SAMP8 mice.
- We lowered dose in males (Study #2).

![Graph showing discrimination index (DI) for SAMR1, Veh, 0.96mpk, and 3.20mpk in SAMP8 mice.](image)

**2 months treatment**
- Tested 2 h after training.

**4 months treatment**
- Tested 2 h after training.

<table>
<thead>
<tr>
<th>Group</th>
<th>SAMR1</th>
<th>Veh</th>
<th>0.96mpk</th>
<th>3.20mpk</th>
</tr>
</thead>
<tbody>
<tr>
<td>DI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>14/15</td>
<td>3/16</td>
<td>16/16</td>
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</tr>
<tr>
<td></td>
<td>5/5</td>
<td>0/5</td>
<td>5/5</td>
<td>4/5</td>
</tr>
</tbody>
</table>
PoC studies in SAMP8 mice

ORY-2001 provides a dose dependent protective effect in the medium-term memory of female mice, compared to age-matched SAMP8 mice.
PoC studies in SAMP8 mice

Dissecting the LSD1 and MAOB components

- MAOB inhibition alone shows a trend on cognitive improvement on the SAMP8 animals but it is not significant
  - $p=0.12$ at 2h
  - $p=0.22$ at 24h

- LSD1 inhibition is therefore crucial to obtain the recovery on cognitive improvement on the SAMP8 animals
PoC studies in SAMP8 mice

Dissecting the LSD1 and MAOB components

- ORY-2001 provides a robust protective effect in the medium and long-term memory of mice, compared to age-matched SAMP8 mice.
- LSD1 inhibition alone is also able to produce an effect but less pronounced.
- Protection is driven by the LSD1 inhibition and not by MAO-B, but the combination with MAO-B inhibition (i.e. a dual compound, ORY-2001) enhances the effect.

**Females**

<table>
<thead>
<tr>
<th></th>
<th>LSD1+MAOB</th>
<th>LSD1 alone</th>
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</thead>
<tbody>
<tr>
<td>SAMR1</td>
<td>Veh</td>
<td>0.96 mpk</td>
</tr>
<tr>
<td>ORY 2001</td>
<td>0.32 mpk</td>
<td>0.96 mpk</td>
</tr>
<tr>
<td>ORY L51</td>
<td>0.1 mpk</td>
<td>0.3 mpk</td>
</tr>
</tbody>
</table>

**SAMP8 female animals (n=8 per group)**
ORY-2001: A possible disease modifier drug

Meta-analysis of cognitive deficit of untreated SAMP-8 mice (historical data)

ORY-2001 restores the discrimination index in SAMP-8 mice
PoC studies in SAMP8 mice - **BIOMARKERS**

We have identified different Hippocampal **biomarkers** upon ORY-2001 treatment:

<50 genes up or down-regulated by > 2-fold female SAMP-8 vs SAMR1 (see also Carter *et al.*)

Chr 4 cluster including *Ccl19* and *Ccl27* is amplified and over-expressed SAMP-8 vs SAMR1 mice

Inflammation genes upregulated in SAMP-8 vs SAMR1 mice

**✓** ORY-2001 potently down-regulated the expression of a subset of genes related to immune reaction and inflammation, including *S100A9* and T-cell receptor β chains in SAMP-8 mice

**✓** ORY-2001 up-regulated genes associated with improved cognitive function, neuroplasticity and memory
**ORY-2001 - PROOF OF CONCEPT IN SAMP8 MICE**

**BIOMARKERS**: We have identified different biomarkers upon ORY-2001 treatment:

- Down-regulation of the pro-inflammatory S100A9 protein by ORY-2001 is particularly interesting, since S100A9 is emerging as an important contributor to inflammation-related neurodegeneration.

- S100A9 was found to be increased in:
  - patients with AD
  - postoperative cognitive dysfunction (POCD)
  - and traumatic brain injury (TBI)

- Knockout or knockdown of S100A9 has been shown to be beneficial to memory in APP/PS1 and Tg2576 models of Alzheimer’s disease.

- S100A9 belongs to the family of calcium-binding S100 proteins.

- It is expressed in granulocytes and at early stages of monocyte differentiation.

- Complexes of S100A8 and S100A9 (S100A8/A9) are expressed and released at inflammatory sites.

- A correlation between serum levels of S100A8/A9 and disease activity has been observed in many inflammatory disorders.
ORY-2001 a possible approach to treat Multiple sclerosis

Experimental Autoimmune Encephalitis (EAE) mice model is a model in which S100A9 has been described to be upregulated

This model is considered a meaningful model for Multiple Sclerosis

To determine the efficacy of ORY-2001 following oral gavage administration for 2 consecutive weeks in mice.

Method:
Female C57BL/6 mice
G1: Vehicle Control
G2: ORY-2001 1.0 mg/Kg, p.o.
G3: ORY-2001 3.0 mg/Kg, p.o.

Parameter to assess:
- Body weight
- Clinical score
- Inflammatory response
- Autoimmune response

Clinical score:
- 0.0, no clinical signs
- 0.5, partial loss of tail tonicity
- 1.0, complete loss of tail tonicity
- 2.0, flaccid tail and abnormal gait
- 3.0, hind leg paralysis
- 4.0, hind leg paralysis with hind body paresis
- 5.0, hind and fore leg paralysis
- 6.0, death

C57BL/6 mice (Six-week old)

Onset of clinical signs

100 µg MOG35-55 sc. In CFA

Early onset reactive treatment

Days
Treatment with ORY-2001 during the effector phase of the disease greatly inhibited the development of EAE and reduced disease incidence and severity.

**ORY-2001 is protective in EAE model**

- All controls developed the disease (from day 14 post immunization), while many of ORY-2001 treated animals remained with no symptoms.

- As EAE model is considered a validated preclinical model of the chronic progressive form of multiple sclerosis, ORY-2001 emerges as a new candidate to treat this disorder.
Next steps of ORYZON in MS

ORY-2001 may be an effective therapeutic agent on Multiple Sclerosis

- The company is working actively on:
  - Dissecting the Molecular MoA
  - Defining the adequate dose scheme
  - Identifying additional biomarkers

- ORY-2001 will be PHASE II ready on 1H2017

- We have analyzed the data with different KOLs and we have got positive feedback regarding the significance of the data

- We have incorporated Dr. Xavier Montalban, a world known KOL in MS, on the Scientific Advisory Board of the company

- We are designing a PHASE-IB/IIA Clinical Study to eventually complement the CDP
ORY-2001 DEVELOPMENT TIMELINE

A Phase I study currently ongoing with 88 healthy volunteers, young and elderly.

Phase I, single center, double blind, parallel, ascending single and multiple dose trial.

---

**TITLE:** A Study to Assess the Safety, Tolerability and Pharmacokinetic of Single and Multiple Oral Doses of ORY-2001 in Healthy Male, Female Subjects and Elderly Population  
**STUDY CODE:** CL01-ORY-2001  
**EUDRACT NUMBER:** 2015-003721-33

---

*Phase I Clinical Trial* in young and elderly healthy volunteers
# ORY-2001 DEVELOPMENT TIMELINE

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<th>2016</th>
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<td>4Q2016</td>
<td>1H2017</td>
<td>2H2017</td>
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**CMC / 4W**  
**Reg Tox**  
**IB / IMPD**  
**CTA**  
**Phase I (SAD)**  
**Phase I (MAD)**  
**9M Reg Tox**  
**Additional Preclinical Work to Broaden CDP**  

**Phase II Dossier**  
**Phase II AD**  
**Phase IB- IIA MS**  
**Other Ind.: HD...**
ORY-2001 CLINICAL & MARKET POTENTIAL

ORY-2001 market capture opportunity above $3 billion.
Further development may include Neuro-inflammatory disorders

ALZHEIMER’S DISEASE
5.4 M people currently affected in US. By 2025 the number of patients will rise to 7.1 million in USA
8.7 million Europeans are also affected and in Asia another potential 10 to 12 million people are diagnosed or suspected to suffer AD.
Drug market projected to reach US $9.5 billion by 2017

HUNTINGTON’S DISEASE
Worldwide prevalence of HD is 5–10 cases per 100,000 persons. There are around 30,000 symptomatic Americans and more than 200,000 at-risk of inheriting the disease
Up to 71,000 patients in Europe.
Drug market projected to reach US$1.3 billion by 2020

MULTIPLE SCLEROSIS
The overall MS market in the U.S. and EU5 is very large at an estimated ~$17B. This is expected to grow to $20.0 billion in 2024, at a compound annual growth rate (CAGR) of 1.5%; even a small share results in high returns

1. Alzheimer's association www.alz.org
2. Alzheimer Europe www.alzheimer-europe.org
3. European Parkinson's Disease Association http://www.epda.eu.com/
8. Global Data
ORY-3001 - the third program of the company

ORY-3001: a third proprietary molecule for orphan diseases

BRIEF-Oryzon Genomics names new compound for preclinical development

Oryzon Genomics SA:

"Names ORY-3001, a specific inhibitor of LSD1, as a candidate for preclinical development for non-oncology indications Source text for Elkon:

Further company coverage: (Gdynia Newsroom)
FINANCIAL HIGHLIGHTS

- €32m raised in the last 12 months (equity+debt)
- Strong balance sheet with €+30m in cash at the end of 1H-2016
- $5 million payment from ROCHE in 2015 ($23m total received in the period 2014-15)
- Secured €2.6M in public aids in 2015
- €20M in debt with low interest rates
  - Repayment terms over either 3-4y or 8-10y (commercial loans or Public R&D loans)
  - Rates from 0-3% (average cost of debt <2%)
  - 1Q-2016: 10.5M non-senior, non-secured debt in 1Q 2016 4-5y term at rates between 1.5%-3.5%
- Current cash burn of €12M annually
- Raised only €31 M in equity since inception
- Spanish GAAP rules adapted partially to IFRS and in readiness for Nasdaq
- Accounts audited by Grant Thornton since 2003
- 35 employees (40 expected by the year’s end)
ORY-1001: LEAD CANCER ASSET

- Complete Phase IIA and report target efficacy
- Roche execute ongoing clinical development plan

ORY-2001: LEAD CNS ASSET

- Begin Phase I patient enrolment
- Complete Phase I dosing safety study in healthy volunteers
- Layout of a multiple Phase II clinical study including potential additional indications

ORY-3001:

- Nomination of Preclinical Candidate

CORPORATE

- Prepare to Dual List on the NASDAQ in the future
THANK YOU VERY MUCH!
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