Transforming care for people with recurring kidney stones

by treating enteric hyperoxaluria

Oxidien™ Pharmaceuticals

Oxidien Pharma: addressing enteric hyperoxaluria with state-of-the-art technology

Enteric hyperoxaluria – unmet medical need (1)

Hyper-oxal-uria (high oxalate in urine) is associated with recurrent kidney stones, inflammation, progression of CKD and ESRF and cause ***significant morbidity***, costly monitoring and interventions.

OX1 – state-of-the-art technology (2)

Next generation, higher efficiency, oxalate-degrading enzyme to degrade insoluble oxalate – first product to demonstrate clinically meaningful effect in healthy volunteers at modest hyperoxaluria as proof-of-mechanism. Demonstrated good safety profile and has received encouraging FDA feedback ahead of phase 2 program. Patents granted or pending in major markets.

Substantial market opportunity (3)

There are currently ***no approved therapies*** adequately treating enteric hyperoxaluria – markets estimated at ***$2B+ US*** and ***$5B+ worldwide***.

Team (4)

Founders/management have deep technology expertise in technology and have assembled a world-class Scientific/Medical Advisory Board and Strategic Advisory Board.

Raising $6 million in series A to get to Phase 2 readiness

Disease Overview
Development Landscape

Where Oxidien Focuses

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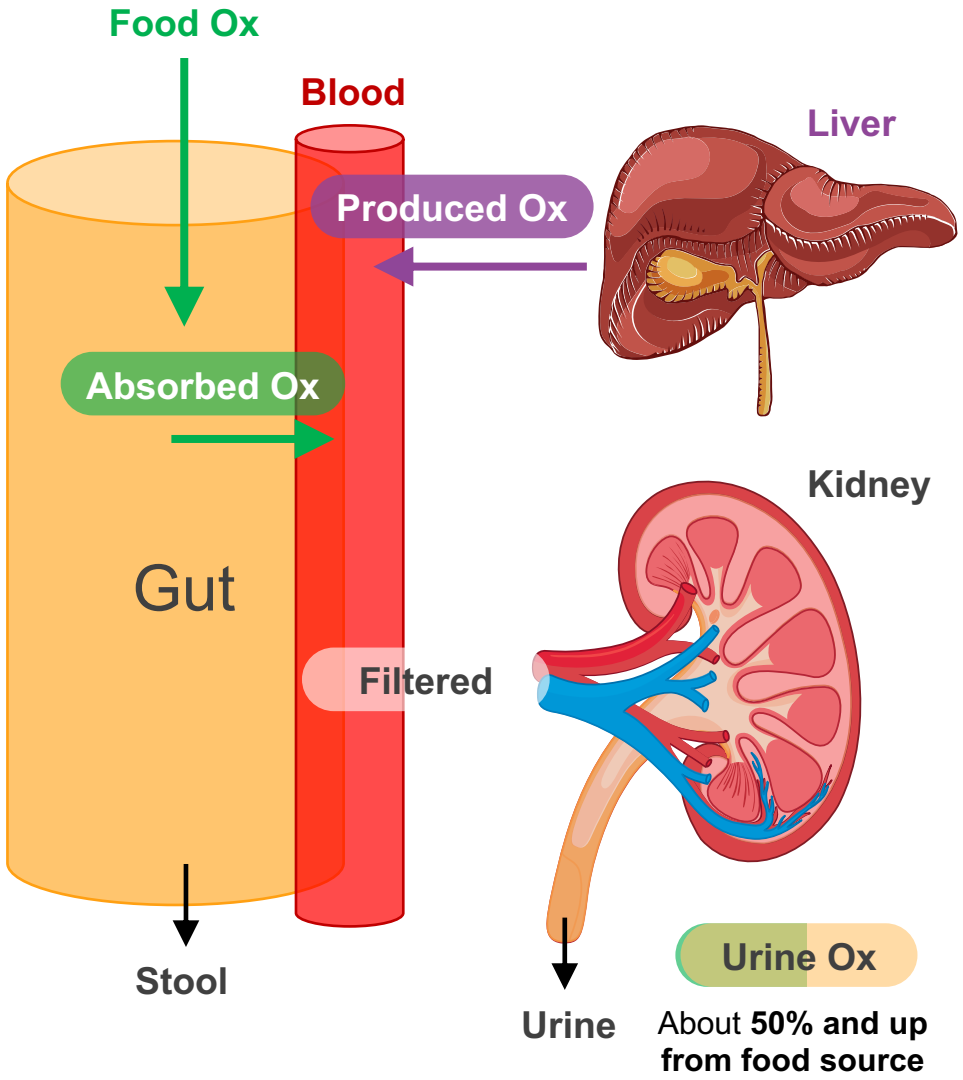
Two key sources of oxalate – same outcome



Simplified scheme of oxalate production and movement in the gut-kidney axis.

1 Oxalate produced in the liver → Primary hyperoxaluria

2 Oxalate absorbed from food → Secondary hyperoxaluria

- **Both are similar in its presentation and symptoms**:
  - ✓ crystallization causing a pro-inflammatory cascade,
  - ✓ oxalate nephropathy,
  - ✓ recurrent nephrolithiasis (stones)

1. Hatch, M., Freel, R.W. Urol Res (2005) 33:1-16
2. Jaeger, P., Robertson, W.G., Nephron Physiol (2004) 98:64-71
3. Bhasin, B., Urekli, H.M., Atta, G. M. *World J Nephrol.* 2015; 4(2): 235-244

Oxalate nephropathy = injury to kidney tubules and interstitial fibrosis due to oxalate deposits
Nephrolithiasis = kidney stones

The hyperoxalurias: diseases with significant morbidity

There are two types of hyperoxaluria:
(1) primary hyperoxaluria (PH) and **(2)** secondary hyperoxaluria (SH)

Primary hyperoxaluria

Sub-indication	Onset	Pathology	Clinical features
Type I, II, III (ultra-rare)	Pediatric	• Autosomal recessive inborn error of metabolism. • Oxalate excretion 90-500mg/24h (up to 10x normal levels)	• Recurrent nephrolithiasis (kidney stones), • nephrocalcinosis, • oxalate nephropathy, • end-stage renal failure (ESRF), • high mortality.

Secondary hyperoxaluria

Sub-indication	Onset	Pathology	Clinical features
Enteric hyperoxaluria (EH) and Idiopathic hyperoxaluria (IH)	Adult	• Oxalate over-absorption due to underlying malabsorptive condition (enteric) or unknown cause (idiopathic). • Oxalate excretion 45-130m/24h ***(up to 3x normal)***	• Recurrent nephrolithiasis (kidney stones), • obstruction, inflammation, • oxalate nephropathy, • loss of kidney function (ESRF), ***(enteric is the more severe form)***

[1] Bhasin, B., Urekli, H.M., Atta, G. M. *World J Nephrol.* 2015; 4(2): 235-244
[2] Ermer, T., Echardt, K., Aronson, P.S., Knauf, F. *Curr Opin Nephrol Hypertens*. 2016; 25(4): 363-371
[3] Waikar, S.S., *JAMA Intern Med*. 2019; 179(4):542-551
[4] Wyatt, C.M., *Kidney Int.* 2020; 97:1070-1073

Nephrocalcinosis = calcification in the kidneys
Oxalate nephropathy = injury to kidney tubules and interstitial fibrosis due to oxalate deposits

We focus initially on Enteric Hyperoxaluria (EH)

Standard of care in enteric hyperoxaluria is mainly dietary recommendations and targeted therapies addressing other urine abnormalities, and, in the case of EH, calcium supplements.

Treatment strategies in Enteric Hyperoxaluria

- Dietary intervention: high fluid intake, ***oxalate restriction***, decreased sodium and fat intake
- Targeted therapies: thiazide diuretics, potassium citrate, ***calcium supplementation***

Enteric hyperoxaluria patients experience recurrent kidney stones and loss of kidney function and ***standard of care does not adequately prevent later stage costly interventions.***

- Shockwave lithotripsy, ureteroscopy, percutaneous nephrolithotomy
- Dialysis, transplantation

There are no approved therapies available for these patients

[1] Bhasin, B., Urekli, H.M., Atta, G. M. *World J Nephrol.* 2015; 4(2): 235-244
[2] Ermer, T., Echardt, K., Aronson, P.S., Knauf, F. *Curr Opin Nephrol Hypertens*. 2016; 25(4): 363-371
[3] Waikar, S.S., *JAMA Intern Med*. 2019; 179(4):542-551
[4] Wyatt, C.M., *Kidney Int.* 2020; 97:1070-1073

Enteric hyperoxaluria patient populations in the U.S. and worldwide

Total potential U.S. patient population approximately 250,000* of which 100,000* are stone formers

5,000*	15,000*	30,000*	50,000*	150,000*
Short Bowel Syndrome	Chronic Pancreatitis	Celiac Disease	Intestinal Bowel Disease, Crohn's Disease and Ulcerative colitis	Roux-en-Y Gastric Bypass

Worldwide estimated patient population approximately 1M EH^ of which 400,000# are stone formers

Estimated enteric hyperoxaluria market opportunity in the US is $2B+ and worldwide $5B+

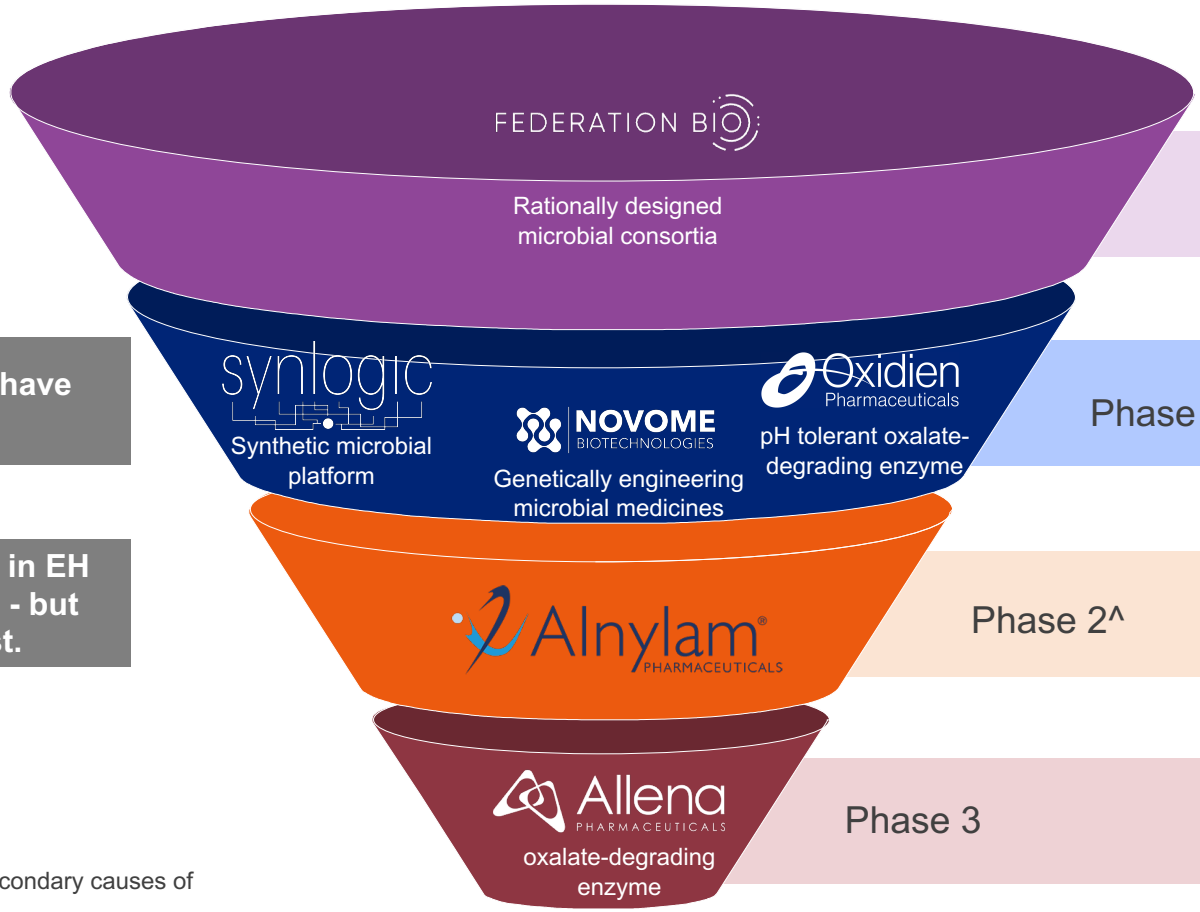
* Poster presented at Kidney week 2019: SA-PO-276. The 52nd Annual Meeting of the American Society of Nephrology. November 2019. Washington, D.C.
^ DR-037 Global Prevalence of Secondary Hyperoxaluria report on file.
# 1,000,000 EH globally x (100,000/250,000) = 400,000 stone formers globally (100,000 in the U.S. and 300,000 in the rest of the world)

Competitive landscape in secondary hyperoxaluria

Two types of modalities (1) oral enzymes (2) microbial platforms

Regulatory pathway for oral enzymes have been clarified.

Only product to show significant effect in EH clinical trials to date is an oral enzyme - but activity in GI-tract has been modest.



GI-tract = gastrointestinal tract

^ Alnylam's clinical trial excludes patients with known secondary causes of elevated urinary oxalate and/or recurrent kidney stones

Advanced clinical programs facilitate for second oral enzyme entrant

OX-1 Product

For Enteric Hyperoxaluria

Oxidien™ Pharmaceuticals

Next-generation enzyme technology for meaningful reduction in urinary oxalate

Novel Enzyme and Mechanism of Action

OX-1 is an oral enzyme.

It is a new and proprietary type of oxalate decarboxylase (OxdC) enzyme, designed to degrade oxalate to natural byproducts in the upper GI-tract.

Product Profile

A single capsule of OX-1 is taken by mouth, with meals, up to three times per day to remove oxalate in the upper GI-tract.

Advantages

High-potency formulation, unique level of stability in the upper GI-tract, much improved affinity for oxalate.

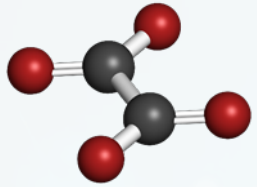
The new and favorable enzymatic profile produce differentiating clinical data with positive effect and good safety profile.

Manufacturing Process and Stability

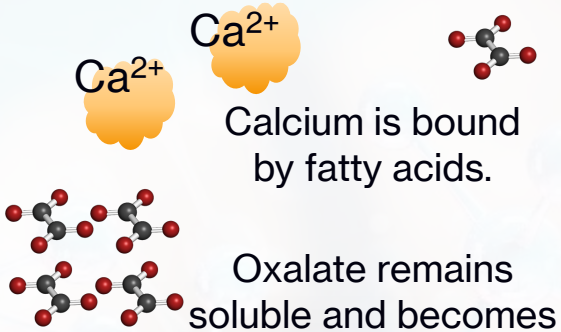
OX-1 is manufactured by recombinant expression in *e.coli*. Initial product candidate is stored at room-temperature.

Mechanism of action: degrading oxalate at its origin

EH Disease Mechanism

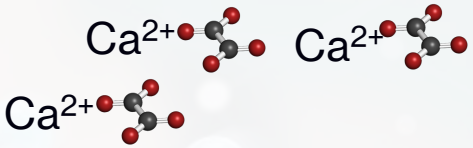


Oxalate is available in the stomach.



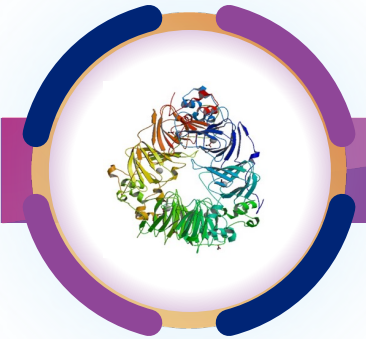
Calcium is bound by fatty acids.

Oxalate remains soluble and becomes hyper-absorbed

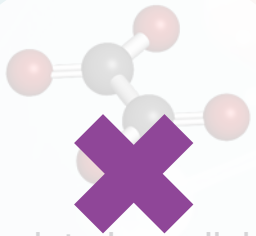


Hyper-absorption leads to calcium oxalate saturation in urine

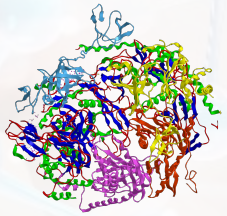
EH = enteric hyperoxaluria



OX-1 Mechanism of Action



Oxalate is available in the stomach.



OX-1 degrades oxalate to formic acid and carbon dioxide

Oxalate removal reduces hyper-absorption and hyperoxaluria.

Expected to reduce kidney stone disease progression.

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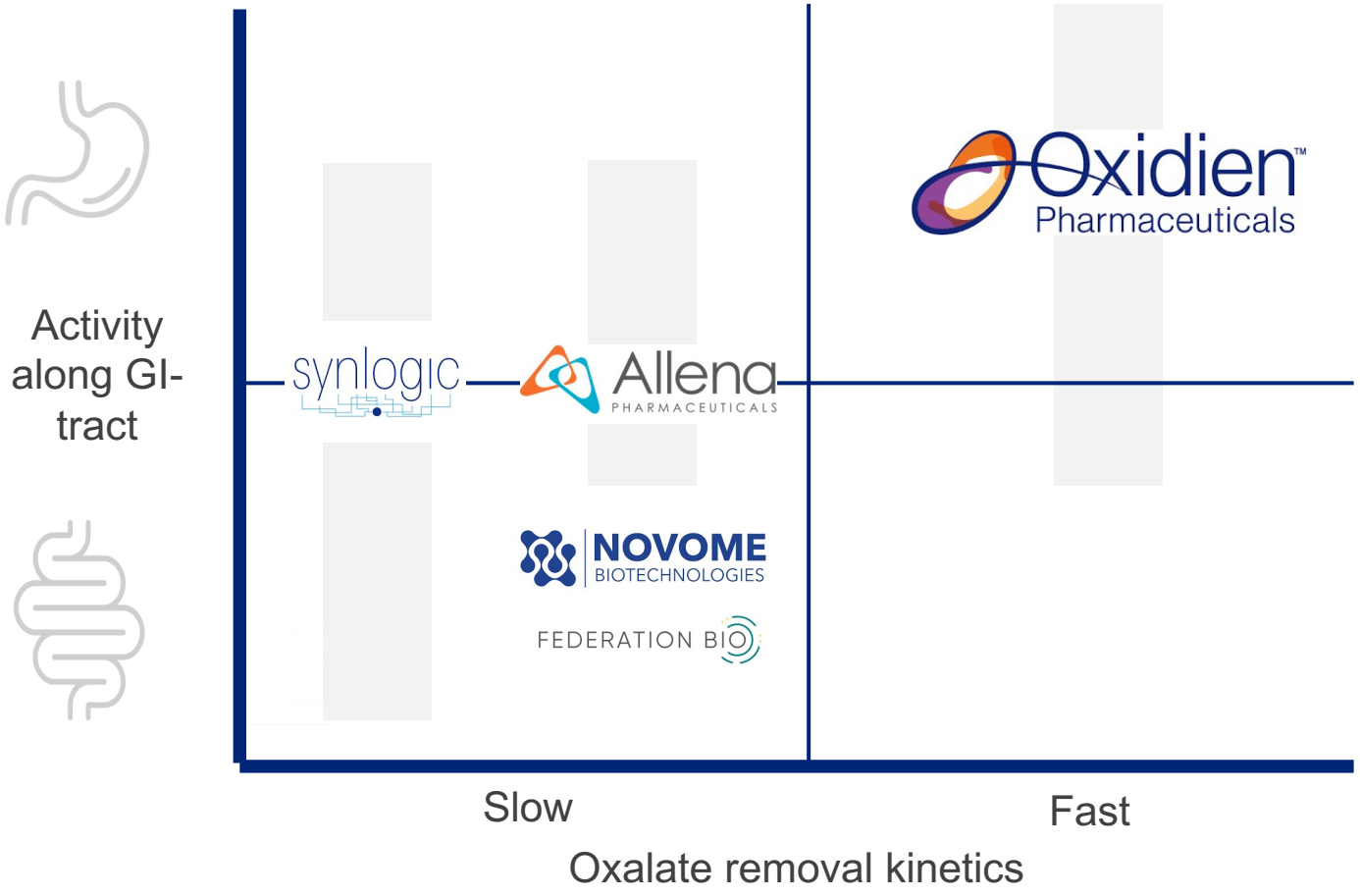
Overcoming challenges with slow kinetics and low stability in the stomach

Most products in development have slow kinetics which limits the applicability across different *in vivo* conditions.



Faster Kinetics 1

Faster kinetics than other products in development allow OX-1 to reduce oxalate in a presence of calcium making it effective in a wider range of *in vivo* conditions.

OX-1 has demonstrated 50-90% reduction in dietary oxalate in a **normal oxalate/normal calcium** diet *in vitro.* Corroborated by FIM study, which demonstrated 56% reduction in dietary oxalate on a high-oxalate low-calcium diet at 3000 U/day dose regimen.

Acid-tolerant 2

The acid-tolerance of OX-1 allows for improved stability in the GI-tract and allows for oxalate degradation where oxalate has high bioavailability (in the stomach).

FIM = First-In-Man
In vivo = in a living system
In vitro = outside of a living system

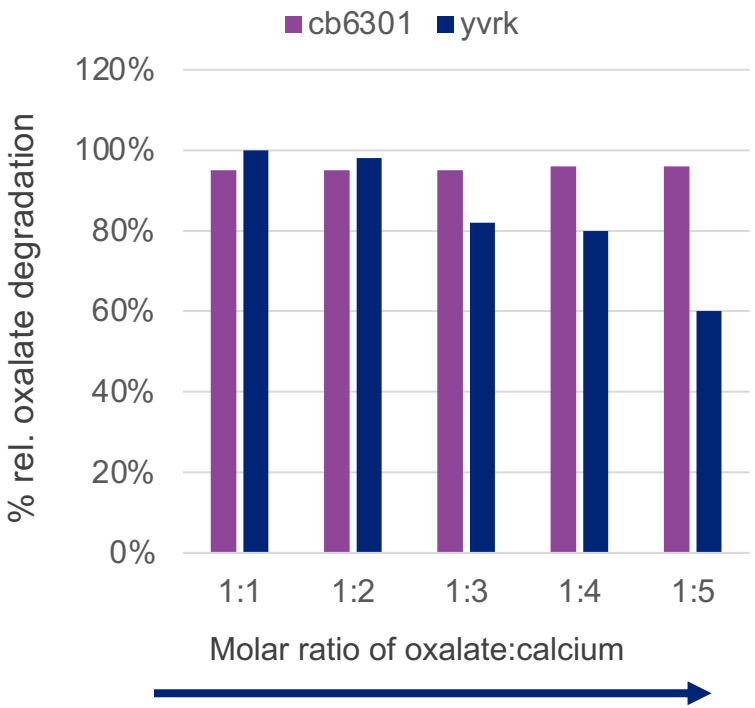
Competitiveness to other oral enzymes

Rate of reaction

	Cb6301 (OX-1)	Yvrk[1]
Fed state [Ox] (mM): approx. **0.5 – 1.0**		
Km (mM)	**0.3**	**16.4**
% relative activity in fed state	**100%**	**3-6%^**
Condition	pH 2.7, 37C (worst case conditions)	pH 4, 26C (best case conditions)

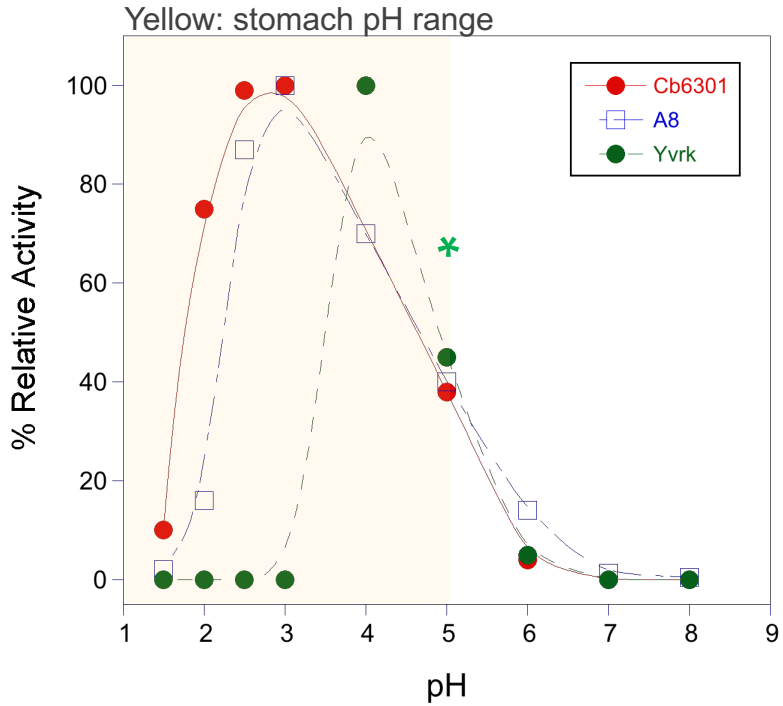
At normal fed state, Yvrk likely exhibits heavily depressed level of activity[2].

Insoluble oxalate degradation



Yvrk see reduced activity with increased calcium.
(Relevant to variety of meal compositions in normal diet.)

pH activity profile



* **At higher pH OX-1 (Cb6301) still exhibit higher rate of reaction (resulting in higher total activity)**

^ (0.5/16.4=3%, 1.0/16.4=6%) relative half of its activity at Km
Km – the oxalate concentration where the enzyme exhibit half it's activity
Cb6301 = OX-1

1.Yvrk is the wild type *B. subtilis* OxDC. Reloxaliase use OxDC from *B. Subtilis.*
2.Thalji, N.K., *et al. Urol* (2011) 78:721.e13-721.e17

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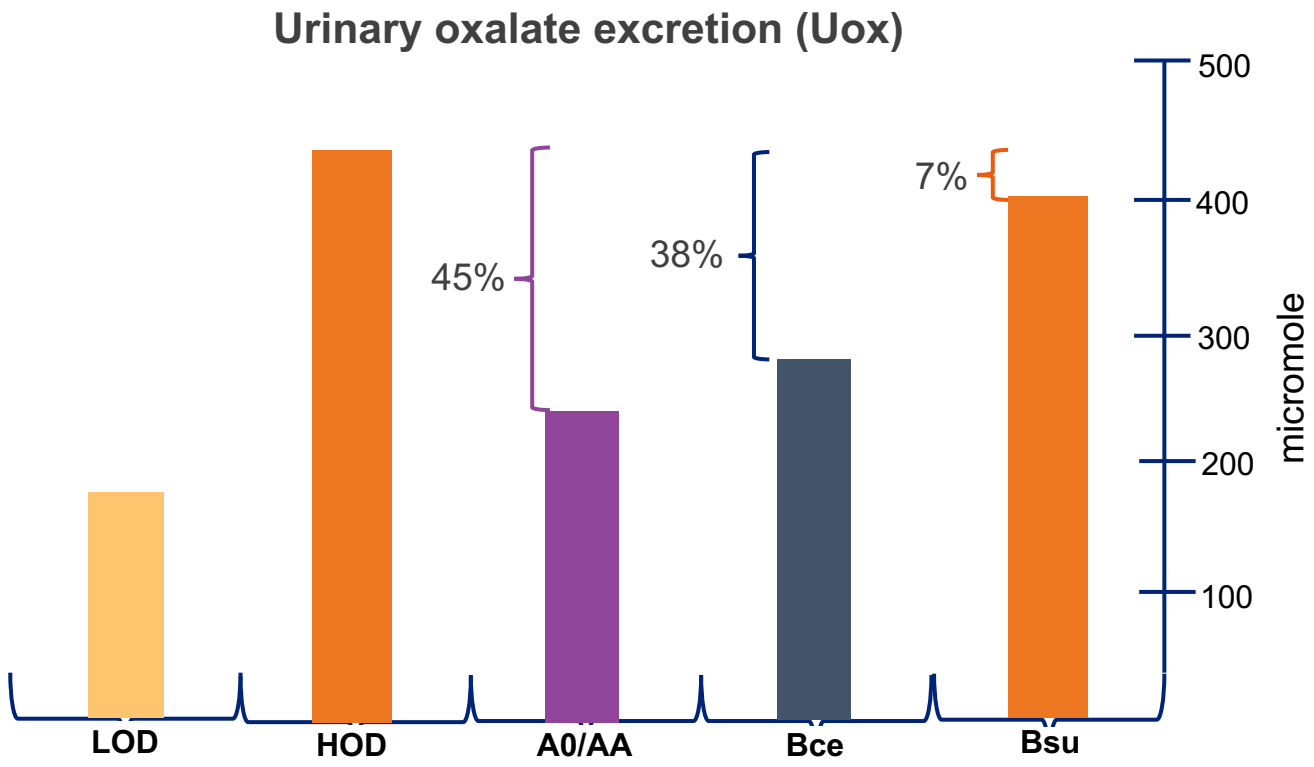
R&D Beagle Study #1

Benchmarking Other Enzymes

- Evaluated three OxDC from: 1) *B. Subtilis* (**Bsu**), 2) *B. cereus* (**Bce**), 3) *Agrocybe aegerita* (**A0**)
- 7 beagle dogs on a controlled oxalate diet during the study. Dosed 250U/meal twice per day. Urine was collected.

Competitor enzyme shows modest effect in side-by-side studies in large animals

HOD: High oxalate diet
Bce: *B. subtilis* enzyme (Oxidien's)
A0: *Agrocybe aegerita enzyme (*Oxidien's)
Bsu: the wild type *B. subtilis* OxDC. Reloxaliase use OxDC from *B. Subtilis*
LOD: Low-oxalate diet



The native *B. subtilis* enzyme reduces less urinary oxalate (7%) when compared side-by-side under identical conditions with A0 and Bce (both Oxidien enzymes), at 45% and 38%, resp.

Acid-stable and high-efficiency (low Km) enzymes differentiate from Bsu in large animal model

R&D Beagle Study #2

PoC study using controlled diet

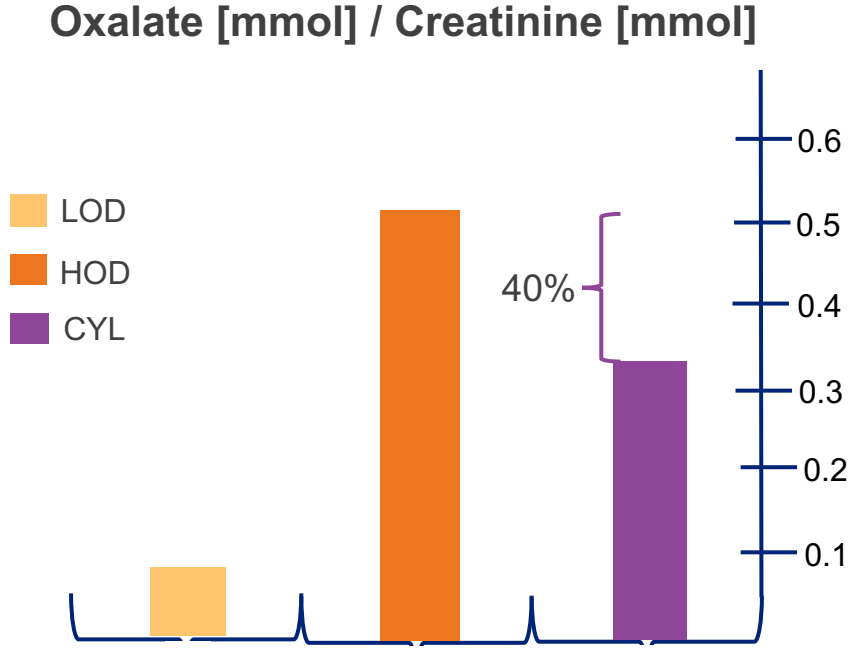
Six beagle dogs dosed two separate doses while on a controlled high oxalate diet and collecting urine.

Results:

- Product was well tolerated.
- OX-1 demonstrated significant reductions in urinary oxalate.
- Majority of gastric readings within pH 2-4.5.

Proof-of-concept study in large animals show 40% reduction in urinary oxalate

LOD=Low oxalate diet (baseline).
HOD=High oxalate diet.
CYL=OX-1 treatment at a low dose of 585U



Statistically significant reduction in urinary oxalate (Uox) (oxalate/creatinine) in beagles on a high-oxalate diet dosed 585U of OX-1 twice per day over four days.

OX-1 show predictable reduction in large animal model, at low dose, consistent with Oxidien's other enzymes

1. Sixteen Day Oral Range-Finding Study of OX1-BC and OX1-CY in Beagle Dogs. (2015). Bioanalytical Systems Inc. (BASi), final report.

Toxicology Studies

Type	GLP	Dose	Species	Design	Route	Duration TX (days)	Results
Genotoxicity Study							
Ames bacterial mutagenicity Protocol #42571 (2016a)	Yes	0.5, 0.75, 1.0, 1.58, 2.5, 5.0, 7.5, 15.8, 50, 158, 500, 1580, 5000 µg/plate	*S. typhimurium*, TA1535, TA1537, TA98, TA100; *E. coli* WP2 uvrA	5 strains 5 positive controls: Sodium azide, ICR 191 Acridine, Daunomycin, Methyl methanesulfonate, 2-Aminoanthracene	---	---	OX-1 was found to be non-mutagenic
Repeat Dose Toxicity Studies							
Repeat Dose Protocol #40954 (2015)	No	Low dose: 118 mg/kg/day Intermediate dose: 235 mg/kg/day High dose: 475 mg/kg/day Vehicle control: 0 All in solution of 50 mM Arginine (pH 9.5) All administered at 10 mL/kg/day	Sprague-Dawley CD IGS Rat	4 groups: 5 male and 5 female rats per group (n=40)	Oral gavage	14	No adverse events at any dose level up to 475 mg/kg/day
Repeat Dose Protocol #41793 (2016b)	Yes	Low dose: 118 mg/kg/day Intermediate dose: 235 mg/kg/day High dose: 475 mg/kg/day Vehicle control: 0 All in solution of 50 mM Arginine (pH 9.5) All administered at 10 mL/kg/day	Sprague-Dawley CD IGS Rat	4 groups: 10 male and 10 female rats per group (n=80)	Oral gavage	90	No adverse events NOAEL = 475 mg/kg/day

NOAEL in sub-chronic study has approximately a 50-fold margin to the highest dose anticipated in phase 2

OX1-CY a.k.a. OX-1

1. Ames bacterial mutagenicity report
2. OX1-CY: A 14-Day Oral Toxicity Study in Rats. (2015). Product Safety Labs (Eurofin), final report.
3. OX1-CY: A 90-Day Oral Gavage Study in Rats. (2016). Product Safety Labs (Eurofin), final report.

First-in-man trial demonstrates safety and biological activity at low dose

Meeting all pre-determined end-points at low dose levels

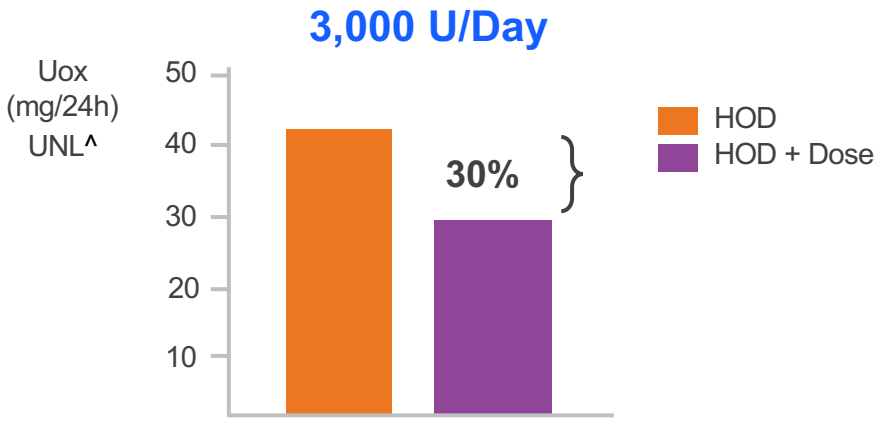


Figure: Significant reduction (p<0.0001) in average urinary oxalate as compared to placebo. ***HOD=High Oxalate Diet.***

UOx / 24h = Urinary oxalate per day
HOD = High Oxalate Diet

Study Design	• 33 normal healthy volunteers • Single U.S. site, in-patient study • Randomized • Double-blind • Placebo-controlled • Diet-controlled (high-oxalate, low-calcium diet) • Cross-over w. 2-day dose periods, 2-day wash-out
Primary end-point **Secondary end-point**	• Within-subject difference in mean 24-hour Uox excretion • Mean difference in 24-hour Uox excretion between product and placebo • Within-subject difference in mean 24-h Uox excretion between dose and placebo • To evaluate safety and tolerability.
Study Implementation	• Day -2 to -1: start of controlled diet • Day 1 to 2: 1st dose period • Day 3 to 4: wash-out • Day 5 6o 6: 2nd dose period • Day 7: follow-up
Results	• Highly statistically significant response (p<0.0001) with 29% reduction in Uox from baseline and 24% treatment difference at 7x lower dose than dose levels of similar class products. • No Serious Adverse Events, no product-related Adverse Event, all Adverse Events mild to moderate.

[1] Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290.

Convincing reduction in urinary oxalate achieves meaningful clinical outcomes and reduces development risk

First-in-man trial demonstrates safety and biological activity at low dose (cont.)

Favorable Safety Profile in Healthy Volunteers

- No Serious Adverse Events
- No product-related Adverse Events (AE)
- All AEs were mild or moderate
- Safety profile in healthy volunteers similar to Allena Pharmaceuticals' currently in second Phase 3 trial

Event Description	OX-1 (active)	Placebo
Musculoskeletal stiffness	0	2
Abdominal pain	1	0
Diarrhoea	1	0
Dyspepsia	0	1
Nausea	1	0
Vomiting	1	0
Headache	1	0
Dysmenorrhea	1	1
Overall	**6 (4 NHV)^**	**4 (3 NHV)**

^ One NHV reported 3 AEs, all occurring in conjunction with constipation

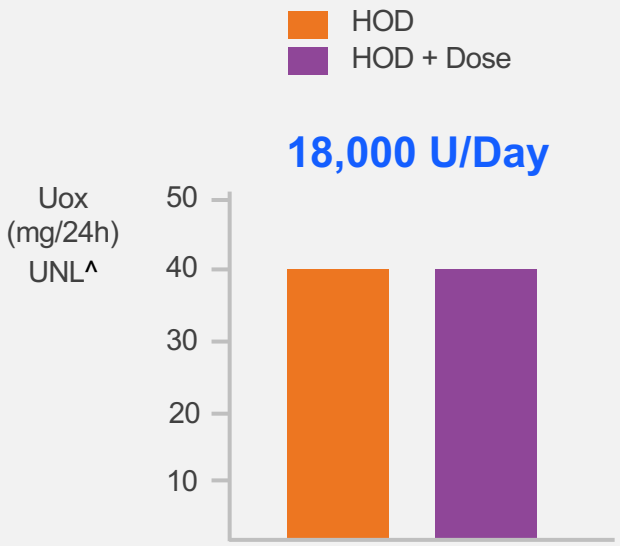
NHV = Normal Healthy Volunteer
AE = Adverse Event

[1] Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290.

Presented studies to date of moderate hyperoxaluria in healthy volunteers

First-in-class ALTU-237, now called ALLN-177*[1]

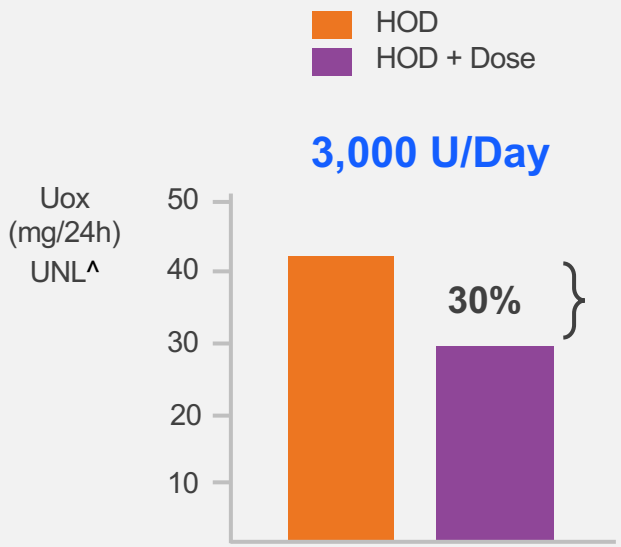
Graph re-created based on public data.
Baseline Uox disclosed as upper limit of normal.



** No substantial or dose-dependent reduction in urinary oxalate levels at the doses administered

OX-1 (2020)[3]

Graph re-created based on the published data.



OX-1 reduced urinary oxalate well into normal range – we attribute this to the increased affinity to the substrate

OX-1 exhibits:

- fast kinetics (activity at lower substrate/oxalate levels).
- acid-tolerance - stability in the stomach.
- removal of over half (56%) of dietary oxalate at 7x lower dose than other products of the same class.
- highly statistically significant response ($p<0.0001$) with 29% reduction in Uox from baseline.

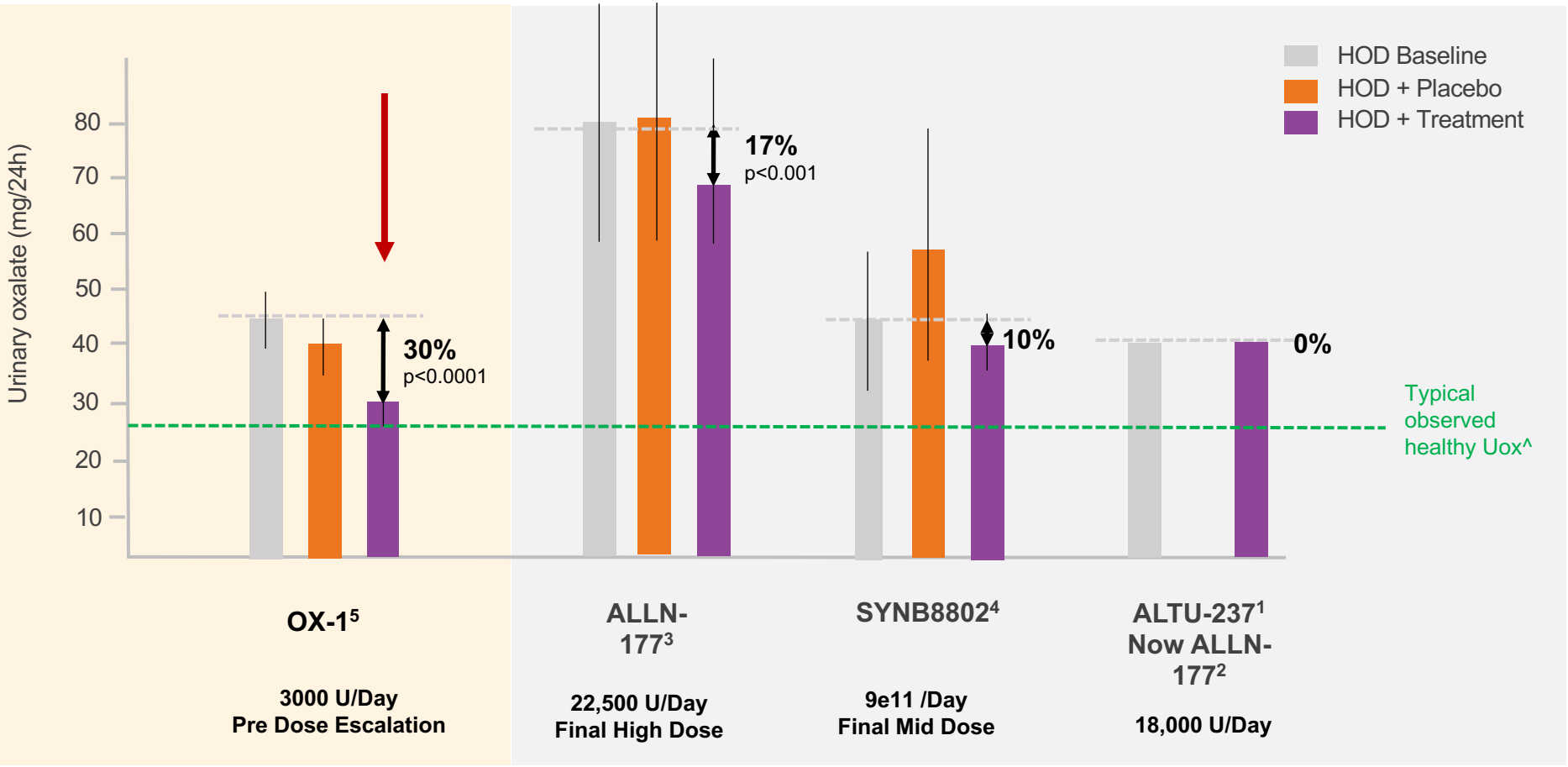
These data:

- proves biological activity in the stomach.
- supports therapeutic concept.
- indicates competitive activity at more severe hyperoxaluria.

* ALTU-237, now called ALLN-177 per Allena S-1
^ ULN-upper limit of normal, normal is considered 40mg/24h urinary oxalate excretion.
1. Atti, K.M., and Grujic, D. Anion Transporters and Oxalate Homeostatis: From Genes to Diseases December 8-9, 2008.
2. Langman, C.B., et al. *Am J Nephrol* 2016; 44:150-158
3. Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290.

OX-1 - only clinical stage product to bring subjects well into normal range

Comparison of all dietary hyperoxaluria studies by industry to date



- OX-1 still has opportunity to evaluate higher doses – competitors have locked in dose.
- ALLN-177 and OX-1 study were cross-over trials. SYNB8802 study had a separate placebo cohort and low power – saw difference in relative effect compared to placebo vs. absolute effect in the treatment arm.

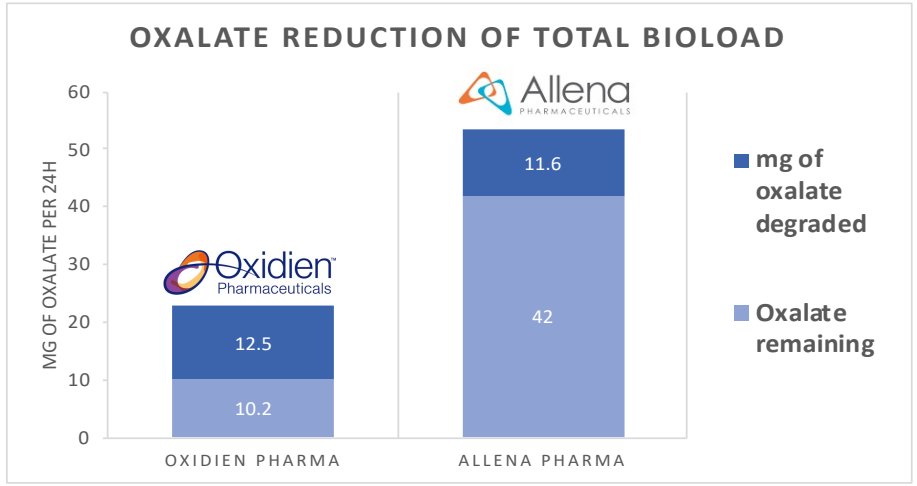
^ Historically Uox in HV is <40mg/24h. Examples: Langman 2016 (27mg), Quintero 2020 (20mg), Captozyme 2018 (28 mg).

1. Atti, K.M., and Grujic, D. Anion Transporters and Oxalate Homeostatis: From Genes to Diseases December 8-9, 2008.
2. Allena Pharmaceuticals S-1. Describes the license of ALTU-237 “now called ALLN-177”
3. Langman, C.B., et al. *Am J Nephrol* 2016; 44:150-158
4. SYBX Corporate presentation Mar 2021 03 25 2021
5. Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290.

Comparison to Allena Pharmaceuticals' Efficacy Data in Healthy Volunteers[2]

	Dose / Day	% Reduction	Effect Rate
Oxidien: OX-1[1]	3,000 *Units*	55%	94% (31/33)
Allena Pharma ALLN-177[2]	22,500 *Units*	22%	63% (19/30)

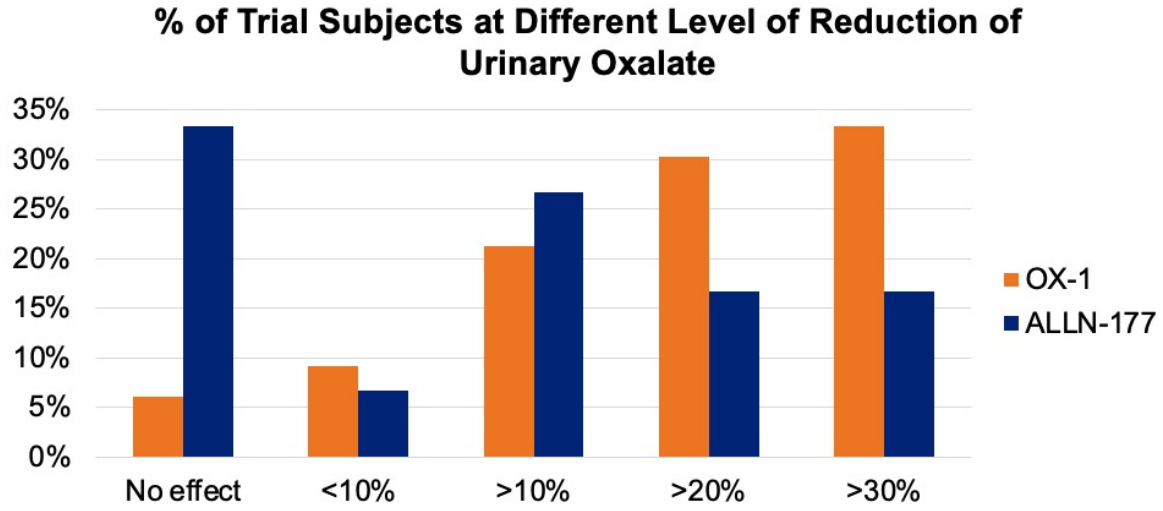
At about 7-fold lower dose, OX-1 demonstrates twice the reduction in oral oxalate bio-load^



^ Oral bio-load = amount of oxalate ingested.
Determined through: [(mg Uox per 24h on high-oxalate diet) - (mg Uox per 24h on high-oxalate diet + dose)] / [(mg Uox per 24h on high-oxalate diet) - (mg Uox per 24h at baseline)]

1. Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290
2. Langman, C.B., et al. *Am J Nephrol* 2016; 44:150-158

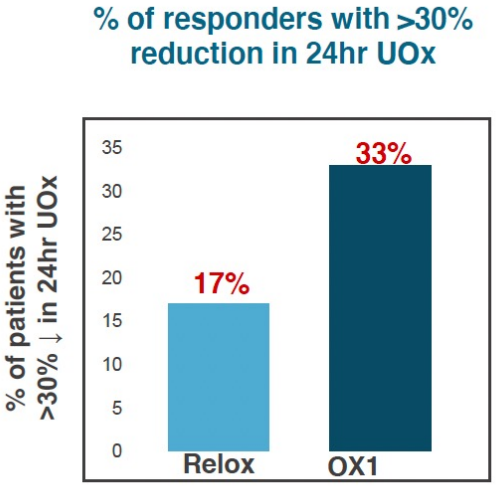
Comparison to Allena Pharmaceuticals' Efficacy Data in Healthy Volunteers[2] (cont.)



Clear differentiation in proportion analysis (secondary end-point in Urirox-1, 2 studies)^

Data is from the same two comparative studies as presented differently on slide 21 but here showing % of trial subjects at different levels of reduction in urinary oxalate.

Note that OX-1 is at more than 7-fold less dose than the comparator.



1. Quintero E. *et al. KIDNEY360* (2020); 1:1284-1290
2. Langman, C.B., et al. *Am J Nephrol* 2016; 44:150-158

Summary: OX-1 clinical experience to date

Safety outcomes

- This first-in-man (FIM) study did not attribute any adverse events to the investigational product (OX-1 in sachet format).
- All adverse events but one were mild, one was moderate, and all were resolved by end of study.
- All subjects completed study and there were no changes to dosing.

Efficacy outcomes

- Statistically significant within-subject difference between product and placebo and product and baseline.
- Statistically significant mean difference between product and placebo and product and high-oxalate baseline ($p<0.0001$) with 29% reduction from baseline and 24% treatment difference.
- There were no changes to any of the other urinary parameters tested, besides oxalate: citrate, calcium, magnesium, creatinine and uric acid.
- The clinical data from this FIM study are corroborated in other clinical data, on file.

Data package to date - meaningful reduction in urinary oxalate at a very low dose portrays a large therapeutic window for study in Phase 2 dosing studies

OX-1 overall development and regulatory profile to date

Strain development, cell banks, assays, toxicology data

- Recombinant strain development successfully completed and cell banks successfully released for initial use.
- Activity assay validated. Purity assay developed (underdoing validation).
- Toxicology studies show NOAEL (no-observed-adverse-effect-level) at 50-fold safety margin as compared to highest dose to be evaluated in the clinic. All tox work required for Phase 2 completed with no tox end-points met.

Manufacturing/ CMC

- Drug substance process successfully transferred to cGMP manufacturer and successfully scaled.
- Sachet product form (used in first-in-man study) ready to be re-formulated into oral dosage form.
- Have R&D stability of spray-dried dispersion (SDD) at room temperature.
- One technical run and two cGMP runs successfully produced at scale, tested, and released.

Clinical data

- First-in-man study completed with sachet form of OX-1. Met all pre-determined end-points in healthy volunteers fed a controlled diet.
- Clinical data prove biological activity in the stomach: a 56% removal of oral bio-load/dietary oxalate resulted in a clinically meaningful 29% reduction ($p<0.0001$) in urinary oxalate from baseline at a daily dose of 3000U.

Regulatory communication

- Receipt of written responses from PIND Type B meeting request.
- The encouraging responses provide additional clarity on the development program and regulatory pathway.
- Agency expressed consideration for “substantial changes in urinary oxalate” as conditional end-point for registrational trial.

CMC, clinical data and regulatory feedback to date provides an encouraging, risk mitigating development path heading into Phase 2

OX-1 for Enteric Hyperoxaluria

Next steps: phase 2 program, CMC and clinical trial

Oxidien™ Pharmaceuticals

Designing phase 2 with a target product profile for enteric hyperoxaluria

Target Patient Population	Initial: adults with enteric hyperoxaluria and recurrent kidney stones with underlying GI disorders and preserved renal function.
Safety	Tolerability consistent with oral enzyme: mild to moderate GI disturbances.
Efficacy	**Initial: Clinically meaningful reduction in urinary oxalate.** Long-term: Reduced kidney stone disease progression (reduced stone formation).
Dosage	**Oral solid dosage form up to 3x per day.**

Phase 2 program overall development plan

Pre-clinical

- Determine PK parameters through a single-dose non-GLP study in 36 rats (male and female).
- Determine safety pharmacology through a single-dose GLP study in 12 dogs during which we will monitor cardiac and respiratory markers up to 24h post dose and include ophthalmology parameters.
- Time schedule: not a critical path item, completed alongside DP development.

DP Development

- Transition from sachet format to oral solid dosage (OSD) with already identified contract manufacturer. Conduct stability studies and complete comparability program.
- Time schedule: critical path item – preparations and assumptions organized to complete in 12 mos. following new capital raise.

Manufacturing

- Transfer drug substance process to a new, pre-determined, manufacturer using our current cell banks. Manufacture new cGMP master and working cell bank.
- Manufacture new drug product (DP) lots and clinical trial materials.
- All manufacturers have been chosen; hence, programs are ready to start.

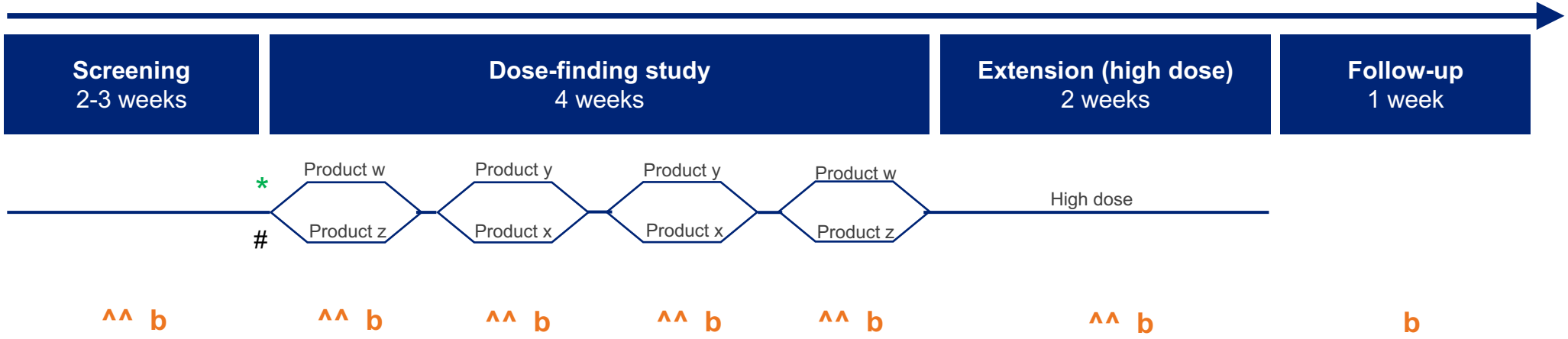
Clinical

- Goal of Phase 2 study: (1) determine dose for a registrational trial, (2) establish sufficient safety database for longer Phase 3 trials, (3) increase understanding for the safety and efficacy in the different sub-populations of EH.
- Planned Phase 2 study design is presented on the next slide.

PK = pharmacokinetic

Development plan phase 2

Placebo-controlled, double-blind, dose-finding cross-over study in enteric hyperoxaluria



Primary End-points	• Percent change in Uox excretion compared to baseline • Safety end-points
Secondary End-points	• Percent change in Uox excretion compared to placebo • Mean change in Uox excretion as compared to placebo, and as compared to baseline • Proportion of subjects with a >30% reduction from baseline in 24-hour Uox excretion • Proportion of subjects with a >25% reduction from baseline in 24-hour Uox excretion

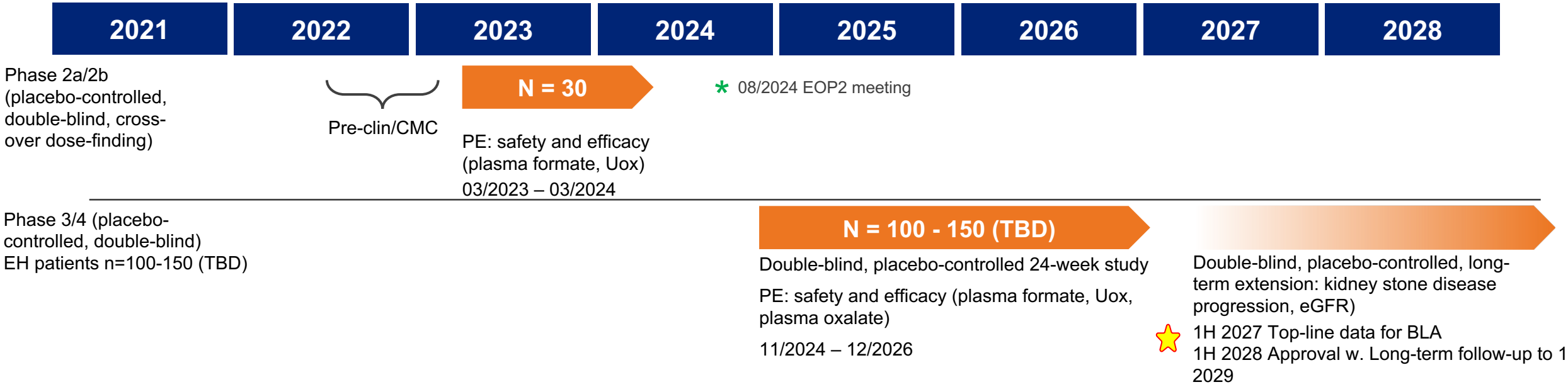
* - Randomization
\# - Graphic is simplified (cross-over has 4 different start doses)

^ - 24-h urine collection
b - blood potassium, formate, plasma oxalate

EH = Enteric hyperoxaluria
Uox = Urinary oxalate (mg/24h)
eGRF = Estimated Glomerular Filtration Rate

GOAL: Successful completion is anticipated to provide appropriate data for phase 3 design and create meaningful value for strategic corporate options

Forward development plan



EH = Enteric Hyperoxaluria
CMC = Chemistry Manufacturing and Controls
PE = Primary Endpoint
EOP = end of phase 2

Corporate Information

Organization and Business

Oxidien™ Pharmaceuticals

Management Team

Helena Cowley, MS, MBA
Chief Executive Officer

- Master's in Bioengineering, MBA from University of Florida.
- 10+ years of entrepreneurial experience; built and led Captozyme from scratch to an exit in 2019.
- Prior with Captozyme and OxThera (consulting).

Mark Mugerditchian
EVP CMC (consulting)

- 40 years in manufacturing, 15+ years as VP or EVP
- Prior successful oral enzyme OSD development
- Past PVP Biologics (sold to Takeda), NovaCardia (sold to Merck)

Loren Miller, PhD
Regulatory Affairs

- 30+ years in regulatory affairs
- Oversight and expertise to 20 approved NDAs
- Past PPD, Glaxo Wellcome, Quintiles

Strategic Advisory Board

Allen R. Nissenson, MD

- Emeritus CMO of DaVita Kidney Care
- Emeritus Professor of Medicine at the David Geffen School of Medicine at UCLA
- Authored 700+ publications in nephrology, dialysis and anemia

John G. Cooper

- 30+ years senior executive experience
- Capital raising and strategic management of development in emerging biotechnology companies.
- Strategic financing for development and commercial programs.

Scientific and Medical Advisory Board

John Lieske, MD
Nephrologist and Professor of Medicine
- Medical Dir. at Renal Testing Lab at Mayo Clinic
- Director of Rare Kidney Stone Consortium
- PI for US Urinary Stone Disease Network Recruiting Site at Mayo Clinic
- Professor of Medicine at Mayo Clinic College of Medicine

Ira Klimberg, MD
Urologist
- 25+ years of experience in clinical practice
- Principal Investigator (PI) or sub-PI on close to 300 trials
- > 75 articles in peer-review, 12 book chapters and monographs

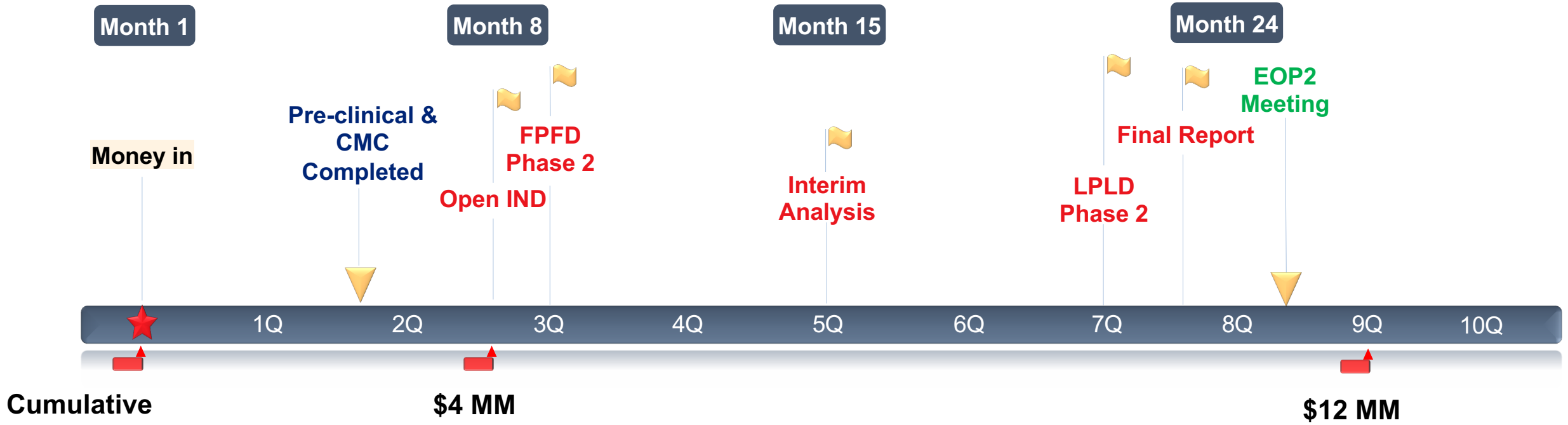
Kyle Wood, MD
Physician and kidney stone researcher
- Practicing at the only multidisciplinary metabolic kidney stone clinic in the southeast U.S.
- Basic, translational and clinical research
- Associate Professor UAB

Kristina Penniston, PhD
Kidney stone researcher
- Clinical nutrition research in kidney stones
- UW Metabolic Stone Clinic
- Coordinator of Cairibu, a multi-center urology disease cooperative research community funded by NIH

Additional consultants:
Melanie Hartsough (pre-clinical): independent consultant, past Biologics Consulting Group, ex-FDA full-time reviewer CDER, CBER.
Wilfredo Rivera-Baez (quality): 20+ experience independent quality consultant, experienced domestic and international GxP auditor

Liquidity Event Opportunity in 24 Months

Prior (Spent) Funding:	• Undilutive: $2.1MM from NIH (SBIR grants) • Dilutive: Series A $3.4MM
Series A Round:	• **$ 4 MM Equity, Preferred**
Series B Round:	• $ 8 MM Equity



CMC = Chemistry Manufacturing and Controls
FPFD = First Patient First Dose
LPLD = Last Patient Last Dose
EOP = End of Phase 2

Opportunity for significant value inflection and strategic corporate options in 24 months

Thank you

Contact:

Helena Cowley
President & CEO

Cell: 352 672 5320
Email: Helena@oxidien.com

Intellectual property protection and exclusivity

"High Efficiency Oxalate-Degrading Enzymes for Degradation of Insoluble and Soluble Oxalate"

United States

Europe

Rest of the World

Patents pending for OX-1 in the United States, European Union, Canada, China, Australia, Korea and India.

Approved and Pending Patent Applications:

- U.S. Patent Application Publication No. 2018/0362955
- European Patent Application Publication No. 3277099
- Canadian Patent Application Publication No. 2984763
- Chinese Patent Application Publication No. 107960105
- Australian Patent Application Publication No. 2016244121
- Korean Patent Application Publication No. 20180044225
- Indian Patent Application Publication No. 201717039146

Strong composition claims pending covering structural elements that produce the characteristics that allow for favorable clinical outcomes.