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Corporate Presentation

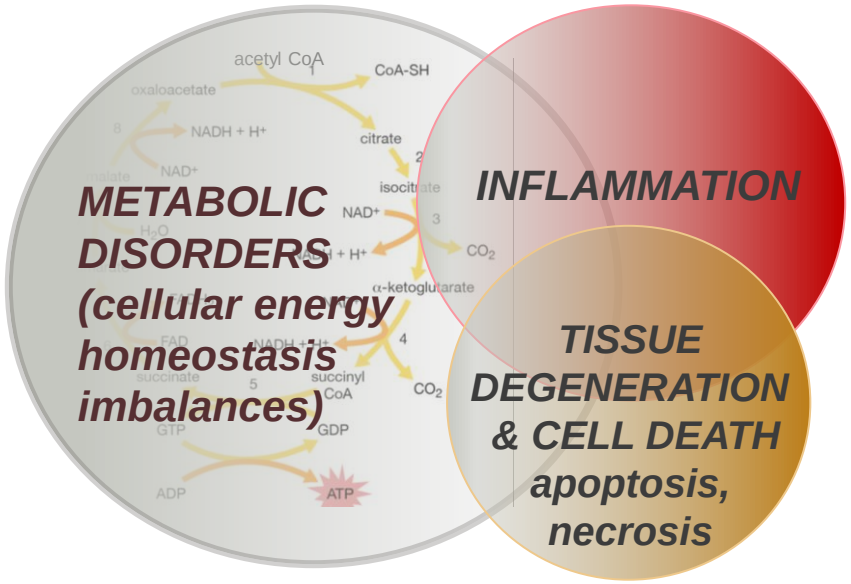
November 2021

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.

Poxel's Investment Highlights

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



- **TWYMEEG® (Imeglimin) approved and launched** for Type 2 Diabetes in Japan
- **Strategic focus on rare metabolic Indications and NASH**
- **Clinical Stage Pipeline** with Global Operations
- **Highly Experienced Management** Team in Metabolic Diseases

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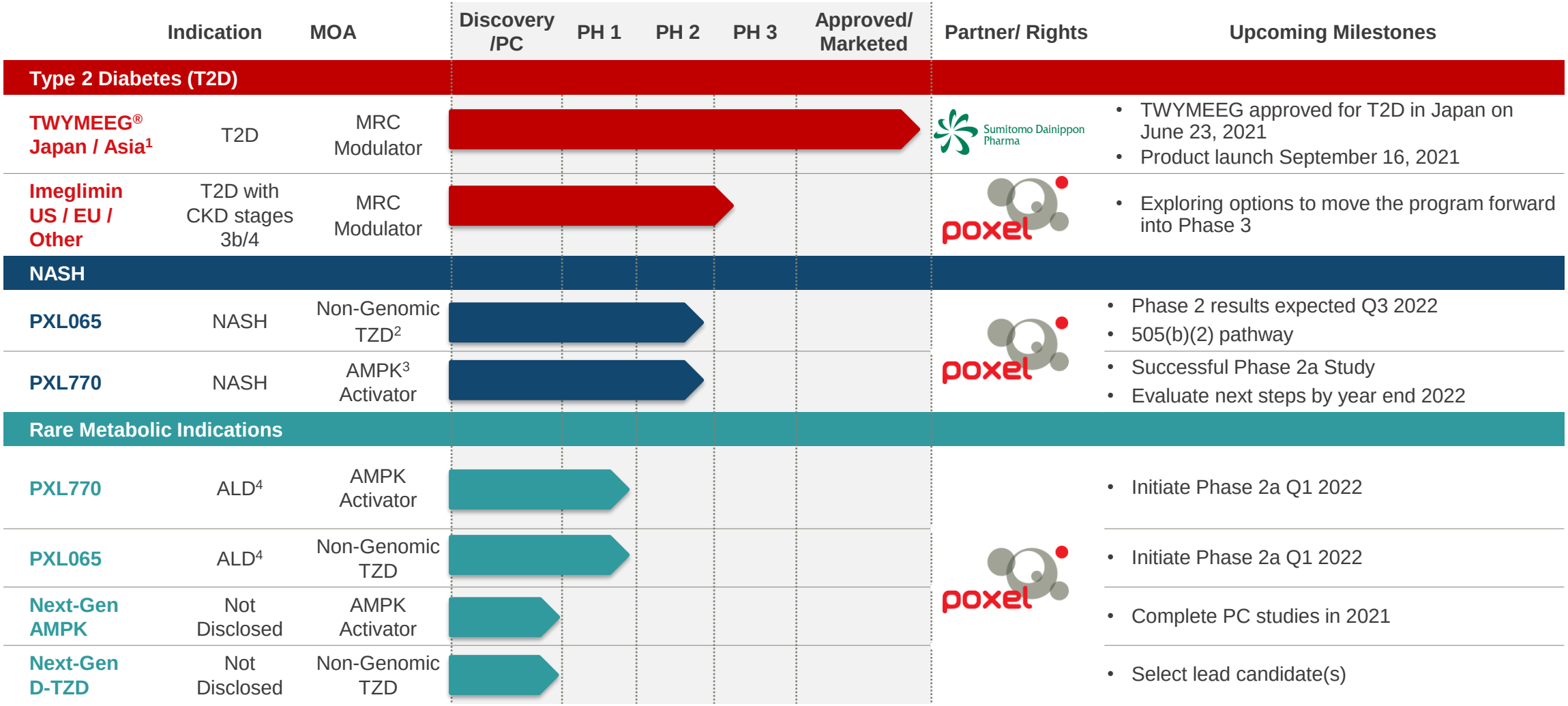
LISTED
EURONEXT

Robust Mid-to-Late Stage Metabolic Pipeline

Focus on Rare Metabolic Diseases and NASH



1. Includes: China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, Philippines, Singapore, Myanmar, Cambodia, Laos. 2. Deuterium-modified thiazolidinediione. 3. AMP-kinase. 4. X-linked **A**dreno**L**euko**D**ystrophy.

A New Chapter to Drive Shareholder Value

Focus on Rare Metabolic Diseases and NASH

Type 2 Diabetes

TWYMEEG®
(Imeglimin)

Partnered in Asia[1] with diabetes market leader in Japan

Sumitomo Dainippon Pharma

- ***Recent cash infusion of EUR~26.7 million triggered by Japan approval***
- ***Japan Launch Sept 2021***
- ***Potential sales-based payments & escalating royalties on net sales***

NASH

PXL065
PXL770

NASH

- ***Phase 2 biopsy data for PXL065 expected in Q3 2022***
- ***Next Steps for PXL770 pending evaluation YE 2022***

D-TZD[2] Platform
AMPK[3] Platform

ALD*

PXL065
PXL770

External Opportunities

- ***Phase 2a Biomarker POC studies in ALD for 065 & 770; results anticipated YE 2022***
- ***Pipeline expansion into new indications***

Rare Metabolic Diseases

1. Japan plus: China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, Philippines, Singapore, Myanmar, Cambodia, Laos.
2. Deuterium-modified thiazolidinediones.
3. AMP-kinase (allosteric activators) /

* X-linked adrenoleukodystrophy (ALD).

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TWYMEEG® (Imeglimin): Approved in Japan

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

- **June 23rd Approval in Japan triggered:**
  - 3rd and final tranche of **EUR 13.5 million** from IPF loan received June 30, 2021
  - Milestone payment of **~EUR 13.2 million (USD 15.8 million)**[2] from Sumitomo Dainippon Pharma in July
- **Japan launch on Sept 16, 2021**
- Potential sales-based payments and escalating royalties on net sales

Business Opportunity Japan: Maximize Product Profile

- Sumitomo Dainippon Pharma #1 diabetes franchise; **FY20 USD 890 million**[4]
- DPP4i's are prescribed to 80% T2D patients[5]
- TWYMEEG can be prescribed as **add on therapy**, on top of DPP4i's, and as **monotherapy**
- TIMES program observed to show robust efficacy with favorable safety and tolerability profile
- The patent estate for Imeglimin extends to 2036 (including potential 5 year patent term extension), with other patent applications ongoing

1. Including: Japan, China, South Korea, Taiwan, Indonesia, Vietnam, Thailand, Malaysia, the Philippines, Singapore, Myanmar, Cambodia, and Laos.
2. Currency exchange rate at the date of the approval (23 June 2021).
3. Currency exchange rate at the date of the agreement (30 Oct 2017).
4. Sumitomo Dainippon Pharma fiscal year April-March
5. IQVIA data FY2016 and NDB data FY2016

Strategy to Pursue Treatments for Rare Diseases

Poxel Molecules can Target Key Nodes in Pathways Driving Several Rare Diseases

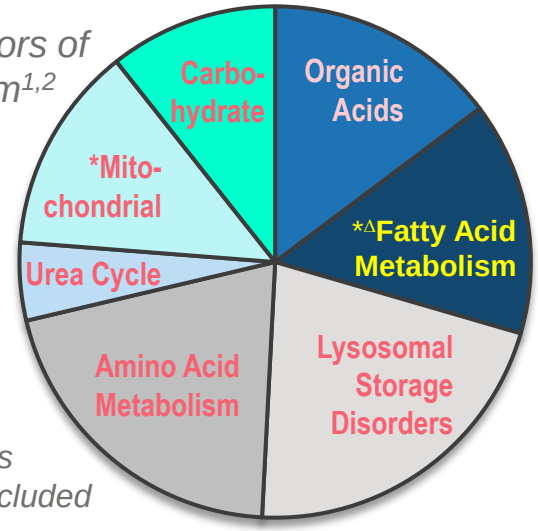
Why Rare Disease ?

- High unmet needs, limited treatment options; > 90% of rare diseases are *without* an FDA approved treatment[1]
- Efficient and lower cost development: faster timelines; higher probability of success; favorable regulatory environment
- Market opportunity:
  - almost 1 in 10 people have rare diseases[3]
  - premium pricing supported by prior orphan drug approvals
  - ability for Poxel to commercialize and capture greater economics

Why Poxel ?

- Scientific fit - more than 1,100 rare diseases have a metabolic basis - "inborn errors of metabolism"[4]; D-TZD and AMPK approaches modulate pathways driving multiple diseases
- Proven R&D capabilities
- Capacity to pursue additional rare disease programs
- Close connections with relevant patient advocacy and KOLs

Inborn Errors of Metabolism[1,2]



** Poxel targeted disorders*
ΔALD included

1. IQVIA Institute for Human Data Science. Orphan Drugs in US: Exclusivity, Pricing and Treated Populations. 2018 Dec. https://www.iqvia.com/-/media/iqvia/pdfs/institute-reports/orphan-drugsin-the -united-states-exclusivity-pricing-and-treated-populations.pdf?_=154844532268
2. CFR 316.20 or Sec 526 of the Orphan Drug Act. https://www.fda.gov/forindustry/developingproductsforraredieasesconditions/howtoapplyfororphanproduct-designation/ucm364750.htm
3. Genetic and Rare Diseases Information Center; National Ctr. Advancing Trans Sciences; FAQs About Rare Diseases; Last updated 11/30/2017. https://rarediseases.info.nih.gov/diseases/pages/31/faqs-about-rare-diseases
4. Genet Med 2019; 21:102-106; 5. Metabolites 2019; 9:242-

Non-Alcoholic Steatohepatitis (NASH)

No Approved Medicines - Poxel has Two Clinical Stage First-in-Class Oral Therapies

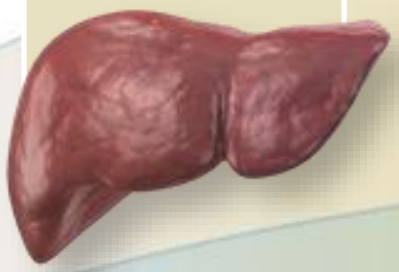
Excessive caloric intake
Sedentary lifestyle

Metabolic syndrome
Dyslipidemia
Type 2 diabetes
Obesity

High Morbidity

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

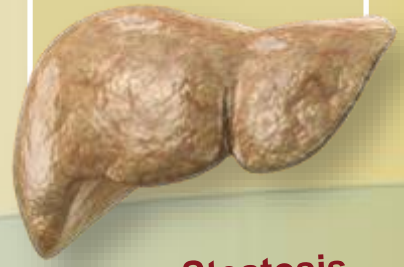
Normal



NAFLD

25% of the general population

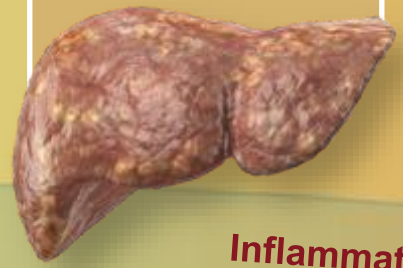
>70% in diabetic & obese patients



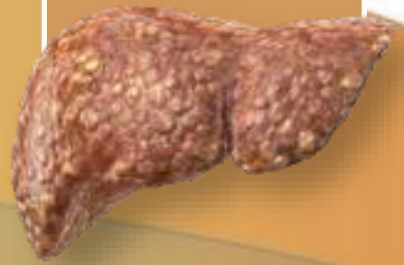
NASH

12% of the general population

25-70% in diabetic and obese patients ≥ 50



Cirrhosis



Fat Deposition › **Steatosis** **Lipotoxicity** › **Inflammation** **Fibrosis** **Collagen Deposition**

Estimated Market Opportunity: >$20B by 2025

J.Hepatology 2018, 68, 362-375. Market Research Engine, Jan. 2020.

Accelerating & Expanding Rare Metabolic Disease Programs

Starting with existing platforms:

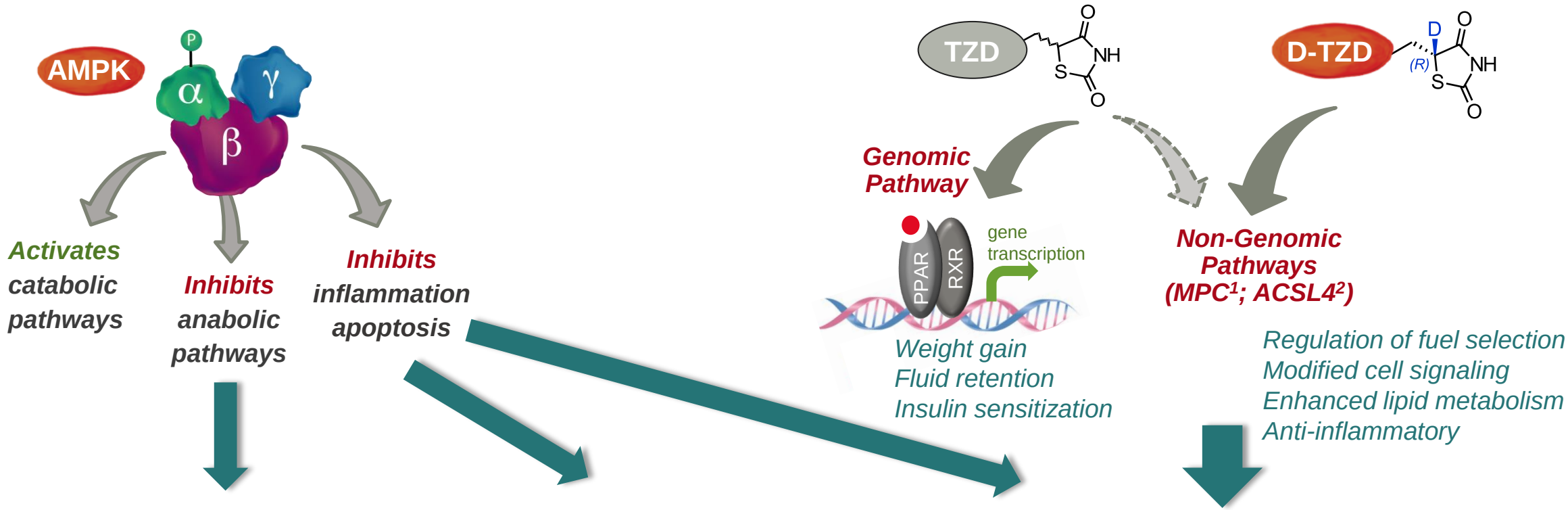
PXL065 - D-TZD's*

PXL770 - AMPK Activator

*Deuterium-modified thiazolidinediones.

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

Two Programs Approaching Clinical Development for ALD



Renal
Diabetic kidney disease
Polycystic kidney disease

Endocrine
Type 2 diabetes
Polycystic ovary syndrome

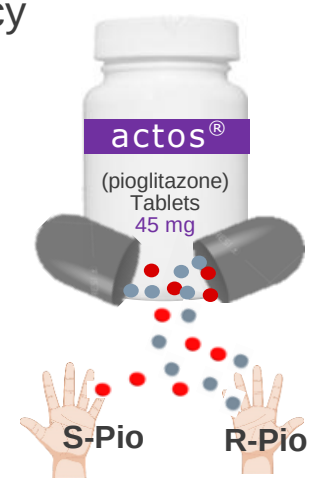
Rare Metabolic
Adrenoleukodystrophy (ALD)
Mitochondrial disorders

1. Mitochondrial Pyruvate Carrier 2. Long chain Acl-CoA Synthetase-4 .

Two First-in-Class Advanced Lead Molecules

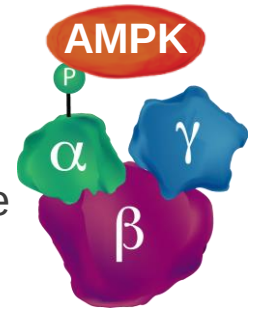
PXL065

- Deuterium stabilized *R*-stereoisomer of pioglitazone*
- Preclinical:
  - no (PPARγ –driven) weight gain/fluid retention
  - metabolic and anti-inflammatory efficacy
- Clinical
  - completed Phase 1
  - confirmed selective *R*-pio exposure
  - good safety profile in >130 human exposures (Phase 1 plus ongoing Destiny-1 NASH trial)
- Composition of matter IP
- 505(b)(2) regulatory path; open IND in ALD/AMN



PXL770

- Proprietary direct allosteric AMPK activator♦
- Preclinical:
  - Metabolic, anti-inflammatory, cytoprotective efficacy in NASH, diabetes, kidney, CV models
- Clinical
  - orally bioavailable; once daily PK profile
  - human target engagement and efficacy demonstrated (diabetes and NAFLD)
  - well tolerated with favorable safety profile>200 human exposures up to12 weeks
- Composition of matter IP



*approved Type 2 diabetes therapy (Actos); Jacques V et al. Hep Comm 2021; implicated in ALD - Brain 2013;136:2432-43

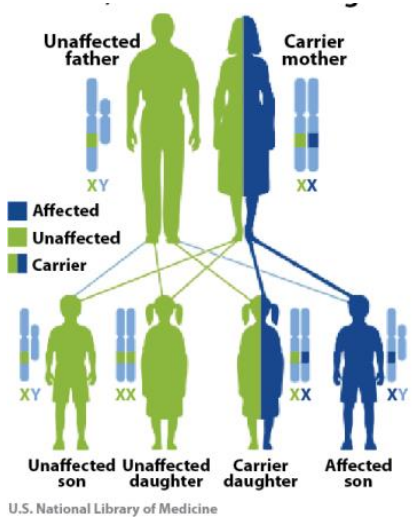
♦ Gluais-Dagorn et al. Hep Comm 2021; implicated in ALD – Weidling I J Neurochem 2016

Adrenoleukodystrophy

A Not-so-Rare Orphan Neurometabolic Disease

Genetics

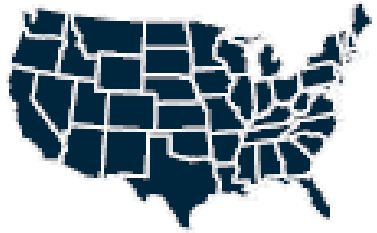
- Monogenic, X-linked mutations in ABCD1 gene
- Gene encodes a transporter present in peroxisomes required for metabolism of very long chain fatty acids (VLCFA)
- *Males more severely affected*



Prevalence

Estimated US Prevalence*

20,000 – 29,000

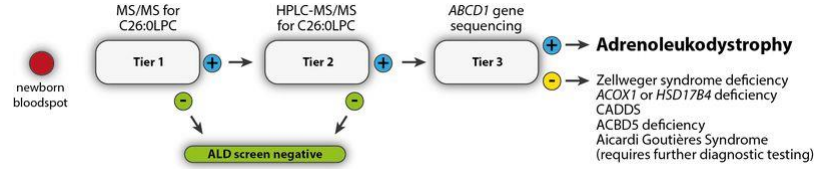


Estimated Global Prevalence*

444,000 – 644,000

Diagnosis

- Newborn screening – increasingly common (now >60% of newborns in US)



- Clinical presentation followed by measurement of VLCFA and genotyping

* Based on published and newborn screening incidence rates of 1/12,000-1/17,000; Bezman L, Ann Neurol 2001; 49:512-17; Kemper AR, Genet Med. 2017; 19:121-26; Schmidt JL, Am J Med Genet 2020; 182A:1906-12; https://rarediseases.org/rare-diseases/adrenoleukodystrophy.

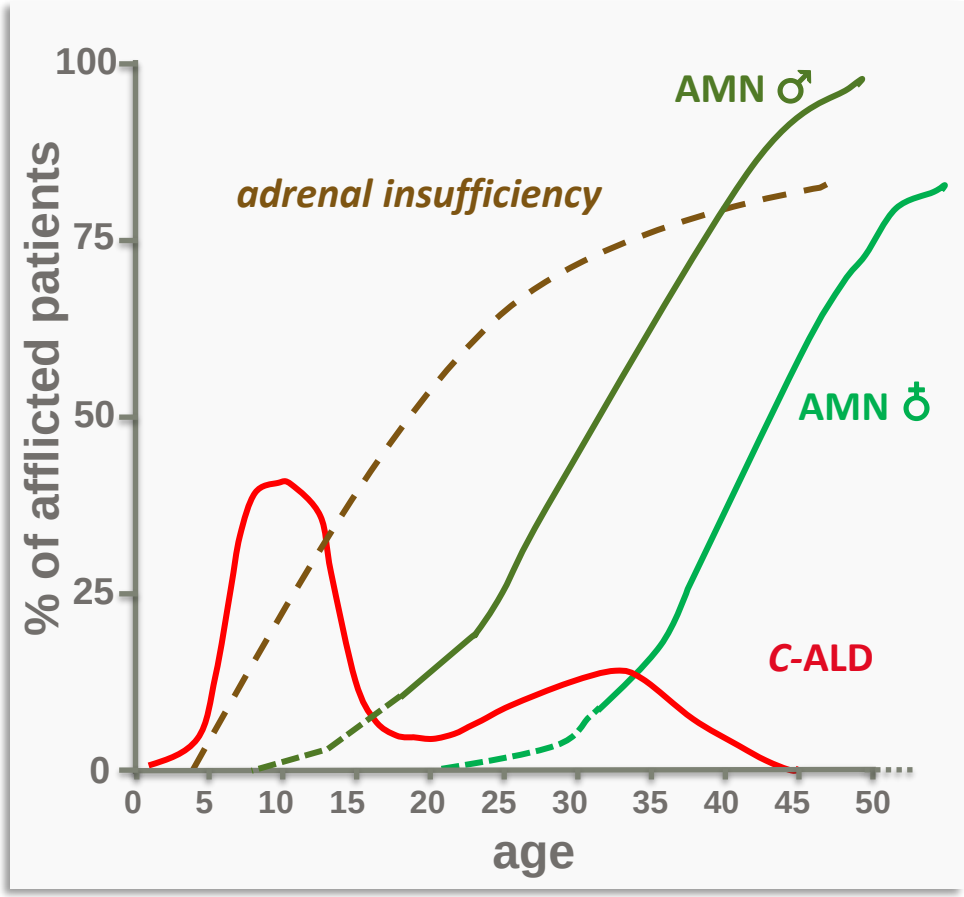
ALD Clinical Features and Disease Course

Two Major Overlapping Subtypes

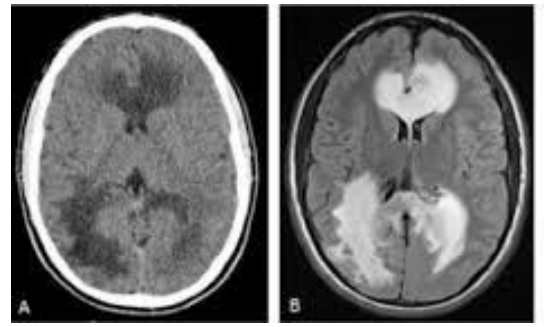
Addison's Disease

Cerebral ALD (C-ALD): Damage to brain white matter; cognitive impairment; loss of vision/hearing; impaired balance-movement; death

Adrenomyeloneuropathy (AMN): Slowly progressive; impaired gait-balance-movement; bladder-bowel dysfunction; also affects women



C-ALD Lesions (MRI)



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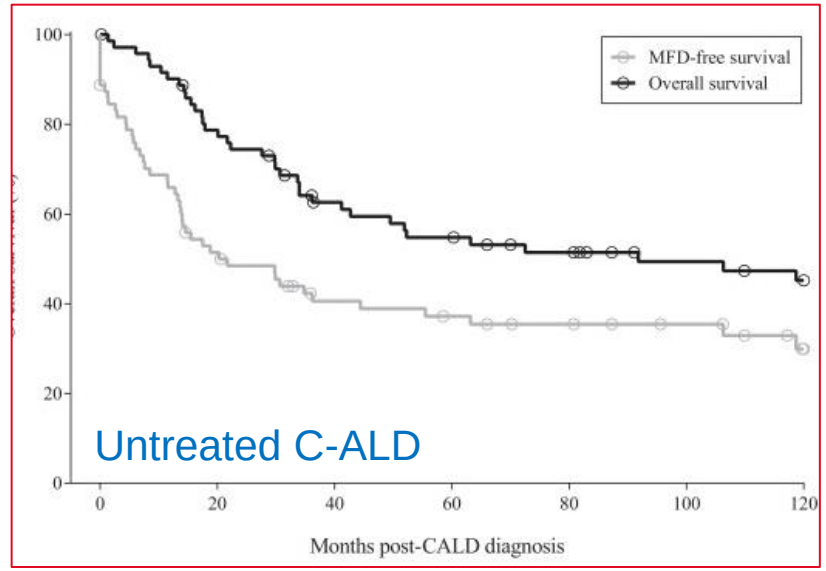
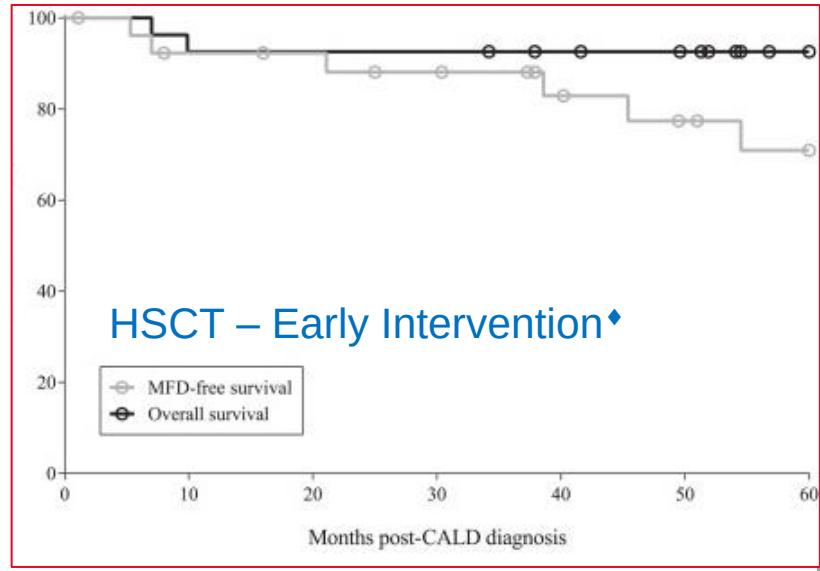
Treatment Approaches

No Approved Pharmaceutical Therapies

- Adrenal insufficiency → glucocorticoid replacement therapy
- Early stage (early onset) C-ALD → HSCT
- Advanced or later onset C-ALD → no effective options
- AMN:
  - physical therapy
  - supportive care
  - major target for future therapies



Untreated C-ALD



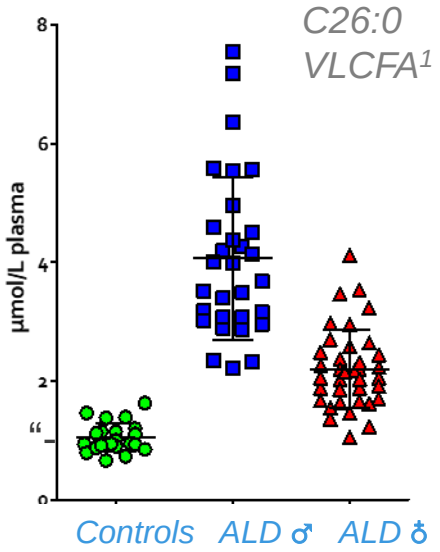
HSCT – Early Intervention♦

♦HSCT Data from Raymond GV. Biol Blood Marrow Trans 2019; 25:538-48. Early intervention = Neuro function score <1; LOES score 0.5-9

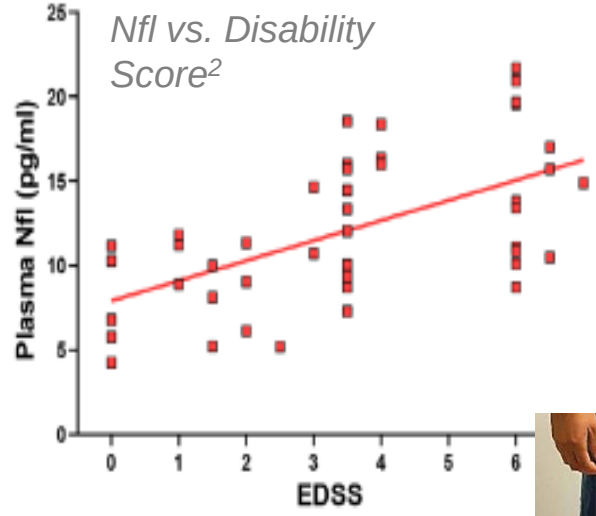
Clinical Trial Measures & Outcomes

Several Potential Surrogates for Longer Term Benefits

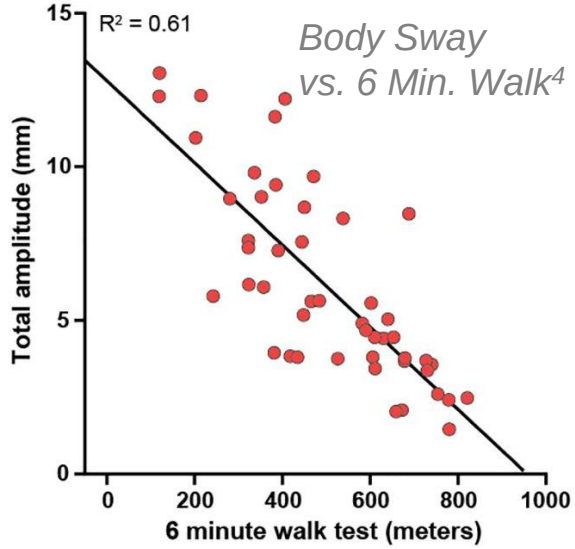
- Biomarkers
  - VLCFA
  - Neurofilament light chain (Nfl)
- MRI imaging (C-ALD)
- Functional tests
  - 6 minute walk; timed up-and-go", others
  - postural body sway
- Disability scores; patient reported outcomes
  - EDSS, ALDS, SSPROM, others



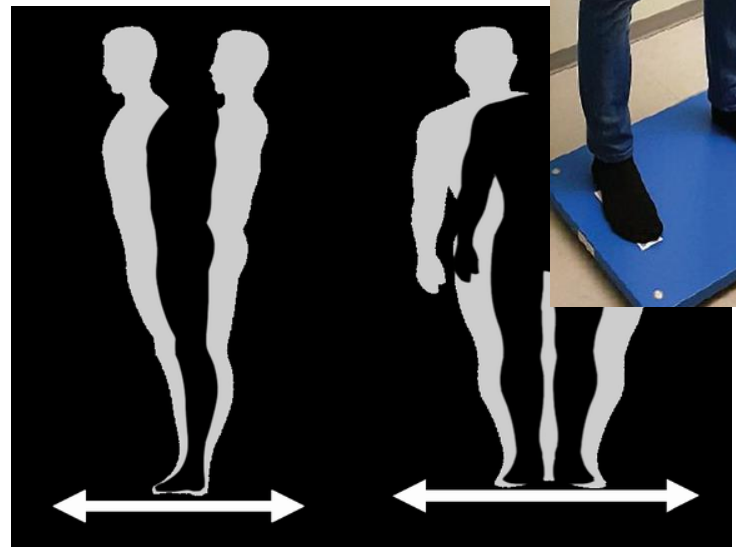
C26:0 VLCFA[1]



Nfl vs. Disability Score[2]

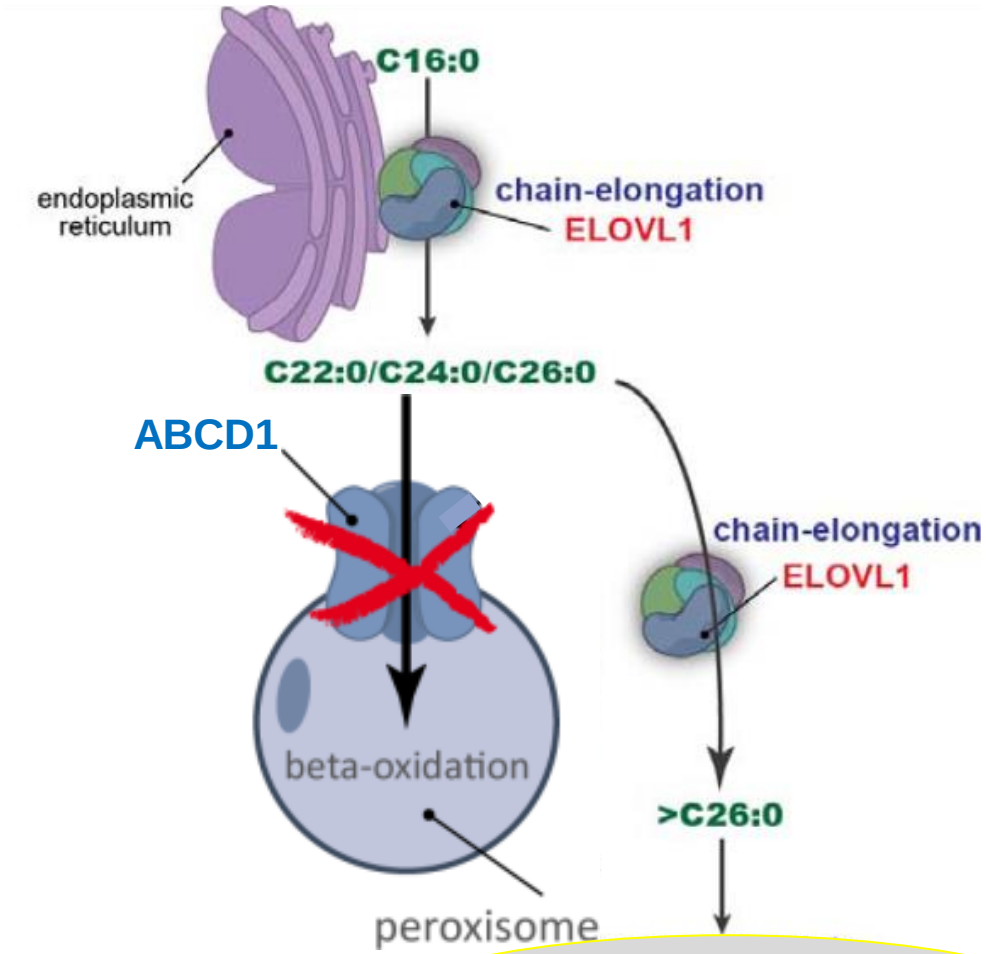


Body Sway vs. 6 Min. Walk[4]



1. Huffnagel et al. 2017 Mol Genet Metab; 122:209-
2. Engelen et al. 2020 Ann Clin Trans Neurol; 7:2127-
3. Engelen et al. 2020 Front Physiol; doi 10.3389/fphys.2020.00786

Poxel Platforms – Potential to Target ALD Pathophysiology



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

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

Defective ABCD1 leads to accumulation of VLCFA in tissues

VLCFA cause axonal degeneration and secondary demyelination

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

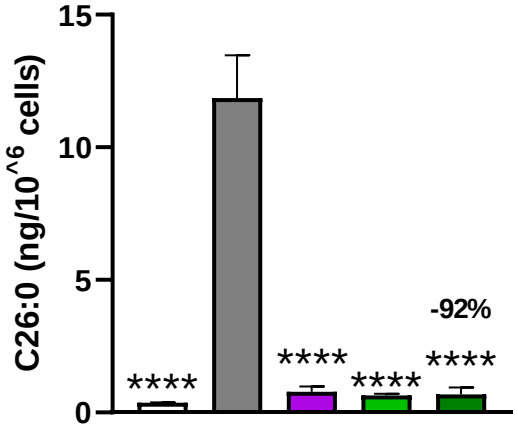
D-TZD's: Rationale and Strong Preclinical Data

- Pioglitazone attenuates neuroinflammation and confers neuroprotection:
  - non-human primates with Parkinson's disease[1]
  - rodent acute brain ischemia[2], spinal cord injury[3]
- Pioglitazone efficacy achieved in ABCD1-null mice[4]
- MPC or ACSL4 inhibition implicated as a therapeutic approach in neurodegeneration[5-8]
- 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
7. Neurosci Lett 2021; 745:135627
8. Brain Behav Immunity 2021; 93:312-

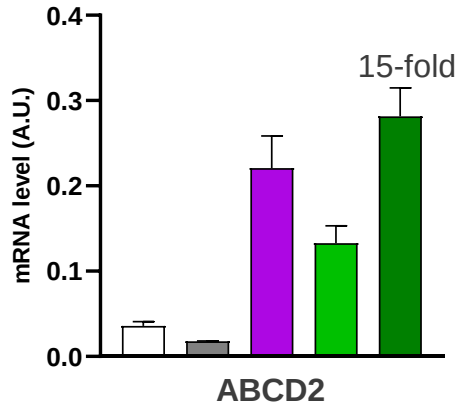
AMN – Fibroblasts Suppression of Elevated VLCFA

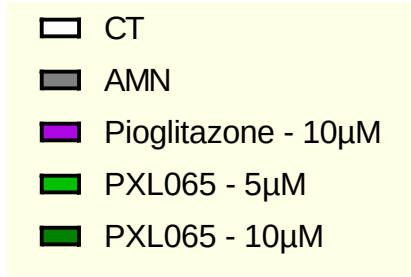
N=3-4



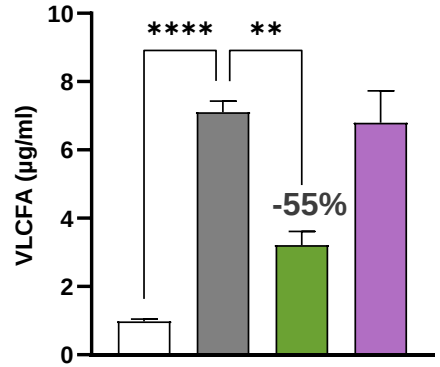
***p<0.01, ****p<0.001

AMN – Fibroblasts Compensatory Transporter Gene Expression





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



N=12-15; Dose =15 mg/kg QD

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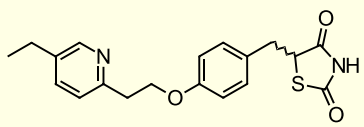
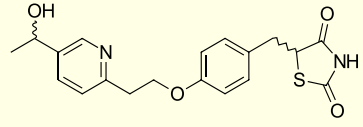
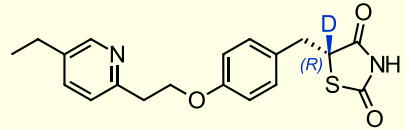
D-TZD's: Clinical Results Support Pursuit of ALD/AMN

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 (M-IV Pio Metabolite)**	 **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

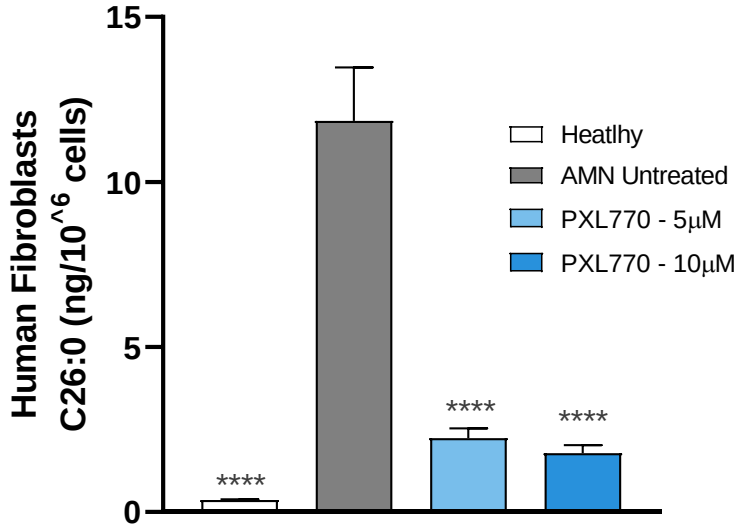
AMPK: Scientific Rationale and Strong Preclinical Data

- Deletion of AMPK in glial cells of ABCD-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-null 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

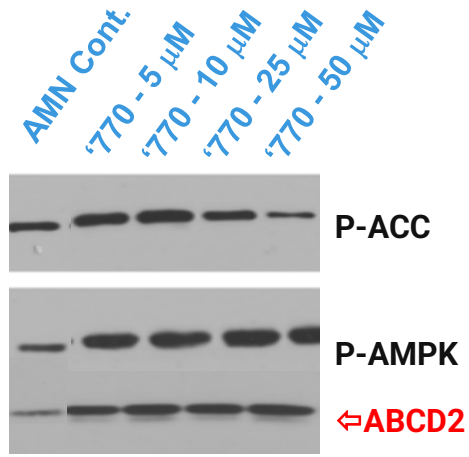
The ABCD's of 5′-Adenosine Monophosphate-activated Protein Kinase and Adrenoleukodystrophy

AMN - Fibroblasts
Supression of Elevated VLCFA

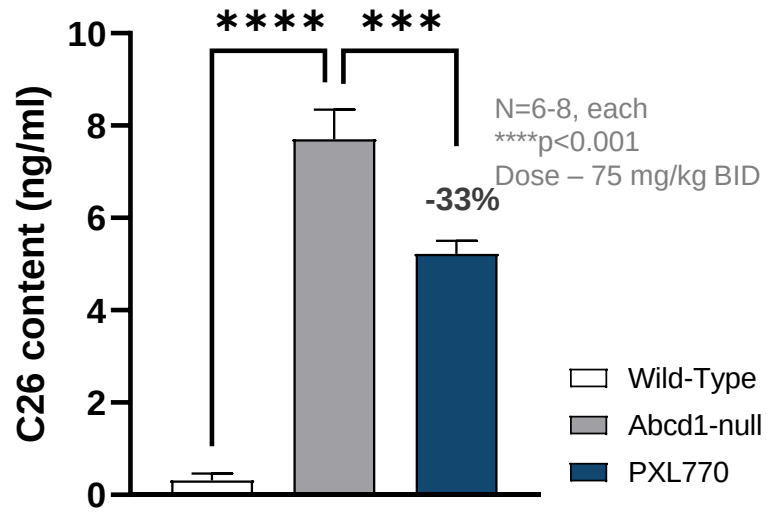


N=3-4

AMN – Fibroblasts
Compensatory Transporter
Protein Expression



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



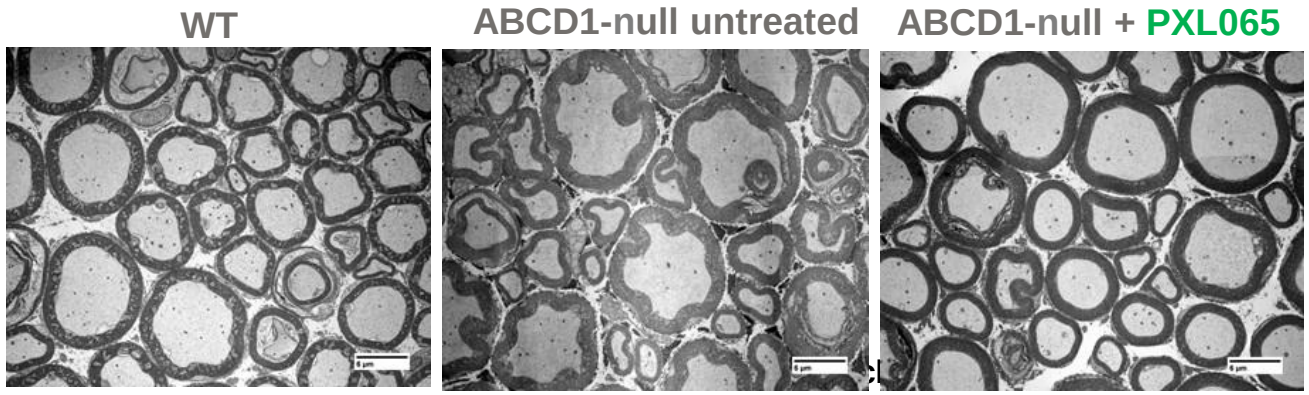
1. Mediators Inflamm 2015; 176983
2. Biochem Biophys Res Comm 2014;445:126-
3. J Neurochem 2016; 138:86-
4. J Neurochem 2016; 138:10-

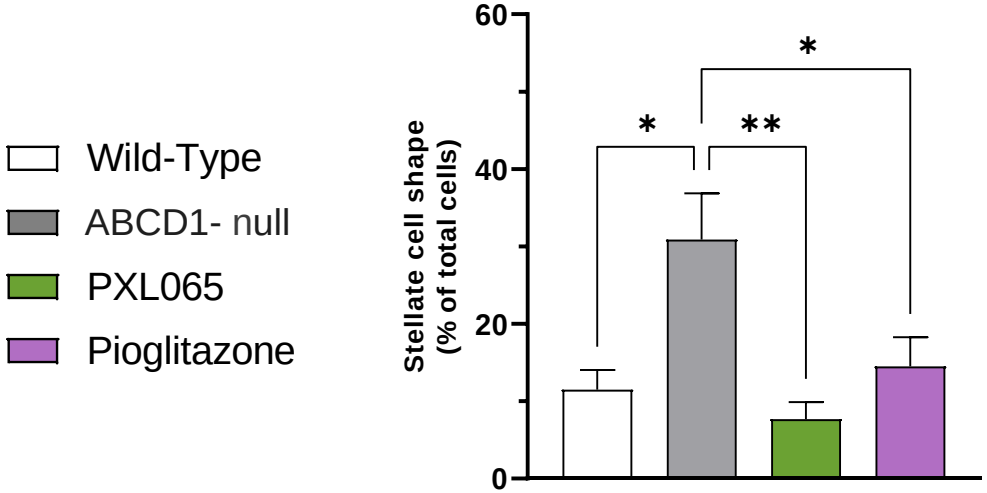
* well accepted indirect AMPK activator; requires metformin concentrations >> clinical exposure levels

Both PXL065 and PXL770 Mediate Neurologic Benefits

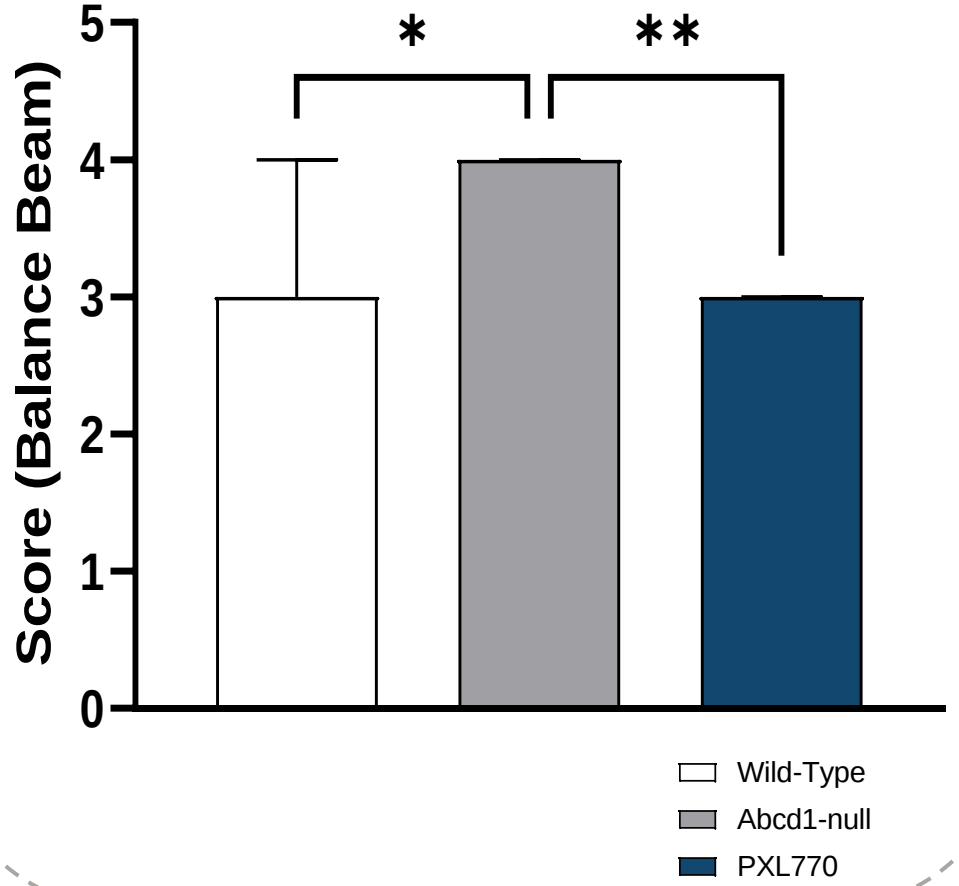
ABCD1-Null Mouse (12 week Treatment)

Electronic Microscopy of Sciatic Nerve





Neurologic Tests (Balance Beam)



*p<0.05; **p<0.01

Poxel Lead Molecules vs. Selected Competitors

Advanced Drug Candidate with Potential for Superior Clinical Results

	PXL065	PXL770	Leriglitazone*	VK0214∇	ABX-002♦
Mechanism	Non-genomic D-TZD	AMPK activator	PPARγ (+ other TZD)	Thyroid receptor β	Thyroid receptor β
Stage	Ph2a – Ready	Ph 2a – Ready	Ph 2b/3	Ph 1b	*Preclinical*
Human ALD Cells	⇩⇩⇩VLCFA ⇧ ABCD2 ⇧ mitochondrial respiration	⇩⇩⇩ VLCFA ⇧ ABCD2 ⇧ mitochondrial respiration	*No VLCFA or ABCD2 effects reported*	*VLCFA not reported* ⇧ ABCD2	*Not reported*
In Vivo ABCD1 Null Mice **Biomarker Signal**	⇩⇩ VLCFA - plasma, brain, spinal cord	⇩⇩ VLCFA - plasma, brain, spinal cord	⇩ VLCFA spinal cord (*plasma not reported*)	⇩ VLCFA plasma, spinal cord	⇩ VLCFA plasma, brain; *spinal cord not reported*
Neuro Histology	Improved	Improved	Improved	*Not reported*	*Not reported*
Neuro-Behavior	Improved	Improved	Improved	*Not reported*	*Not reported*
Other Comments	Clinical safety: >130 exposures plus 505(b)(2)	Clinical safety: (>200 exposures)	+ results in Ph2b/3 *weight gain, edema*	Phase 1 completed	*No clinical experience*

* Rodriquez-Pascau Science Trans Med 2021; Am Acad Neuro (AAN) oral presentation 2021; Minoryx 2021 press release

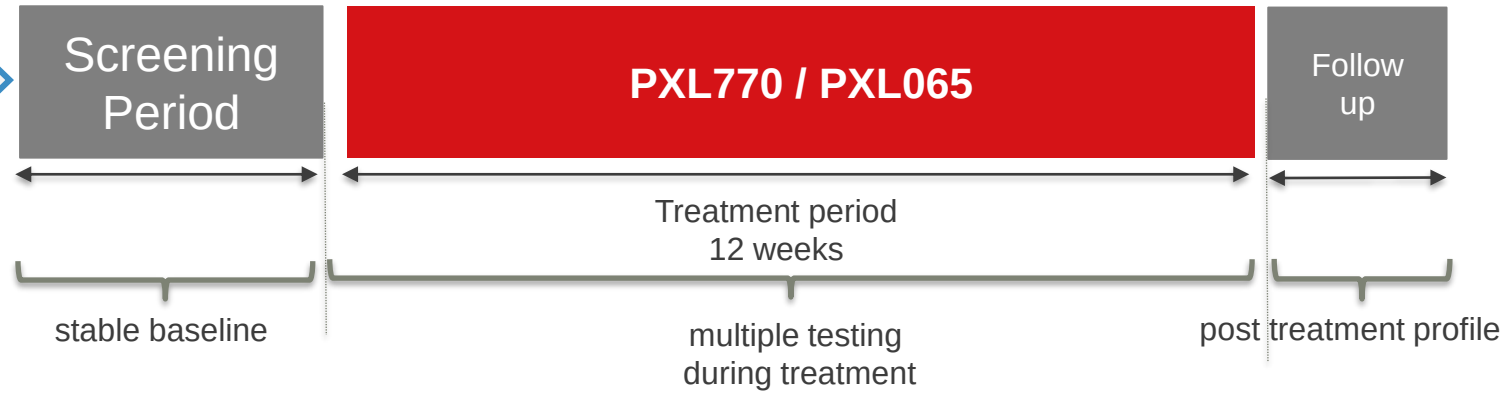
∇ Viking corporate presentation 2021; ♦Autobahn AAN Poster 2021

Planned Phase 2a Studies in ALD/AMN

PXL770 and PXL065 in Two Separate Identical Studies

Key inclusion criteria

- Males with AMN
- Age 18-65
- No active cerebral disease
- 12 patients each



Endpoints

- VLCFA – biomarker and hallmark of disease – drives pathology
- Neurofilament light chain – validated biomarker of neuronal damage
- Other / exploratory biomarkers
- PK
- Safety

Phase 2a initiation 1Q22 – completion 4Q22
Phase 3 Pivotal trial(s) to begin in 2023

ALD Opportunity Summary

High Unmet Needs, Blockbuster Market Potential

- **Blockbuster market opportunity**
  - US prevalence of 20,000-29,000; Global prevalence of 444,000 – 644,000
  - Ability for premium pricing based upon other orphan drugs with similar prevalence
  - Potential Regulatory designations:
    - US: Orphan (7 years exclusivity), Fast Track, Breakthrough, Priority Review
    - EU: Orphan (10 years exclusivity), PRIME
- **Expedited Clinical Development**
  - Established safety profiles of PXL065 (with 505b2) and PXL770 mitigate risk & may reduce clinical development timelines
  - Data from ALD preclinical models for PXL065 and PXL770 suggest significant impact on key biomarkers (VLCFA, neurofilament light chain)
  - Potential for accelerated approval based upon biomarkers
- **Community Engagement**
  - Established relationships with Key Opinion Leaders
  - Collaborations with important patient advocacy groups

NASH

PXL065

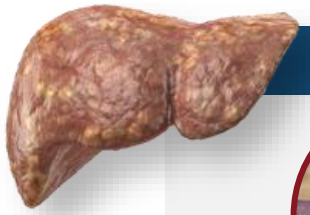
Non-Genomic Pathway D-TZD Modulator for Treatment of NASH

Utilizing the 505(b)(2) Regulatory Pathway

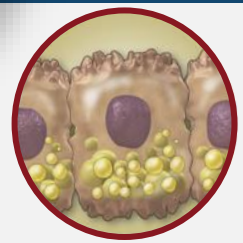
PXL770

Direct AMPK Activator

PXL770 and PXL065: Novel, First-in-Class Product Candidates



HALLMARKS OF NASH

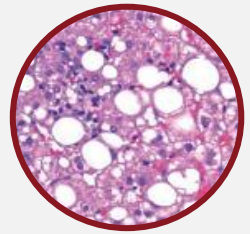


Lipid accumulation in hepatocytes

Steatosis

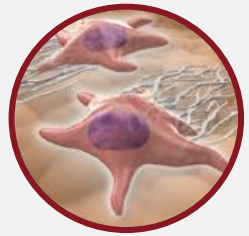
Immune cells (macrophages - MΦ)

Inflammation



Cellular damage-death

Ballooning



Hepatic stellate cell activation

Fibrosis

- **First-in-Class - Novel Mechanisms**
  - ability to target multiple hallmarks of NASH
- **Clinical validation**
  - positive Phase 2a results (‘770)
  - derived from pioglitazone – proven NASH benefits (‘065)
- **Daily oral administration**
  - combinable with other approaches
- **Innovative development approaches**
  - focus on patients with co-existing diabetes (‘770)
  - 505(b)(2) regulatory path (‘065)

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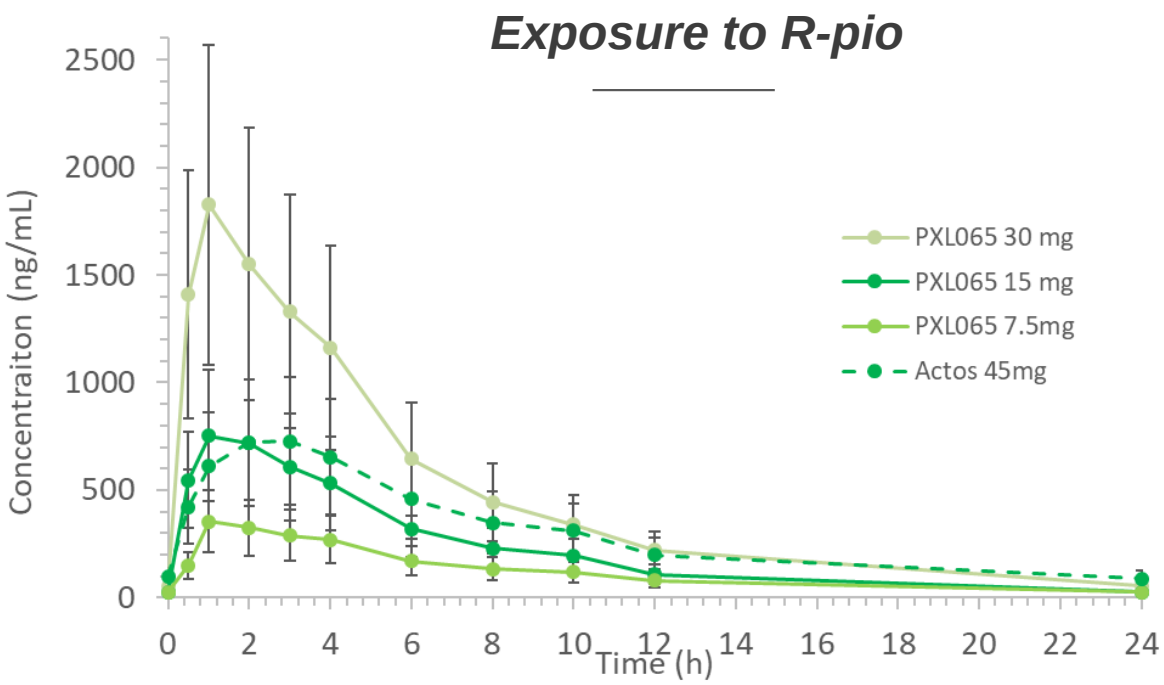
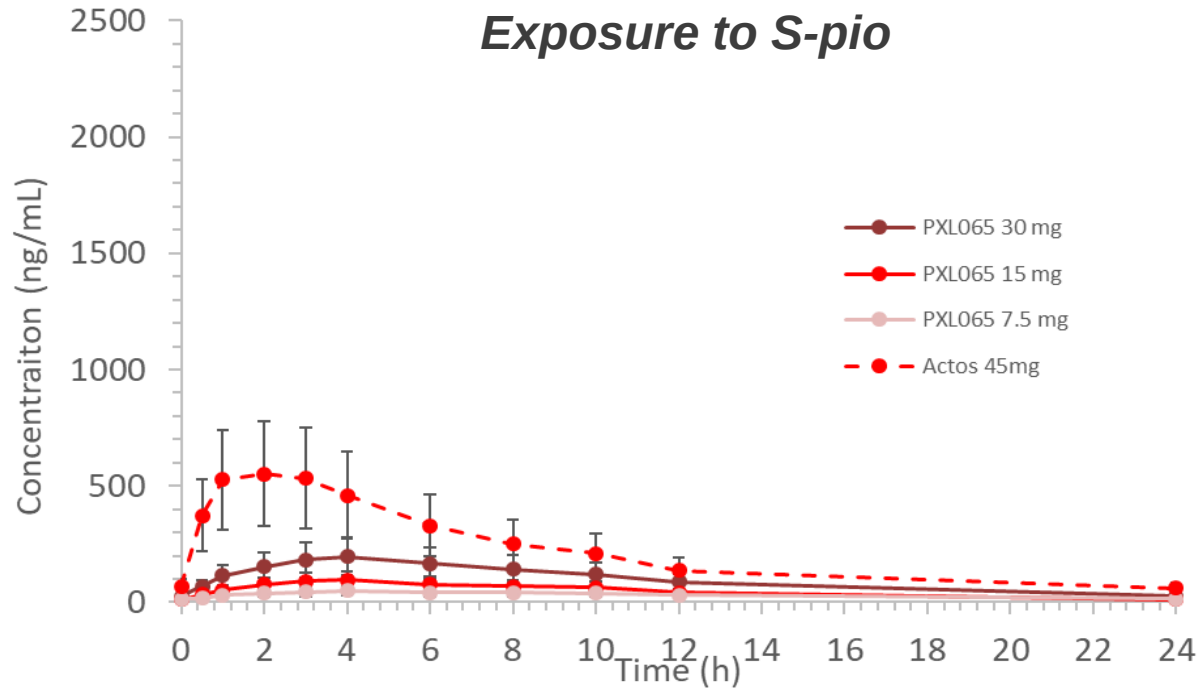
PXL065 Phase 1 Study Results

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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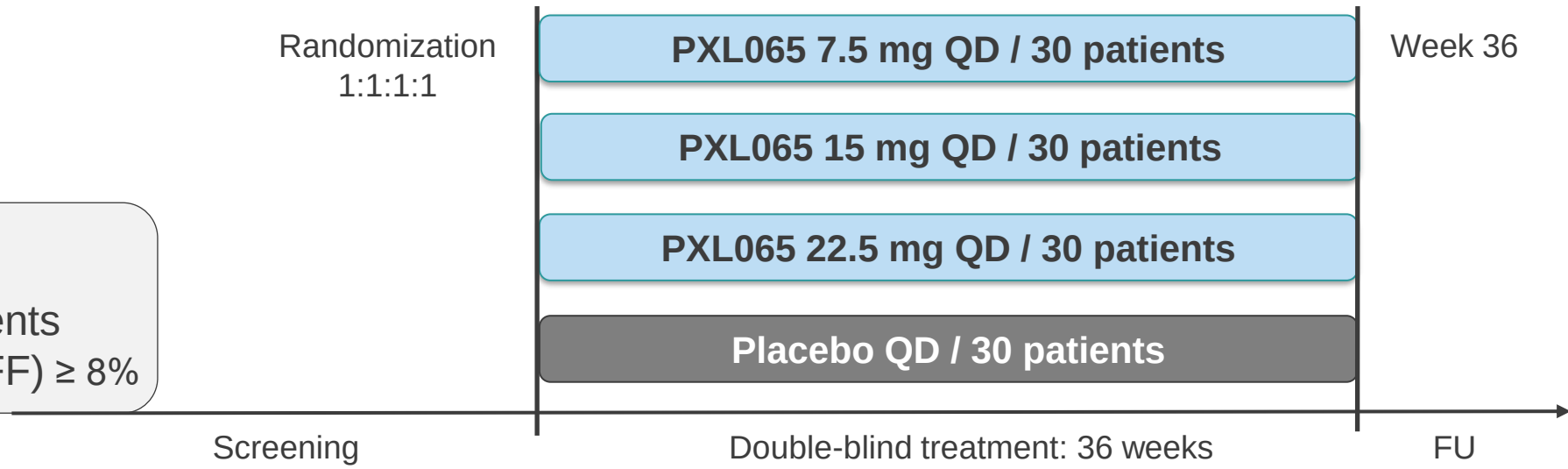
PXL065 Ongoing Phase 2 in Biopsy-Proven NASH Patients

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

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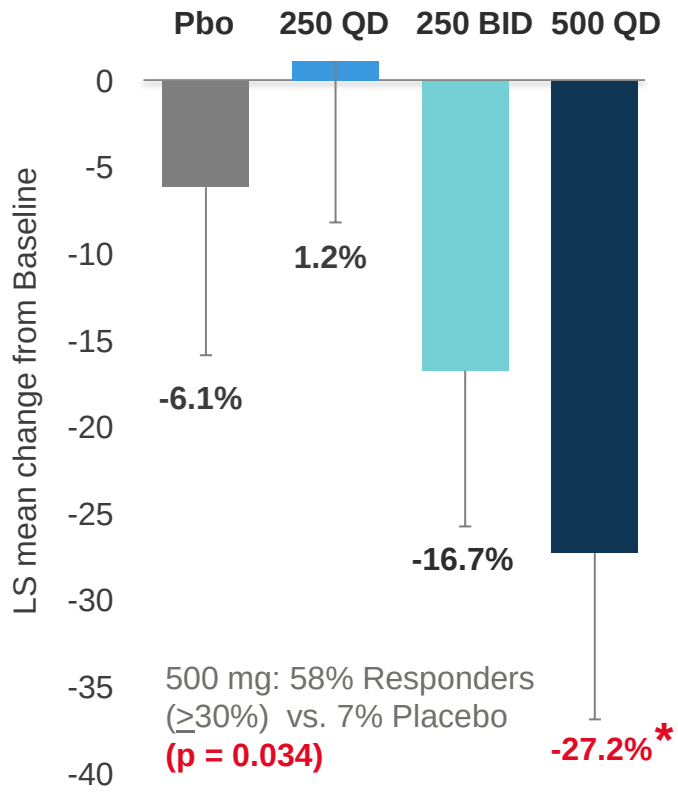
PXL770 – NASH Summary

Phase 2a – Results in NAFLD Patients with Diabetes

- First direct AMPK activator studied in human disease
- Well tolerated, 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

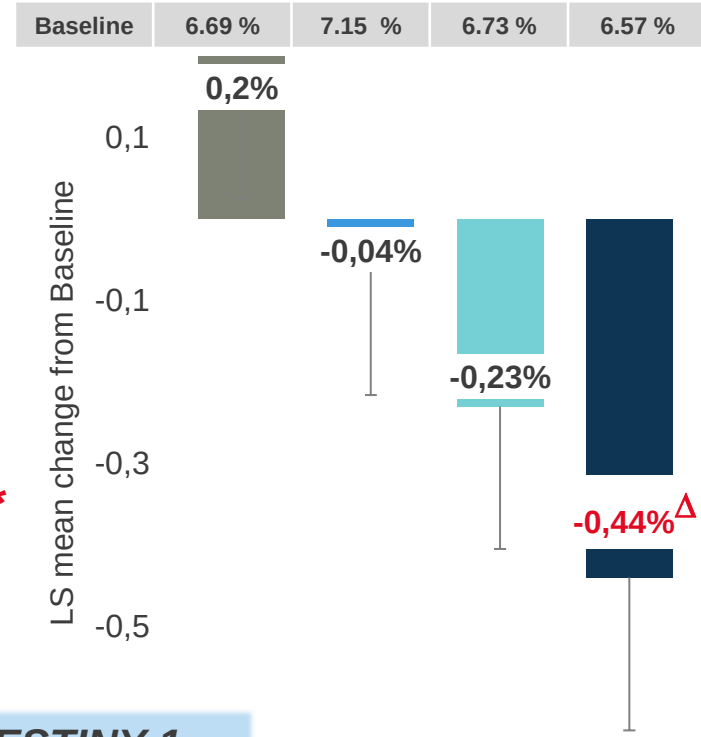
Liver Fat Content

(% Change from baseline)



STAMP NAFLD

HbA1C *(% Change from baseline)*



Future development in NASH to be evaluated pending PXL065 DESTINY-1 Phase 2 data in NASH and Phase 2 POC results in ALD

* p = 0.004 vs. baseline
Δ p = 0.01 vs. placebo

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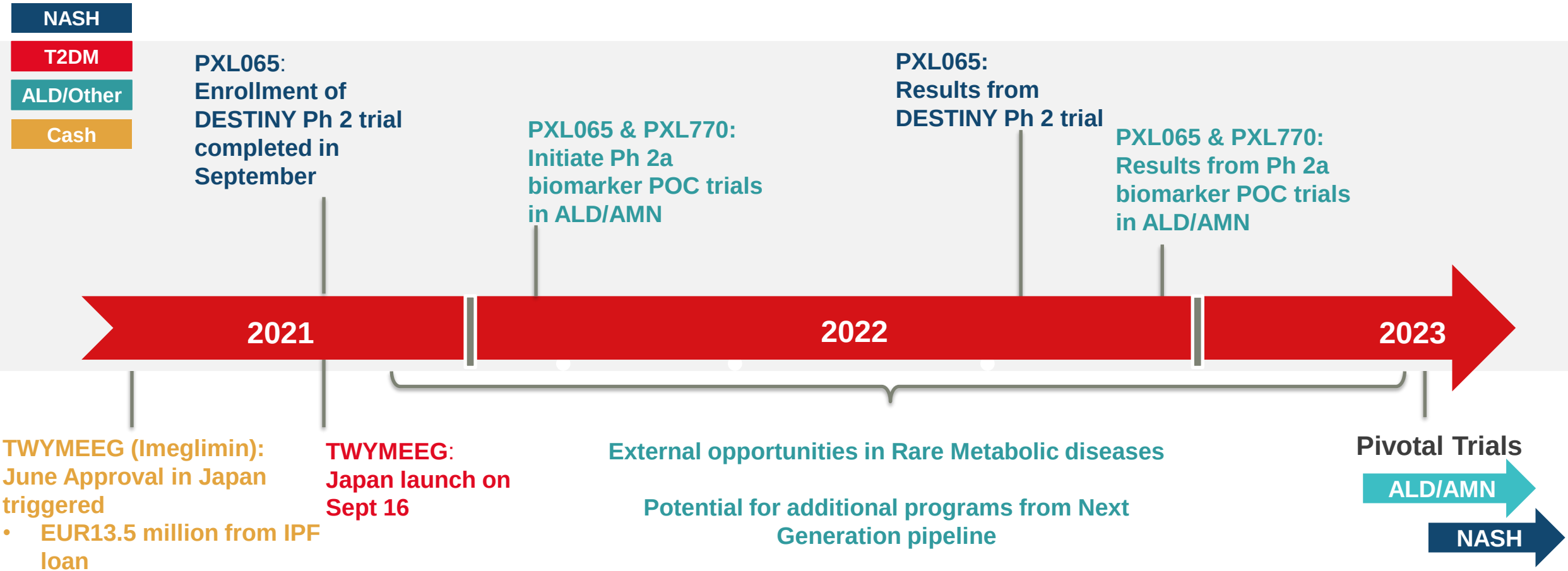
Upcoming Milestones

Near-Term Milestones to Drive Poxel's Growth



1. Currency exchange rate at the date of the approval (23 June 2021).

Summary and Investment Highlights

- **TWYMEEG® (Imeglimin) approved** for Type 2 Diabetes in Japan
  - Sumitomo Dainippon Pharma, #1 diabetes company in Japan, launch Sept 2021
  - Potential sales-based payments and escalating royalties on net sales
  - US/Europe: exploring options to move the program forward into Phase 3
- **Strategic focus on rare metabolic indications and NASH**
  - ALD: PXL065 and PXL770 Phase 2a biomarker Proof-of-Concept results by year end 2022; potential to advance into pivotal trial
  - NASH: PXL065 Phase 2 results anticipated Q3 2022; option to advance either PXL065 or PXL770 as oral, first-in-class addressing large market opportunity
- **Cash & cash equivalents: EUR 37.2 million (USD 43.2 million) as of 9/30/2021**
  - EUR 13.2 million[1] approval milestone received in Q3 2021 from Sumitomo Dainippon Pharma
- **Highly experienced management team** with extensive metabolic R&D and business expertise & track record in US, EU and Japan

Appendix

Key Financial & Shareholder Information

Market data

Ticker: POXEL

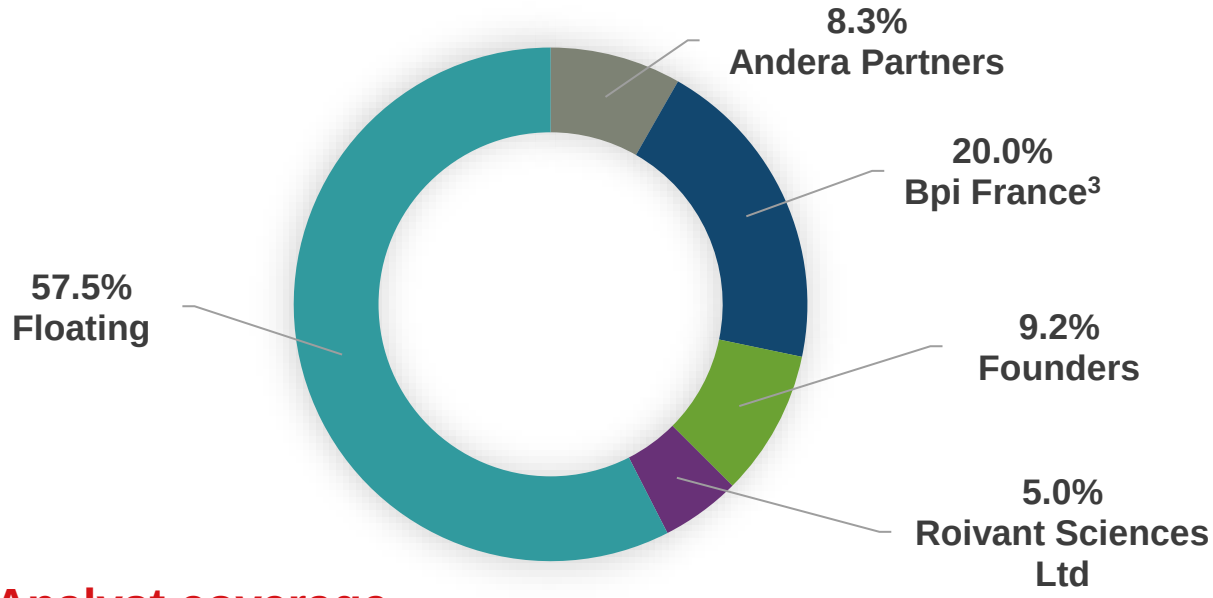
ISIN: FR0012432516

Number of shares: 28,703,692[1]

Key financials

- As of 09/30/21 cash & cash equivalents: **EUR 37.2 million (USD 43.2 million)**

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. As of November 2, 2021.
2. At the date of the presentation, based on the Company's knowledge.
3. And affiliates.