Disease Modifying Treatments for Diabetic Eye Disorders

Company presentation – April 2019
Forward-looking statement

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Oxurion (Euronext: OXUR) – Investment Highlights

- Forging new directions in diabetic eye disease therapies
  - Targeting multiple disease-modifying pathways
  - Enhancing & going beyond vascular endothelial growth factor (VEGF) inhibition
- Near-term value drivers: robust pipeline and strong R&D engine
  - 3 distinct proprietary programs / 4 clinical trials
  - 4 key data readouts by end of 2019
- End-to-end proven ability to discover and develop innovative ophthalmology therapies
  - Jetrea® (ocriplasmin) - First-in-Class therapeutic for treatment of symptomatic vitreomacular adhesion & traction
  - +30,000 patients treated globally
- Oncurious – the Oncology Subsidiary:
  - 1 Phase II ongoing in medulloblastoma + optionality of broad immuno-oncology pipeline from VIB
- Solid financial position
  - €85.1m cash on hand at December 31, 2018
  - 80 employees globally (1/3 are MDs/PhDs), HQ in Leuven (BE), US office in Iselin, NJ
Diabetic Retinopathy Market & Unmet Needs
Diabetic Retinopathy is a Serious Sight-Threatening Disease

Diabetic retinopathy (DR) is a chronic, progressive, sight-threatening, and life-altering disease.
Diabetic Macular Edema is a Severe Complication of DR

Diabetic macular edema (DME) is an accumulation of fluid in the macula - part of the retina that controls our most detailed vision abilities - due to leaking blood vessels.
Diabetic Retinopathy is a Major Public Health Concern Globally

DR is a progressive disease classified by severity, whereas DME can occur at any stage of DR.

People with diabetes (HbA1c > 7%)

450 Mio people with diabetes

150 Mio people with any diabetic retinopathy*

incl. 50 Mio people with vision-threatening disease**

Abbreviation(s): DR, diabetic retinopathy; NPDR, non-proliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy; DME, diabetic macular edema

* Any diabetic retinopathy is defined as the presence of non-proliferative diabetic retinopathy, proliferative diabetic retinopathy, diabetic macular edema, or any combination thereof.

** Vision-threatening diabetic retinopathy is defined as the presence of proliferative diabetic retinopathy and/or diabetic macular edema.
Major Unresolved Challenges in DME Management

Although anti-VEGF therapy is the mainstay of treatment in DME, finding ways to significantly improve treatments outcomes remain unresolved challenges.

- **Efficacy** | Higher gain on visual acuity & visual function
- **Durability** | Longer sustained treatment response
- **Safety** | Better long-term profile and/or risk of complications

Decrease treatment burden
Significant Unmet Needs for Better Treatment Outcomes in DME

More than 50% of patients have an unsatisfactory early visual response with anti-VEGF therapies, without clinically meaningful visual improvement in the majority of cases over the time.

**Early vision response at 3 months**

- 33-49% \( \geq 10 \) letters
- 25-30% 5-9 letters
- 26-37% \(< 5 \) letters

**Long-term vision change at 2 years**

- Majority of patients maintain satisfactory vision response, but 16-23% decline \(< 10 \) letters
- 53-78% of patients remain with unsatisfactory vision response \(< 10 \) letters

Note: clinically satisfactory vision response is defined by a visual gain \( \geq 10 \) letters

Abbreviation(s): DME, diabetic macular edema; VEGF, vascular endothelial growth factor

**Oxurion Programs Tackling Main Diabetic Retinopathy Hallmarks**

Tackle multiple hallmarks of Diabetic Retinopathy by differentiated mechanisms of action

<table>
<thead>
<tr>
<th>OXUR program</th>
<th>Hallmarks of DR</th>
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<tbody>
<tr>
<td></td>
<td>Inflammation</td>
</tr>
<tr>
<td>THR-317</td>
<td>++</td>
</tr>
<tr>
<td>(PIGF inhibitor)</td>
<td></td>
</tr>
<tr>
<td>THR-149</td>
<td>+</td>
</tr>
<tr>
<td>(Plasma kallikrein inhibitor)</td>
<td></td>
</tr>
<tr>
<td>THR-687</td>
<td>++</td>
</tr>
<tr>
<td>(Integrin antagonist)</td>
<td></td>
</tr>
</tbody>
</table>

+= level of biological activity in preclinical animal models

Abbreviation(s): DR, diabetic retinopathy; PIGF, placenta growth factor
Oxurion Pipeline Addresses Limitations of Standard of Care

Oxurion is addressing unresolved SoC challenges by developing next-generation treatments for diabetic eye disorders.

<table>
<thead>
<tr>
<th>OXUR program</th>
<th>Unresolved challenges</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improve treatment outcomes</td>
<td>Improve efficacy and safety profile of anti-VEGF therapy (safer maintenance therapy)</td>
</tr>
<tr>
<td>Decrease treatment burden</td>
<td>Complementing anti-VEGF efficacy (independent pathway tackling edema)</td>
</tr>
<tr>
<td>THR-317 PIGF inhibitor</td>
<td>THR-149 Plasma kallikrein inhibitor</td>
</tr>
<tr>
<td>THR-687 Integrin antagonist</td>
<td>Alternative to anti-VEGF therapy (higher efficacy through multiple points of attack)</td>
</tr>
</tbody>
</table>

Abbreviation(s): VEGF, vascular endothelial growth factor
Oxurion Clinical-Stage Programs

Clinical-stage programs are based on distinct mechanisms of action for better treatment outcomes

<table>
<thead>
<tr>
<th>Year</th>
<th>Phase 1/2a</th>
<th>Phase 2 (DME)</th>
<th>Phase 2 (MacTel 1)</th>
<th>Phase 1 (DME)</th>
<th>Phase 1 (DME)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2018</td>
<td>THR-317-002</td>
<td>THR-317-003</td>
<td>THR-149-001</td>
<td>THR-149-001</td>
<td>THR-687-001</td>
</tr>
<tr>
<td>2019</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Abbreviation(s): DME, diabetic macular edema; MacTel, macular telangiectasia
THR-317 : anti-PIGF (Placental growth factor)
THR-317 : Humanized mAb Against Human PlGF

PIGF (Placental growth factor) *

- Member of the VEGF-family signaling via binding to VEGFR-1, NRP-1, NRP-2 but not to VEGFR-2
- Expressed in many tissues, including the retina (e.g. endothelial cells, glial cells)
- Key molecule in pathological angiogenesis, edema and inflammation
- DR is a progressive disease... Increasing levels of PIGF correlate with disease severity

THR-317

- Humanized monoclonal antibody against PIGF (IgG1 format)
- Affinity for PIGF in the low picomolar range

THR-317 Shows Unique Properties Compared to anti-VEGFs*  
Equimolar comparison between anti-PIGF versus anti-VEGF

- **Vascular stabilization**
- **Inflammation**
- **Fibrosis**
- **Neurodegeneration**

**THR-317**

**Pericyte coverage**  
(mouse CNV)

<table>
<thead>
<tr>
<th></th>
<th>α-SMA positive area (µm²)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>vehicle</td>
<td>200 ± 5</td>
<td>150 ± 5</td>
<td>100 ± 5</td>
<td></td>
</tr>
<tr>
<td>anti-PIGF</td>
<td>350 ± 5</td>
<td>300 ± 5</td>
<td>250 ± 5</td>
<td></td>
</tr>
<tr>
<td>anti-VEGF</td>
<td>400 ± 5</td>
<td>350 ± 5</td>
<td>300 ± 5</td>
<td></td>
</tr>
</tbody>
</table>

**Collagen deposition**  
(mouse CNV)

<table>
<thead>
<tr>
<th></th>
<th>Mean % Col1a positive area/ total laser spot area</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>30 days after laser</td>
<td>20 ± 2</td>
<td>15 ± 2</td>
<td>10 ± 2</td>
<td></td>
</tr>
</tbody>
</table>

**F4/80 positive cells**  
(mouse STZ)

<table>
<thead>
<tr>
<th></th>
<th>Mean number F4/80 positive cells/retinal area</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>W8 after diabetes onset</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-DR</td>
<td>20 ± 2</td>
<td>15 ± 2</td>
<td>10 ± 2</td>
<td></td>
</tr>
<tr>
<td>DR</td>
<td>*** 25 ± 5</td>
<td>*** 20 ± 5</td>
<td>*** 15 ± 5</td>
<td></td>
</tr>
</tbody>
</table>

**RGC apoptosis**  
(Swiss mouse PDE6b (R560C))

<table>
<thead>
<tr>
<th></th>
<th>Number of apoptotic ganglion cells/retinal area</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>6 weeks after treatment start</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-DR</td>
<td>20 ± 2</td>
<td>15 ± 2</td>
<td>10 ± 2</td>
<td></td>
</tr>
<tr>
<td>DR</td>
<td>*** 40 ± 5</td>
<td>*** 35 ± 5</td>
<td>*** 30 ± 5</td>
<td></td>
</tr>
</tbody>
</table>

Van Bergen et al. 2018 Prog. Retin. Eye Res. Published online 30 October (review article)
THR-317 Offers Additional Benefits Over Anti-VEGF Therapy

Anti-PIGF differentiates itself from pure anti-VEGF approach*

- **THR-317**
  - Inhibition: Neovascularization, leakage
  - No impact: Neurodegeneration
  - Inhibition: Scarring

- **Anti-VEGF**
  - Inhibition: No impact
  - Inflammation: No impact
  - Neurodegeneration: Negative impact

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THR-317 Program: Clinical Development Plan Status

• **THR-317-001**
  - Phase 1 / 2a evaluating THR-317 for treatment of DME patients
    - Study completed
    - Positive Topline Data Day 90 and Day 150 - safe and clinical activity

• **THR-317-002**
  - Phase 2 evaluating THR-317 combo therapy with ranibizumab (Lucentis) for DME
    - Enrollment completed – Initial data planned for Q3 2019

• **THR-317-003**
  - Phase 2 evaluating THR-317 for treatment of MacTel1 patients
    - Currently enrolling patients
THR-317-001: DME Phase I/II Study Design

Single-masked, multicentre study to evaluate the safety and efficacy ClinicalTrials.gov Identifier: NCT03071068

![Study Design Diagram]

**Total n = 49 patients**

- IVT administration
- Age > 18 years
- Treatment naïve n= 40
- Anti-VEGF poor responders n=9
- Randomized 1:1
- Centre-involved DME; CST ≥ 320 µm
- BCVA ≤ 20/40 and ≥ 20/320

**Study Treatment**

- DAY 0
- DAY 30
- DAY 60

**Follow-up**

- DAY 90
- DAY 150

**Incidence of acute ocular adverse events**

- 4 mg THR-317
- 8 mg THR-317

**Primary outcome measures**

- Incidence of systemic and ocular (S)AEs

**Secondary outcome measures**

- Proportion of subjects with a ≥ 15 ETDRS letters gain in BCVA from baseline
- Mean change from baseline in BCVA
- Mean change from baseline in CST-OCT
## THR317-001 Primary Endpoint: THR-317 Safe and Well-Tolerated

### Acute Adverse Events * in the Study Eye in All Treated Subjects

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>THR317 4mg Across Injections</th>
<th>THR317 8mg Across Injections</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 subjects, 3 events</td>
<td>4 subjects, 4 events</td>
</tr>
<tr>
<td>Conjunctival haemorrhage</td>
<td>1</td>
<td>1**</td>
</tr>
<tr>
<td>Ocular hyperaemia</td>
<td>0</td>
<td>1**</td>
</tr>
<tr>
<td>Conjunctivitis</td>
<td>0</td>
<td>1**</td>
</tr>
<tr>
<td>Eye pain</td>
<td>0</td>
<td>1**</td>
</tr>
<tr>
<td>Visual impairment</td>
<td>2***</td>
<td>0</td>
</tr>
</tbody>
</table>

* Onset Up to Day 7 Post-Injection  
** AE was deemed drug-related by the Investigator  
*** One of these AEs was deemed drug related by the Investigator

None of the acute AEs in Study Eye were SAEs, and all were of mild intensity. All acute AEs in the study eye were resolved within 1 to 31 days after onset.
THR-317-001: Top Line Efficacy Results Day 90 & Day 150

Proportion of Subjects with ETDRS Letters Gained from Baseline (anti-VEGF treatment naïve)
THR-317-001: Top line results Day 90/ Day 150
Mean Change in Central Subfield Thickness from Baseline (anti-VEGF Treatment Naïve)
THR-317-001: Positive Topline Data and Next Steps

THR-317 is safe and well tolerated - No dose-limiting (ocular) toxicities or relevant safety events were reported at either dose level.

Clinical activity: 30% of the anti-VEGF treatment naïve study subjects treated with THR-317 in the 8mg group showed a ≥15 letters (3 lines or more) gain in Best Corrected Visual Acuity (BVCA) from baseline at Day 90, versus 5.3% in the 4mg group.

Durability of clinical activity: 10% of the anti-VEGF treatment naïve study subjects treated with THR-317 in the 8mg group still showing a ≥15 letters (3 lines or more) gain in Best Corrected Visual Acuity (BVCA) from baseline at Day 150.

Data supported immediate initiation of a next study evaluating THR-317 in combination with an anti-VEGF (THR-317-002).
THR-317-002: Phase 2 Study in THR-317 + ranibizumab for DME

Randomized, single-masked, active-controlled, multicentre study

ClinicalTrials.gov Identifier: NCT03499223

Total n = 72 patients

- IVT administration
- Age > 18 years
- Treatment naïve n= 48
- Anti-VEGF poor responders n=24
- Randomized 2:1
- Centre-involved DME; CST ≥ 320 µm
- BCVA ≤ 20/40 and ≥ 20/320

Study Treatment

- DAY 0
- DAY 28
- DAY 56

Follow-up

- DAY 84
- DAY 140

Primary outcome measures

- Incidence of systemic and ocular adverse events
- Change from baseline in BCVA
- Change from baseline in CST

Secondary outcome measures

- Change from baseline in BCVA
THR-317-003: Idiopathic Macular Telangiectasia Type 1 (MacTel 1)

- Rare, congenital or developmental form of idiopathic macular telangiectasia
- Predominantly in males, ~ 40 – 50 years
- Typically unilateral
- Different clinical manifestations:
  - Prominent, easily visible microvascular ectasia
  - Increased tortuosity of the macular capillary network
  - Minimal retinal ischemia
  - Absence of neovascularization
  - Lipid deposition
  - Absence of pigment proliferation
  - Macular edema and exudation
- No approved treatment for MacTel 1
- Off-label, aflibercept works better than ranibizumab
Macular Telangiectasia Type 1 (MacTel 1)

Rationale for a potential role of PlGF in MacTel 1

Preclinical observations

- Overexpression of PlGF in rat results in retinal vessel abnormalization manifested by tortuosity, dilation and capillary aneurysms, hallmarks of MacTel1. In contrast, VEGF induce preretinal neovessels

Clinical observations

- Significantly higher level of PlGF in MacTel1 eyes compared with healthy control subjects. No change in VEGF level in these patients
- Aflibercept shows effect in patients refractory or non-responder to bevacizumab or ranibizumab

The combination of biological and clinical evidence strongly suggests that PlGF is involved in the pathogenesis of MacTel type 1
THR-317-003: Phase 2 THR-317 for MacTel1 Study Design

Open-label, multi-centre study ClinicalTrials.gov Identifier: NCT03669393

Total n = 10 patients

- IVT administration
- Age > 18 year
- Macular edema due to MacTel1 with CST >300 µm (OCT)

Primary outcome measures
- Change from baseline CST-OCT by study visit
- Change from baseline area of cystoid spaces by study visit
- Change from baseline in BCVA by study visit
- Incidence of systemic and ocular adverse events from day 0 to 140

Secondary outcome measures
- Change from baseline in CST-OCT

Study Treatment

- Day 0
- Day 28
- Day 56
- Day 84
- Day 140

Follow-up

- 8 mg THR-317
THR-149 : Plasma Kallikrein Inhibitor
Preclinical evidence
- PKal mediates vascular hyper-permeability, leukostasis, cytokine production, retinal thickness and microaneurysm
- PKal inhibition significantly reduces retinal vascular leakage in a diabetic mouse model

Clinical evidence
- Upregulation of intraocular plasma kallikrein contributes to a VEGF independent mechanism of DME progression
- Retinal expression of Bradykinin-1 receptor is increased in DME patients
THR-149: Plasma Kallikrein Inhibitor for Diabetic Macular Edema

Highly Potent Selective and Stable Peptide targeting Plasma Kallikrein

 THR-149 (K_i PKal = 0.4 nM)
Plasma kallikrein (PKal) is a key driver in diabetic macular edema
PKal inhibitors have the potential as a stand-alone treatment in poor responders to Standard of Care or in combination with anti-VEGF for all DME patients
THR-149 Demonstrates a Strong Anti-Edema Effect in vivo
Rat paw edema model (carrageenan-induced)

**Treatment:** intraperitoneal injection after carrageenan injection

<table>
<thead>
<tr>
<th>Test compound</th>
<th>THR-149</th>
<th>3 – 10 – 30 mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative control</td>
<td>Vehicle</td>
<td>NA</td>
</tr>
<tr>
<td>Positive control</td>
<td>Indomethacin</td>
<td>5 mg/kg</td>
</tr>
</tbody>
</table>

Mean ± SEM
N= 10
* P< 0.05
THR-149 Program: Key Take-Aways
Peptide Targeting Plasma Kallikrein for Diabetic Macular Edema

- Plasma Kallikrein (Pkal) is a clinically well validated target for edema, inflammation and the prevention of microhemorrhages
- THR-149 is a stable peptide, potent, selective and reversible inhibitor of Pkal
- Acquired from a partnership with UK-based Bicycle Therapeutics – Oxurion owns all rights to Pkal inhibitors
- The mechanism of action of THR-149 is independent of that of anti-VEGF, allowing to target:
  - DME patients who are non/poor-responders to anti-VEGFs as a monotherapy and/or
  - DME patients who are “naïve” to anti-VEGFs, in combination with anti-VEGFs
- Phase 1 DME study currently enrolling patients
- Data anticipated for end of H2 2019
THR-687: Pan-RGD Integrin Antagonist
THR-687: a Pan RGD Integrin Antagonist

Integrin Antagonists work both upstream and downstream of VEGF, hence they have a broad efficacy

- Inhibition of integrins targets multiple processes involved in pathological angiogenesis and vascular leakage unlike anti-VEGF treatment
- THR-687 is a novel, potent RGD integrin antagonist
- Pharmacological profiling of THR-687 indicates a good safety profile
- THR-687 has a broad therapeutic potential
  - Diabetic retinopathy (DR) with and without DME
  - Wet (Neovascular) AMD
THR-687 Potently Inhibits VEGF-Induced Leakage

Mouse VEGF-induced leakage model (C57BL/6J)

IVT injection of human VEGF (300 ng/1 µL per eye)

Vehicle
Aflibercept
THR-687 (15mg/kg IP, 1h prior VEGF + 50µg IVT)

Read-out: mouse serum albumin (MSA)/mg retina samples

 THR-687 potently inhibits retinal permeability in a mouse VEGF-induced leakage model
THR-687: Potent Inhibition of Angiogenesis-Driven Leakage in Cynomolgus Monkey CNV Model

THR-687 potently inhibits angiogenesis-induced leakage in a monkey CNV model
THR-687: Key Take-Aways and Next Steps

- THR-687 is a novel and potent pan-RGD integrin antagonist in-licensed from Galapagos NV
- THR-687 has a broad therapeutic potential
  - Diabetic retinopathy with and without DME
  - Wet AMD
- Sizeable target populations
- **Phase 1 DME study currently enrolling patients**
- Data anticipated for end of H2 2019
3 New Clinical-Stage DME Programs with Distinct Mechanisms of Action and Data Readouts in H2 2019

<table>
<thead>
<tr>
<th></th>
<th>2018</th>
<th>2019</th>
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<tbody>
<tr>
<td>H1</td>
<td>H2</td>
<td>H1</td>
</tr>
<tr>
<td>Phase 1/2a</td>
<td>THR-317-002</td>
<td>THR-687-001</td>
</tr>
<tr>
<td></td>
<td>Phase 2 (DME)</td>
<td>Phase 1 (DME)</td>
</tr>
<tr>
<td>THR-149-001</td>
<td>Phase 1 (DME)</td>
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</table>
Financials
Ourion NV: Key Financial Data Summary

- Equity investment of €10 million by Novartis Pharma AG in Oxurion capital
- €85.1 million in cash and investments at end-December 2018 (€115.1 million at end-December 2017)
- +85% owner of Oncurious NV (VIB venture partner)
- 80 employees globally, HQ in Leuven (BE), US office in Iselin, NJ
- Listed on Euronext Brussels (IPO 2006: OXUR)
Oxurion (OXUR) Shareholder Structure and Cash Position

- Novartis Pharma AG
- Mr. Thomas M. Clay and entities controlled by him
- Mrs. Lavinia D. Clay
- Baron Philippe Vlerick and entities controlled by him
- Public

- Listed on Euronext Brussels: OXUR
- Cash position Q4 2018: € 85.1 M
Oxurion NV: Board of Directors

Thomas Clay, Non-Executive, Independent Director, Chairman
Vice-President of East Hill Management Company, LLC and Chairman and CEO of Golden Queen Mining Co., Ltd.

Patrik De Haes, MD, (ViBio BVBA), Chief Executive Officer, Executive Director

Dr David Guyer MD, Non-Executive Director
A long standing member of the US retina community. Co-Founder and Executive Chairman of Ophthotech Corporation. He was previously the CEO of Ophthotech.

Adrienne Graves, PhD, Non-Executive Director
Brings over 25 years in global ophthalmology leadership experience to Oxurion. Board member of multiple companies and organizations including Akorn Inc., Nicox, the American Society of Cataract and Refractive Surgery, the Glaucoma Research Foundation, and the American Academy of Ophthalmology. Former CEO of Santen in the US.

Emmanuèle Attout (Investea BVBA), Non-Executive, Independent Director
Audit partner at PwC from 1994 to 2014, in charge of audits of a range of clients including banks, insurance companies, investment funds and asset managers.

Baron Philippe Vlerick, Non-Executive, Independent Director
Owner, Chairman and CEO of several businesses in Belgium and abroad.
Oxurion Business Model: An Unique Ophthalmology Platform

Leverage unique Back-of-the-eye expertise and capabilities to identify, acquire and develop best-in-class molecules addressing high unmet needs

- mAb, scAb, nanobody generation
- Peptide library screening
- Compound library screening
- Medical chemistry
- “.....omics” technologies (e.g. single cell retina transcriptomics)
- Drug Formulation & delivery technologies

Unique Disease Platform
✓ Back of the eye expertise
✓ Drug targets / pathways
✓ Translational expertise
✓ Clinical Development track-record
✓ Dedicated R&D team (30 FTEs)

First- or Best-in-Class clinical drug candidates for complex retinal diseases with large unmet needs
4 Clinical studies recruiting Disease Modifying Treatments Unmet Medical Needs in DR/DME

- THR-317-002 (anti-PIGF) – DME
- THR-317-003 (anti-PIGF) – MacTel1
- THR-149-001 (PKal inh) – DME
- THR-687-001 (Pan RGD integrin) – DME

Data in second half of 2019
An Oxurion venture developing next immuno-oncology therapies
Recent Oncurious Development – Next Generation Immuno-Oncology

Early-stage but exciting pipeline of next-generation immuno-oncology drugs targeting a broad spectrum of cancers

- Phase 1/2a clinical trial evaluating TB-403 for relapsed medulloblastoma with the Support of the US Consortium Beat Childhood Cancer
- Oncurious and VIB developing pipeline of 5 next-generation immuno-oncology compounds targeting a broad spectrum of cancers: preclinical PoC by end of 2019
- Oncurious venture partners: VIB / Oxurion hold 15% and 85% respectively
JETREA®, a unique treatment option for Vitreo-Macular Traction (VMT), a rare and life-altering vitreo-retinal disorder
Jetrea®: a unique First-in-Class Drug for symptomatic VMA Treatment

- Jetrea (ocriplasmin), the only approved pharmacologic agent for the treatment of symptomatic vitreomacular adhesion/ vitreomacular traction
- Administered to ~30,000 patients worldwide since 2013 and tracked in a robust safety database
- Post marketing data enables targeting specific patients with the highest clinical benefit and chances of success
- New pre-diluted ready-to-use (RTU) formulation enabling easy administration by physicians
- Jetrea benefits from strong IP protection and a proprietary manufacturing process, creating high barriers to competitive entry
- Website: www.jetrea.com
- Operated by focused global business unit – objective is profitable growth and continued access for patients
Jetrea Global Rights with Oxurion
Building on positive experience of JETREA® loyal users and a 360° evidence based approach

JETREA® is a unique, early stage, first line, cost-effective pharmacological option to treat symptomatic VMA/VMT in selected patients, providing sustained and improved patient visual outcomes and preventing disease progression/worsening of visual function.

Abbreviation(s): VMT, vitreomacular traction; ERM, epiretinal membrane; DME, diabetic macular edema; BCVA, best-corrected visual acuity; VFQ-25, visual function questionnaire-25 items; RCT, randomized controlled trial; NICE, national institute for health and care excellence; HAS, high authority for health; G-BA, federal joint committee; HTA, health technology assessment.