Corporate Presentation

June 2021

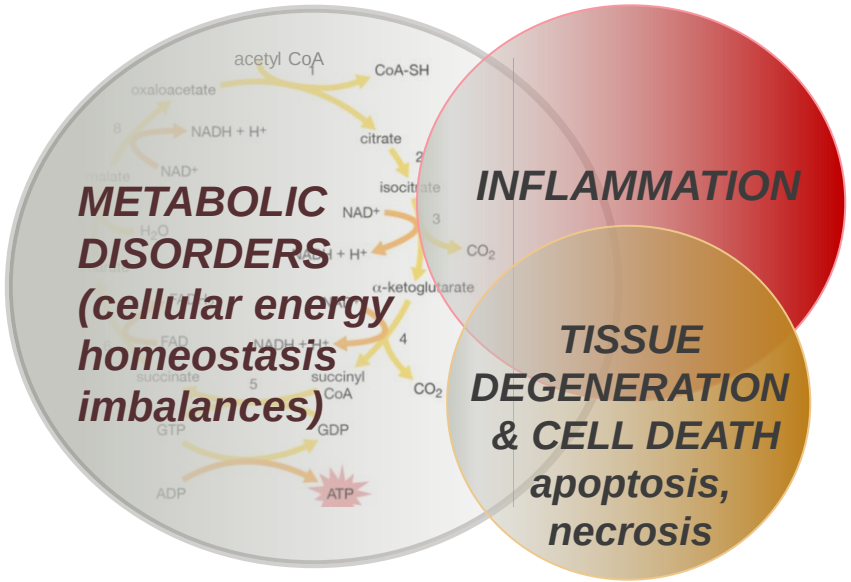
Disclaimer

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.

In the context of the COVID-19 outbreak, which was declared a pandemic by the World Health Organization (WHO) on March 12, 2020, the Company is regularly reviewing the impact of the outbreak on its business. As of the date of this presentation, and based on publicly available information, the Company has not identified the occurrence of any material negative effect on its business due to the COVID-19 pandemic that remains unresolved. However, the Company anticipates that the COVID-19 pandemic could have further material negative impact on its business operations. The worldwide impact of COVID-19 may notably affect the Company’s internal organization and efficiency, particularly in countries where it operates and where confinement measures are implemented by the authorities. In addition, COVID-19 may impact market conditions and the Company’s ability to seek additional funding or enter into partnerships. Particularly, delays in the supply of drug substance or drug products, in the initiation or the timing of results of preclinical and/or clinical trials, as well as delays linked to the responsiveness of regulatory authorities could occur, which could potentially have an impact on the Company’s development programs and partnered programs. The Company will continue to actively monitor the situation.

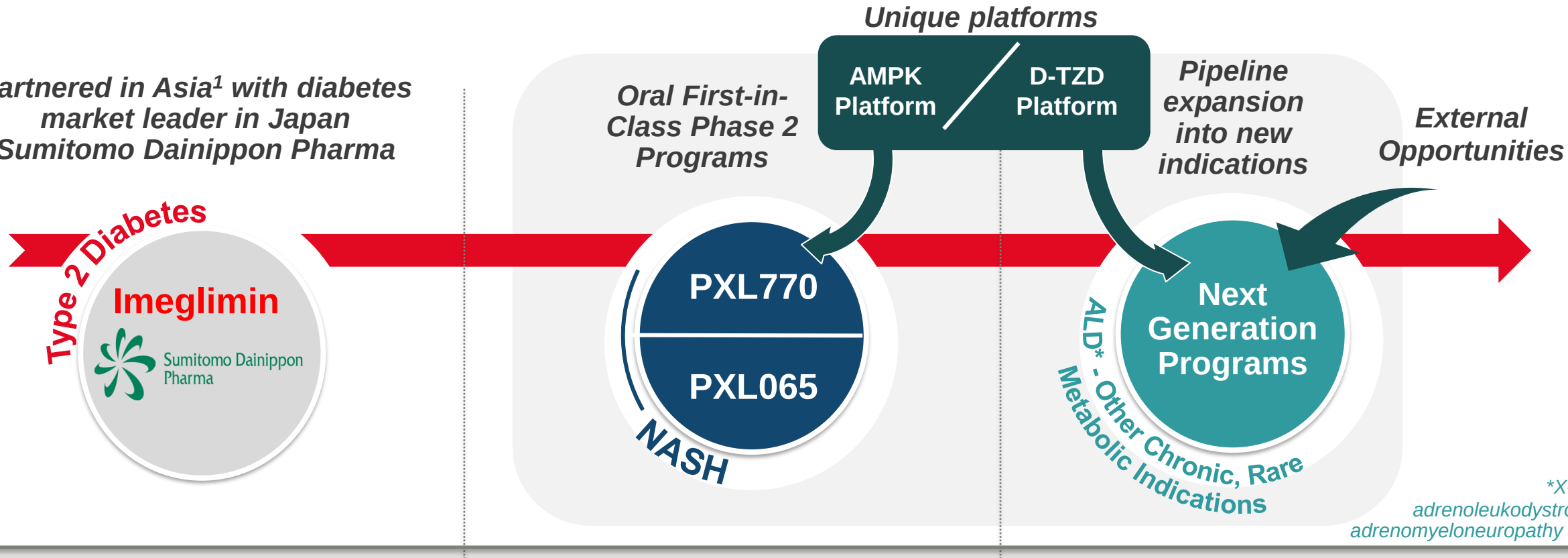
Poxel's Mission and Vision

To discover, develop and commercialize innovative therapies for patients suffering from serious chronic diseases with underlying metabolic pathophysiology



Three Pillars of Poxel's Strategy

First-in-Class Programs Leading to Key Value Inflection Points



Expected approval in 2021[2] triggering milestones

Phase 3 ready partnership opportunity in US/EUR

Phase 2 biopsy data for both programs in 2022-2023

Combination potential

New clinical programs in next 12-24 months

Further strengthening product pipeline

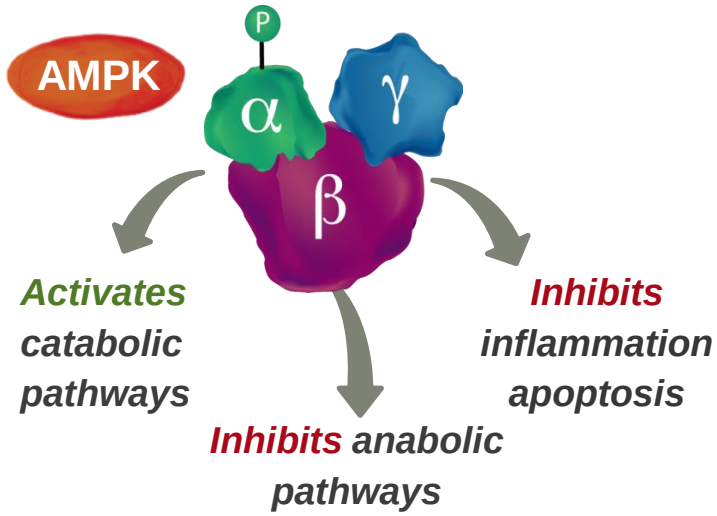
1. Including: Japan, China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos. 2. Sumitomo fiscal year April-March.

Poxel Platforms and Molecules Target Key Nodes that Regulate Cellular Energy Homeostasis

Multiple Entry Points Available to Intervene in Metabolic Diseases

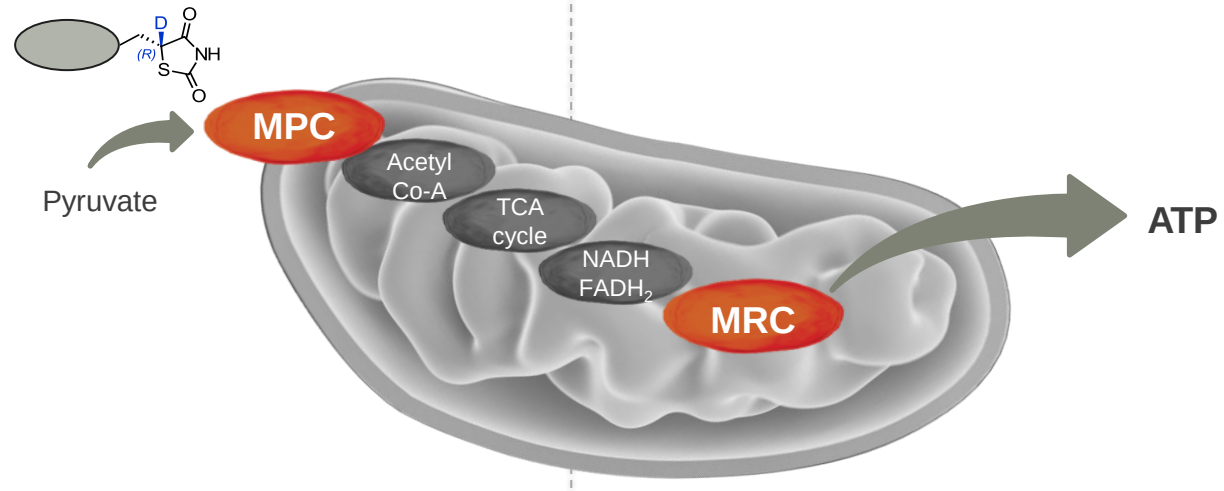
NASH Other

AMP-activated Protein Kinase (AMPK) Activator Platform
cellular *energy sensor* :
reduces liver fat, increases insulin sensitivity, decreases inflammation



NASH Other

Deuterium-Stabilized TZD* Platform *non-genomic* pathway *modulators* of mitochondrial pyruvate carrier – a key fuel *gate-keeper* :
promotes fat utilization, increases insulin sensitivity, decreases inflammation



T2D

Imeglimin – modulates mitochondrial respiratory chain (MRC), cell's *energy producing* machine: improved islet β-cell function; insulin sensitization; cardiorenal benefits; several other disease opportunities

* Thiazolidinediones (glitazones) – operate via PPARγ and non-genomic pathways including MPC (mitochondrial pyruvate carrier).

Robust Mid-to-Late Stage Metabolic Pipeline

	Indication	MOA	Discovery/PC	PH 1	PH 2	PH 3	NDA review	Partner/ Rights	Upcoming Milestones
Type 2 Diabetes (T2D)									
Imeglimin Japan / Asia[1]	T2D	MRC Modulator						Sumitomo Dainippon Pharma	▪ Target approval mid-2021 in JP ▪ Target product launch in 2021[2]
Imeglimin US / EU / Other	T2D with CKD stages 3b/4	MRC Modulator						poxel	▪ Exploring options to move the program forward into Phase 3
NASH									
PXL770	NASH with T2DM	AMPK Activator						poxel	▪ Initiate Phase 2b study in 2H 2021
PXL065	NASH	Non-Genomic TZD						poxel	▪ Phase 2 results mid-2022 ▪ 505(b)(2) pathway
PXL007 (EYP001)	Hepatitis B / NASH	FXR Agonist						ENYO PHARMA	▪ Complete Ph 2a program by Enyo Pharma mid-2021
Other Chronic and Rare Metabolic Indications									
Next-Gen AMPK	ALD/AMN, other	AMPK Activator						poxel	▪ Complete PC studies in 2021 ▪ Select lead candidate(s)
Next-Gen D-TZD	ALD/AMN other	Non-Genomic TZD							

1. Including: China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos. 2. Sumitomo fiscal year April-March.

Type 2 Diabetes

Imeglimin

Key Partnership for Japan & Asia

Sumitomo Dainippon Pharma

First in a New Class of Potential Anti-diabetic Therapies with a Differentiated Mechanism of Action

Expect Regulatory Approval/Launch in Japan in 2021

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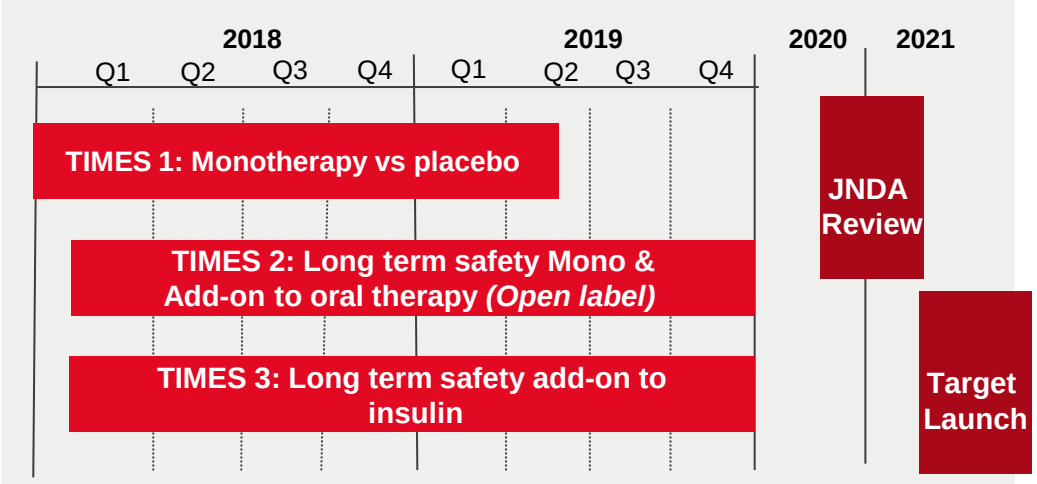
Imeglimin: Novel Mechanism - Nearing Approval in Japan

Partnered in Asia[1] with Diabetes Market Leader, Sumitomo Dainippon Pharma

- Successful Completion of Phase 3 Program in Japan
- J-NDA approval triggers **milestone payment of ~€13.8M ($16.9M)**[2] and ability to draw down **€13.5M** from IPF loan
- **Target launch expected in 2021**[3]; Future potential **development milestone payments and sales-based payments** of up to **approx. $237M**[4] and double-digit escalating royalties



Business Opportunity Japan: Maximize Product Profile

- Sumitomo #1 diabetes franchise; Guidance **FY20 $900M**[3]
- DPP4i's are prescribed to 80% T2D patients[5]
- Limited treatment options for selected populations, including elderly and patients with renal impairment
  - *elderly patients account for ~60% of T2D in Japan*
- TIMES program observed to show ***robust efficacy with favorable safety and tolerability profile***

1. Including: Japan, China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos.
2. Based on the JPY/€ exchange rate at December 31, 2020.
3. Sumitomo fiscal year April-March.
4. Currency exchange rate is at the date of the agreement.
5. IQVIA data FY2016 and NDB data FY2016.

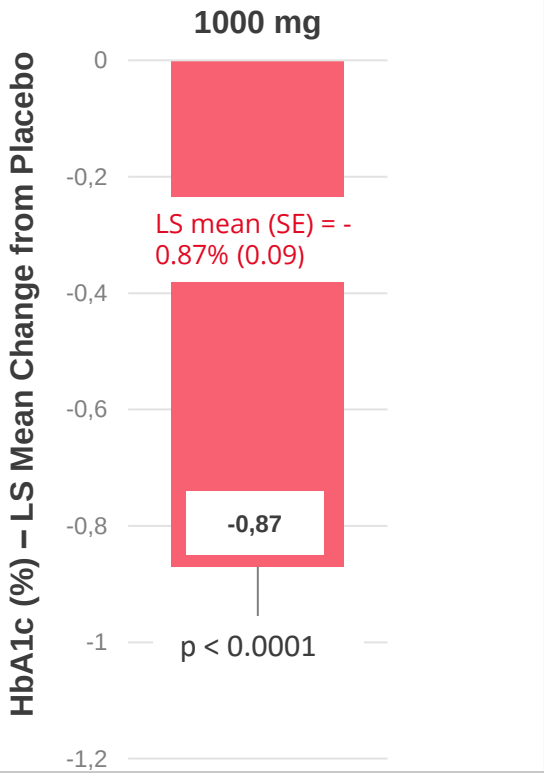
Imeglimin Phase 3 TIMES Program Overview (N=1,142)

Robust and Consistent Efficacy in Monotherapy and as an Add-on Therapy

TIMES 1* Monotherapy

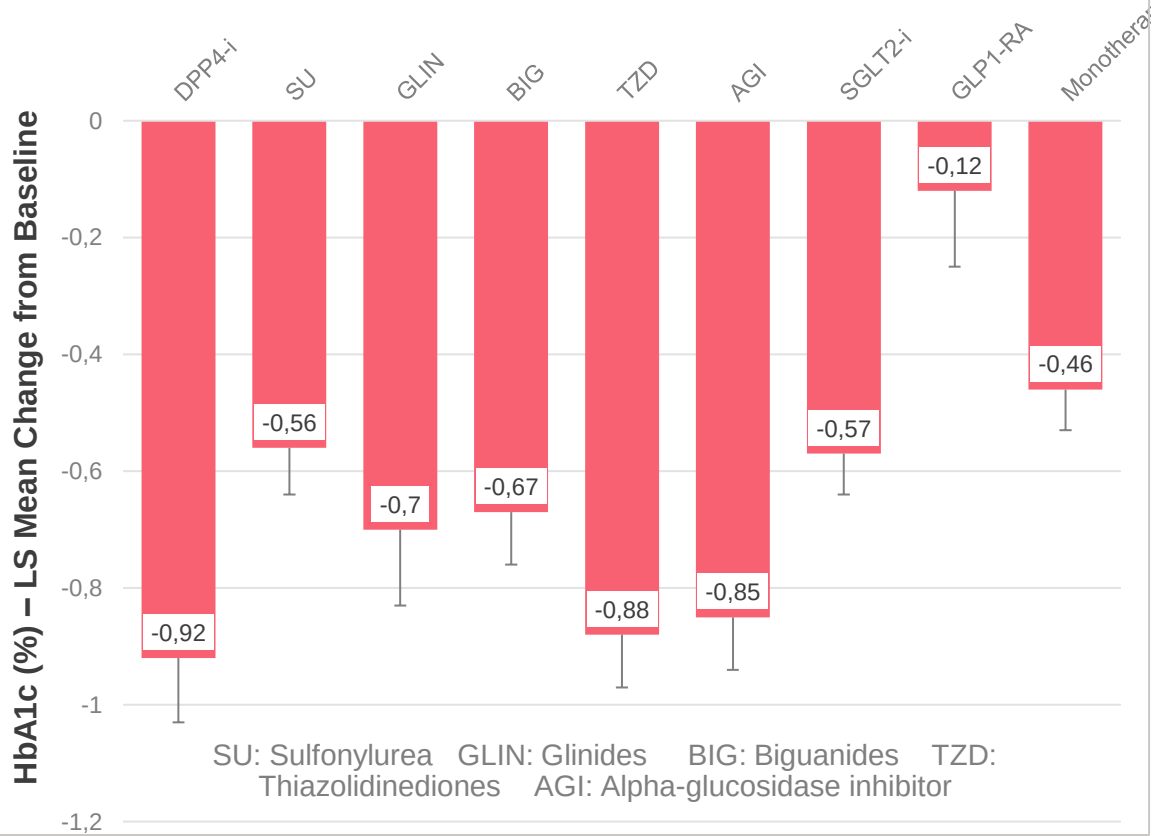
Change in HbA1c – 24 Weeks

Patients (n)	Placebo (N=107)	Imeglimin (N=106)
HbA1c (%), mean (SD)	7.93 (0.684)	7.99 (0.764)



TIMES 2 As an Add-on to Standard of Care

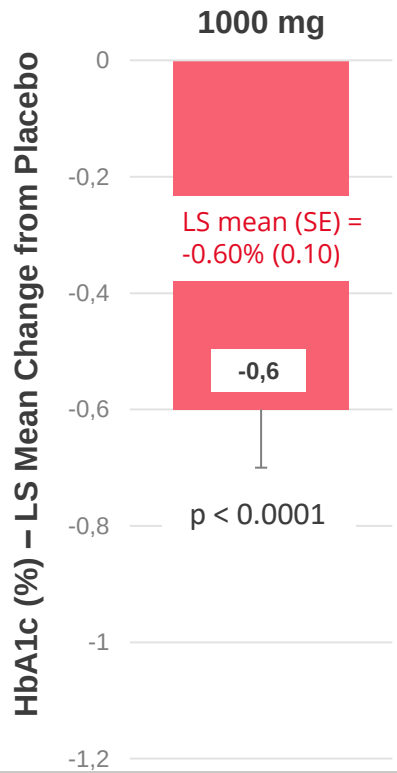
Change in HbA1c (vs baseline) – 52 Weeks – 714 patients



TIMES 3 Combination with Insulin

Change in HbA1c – 16 Weeks

Patients (n)	Placebo (N=107)	Imeglimin (N=108)
HbA1c (%), mean (SD)	8.8 (0.8)	8.7 (0.7)



*European Association for the Study of Diabetes meeting 2019.

Business Opportunity for Imeglimin in US, EU, Other Countries[1]

- Data, materials, information, IP, and FDA regulatory filings transferred from Metavant[2] to Poxel
- Exploring options to pursue for **T2D patients with chronic kidney disease stages 3b/4** (CKD 3b/4)

Commercial opportunity

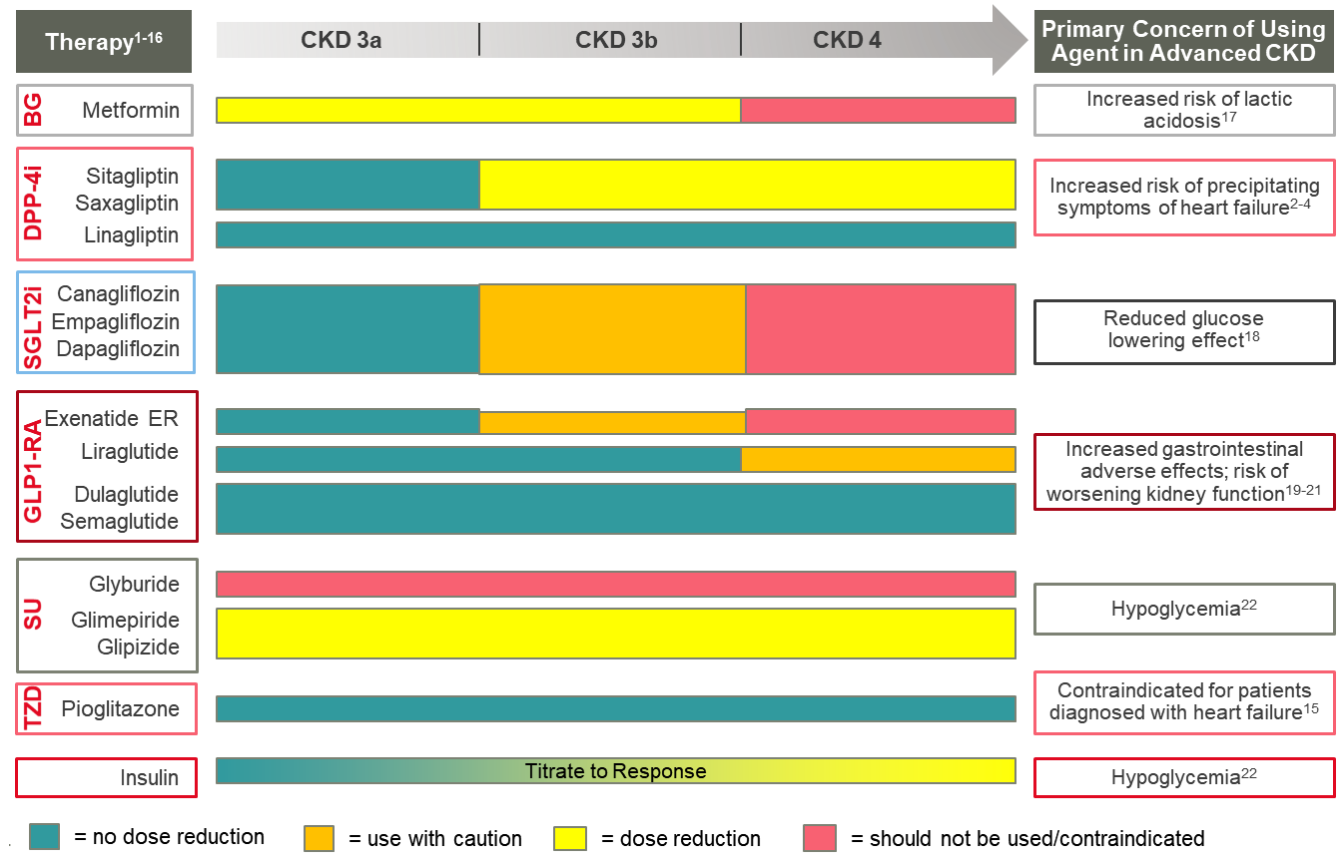
- Diabetes is the most common cause of CKD
- 10% of T2D patients / ≈2.4 million adults in U.S.[3]
- Underserved patient population

Clinical Evidence

- PK/PD data in CKD3b/4 Caucasian patients, showing adequate PK profile, good safety and efficacy profile
- Phase 3 data in JP confirming efficacy and safety profile in CKD3
- Safety database of >2500 patients

Path to Approval discussed with FDA

- End of Ph2 meeting completed 2020
- Pivotal and supportive trials discussed with FDA
- Ability to leverage Phase 3 data in JP to ensure sufficient patients exposure



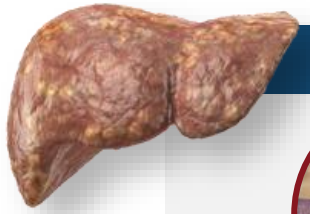
1. Not covered by the Sumitomo Dainippon Pharma agreement. 2. A subsidiary of Roivant Sciences. 3. Centers for Disease Control and Prevention (CDC).NHANES; 2015-2016; 4-22 – see Appendix.

NASH Programs

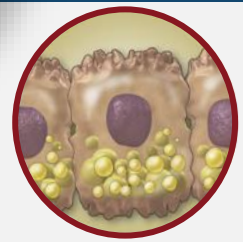
PXL770 - Direct AMPK Activator

PXL065 – Deuterium-stabilized *R*-pioglitazone

PXL770 and PXL065: NASH Value Proposition



HALLMARKS OF NASH

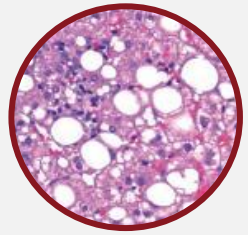


Lipid accumulation in hepatocytes

Steatosis

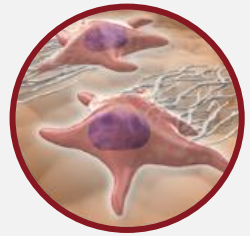
Immune cells (macrophages - MΦ)

Inflammation



Cellular damage-death

Ballooning



Hepatic stellate cell activation

Fibrosis

- **First-in-Class - Novel Mechanisms**
  - ability to target multiple hallmarks of NASH
  - high unmet need; large market opportunity
- **Clinical validation**
  - positive Phase 2A results (‘770)
  - derived from pioglitazone – proven NASH benefits (‘065)
- **Daily oral administration**
  - combinable with other approaches
- **Differentiated development approaches**
  - potential to also treat co-existing diabetes
  - streamlined development - 505(b)(2) regulatory path (‘065)

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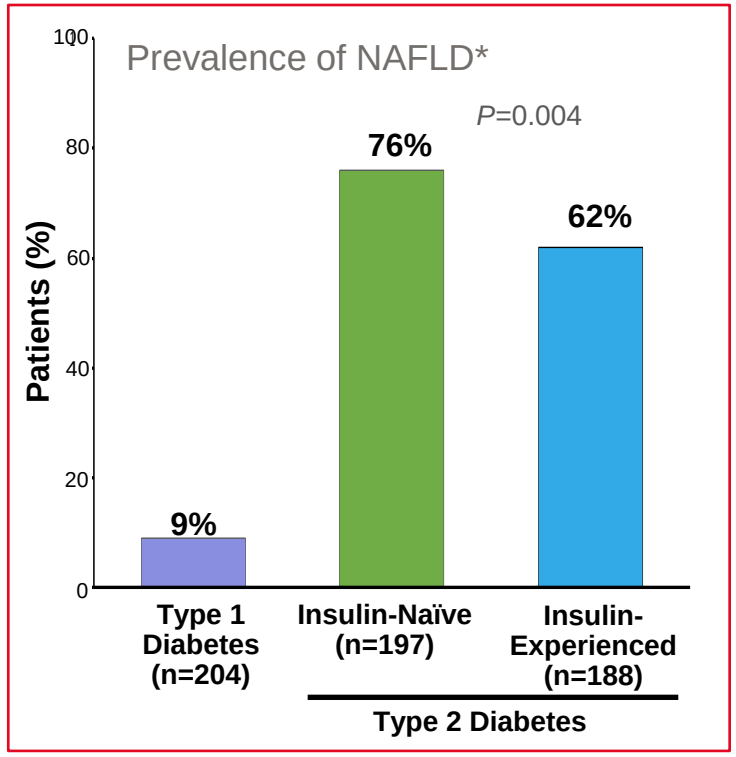
NASH and Type 2 Diabetes – Strong Clinical Overlap

NASH with T2D - High Prevalence and Greater Unmet Medical Need

- Approximately 40-50% of NASH patients have coexisting T2D[1]
- High prevalence of NAFLD (>60-70%) and NASH (26%) in T2D patients[2,3]



*NAFLD ≥ 6% hepatic fat fraction by MRI; data based on post-hoc analysis from 4 Phase III trials (n=589)

- Insulin resistance greater in patients with both NASH and T2D vs. either alone[4-6]
- 15% of patients with T2D have undiagnosed clinically significant fibrosis (F2-F4)[7]
- Clinical burden of NASH in patients with T2D greater than broader NASH population[1,6,8]
  - Progression of fibrosis
  - Worse CVD morbidity and mortality
- Economic burden for the group with prevalent NASH and T2D estimated $642 billion[8]

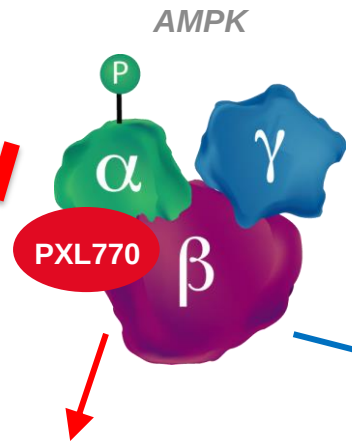
1. Younossi ZM et al; *Hepatology* 2016.
2. Cusi et al, *Diabetes Obes Metab.* 2017.
3. Portillo/Cusi et al, *J Clin Endocrinol Metab 2015.*
4. Cusi K, *Diabetes Care* 2020.
5. Bril/Cusi et al, *Hepatology* 2017.
6. Gastaldelli A & Cusi K, *JHEP Reports* 2019.
7. Lomonaco/Cusi, *Diabetes Care* (in press, 2021).
8. Younossi ZM et al, *Diabetes Care* 2020.

PXL770 is a Direct AMP Kinase Activator

Mechanism, Preclinical Profile, Phase I Summary

Overnutrition (metabolic syndrome, NASH, Type 2 Diabetes)

Caloric Restriction, Exercise



***Activates* catabolic pathways**

↑ fatty acid oxidation
↑ glucose uptake

***Inhibits* anabolic pathways**

↓ de novo lipogenesis (DNL)
↓ adipose tissue lipolysis

***Other* benefits**

- Reduces inflammation
  - ↓ macrophage and dendritic cell activation
  - ↓ pro-inflammatory cytokines
- Blocks hepatic stellate cell activation

Phase I Clinical Summary:

- 132 healthy subjects; good tolerability, low incidence of AE's; acceptable PK
- Ph1b NAFLD study (n=20; 4 weeks): evidence of target engagement (suppression of DNL); improved glucose tolerance; insulin sensitization

AMPK - potential to target core drivers of NASH and to improve key cardiometabolic risk factors

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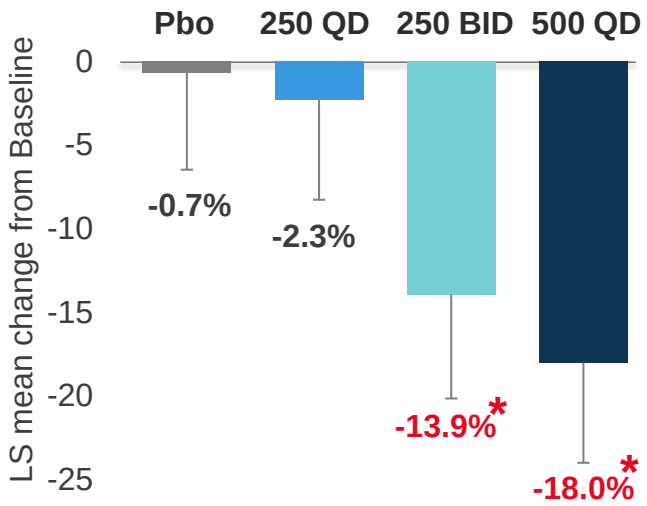
PXL770 Successful Phase 2a Results

Statistically Significant Results and Greater Efficacy in Patients with Diabetes♦

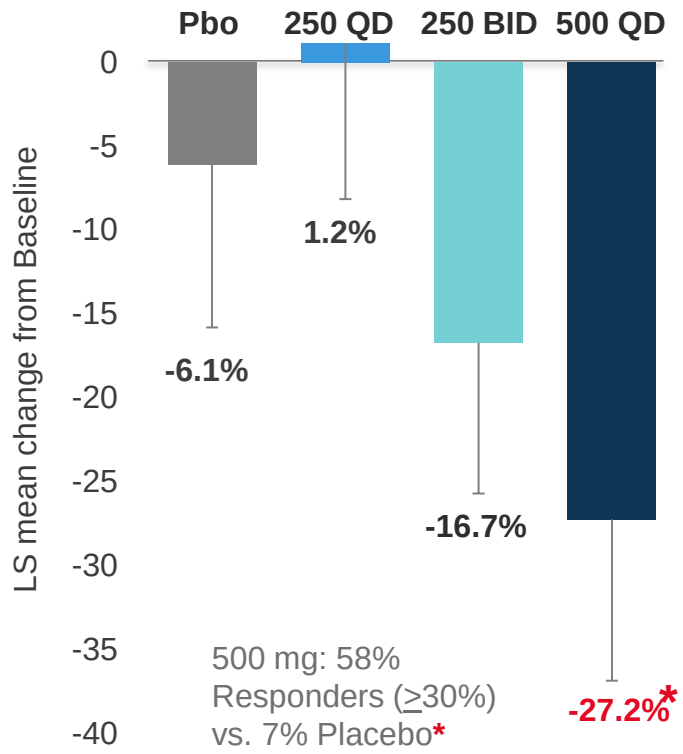
Liver Fat Content *(% Change from baseline)*

NAFLD with / without T2DM



500 mg: significant ALT reduction*

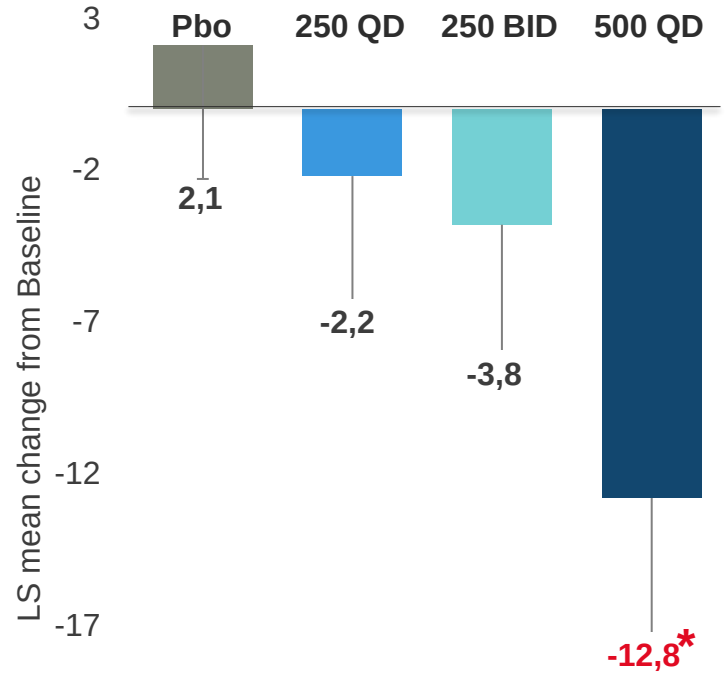
T2D subgroup



500 mg: 58% Responders (≥30%) vs. 7% Placebo*

ALT *(IU/L Change from baseline)*

T2D subgroup



500 mg: significant AST reduction*

♦ 41-47% of each cohort

*p values 0.044-0.0036

Meaningful Glycemic Benefits in Patients with (Well Controlled) T2D:

- ***-0.64% HbA1c*; -21 mg/dL* fasting glucose vs. placebo***
- ***evidence of insulin sensitization***

PXL770 Profile

Phase 2A Efficacy Results (in T2D Subgroup) vs. Selected Oral Competitors#

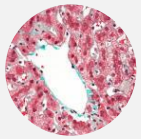
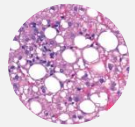
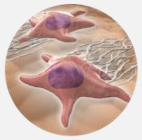
	PXL770◊ T2DM	Galmed Aramchol[1]	Madrigal Resmetirom[2]	Viking VK2809[3]	Intercept OCA [4,5]	Enanta EDP-305[6]	Metacrine MET409[7]
	AMPK	SCD1	THR-β	TR	FXR	FXR	FXR
Relative % LFC decrease vs. baseline	**-27.2**	**-12.6**	**-32.9**	**-53-60**	**-**	**-30.5**	**-37-55**
Relative % LFC decrease vs. placebo	**-21.1**	**-20**	**-22.5**	**-40-50**	**-17[3]**	**-18.6**	**-31-49**
Decrease in ALT (IU/L) vs. placebo	**-14. 9**	**-8.6***	**-3.0***	**-6.2***	**No change[4]**	**-12.5**	**-**
Decrease in HbA1c (%) vs. placebo	**-0.64**	**No effect**	**No effect**	**?**	**?**	**?**	**?**
Potential liabilities	**Mild GI**		**Mild GI**	**Potential QOD Dosing**	**Pruritus ↑LDL BBW for liver failure**	**Pruritus ↑LDL**	**Pruritus ↑LDL CYP3A4 inhibition**

1. Safadi R et al Clin Gastro & Hep 2014 (12 week Ph2a)
2. 12 week results; Tables 2,4 – Harrison SA et al. Lancet 2019 https://doi.org/10.1016/S0140-6736(19)32517-6;
3. Viking Corporate Presentation AASLD 2019 [12 week results]
4. Intercept presentation & Gastroenterology 2019;156:88–95. ALT in FLINT trial at 12 wks; MRI-PDFF results in smaller cohort from FLINT trial (40 pts treated with OCA)
5. Mudaliar S et al. Gastroenterology 2013;145:574–582 [6 week Ph2 study *in NAFLD pts with T2DM*]
6. Enanta presentation – 21% discontinuation due to "pruritus generalized" at 2.5 mg dose
7. Metacrine 2020 EASL poster presentation – 50/80 mg 12 wk results; net increase ALT with 50 mg at 12 wks vs decrease ALT with 80 mg; 16-40% pruritus; 24% increase LDL at 80 mg

◊ 500 mg QD group
 # Competitor data for 12 week treatment time points (except where noted if not available)
*** Not stat significant or stats not reported**

PXL770 - Translation of AMPK Activation Approach

Remaining Hypotheses to be Addressed in Phase 2b

	Rodent (*in vivo*)	Human Cells (*in vitro*)	NASH / NAFLD Patient
 **Steatosis**	✓ ↓ steatosis score; ↓ liver lipids; ↓ de novo lipogenesis	✓ ↓ de novo lipogenesis	✓ ↓ de novo lipogenesis; ↓ liver fat mass
 **Inflammation**	✓ ↓ inflammation score; ↓ liver leukocytes; MCP1 (+ other)	✓ ↓ cytokine secretion (macrophage)	*Pending Phase 2b*
 **Ballooning**	✓ ↓ ballooning score	no model	✓ ↓ ALT / AST *Pending Phase 2b*
 **Fibrosis**	✓ ↓ fibrogenesis	✓ ↓ stellate cell activation	*Pending Phase 2b*
↓ Insulin Resistance	✓ improved OGTT; ↑ glucose infusion rate (clamp) ↓ HbA1c	✓ ↑ glucose uptake (muscle cells)	✓ improved OGTT, HOMA-IR, Matsuda; ✓ ↓ HbA1c

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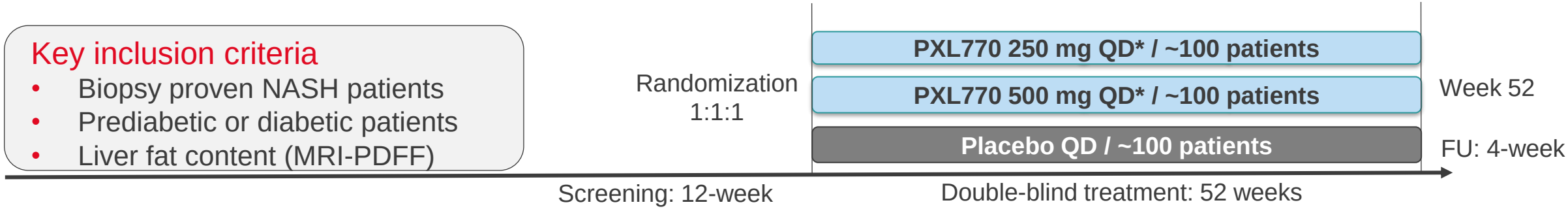
PXL770 – Safety, Conclusions, Next Steps

- First direct AMPK activator studied in human disease – results support progression of development and pursuit of other indications
- Well tolerated, with acceptable safety profile
- Target engagement established with improvements in multiple NASH-related parameters
- Greater response in patients with T2D - opportunity to target a large (45-50%) high risk subpopulation
  - consistent with lower endogenous AMPK “tone” hypothesis
  - additional glycemic benefits with improved insulin sensitivity
- Planning underway for initiation of NASH Phase 2b – focusing on T2D patients



*new tablet formulation - human PK confirmed higher/optimized exposure

NASH

PXL065

Proprietary Program

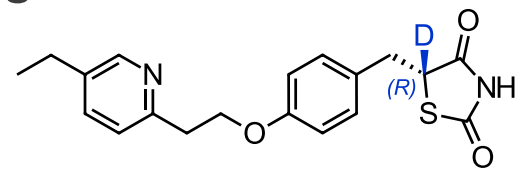
Non-Genomic Pathway D-TZD Modulator for Treatment of NASH

Utilizing the 505(b)(2) Regulatory Pathway

PXL065: Leveraging the Benefits of Pioglitazone

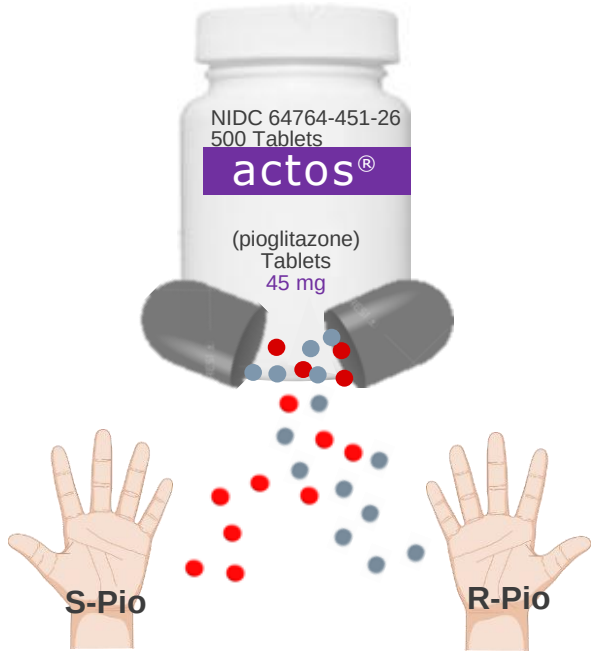
With Reduced PPARγ Activity

- Pioglitazone used in T2D[1,2] – most extensively studied molecule in NASH – multiple trials[3]
  - Recommended for NASH by AASLD & EASL Practice Guidelines[4]
  - Currently prescribed by ~14% of physicians for biopsy-proven NASH patients[5]
  - Limited use due to PPARγ-related side effects: weight gain, fluid retention, bone loss
- Pioglitazone is a mixture of 2 stereoisomers with dramatically different properties
- PXL065 is the deuterium-stabilized R-stereoisomer



S-Pioglitazone (stabilized)

- Strong PPARγ agonist
- Undesired side effects:
  - Weight gain
  - Fluid retention



PXL065 (stabilized R-pio)

- Very weak PPARγ agonism
- Operates via non-genomic pathways*
- Retains NASH activity in models

Composition of Matter IP

505(b)(2) Regulatory Path

1. Takeda 2014. https://www.takeda.com/newsroom/newsreleases/2014.
2. Diab Vasc Dis Res. 2019, 16(2), 133-143.
3. Ann Intern Med. 2016, 165(5), 305-315.
4. J Hepatol. 2016, 64(6),1388-402; Hepatology 2018, 67, 328-357.
5. Therap Adv Gastroenterol. 2016, 9(1), 4-12.

* Including inhibition of MPC – mitochondrial pyruvate carrier.

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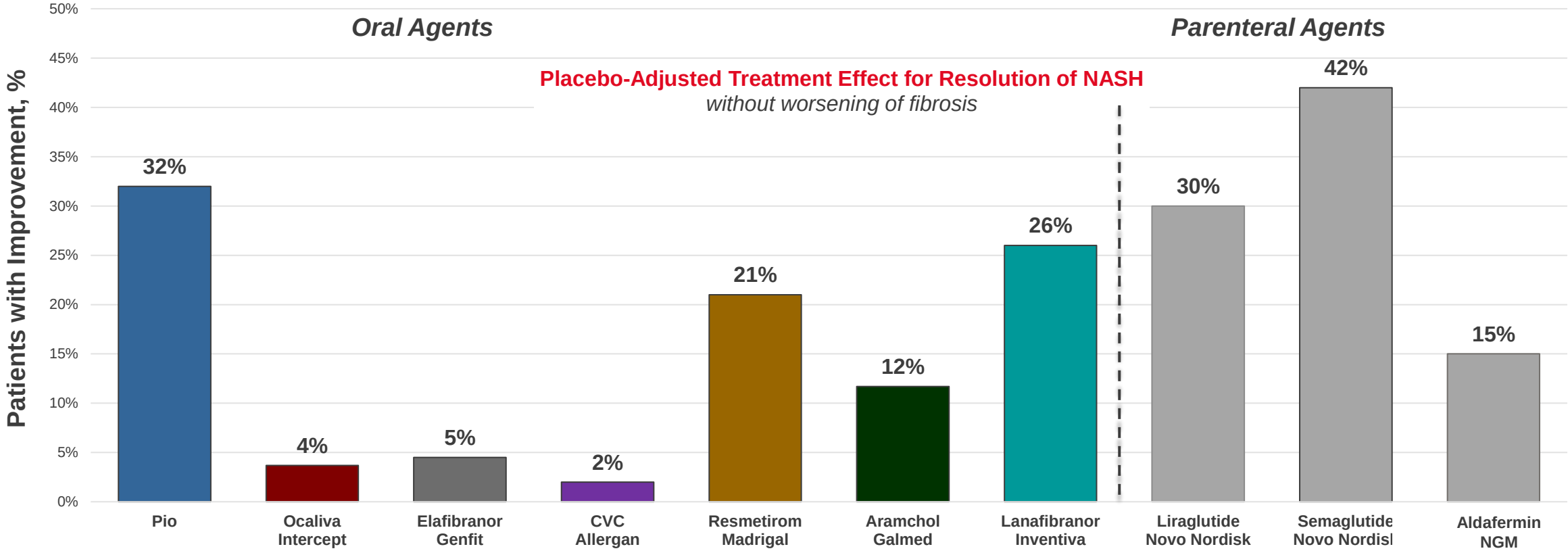
Pioglitazone Demonstrated Strong Efficacy in NASH Trials

Comparison vs. Other Agents in Development



NOTE: No head-to-head trials conducted.
Pio Cusi Phase 4 trial (30➝45 mg, 18 mos) - Ann Intern Med. 2016, 165, 305-315.
Ocaliva REGENERATE Phase 3 trial (25 mg, 18 mos) - Lancet. 2019, 394, 2184-2196
Elafibranor RESOLV-IT Phase 3 trial (120 mg, 52 wks/) – Press release May 11, 2020
CVC (Cenicriviroc) CENTAUR Phase 2 trial (150 mg, 2 yrs) – Hepatology 2020, Jan 13 epub
Resmetirom (MGL-3196) Phase 2 trial (80 mg +/- 20 mg, 36 wks) – Lancet 2019 394:2012-24.

.
Aramchol Phase 2 trial (600 mg, 52 wks) – press release June 12, 2018. .
Lanafibranor Phase 2 trial (1200 mg, 24 wks, ITT population) – Press release Jun 15, 2020
Liraglutide Phase 2 trial - The Lancet, 2016, 387(10019), 679–690
Semaglutide Phase 2 trial (0.4 mg, 72 wks) – Newsome et al NEJM Nov 19, 2020
Aldafermin (NGM282) Phase 2 trial (1 mg, 24 wks, cohort 4) - Press release Feb 25, 2020.

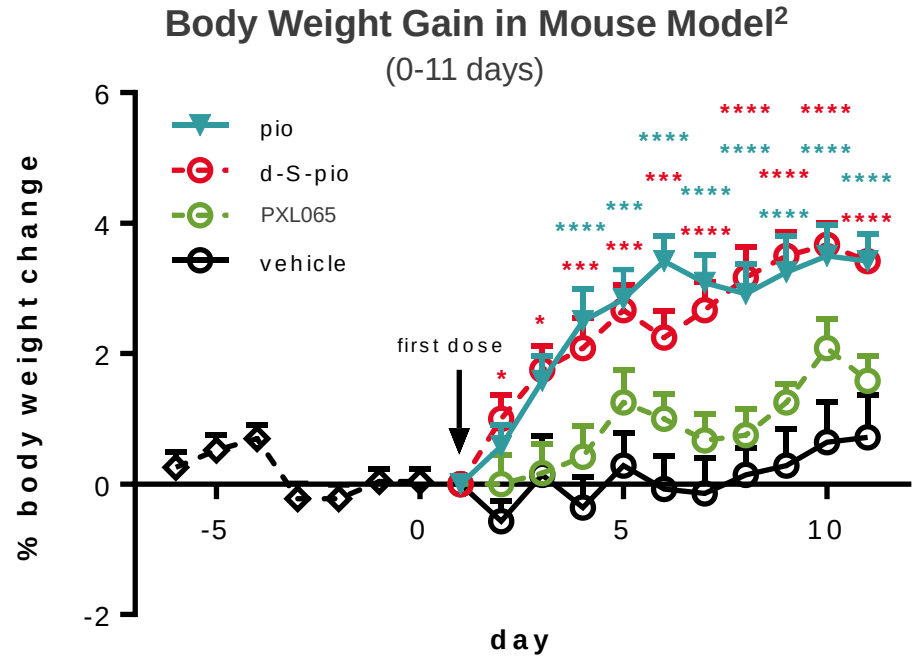
Meta-analysis OR >10 for improvement in advanced fibrosis[1]

1. Musso et al. Hepatology 65:1051-68, 2017.

PXL065 Profile in NASH Preclinical Models

PXL065 (R-Pio) Retains Benefits of Pio; S-Pio Drives Weight Gain and Fluid Retention

NASH Rodent Models[1] Functional Parameters	Pio	PXL-065
↓ Hepatic Triglycerides	✓	✓
↓ Hepatic Free Fatty Acids	✓	✓
↓ Hepatic Cholesterol	✓	✓
↓ Hepatic Steatosis	✓	✓
↓ Hepatic Inflammation	✓	✓
↓ Hepatic Ballooning	✓	✓
↓ Hepatic Fibrosis	✓	✓
↑ Weight Gain	✓	-
↑ Fluid Retention	✓	-

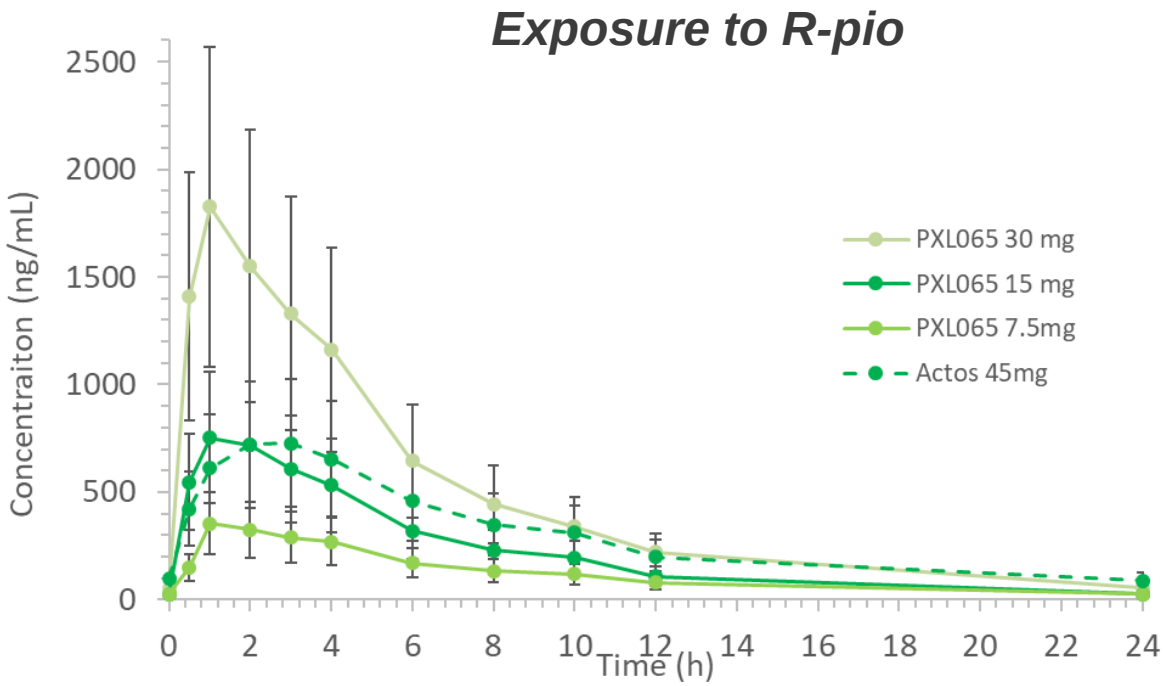


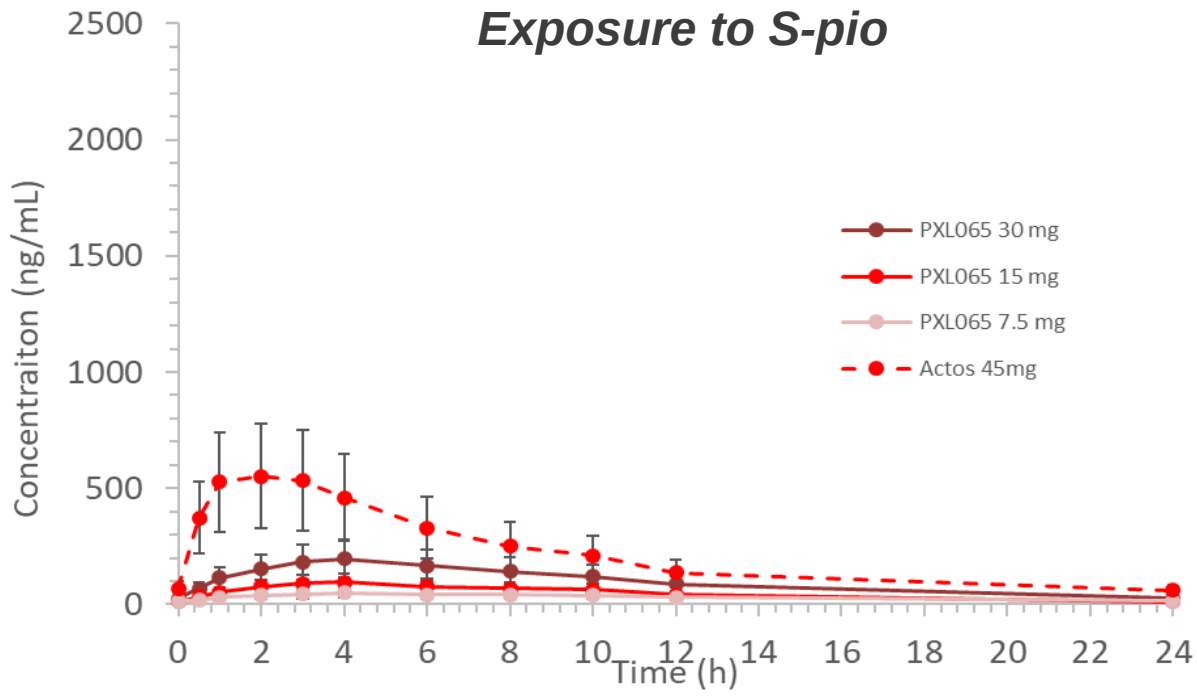
1. NASH rodent models selected based on literature: *C57BL/6J mouse model of weight gain & edema* (Nat Med 2005, 11, 861-866) and methionine-choline deficient (MCD) model of NASH (Lab Investig. 2007, 87, 56-65). Additional choline deficient (CD) model of NASH was validated with RenaSci. In MCD model both pio and PXL065 reduced ballooning. d-S-pio was only run in the CD model where no effect on ballooning with any compound was observed.

2. Weight gain measured in C57BL/6J mouse model. Pioglitazone dosed at 30 mg/kg, d-S-pio and PXL065 dosed at 15 mg/kg. Statistical significance determined by 1-way (total day 11) or 2-way (% by day) ANOVA with Dunnett's post-test average ± SEM; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $P < 0.0001$.

PXL065 Ph1 Study Results

15 mg vs. 45 mg Actos®[1]: Similar R-Pio Exposure; S-Pio Exposure Decreased ~5-fold





- Single (SAD) and repeated (Phase 1b) oral dose studies completed
- Stabilization and sustained higher exposure to R-pio (limited conversion to S-pio)
  - PK dose proportionality; no food effect
  - Tablet formulation qualified in Phase 1b study
- Well tolerated at all doses tested

AASLD
AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASES

1. Actos is the branded version of pioglitazone and a registered trademark of Takeda Chemical Industries, Ltd.

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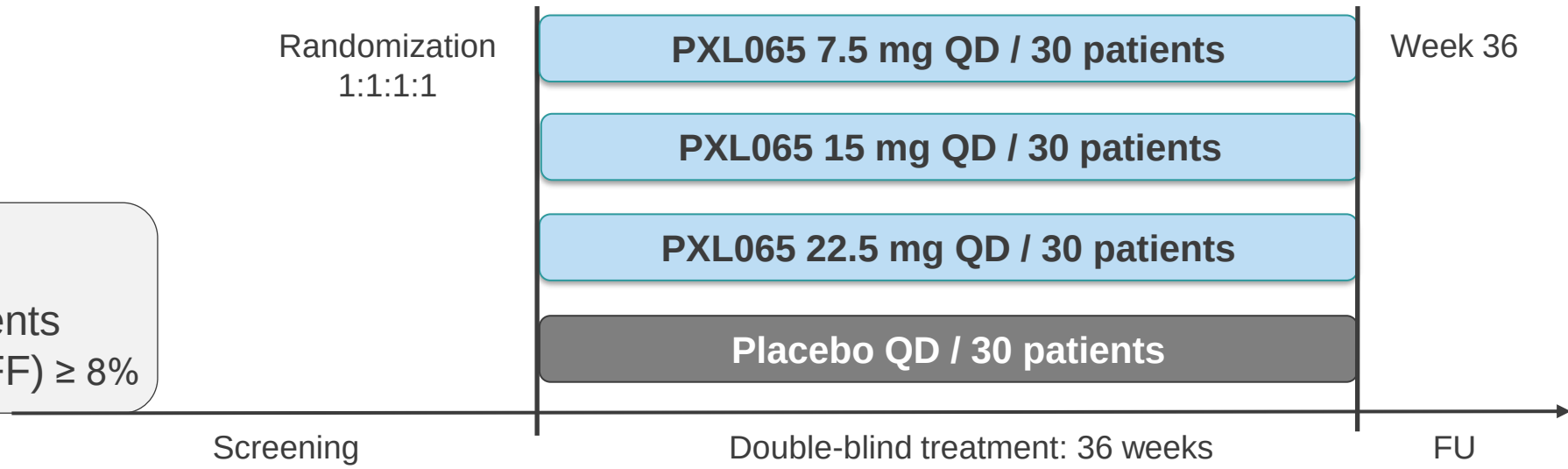
PXL065 Ongoing Phase 2 in Biopsy-Proven NASH Patients

Single Streamlined Study - 505(b)(2) Pathway; Designed to Select Ph3 Dose(s)

DESTINY 1

Key inclusion criteria

- Biopsy-proven NASH patients
- Liver fat content (MRI-PDFF) ≥ 8%



Primary Endpoint

- Relative change in liver fat content (MRI-PDFF)

Secondary Endpoints

- Liver histology: NASH resolution without worsening of fibrosis
- Liver enzymes
- Metabolic parameters
- Biomarkers, Safety, PK

Additional Opportunities

Pipeline Expansion

Chronic and Rare Metabolic Indications

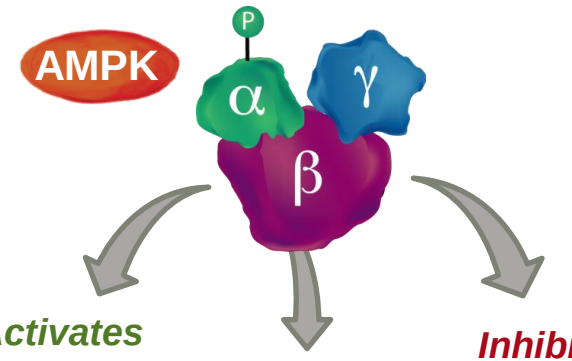
Next Generation AMPK Activators

Next Generation D-TZD's*

*Deuterium-modified thiazolidinediones.

Harnessing AMPK and D-TZD Platforms to Address Diseases with Metabolic Pathophysiology

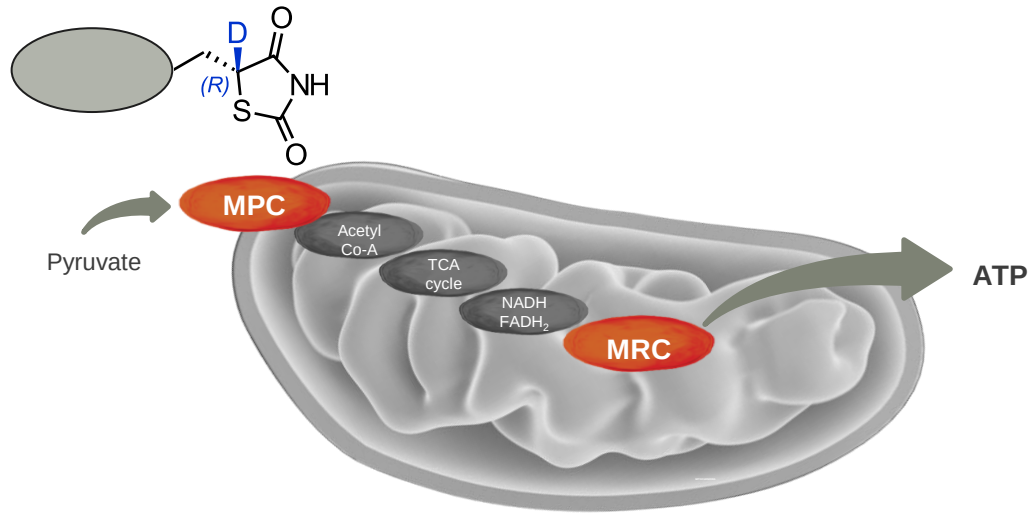
Next Generation Programs Approaching Clinical Development (Both Platforms)



Activates ***catabolic pathways***

Inhibits ***anabolic pathways***

Inhibits ***inflammation apoptosis***



Renal

Diabetic kidney disease

Polycystic kidney disease

Endocrine

Type 2 diabetes

Polycystic ovary syndrome

Rare Metabolic

Adrenoleukodystrophy (ALD, AMN)

Mitochondrial disorders

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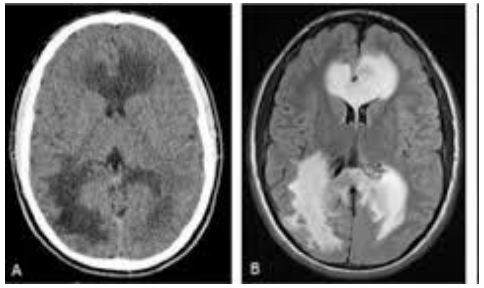
Adrenoleukodystrophy

Orphan Neurometabolic Disease with High Unmet Need

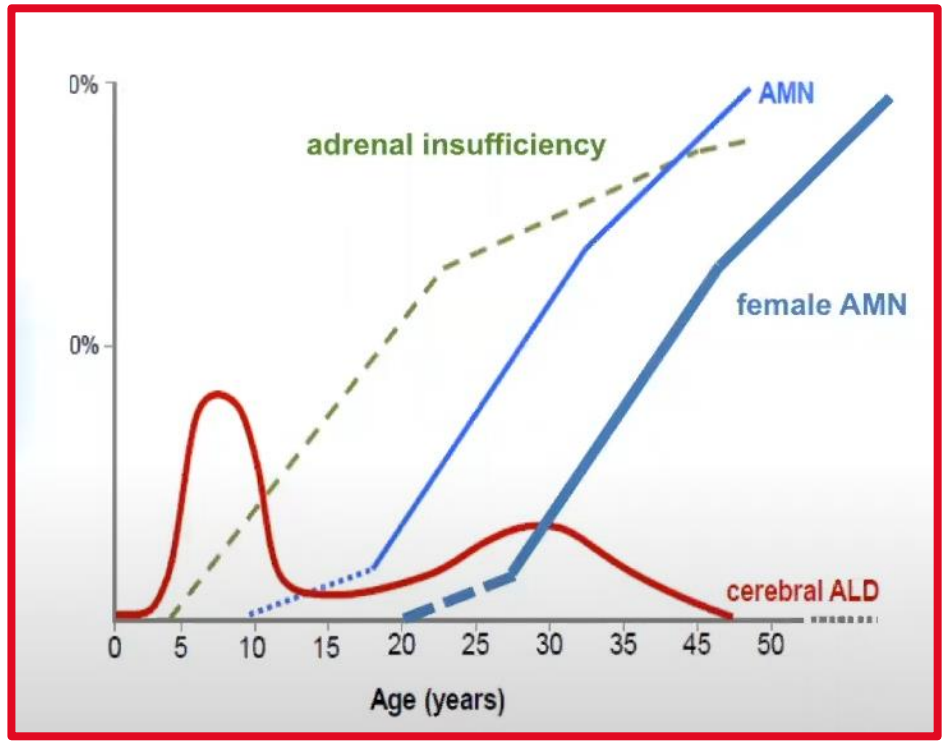
ALD AMN

- X-linked mutations in ABCD1 gene; defective metabolism of long chain fatty acids (VLCFA) leads to inflammation; cellular-myelin damage
  - ≈10,000-20,000 patients in U.S.*; increasing diagnosis based on newborn screening
- Two overlapping syndromes – cerebral adrenoleukodystrophy (C-ALD); adrenomyeloneuropathy (AMN) - plus adrenal insufficiency
- No approved therapies; hematopoietic stem cell transplant (HSCT) often used in early C-ALD

C-ALD Lesions (MRI)



- *Addison's disease*
- *Damage to brain white matter; cognitive impairment; loss of vision/hearing; impaired balance-movement; death*
- *Slowly progressive; impaired gait-balance-movement; bladder-bowel dysfunction; also affects women*



*https://rarediseases.org/rare-diseases/adrenoleukodystrophy

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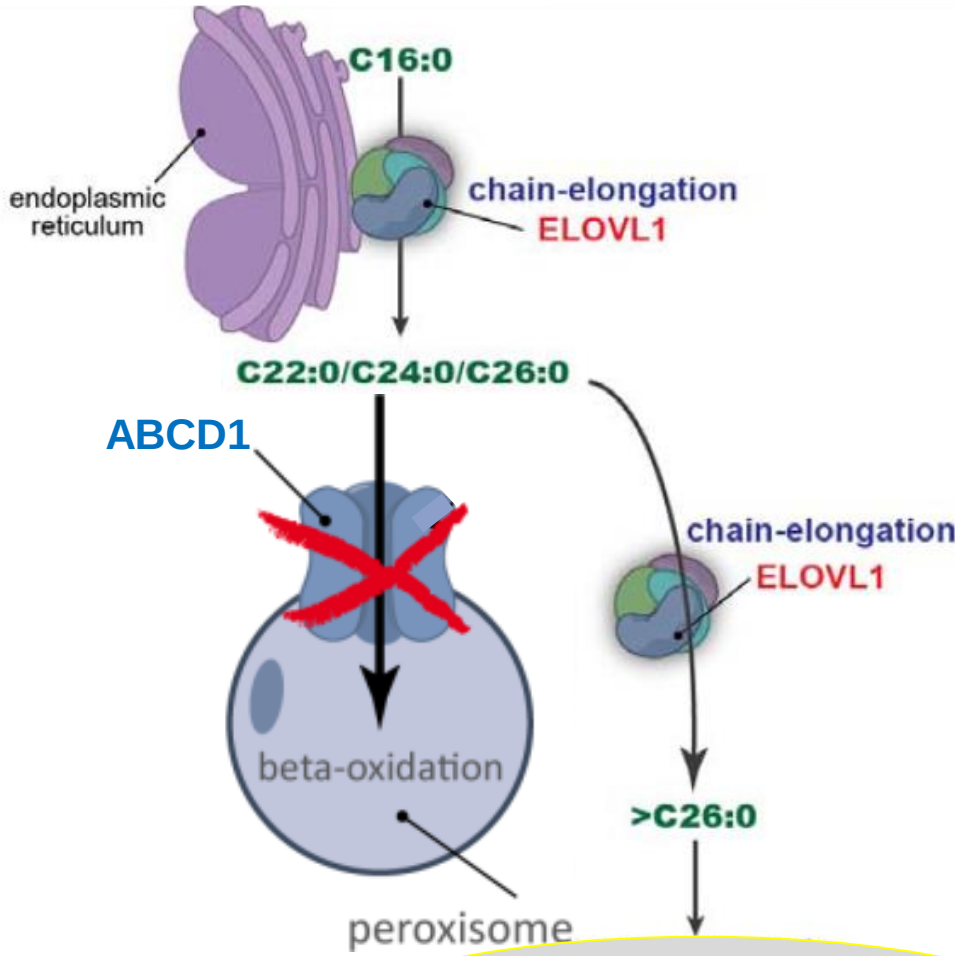
Molecular Pathophysiology

Potential Benefits of D-TZDs or AMPK Activators

ALD AMN



VLCFA accumulation:, inflammation, mitochondrial dysfunction, cell death
Axonal Degeneration

ABCD1: Transports VLCFA into peroxisome for degradation (ABCD2 can serve as an alternative peroxisomal transporter)[1]

ALD / AMN:
Defective ABCD1 leads to accumulation of VLCFA in tissues

High VLCFA levels disrupt cell membranes: mitochondrial dysfunction, reactive oxygen species, inflammatory demyelination in brain, motor neuron deterioration

D-TZD's and AMPK activators: modulate lipid metabolism and mitochondrial function, mediate anti-inflammatory effects; inhibit apoptosis

VLCFA – very long chain fatty acid, Graphic adapted from:
https://adrenoleukodystrophy.info/mutations-biochemistry/origin-and-metabolism-of-vlcfa

1. Hum Mol Gen 2004; 13:2997-3006; Biomed Res Int 2016: 6786245

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D-TZD's: Clinical Results Support Pursuit of ALD/AMN

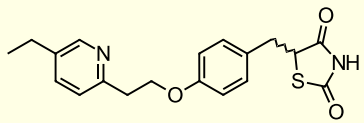
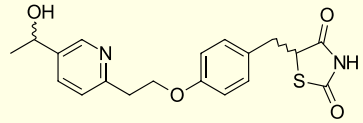
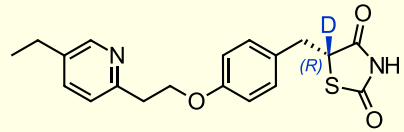
ALD AMN

Leriglitazone - Human PoC with PPARγ - Related AEs

- Phase 2/3 trial in adult AMN patients (n=116; 96 week)[Δ]
- Primary Endpoint: 6 min walk test - *Failed (*differences "observed in early symptomatic pts")
- Secondary / Exploratory: Body Sway - *Significant (p=0.036; p=0.003) improvements*

  SSPROM & EDSS – *Positive effect*

  Cerebral ALD - *Positive effect*

	 **Pioglitazone**	 **Leriglitazone (MIN-102)**	 **PXL065**
MoA	PPARγ agonist & Non-genomic effects (MPC, other)*	PPARγ agonist & MPC inhibition**	Minimal PPARγ activity Non-genomic effects (MPC, other)*
Relationship to Pio	Parent molecule	M-IV metabolite of Pio	R-Pio (1/2 of pio mixture)
Known or expected side effects PPARγ)	weight gain (≈3 kg), edema, & risk of bone fracture	weight gain (5.8 kg[Δ]), edema[Δ]	No significant PPARγ–related side effects expected

PXL065 and other D-TZD's:
Potential for superior efficacy with reduced side effects

[Δ]Minoryx press release Feb. 2021; Am Acad Neurology 2021 presentation; *Both Pio isomers have similar mitochondrial pyruvate carrier (MPC) activity; **in-house data and results reported in Minoryx patent WO 2019/234690

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D-TZD's: Additional Rationale and Strong Preclinical Data

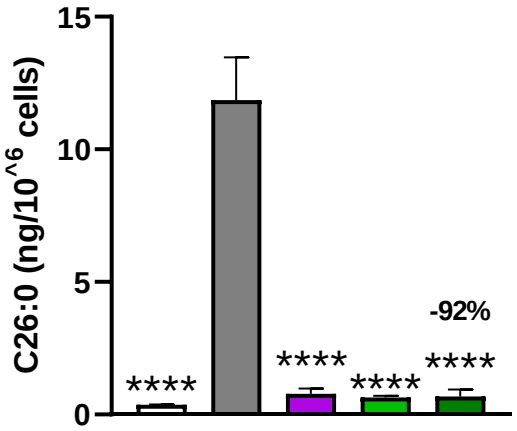
ALD AMN

- Pioglitazone attenuates neuroinflammation and confers neuroprotection:
  - non-human primates with Parkinson's disease[1]
  - rodent acute brain ischemia[2], spinal cord injury[3]
- Efficacy achieved in ABCD1-null mice with Pioglitazone[4]
- MPC inhibition implicated as a therapeutic approach in neurodegeneration[5,6]
- *PXL065 is active in ALD/AMN patient-derived cells and in ABCD1 null mice:*

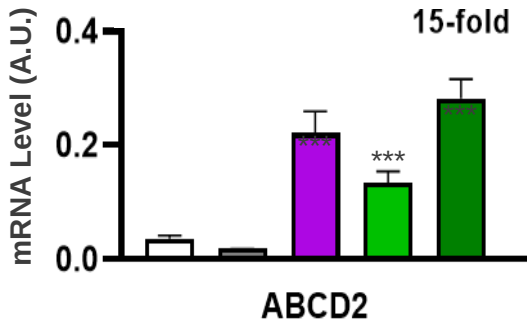
1. J Neuroinflamm 2011; 8:91
2. Exp Neurol 2009; 216:321-
3. Exp Neurol 2017; 293:74-
4. Brain 2013;136:2432-43
5. Sci Trans Med 2016; 8:368ra174
6. Neural Regen Res 2017;12:1807-8

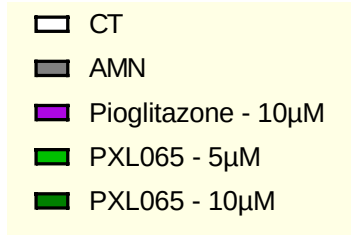
AMN – Fibroblasts Suppression of Elevated VLCFA

N=6-8



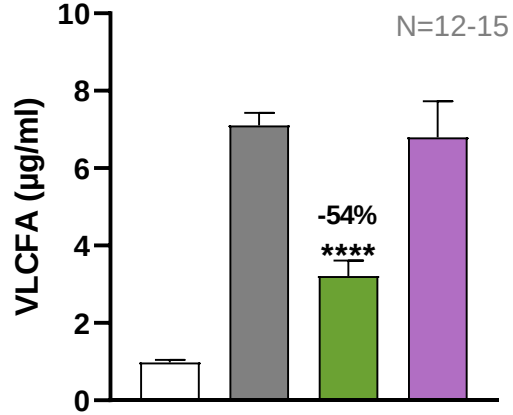
AMN – Fibroblasts Compensatory Transporter Gene Expression





ABCD1 Null Mouse Model Spinal Cord Suppression of Elevated VLCFA (C26:0)

N=12-15



***p<0.01, ****p<0.001

Next Steps – including clinical development plans – under finalization

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AMPK: Scientific Rationale and Strong Preclinical Data

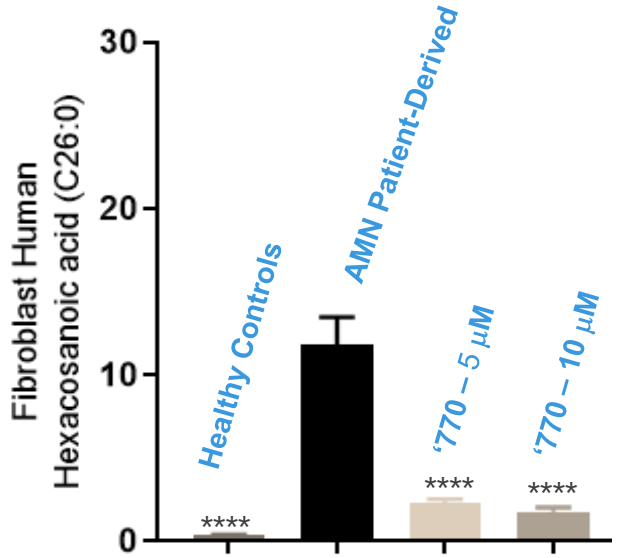
ALD AMN

- Deletion of AMPK in glial cells of ABCD1-null mice → mitochondrial dysfunction / low ATP[1]
- Reduced AMPK in patient-derived cells and brain tissue from ALD patients[2,3]
- AMPK activation with metformin* elevates ABCD2 levels in patient cell lines and ABCD1-KO mice[3,4]
- *PXL770 is active in ALD/AMN patient-derived cells and in ABCD1 null mice:*

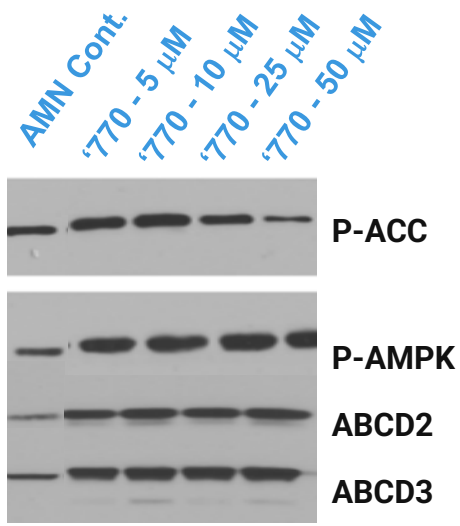
J Neurochem. 2016 Jul; 138(1): 10–13. PMID
Published online 2016 Mar 15. doi: 10.1111/jnc.13594

The ABCD's of 5′-Adenosine Monophosphate-activated Protein Kinase and Adrenoleukodystrophy

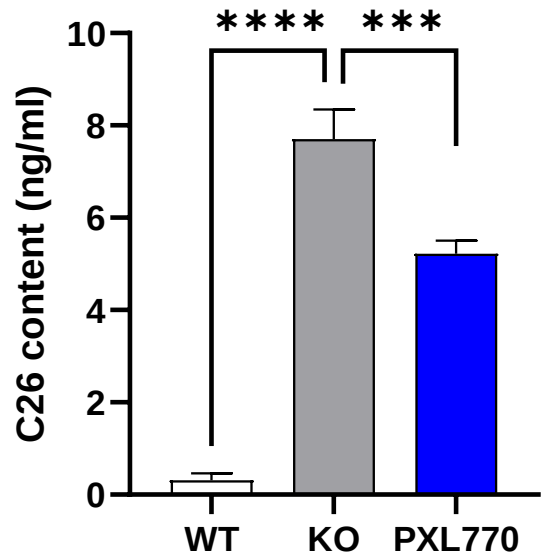
AMN – Fibroblasts Suppression of Elevated VLCFA



AMN – Fibroblasts Compensatory Transporter Protein Expression



ABCD1 Null Mouse Model Spinal Cord Suppression of Elevated VLCFA (C26:0)



N=12-15
****p<0.001

1. Mediators Inflamm 2015; 176983
2. Biochem Biophys Res Comm 2014;445:126-
3. J Neurochem 2016; 138:86-
4. J Neurochem 2016; 138:10-

Next Steps – including clinical development plans – under finalization

* well accepted indirect AMPK activator; requires metformin concentrations >> clinical exposure levels

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AMPK Activation to Treat Renal Diseases

DKD

Diabetic Kidney Disease

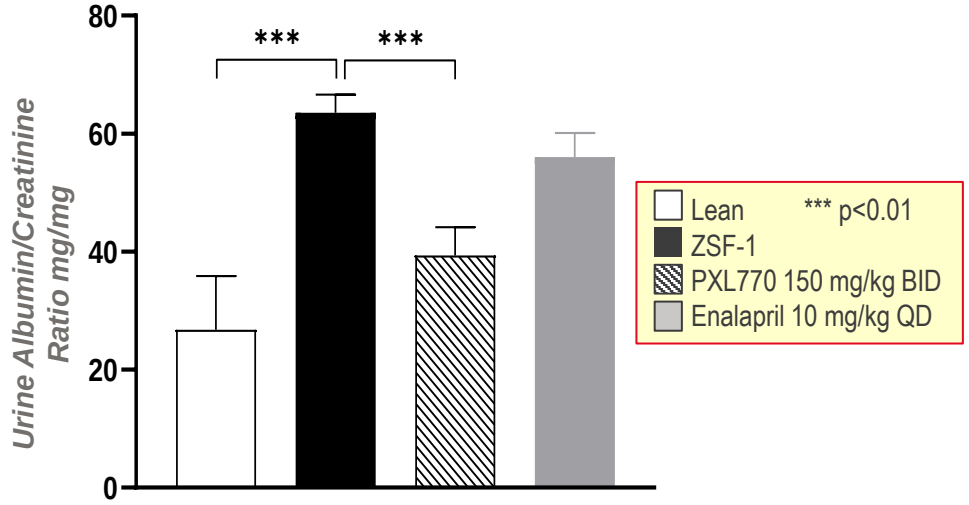
- Multiple pathways engaged; anti-inflammatory, anti-apoptotic, anti-fibrotic effects of AMPK[1]
- AMPK activity is ***reduced*** in human/rodent DKD tissue samples[2]
- ***Preclinical efficacy*** reported with indirect and direct AMPK activation[3]

PKD

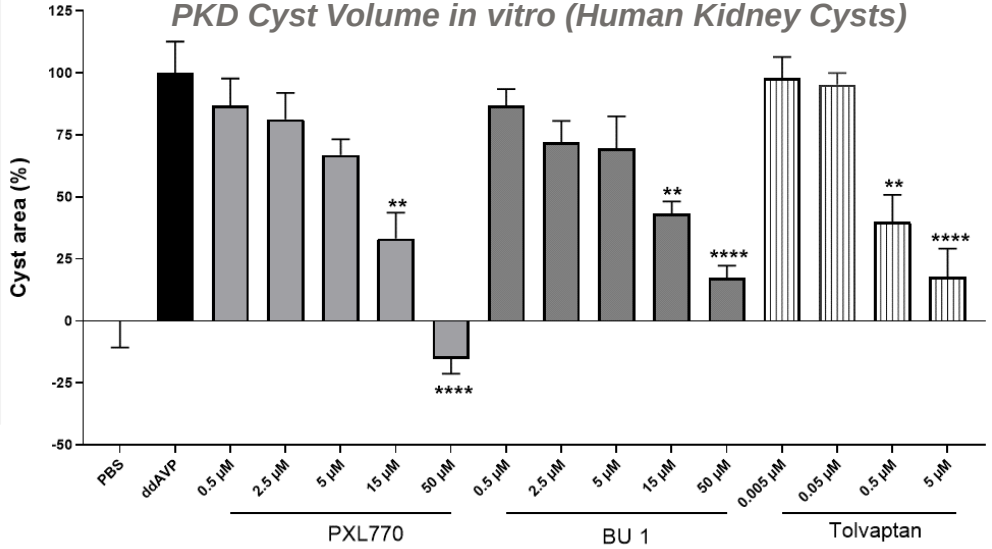
Polycystic Kidney Disease

- Autosomal dominant; fourth leading cause of CKD
- Significant unmet medical need
- ***AMPK activation validation***:
  - AMPK pathways linked to pathophysiology (*eg* mTOR[4]; CFTR[5])
  - *In vivo* efficacy with both indirect and direct AMPK activators[6]

PXL770 Improves Kidney Function in ZSF1 Rat Model of DKD



PXL770 & Next Gen. AMPK Activator (BU1) Reduce PKD Cyst Volume in vitro (Human Kidney Cysts)



** p<0.01, ****p<0.001 vs ddAVP (desmopressin) N=3-8

1. Curr Op Nephrol 2017, 26:375-83; Curr Drug Targets 2018; 19:709-20
2. J Clin Invest 2013; 123:4888-99
3. J Med Chem 2016; 59:8068-81; JPET July 2019; JPET 2017; 361:303-311
4. mammalian target of Rapamycin
5. cystic fibrosis transmembrane conductance regulator
6. J Clin Invest 2001; 108:1167-74; PNAS 2011;108: 2462–67; Sci Rep 7: 7161, 2017; EBioMed 47:436-45, 2019

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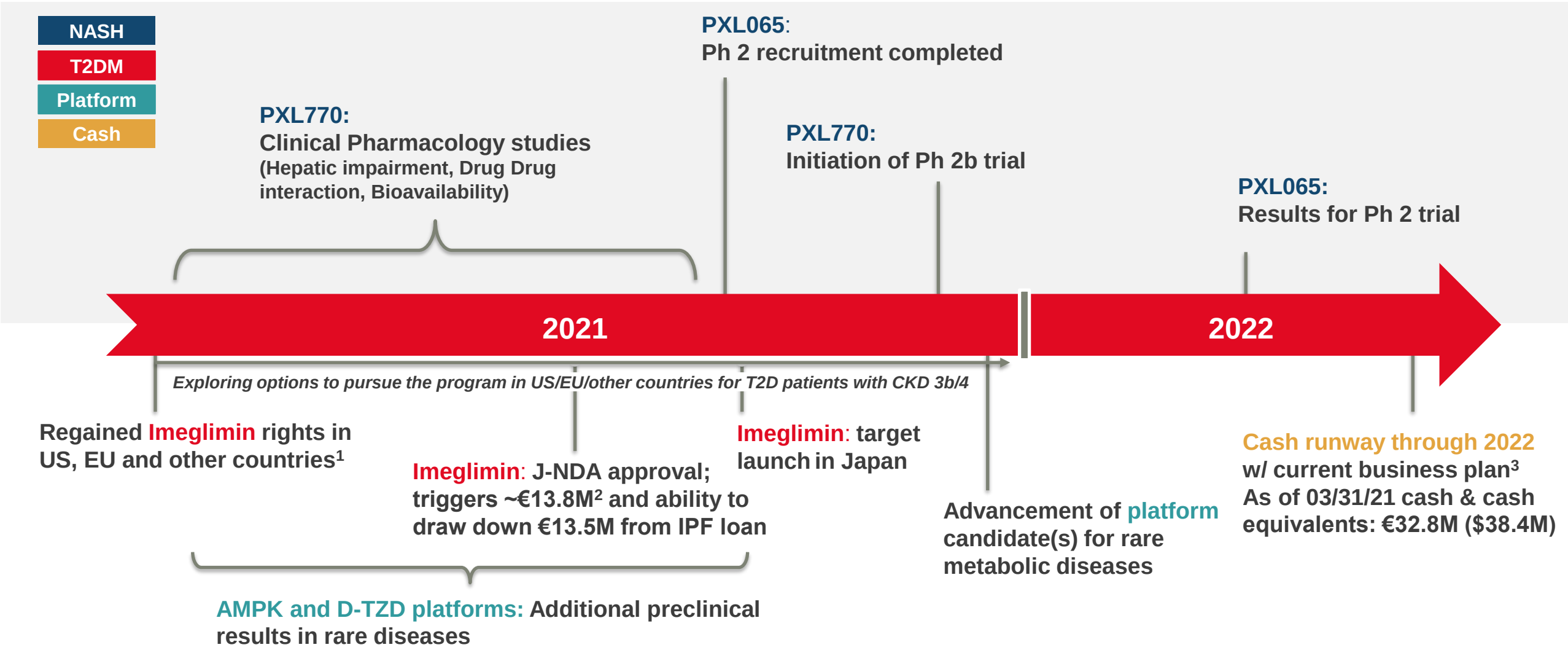
Upcoming Milestones

Near-Term Milestones to Drive Poxel's Growth



1. For countries not part of the DSP agreement. 2. Based on the JPY/€ exchange rate at December 31, 2020. 3. Taking into account ~€13.8M milestone payment by Sumitomo Dainippon Pharma, and €13.5M from IPF loan, which are both subject to the marketing approval of Imeglimin in Japan, expected in 2021 (Sumitomo Dainippon Pharma's fiscal year), and not including the financing of the Ph.2b study for PXL770.

Summary and Investment Highlights

- **Near Term Product Approval** anticipated for T2D in Japan with Sumitomo Dainippon Pharma, #1 diabetes company in Japan
- **Robust Clinical Pipeline in NASH** with two oral, first-in-class Phase 2 programs with significant near term milestones and addressing large market opportunities
- **Pipeline Expansion** focused on next generation programs targeting chronic and rare metabolic indications including adrenoleukodystrophy
- **Highly Experienced Management Team** with extensive metabolic R&D and business expertise & track record in US, EU and Japan
- **Listed on Euronext Paris** with global presence in France, US and Japan
- **Cash Runway through 2022**[1]**;** Cash & Equiv. €32.8M ($38.4M) as of 03/31/21

1. Taking into account ~€13.8M (based on the JPY/€ exchange rate at December 31, 2020) milestone payment by Sumitomo Dainippon Pharma, and €13.5M from IPF loan, which are both subject to the marketing approval of Imeglimin in Japan, expected in 2021 (Sumitomo Dainippon Pharma's fiscal year), and not including the financing of the Ph.2b study for PXL770.

Appendix

Key Financial & Shareholder Information

Market data

Ticker: POXEL

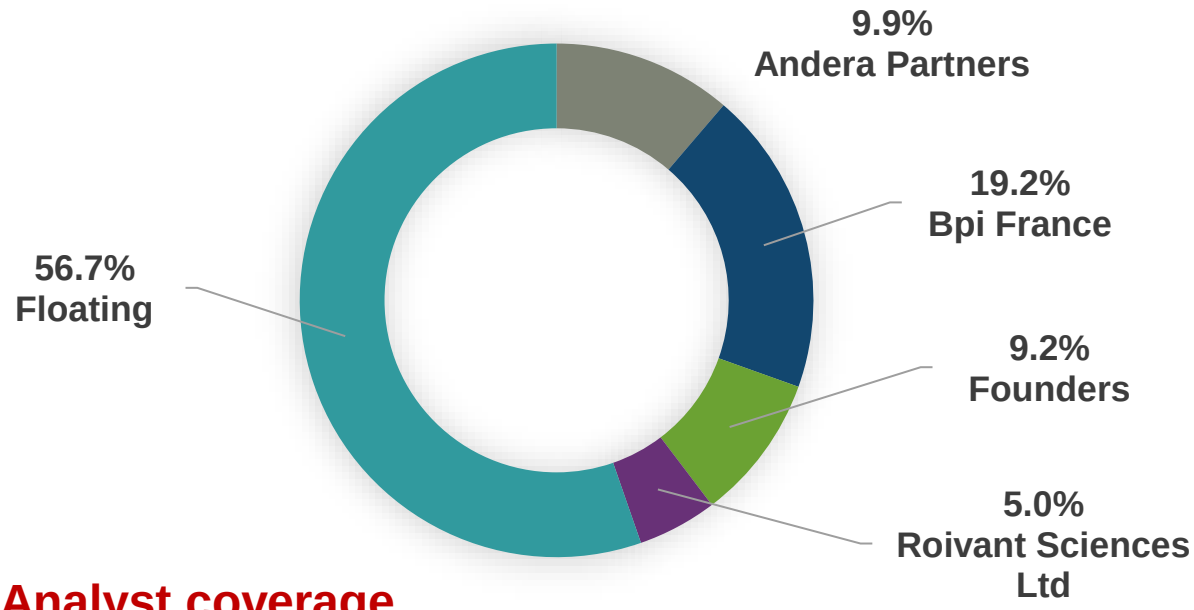
ISIN: FR0012432516

Number of shares: 28,611,254[1]

Key financials

- As of 03/31/21 cash & cash equivalents: **32.8 million (USD 38.4 million)**
- Cash runway extends through 2022 based on our current business plan[3]

Shareholder ownership[2]



Analyst coverage

Bryan Garnier	Jean-Jacques Le Fur
Degroof Petercam	David Seynnaeve
Jefferies	Lucy Codrington
JMP Securities	Jason Butler
Oddo	Martial Descoutures

1. At January-end 2021. 2. At the date of the presentation, based on the Company's knowledge. 3. Taking into account ~€13.8M (based on the JPY/€ exchange rate at December 31, 2020) milestone payment by Sumitomo Dainippon Pharma, and €13.5M from IPF loan, which are both subject to the marketing approval of Imeglimin in Japan, expected in 2021 (Sumitomo Dainippon Pharma's fiscal year), and not including the financing of the Ph.2b study for PXL770.

Leadership Team

Highly Experienced Management Team; Extensive R&D and Metabolic Expertise

Based in France

Thomas Kuhn (Pharm D, MBA)
Chief Executive Officer (CEO) and Co-founder

Sébastien Bolze (Pharm D, PhD)
Executive Vice President, Chief Operating Officer (COO), Co-founder

Sophie Bozec (PhD)
Senior Vice President, R&D Pharmacology, Co-founder

Anne Renevot
Executive Vice President, Chief Financial Officer (CFO)

Pascale Fouqueray (MD, PhD)
Executive Vice President, Clinical Development & Regulatory Affairs, Co-founder

Quentin Durand
Executive Vice President, Chief Legal Officer

Based in the US

Noah Beerman (MBA)
Executive Vice President, Business Development & President, US Operations

David Moller (MD)
Executive Vice President, Chief Scientific Officer (CSO)

Based in Japan

Takashi Kaneko (MD, PhD)
Senior Vice-President Medical & President of Poxel Japan K.K.

References for Slide 10

1. Glucophage [package insert]. Princeton, NJ: Bristol-Myers Squibb Company; 2018
2. Januvia [package insert]. Whitehouse Station, NJ. Merck & Co, Inc.; 2019
3. Onglyza [package insert]. Wilmington, DE. AstraZeneca Pharmaceuticals; 2019
4. Tradjenta [package insert]. Ridgefield, CT. Boehringer Ingelheim Pharmaceuticals; 2019
5. Invokana [package insert]. Titusville, NJ. Janssen Pharmaceuticals; 2020
6. Jardiance [package insert]. Ridgefield, CT. Boehringer Ingelheim Pharmaceuticals; 2020
7. Farxiga [package insert]. Wilmington, DE. AstraZeneca Pharmaceuticals; 2020
8. Bydureon [package insert]. Wilmington, DE. AstraZeneca Pharmaceuticals; 2019
9. Victoza [package insert]. Plainsboro, NJ. Novo Nordisk; 2019
10. Trulicity [package insert]. Indianapolis, IN. Eli Lilly and Company; 2019
11. Ozempic [package insert]. Plainsboro, NJ. Novo Nordisk; 2019
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13. Amaryl (glimepiride) [package insert]. Bridgewater, NJ. Sanofi-Aventis; 2018
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