Disclaimer

Some of the statements contained in this presentation constitute forward-looking statements. Statements that are not historical facts are forward-looking statements. Forward-looking statements generally can be identified by the use of forward-looking terminology such as “may”, “will”, “expect”, “intend”, “estimate”, “anticipate”, “believe”, “continue” or similar terminology. These statements are based on the Company’s current strategy, plans, objectives, assumptions, estimates and projections. Investors should therefore not place undue reliance on those statements. The Company makes no representation, warranty or prediction that the results anticipated by such forward-looking statements will be achieved, and such forward-looking statements represent, in each case, only one of many possible scenarios and should not be viewed as the most likely or standard scenario. Forward-looking statements speak only as of the date that they are made and the Company does not undertake to update any forward-looking statements in light of new information or future events. Forward-looking statements involve inherent risks and uncertainties. The Company cautions that a number of important factors could cause actual results to differ materially from those contained in any forward-looking statement.
Poxel: A Mid-to-Late Stage Metabolic-Focused Opportunity

- Targeting key pathways of cellular energy to treat chronic metabolic diseases
- Three mid-to-late stage candidates with differentiated strategies
- Multiple near-term catalysts 4Q 2019/2020
- Euronext Paris

**Imeglimin**
- First in class
- Mitochondrial bioenergetics

**PXL770**
- First in class
- Direct AMPK activator

**PXL065**
- MPC inhibitor
- Single isomer of pioglitazone

**Additional Opportunities**
- Other metabolic diseases

**Other Metabolic Diseases**

- High-value, global partnerships
- Novel mechanism with franchise potential
- Fast-to-market strategy via 505(b)(2) pathway
- Exploring complementary mechanisms in complex multifactorial diseases
- Cash & equiv. EUR 36.8M (USD 40.1M) as of 9/30/19; bond loan financing of up to EUR 30M* extends cash runway into 2022
- Based in Lyon, France with subsidiaries in Boston and Tokyo

*Obtained in November 2019
### Metabolic Pipeline

**Well-Diversified Pipeline with Mid-to-Late Stage Programs**

<table>
<thead>
<tr>
<th>Indication</th>
<th>MOA</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Partner/Rights</th>
<th>Upcoming Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type 2 Diabetes</strong></td>
<td></td>
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</tr>
<tr>
<td>Imeglimin Japan / Asia*</td>
<td>Type 2 Diabetes (T2D)</td>
<td>Mitochondrial Bioenergetics</td>
<td></td>
<td></td>
<td></td>
<td>Japan / Asia*</td>
<td>▪ Ph 3 completion; Ph 3 results for TIMES 2 and TIMES 3 36-week portion 4Q 19 &lt;br&gt;▪ Target JNDA submission 2020 &lt;br&gt;▪ Target product launch 2021</td>
</tr>
<tr>
<td>Imeglimin US / EU / Other**</td>
<td>TD2 patients with CKD stages 3b/4</td>
<td>Mitochondrial Bioenergetics</td>
<td></td>
<td></td>
<td></td>
<td>US / EU / Other**</td>
<td>▪ FDA mtg prior to initiating Ph 3 in T2D patients w/ CKD in 2020</td>
</tr>
<tr>
<td><strong>NASH</strong></td>
<td></td>
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<tr>
<td>PXL770</td>
<td>NASH</td>
<td>Direct AMPK Activator</td>
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<td></td>
<td></td>
<td>▪ PK/PD results 1Q 20 &lt;br&gt;▪ Ph 2a results 2Q 20</td>
</tr>
<tr>
<td>PXL065</td>
<td>NASH</td>
<td>MPC Inhibition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>▪ Ph 1b results 4Q 19 &lt;br&gt;▪ Utilize 505(b)(2) pathway &lt;br&gt;▪ Phase 2 initiation 2Q 20</td>
</tr>
<tr>
<td>PXL007 (EYP001)</td>
<td>Hepatitis B / NASH</td>
<td>FXR Agonist</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>▪ Complete Ph 2 program by Enyo Pharma 2H 20</td>
</tr>
<tr>
<td><strong>Other Metabolic Diseases</strong></td>
<td></td>
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<tr>
<td>Poxel / DeuteRx Programs</td>
<td>Metabolic (AMN / ALD, NASH, etc.)</td>
<td>Direct AMPK Activator / MPC Inhibition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>▪ Complete preclinical studies 4Q19-1H 20</td>
</tr>
</tbody>
</table>

*including: China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos.

**countries not covered in the Sumitomo Dainippon Pharma agreement.**
Imeglimin
First in a New Class of Potential Anti-diabetic Treatments with a Differentiated Mechanism of Action
### Imeglimin Global Partnerships

**Combined Potential Total Deal Value >$850 Million**

<table>
<thead>
<tr>
<th>Partner</th>
<th>Imeglimin Rights</th>
<th>Deal Value</th>
<th>Development Status</th>
</tr>
</thead>
</table>
| **Sumitomo Dainippon Pharma** | Development and commercialization rights in Japan, China, South Korea, Taiwan and 9 other Southeast Asian countries* | • Upfront payment: $42M  
• Future potential development milestone payments and sales-based payments of up to approx. $257M  
• Double-digit escalating royalties | • Ph 3 TIMES program (TIMES 1, TIMES 2 and TIMES 3) in T2D patients  
• Positive results for TIMES 1 and TIMES 3 16-week data  
• TIMES 2 and TIMES 3 36-week results anticipated 4Q19  
• JNDA target submission in Japan 2020 |
| **Roivant Sciences** | Development and commercialization in the U.S., Europe, and other countries** | • Upfront payment: $35M\(^1\) Equity Investment: $15M at €8.5/share  
• Future potential development and regulatory milestone payments and sales-based payments of up to $600M  
• Double-digit escalating royalties | • Positive results reported for PK/PD trial in T2D patients with CKD stages 3b/4  
• FDA meeting prior to initiating Ph 3 program |
| **Metavant** | Poxel and Roivant will decide on a potential co-promotion prior to commercialization |                                                                                               |                                                                                      |

*including: Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos.
**countries not covered in the Sumitomo Dainippon Pharma agreement 1. Poxel contributing $25M (~€20M) to development program over a 2-year period.
Imeglimin Development Strategy for Japan

Targeting a JNDA Submission in 2020

- Target launch anticipated in Japan in 2021
- Phase 3 TIMES program includes over 1,100 patients
- Positive results reported from TIMES 1 and TIMES 3 (16-week portion)

*Sumitomo Dainippon Pharma is development and commercialization partner for Japan, China and 11 other East and Southeast Asian countries*

* including: South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos.
Imeglimin Phase 3 TIMES 1 Trial in Japan Met Primary and Key Secondary Endpoints (N=213)

Phase 3, 24-week, randomized, double-blind, placebo-controlled, monotherapy trial to evaluate the efficacy, safety and tolerability of Imeglimin administered orally in Japanese patients with type 2 diabetes.

- Statistically significant HbA1c reduction with demonstrated efficacy and an observed safety and tolerability profile similar to placebo (n=213)

- TIMES 1 results are consistent with Phase 2b results observed in Japan

European Association for the Study of Diabetes meeting 2019
Imeglimin Phase 3 TIMES 3 16-week Portion Met Primary Endpoint with Favorable Safety and Tolerability Profile (n=215)

Phase 3, 16-week, double-blind, placebo-controlled, randomized trial with a 36-week open-label extension period to evaluate the efficacy and safety of Imeglimin in combination with insulin in Japanese patients with type 2 diabetes and inadequate glycemic control on insulin therapy.

- TIMES 3 16-week results met its primary endpoint of a reduction in HbA1c
- Safety and tolerability profile observed similar to placebo
  - A similar number of patients experienced hypoglycemia with Imeglimin compared to placebo
  - No severe hypoglycemia events observed
- TIMES 3 36-week open-label results expected around the end of 2019

Change in HbA1c – 16 Weeks

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>Placebo (N=107)</th>
<th>Imeglimin (N=108)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c (%), mean (SD)</td>
<td>8.8 (0.8)</td>
<td>8.7 (0.7)</td>
</tr>
</tbody>
</table>

LS mean (SE) = -0.60% (0.10)
Imeglimin
US/Europe Strategy
Metavant to develop Imeglimin first specifically to treat patients with type 2 diabetes with chronic kidney disease (CKD) stages 3b/4¹

1. CKD stage 3b = eGFR 30-44 ml/min/1.73 m² inclusive; CKD stage 4 = 15-29 ml/min/1.73 m² inclusive.

   • Positive results reported for PK/PD trial in this patient population July 2019
     • Favorable safety and tolerability profile observed
     • PK/PD data consistent with previous Poxel data

   • Metavant will meet with regulatory authorities to discuss Phase 3 program in this patient population

   **Imeglimin TIMES 1 and Phase 2 data (Japan, US and Europe) was observed to be well tolerated and demonstrated similar efficacy in patients with impaired renal function compared to patients with normal renal function**

**Patients with type 2 diabetes and CKD stages 3b/4**

1. Diabetes is the most common cause of chronic kidney disease
2. Approximately 2.4 million adults in the US²
3. Patients have increased cardiovascular risk and challenging glucose management

**Underserved patient population**

1. Many approved therapies require dose reduction or are not recommended in the presence of kidney disease
2. Insulin and insulin secretagogues are the most commonly used therapies at suboptimal doses to prevent risk of hypoglycemia
3. We believe there is a need for a new treatment providing a strong efficacy and safety profile with no hypoglycemia risk

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Limitations of Current Therapies to Treat T2D by Kidney Disease Stage Drives Metavant Focus for Imeglimin

<table>
<thead>
<tr>
<th>Therapy1-16</th>
<th>CKD 3a</th>
<th>CKD 3b</th>
<th>CKD 4</th>
<th>Primary Concern of Using Agent in Advanced CKD</th>
</tr>
</thead>
<tbody>
<tr>
<td>BG Metformin</td>
<td></td>
<td></td>
<td></td>
<td>Increased risk of lactic acidosis17</td>
</tr>
<tr>
<td>DPP-4i Sitagliptin Saxagliptin Linagliptin</td>
<td></td>
<td></td>
<td></td>
<td>Increased risk of precipitating symptoms of heart failure2-4</td>
</tr>
<tr>
<td>SGLT2i Canagliflozin Empagliflozin Dapagliflozin</td>
<td></td>
<td></td>
<td></td>
<td>Reduced glucose lowering effect18</td>
</tr>
<tr>
<td>GLP1-RA Exenatide ER Liraglutide Dulaglutide Semaglutide</td>
<td></td>
<td></td>
<td></td>
<td>Increased gastrointestinal adverse effects19-21</td>
</tr>
<tr>
<td>SU Glyburide Glimepiride Glipizide</td>
<td></td>
<td></td>
<td></td>
<td>Hypoglycemia22</td>
</tr>
<tr>
<td>TZD Pioglitazone</td>
<td></td>
<td></td>
<td></td>
<td>Contraindicated for patients diagnosed with heart failure15</td>
</tr>
<tr>
<td>Insulin</td>
<td></td>
<td></td>
<td></td>
<td>Hypoglycemia22</td>
</tr>
</tbody>
</table>

Please note that references for this slide are in the Appendix section.
PXL770 and PXL065
Two Differentiated Drug Candidates with Complementary Mechanisms of Action for the Treatment of NASH
Progression of Non-Alcoholic Fatty Liver Disease (NAFLD)

- **Excessive caloric intake**
  - Sedentary lifestyle

- **Metabolic syndrome**
  - Dyslipidemia
  - Type II diabetes
  - Obesity

- **Steatosis**
  - Normal
  - 25% of the general population
  - >70% in diabetic & obese patients

- **Lipotoxicity**
  - NAFLD
  - 12% of the general population
  - 25-70% in diabetic and obese patients ≥ 50

- **Inflammation**
  - NASH
  - >70% in diabetic & obese patients

- **Fibrosis**
  - Cardiovascular events (leading cause of death)
  - Hepatic impairment
  - Hepatocellular carcinoma

- **High Morbidity**
  - High Morbidity
  - Cardiovascular events (leading cause of death)
  - Hepatic impairment
  - Hepatocellular carcinoma

- **Collagen Deposition**
  - Cirrhosis

- **Fat Deposition**
  - Non-Alcoholic Fatty Liver Disease (NAFLD)
PXL770

Direct AMPK Activator for the Treatment of NASH
Why Activating AMPK is of Interest for Metabolic Disorders

Adapted from:
Clinical Science (2012) 122, 555-573
Molecular Cell Biology Volume 13 | April 2012 | 251
PXL770: Targeting Master Regulator of Cellular Energy for NASH

- In addition to monotherapy, PXL770 has the potential to be combined with other clinical candidates in development for NASH targeting other pathways.

Metabolic Impact
- Induces a metabolic switch toward preferential fat oxidation, which is a breakdown of triglycerides stored in fat cells, and free fatty acids in the blood for energy.
- Improves overall metabolic profile by increasing insulin sensitivity, glucose tolerance and muscle glucose uptake.

Beneficial Effects for NASH
- Decreases liver steatosis by inhibiting free fatty acids from traveling from the adipose tissue to the liver and hepatic de novo lipogenesis.
- Reduces adipose tissue and liver inflammation.
- Decreases profibrogenic pathways.

PXL770 directly activates AMPK, an energy sensor which controls multiple metabolic, inflammatory and profibrogenic pathways.
PXL770: Expected Effects for Treating NASH
A Direct AMPK Activator

Effects in Adipose Tissue:
- Low grade inflammation
- Lipolysis
- Peripheral insulin resistance

Effects in Muscle:
- De novo lipogenesis

Effects in Hepatocytes:

Effects in Mitochondria:
- Mitochondrial integrity
- Mitochondrial functions

Effects in Liver:
- Steatosis
- Chronic inflammation
- Effects in Hepatic Stellate Cells
- Fibrogenesis
- Hepatic stellate cell activation

NASH

Low grade inflammation
Lipolysis
Peripheral insulin resistance
De novo lipogenesis
Effects in Hepatocytes
Effects in Mitochondria
Effects in Liver
Effects in Adipose Tissue
NASH

Steatosis
Chronic inflammation
Effects in Hepatic Stellate Cells
Fibrogenesis
Hepatic stellate cell activation
Mitochondrial integrity
Mitochondrial functions
PXL770: Promising Preclinical Data Shows Potential to Treat Underlying Root Causes of NASH (1/2)

Data Observed Correlates to Metabolic Impact and Beneficial effects for NASH

- Improve liver steatosis and NAS in a diet induced obesity biopsy-proven NASH mouse model

- Decreases adipose tissue and liver inflammation markers in a diet induced obesity biopsy-proven NASH mouse model

- Decreases profibrogenic pathways in a diet induced obesity biopsy-proven NASH mouse model

PXL770: Promising Preclinical Data Shows Potential to Treat Underlying Root Causes of NASH (2/2)

Data Observed Correlates to Metabolic Impact and Beneficial effects for NASH

• Improves metabolic syndrome associated with NASH
  – Improves glycemia and lipids in metabolic rodent models:
    ▪ Insulin sensitivity
    ▪ Glycemic control: basal glycemia, glucose tolerance and HbA1c
    ▪ Circulating lipids (TGs, FFAs)

• Induces a metabolic switch toward preferential fat oxidation

![Insulin Sensitivity Graph](image)

![Fat Oxidation Graph](image)

*P<0.0001 vs. veh group.
WD: Whole Day
PXL770: Positive Phase 1 Study Results (n=124)

Single ascending and multiple ascending doses from 30 mg up to 500 mg

• Good linear PK profile:
  - Cmax and area under the curve (AUC) increased dose-dependently following oral single administration
  - After multiple administrations, increased plasma exposure was dose-dependent up to 375 mg and less than dose-proportional at 500 mg

• Good safety and tolerability profile:
  - No serious adverse events, no severe adverse event or adverse event leading to study discontinuation
  - Very good tolerability with low placebo-like incidence of treatment emergent adverse events
  - No effect on ECG parameters, and specifically, no prolongation of the corrected QT interval
PXL770: Phase 2a Program Initiated for NASH

The Phase 2a 12-week, multicenter, randomized, double-blind, placebo-controlled, parallel group trial for PXL770 was initiated in April 2019

- Objective is to demonstrate PXL770’s potential in NASH and validate the hypothesis for AMPK activation more broadly
- Phase 2a trial will assess efficacy and safety in approximately 100 patients who likely have NASH

Results expected 2Q 20

A separate four-week PK/PD study of PXL770 was initiated in August 2019

- Expected to enroll approximately 16 patients per dose
- Primary objective is to assess full PK profile of PXL770 in nonalcoholic fatty liver disease (NAFLD) patients who likely have NASH and evaluate efficacy biomarkers, safety and tolerability

Results expected in the 1Q 20
PXL770: Development Strategy for NASH

**Phase 2a Program**

- Phase 2a efficacy and safety trial
  - ~100 patients with NAFLD (likely NASH)
  - 12-week treatment
  - Primary endpoint: change in liver fat mass based on MRI-PDFF
  - Assessment of effect on inhibition of lipolysis and hepatic *de novo* lipogenesis and safety
- PK/PD trial to evaluate PK profile and effects on hepatic and metabolic parameters
- Phase 2b in biopsy proven NASH patients
PXL065

MPC Inhibitor for the Treatment of NASH Utilizing the 505(b)(2) Regulatory Pathway
Pioglitazone is a mixture of 2 stereoisomers with dramatically different properties

**S-Pioglitazone (stabilized)**
- MPC inhibitor
- Strong PPARγ agonist
- Undesired side effects:
  - Weight gain
  - Fluid retention

**PXL065 (stabilized R-pioglitazone)**
- MPC inhibitor
- Very weak PPARγ agonist
- Anti-inflammatory
- NASH efficacy

---

**Pioglitazone has been approved since 1999 for T2D**
- >30 million patient years of exposure

**Pioglitazone has been extensively studied and is efficacious for NASH**
- Achieved “Resolution of NASH without worsening of fibrosis” in Phase 4 trial
- Only drug recommended for biopsy-proven NASH by AASLD & EASL Practice Guidelines
- Currently prescribed by ~14% of physicians for biopsy-proven NASH patients
- Limited use due to PPARγ-related side effects: weight gain, fluid retention, bone loss

---

4. Therap Adv Gastroenterol. 2016, 9(1), 4-12
Pioglitazone Observed to be Efficacious for NASH
Use Limited by Weight Gain

Resolution of NASH
without worsening of fibrosis

Pio Cusi Phase 4 trial (45 mg, 18 mos) - Ann Intern Med. 2016, 165(5), 305-315 (only completers with definite NASH at baseline). Patients on placebo benefited from 4% weight loss due to hypocaloric diet.

Ocaliva REGENERATE Phase 3 trial (25 mg, 18 mos), Intercept press release Feb 19, 2019

CVC (Cenicriviroc) CENTAUR Phase 2b trial (150 mg, 1 yr/12 mos) – Hepatology 2017 (doi: 10.1002/hep.29477)

Elafibranor Phase 2 trial (120 mg, 52 wks/12 mos) – Gastroenterology, 2016, 150(5), 1147-1159

Liraglutide Phase 2 trial (0.6 increased to 1.8 mg sc weekly 48 wks) - The Lancet, 2016, 387(10019), 679–690

MGL-3196 Phase 2 trial (36 wks/8 mos) – press release May 31, 2018. Results from per protocol, not intent to treat (ITT) population.

Aramchol Phase 2 trial (600 mg, 52 wks) – press release June 12, 2018. No effect on "Fibrosis without worsening of NASH".
PXL065: Inhibits Mitochondrial Pyruvate Carrier (MPC)
Without PPAR\(\gamma\) Agonist Activity from S-Stereoisomer

**MPC Inhibition in HepG2 Cells**

<table>
<thead>
<tr>
<th>Conc (µM)</th>
<th>Pyruvate-driven respiration (% control)</th>
<th>IC(_{50})</th>
</tr>
</thead>
<tbody>
<tr>
<td>PXL065 (R-Pio): 6.5 µM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d-S-Pio: 8.5 µM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pioglitazone: 6.8 µM</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**PPAR\(\gamma\) Agonist Activity**

![Graph showing PPAR\(\gamma\) activation](image)

PPAR\(\gamma\) activation in fluorescence-based TRAP220 coactivator recruitment assay. Results are expressed as % of response of positive control (10µM rosiglitazone).
PXL065: Expected Effects for NASH
A Mitochondrial Pyruvate Carrier (MPC) Inhibitor

Effects in Mitochondria
- Mitochondrial integrity
- Mitochondrial functions

Effects in Liver
- Steatosis
- Chronic inflammation
- Fibrosis
- Neoglucogenesis

Effects in Adipose Tissue
- Peripheral insulin resistance

Effects in Muscle
Liver NAS and Fibrosis Measured in CD and MCD Mouse Models

Liver histopathology on day 43 in mice fed a Choline Deficient (CD) or an Methionine/Choline Deficient (MCD) diet, Pioglitazone (30 mg/kg/day) or PXL065 (15 mg/kg/day), Wilcoxon rank sum test vs vehicle; *p < 0.05, **p < 0.005, ***p < 0.001
PXL065: Phase 1a Results
Favorable Tolerability and Pharmacokinetics (PK)

Single oral dose of PXL065 (7.5, 22.5 and 30 mg) or Actos®1 (45 mg) in healthy subjects, 18-40 yrs

Tolerability
- PXL065 was observed to be well-tolerated at all doses tested

PK Results
- Observed stabilization of d-R-pio, with limited interconversion to S-pio, at all doses tested
- Relative exposure (AUC) to R-pio/S-pio increased ~3x
- Dose-proportional up to 22.5 mg
- PK/PD simulation predicts 15 mg PXL065 ≥ efficacy as 45 mg pio without weight gain

1. Actos is the branded version of pioglitazone and a registered trademark of Takeda Chemical Industries, Ltd
### PXL065: Phase 1b MAD Study (n=30)

A Phase 1, Double-blind, Randomized, Placebo-controlled Study to Evaluate the Safety, Tolerability, and Pharmacokinetics of Repeated Doses of PXL065 in Healthy Subjects versus Actos®

<table>
<thead>
<tr>
<th>Screening</th>
<th>Double-blind Treatment Period</th>
<th>Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 weeks</td>
<td>Single dose</td>
<td>1 day</td>
</tr>
<tr>
<td></td>
<td>Wash-out</td>
<td>2 days</td>
</tr>
<tr>
<td></td>
<td>Repeated dose</td>
<td>7 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 days</td>
</tr>
</tbody>
</table>

- **Actos® 45 mg (n= 6)**
- **PXL065 7.5 mg, pbo (3:1 active:pbo, n = 8)**
- **PXL065 15 mg, pbo (3:1 active:pbo, n = 8)**
- **PXL065 30 mg, pbo (3:1 active:pbo, n = 8)**

- Phase 1b will support dose selection for a pivotal study; topline results expected in 4Q 19
- Primary objective: Evaluate safety and tolerability
- Secondary objectives (PK):
  - Assess and compare relative exposures to R- and S-pioglitazone after PXL065 vs Actos®
  - Evaluate dose proportionality and intra-subject variability

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1. Actos is the branded version of pioglitazone and a registered trademark of Takeda Chemical Industries, Ltd.
505(b)(2) Regulatory Pathway

1992 FDA guidance document “Development of New Stereoisomeric Drugs”

- Streamlined development pathway is expected for single enantiomers of approved racemic drugs
- Existing nonclinical data from the racemate can be relied upon to support the safety of the single enantiomer, and an abbreviated pharmacology and/or toxicology evaluation and initial clinical characterization may be pursued (Section IV of FDA, 1992)

Ability to rely on data generated by others in:

- Product label for parent drug
- Published literature

Potential opportunities to bridge to data from parent drug

- Fewer animal toxicity studies
  - Example: 28 day and 90-day studies in 1 species instead of 2
  - Example: no need for 2-year rat carcinogenicity study
- Potential for fewer clinical trials for submission of NDA
Following the FDA meeting and review of the top-line Phase 1b MAD trial results (expected 4Q 19), Poxel plans to initiate a Phase 2 trial 2Q 20 in biopsy-proven NASH patients.

Primary objective is to identify dose or doses for a Phase 3 registration trial.

Poxel anticipates evaluating three doses of PXL065 compared to placebo in biopsy-proven NASH patients and using several endpoints, which may include:

- MRI-PDFF
- Alanine aminotransferase (ALT)
- Histology (biopsy to assess measures of NASH efficacy in the liver)
- Body weight gain assessment

Finalization of the design for the Phase 2 plan is underway with input from scientific experts, advisors and KOLs, and an update will be provided once the plan is formalized.
Summary of Upcoming Milestones
Significant Upcoming Milestones for 2019/2020

Imeglimin (Type 2 Diabetes)

- Phase 3 full results for TIMES 3 (36-week open label) (4Q19)
- Phase 3 TIMES 2 results (4Q19)
- Metavant to meet with FDA prior to initiating Phase 3 program in T2D patients with CKD stages 3b/4
- Target NDA submission in Japan (2020)
- Imeglimin target launch in Japan (2021)

PXL770 (NASH)

- PK/PD data results (1Q20)
- Phase 2a data results (2Q20)
- Presentations and scientific papers published for PXL770 (2019/2020)
- Global NASH Congress 2020 (1Q20)

PXL065 (NASH)

- Completion of Phase 1 program (4Q19)
- NASH-TAG conference presentation (1Q20)
- Initiation of Phase 2 clinical program in NASH (2Q20)

Preclinical data on other metabolic diseases (2019/2020)
Thank You