REATA PHARMACEUTICALS

4th Quarter and Full Year 2020 Financial Results and Update on Clinical Development Programs

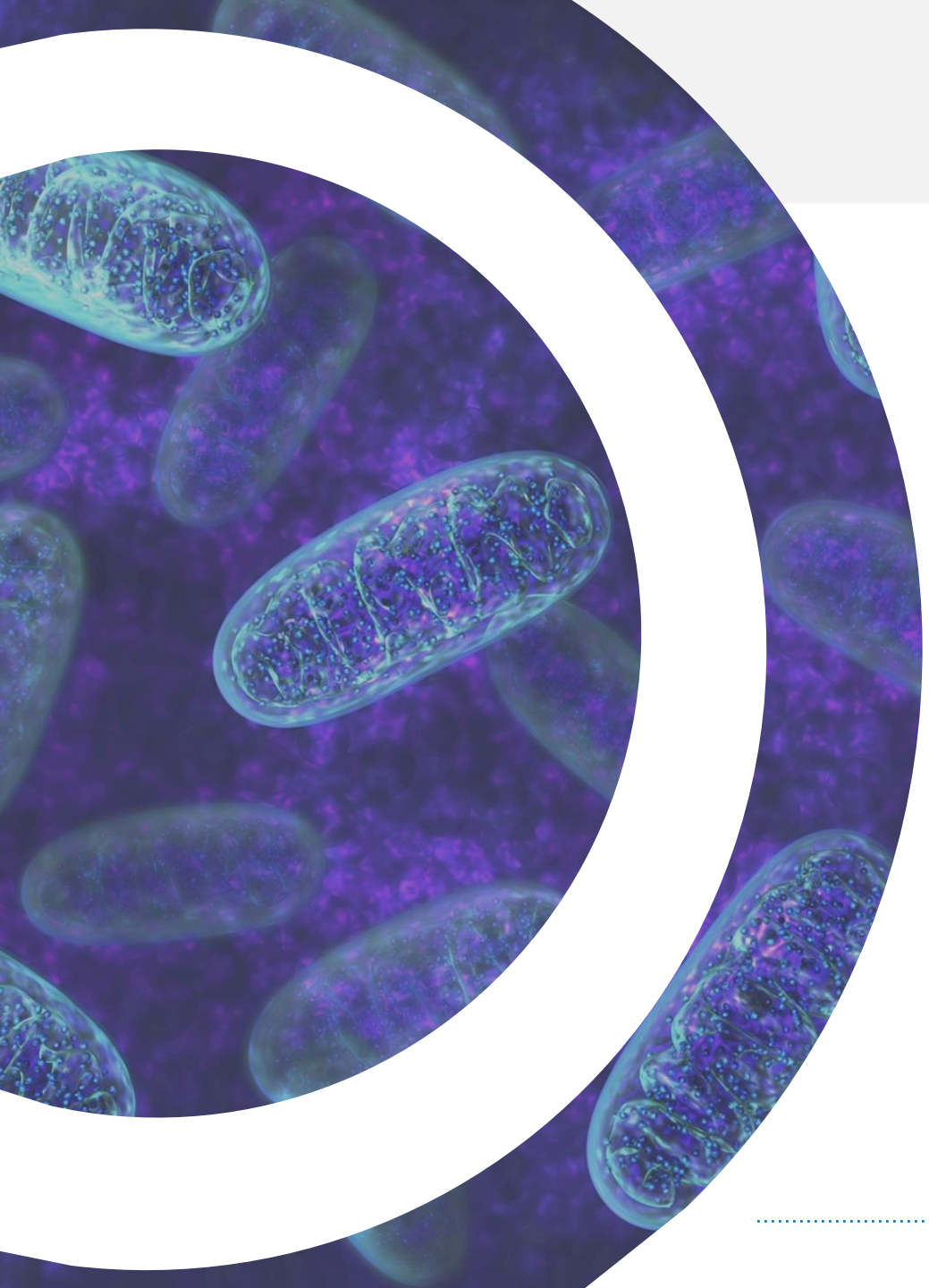
March 1, 2021

Forward-Looking Statements

This presentation contains certain “forward-looking” statements that are made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. All statements, other than statements of historical or present facts, are forward-looking statements, including statements regarding our future financial condition, future revenues, projected costs, prospects, business strategy, and plans and objectives of management for future operations, including our plans to submit for regulatory filings. In some cases, you can identify forward-looking statements by terminology such as “believe,” “will,” “may,” “might,” “estimate,” “continue,” “anticipate,” “intend,” “target,” “project,” “model,” “should,” “would,” “plan,” “expect,” “predict,” “could,” “seek,” “goal,” “potential,” or the negative of these terms or other similar terms or expressions that concern our expectations, strategy, plans, or intentions. These statements are based on our intentions, beliefs, projections, outlook, analyses, or current expectations using currently available information, and are not guarantees of future performance, and involve certain risks and uncertainties. Although we believe that the expectations reflected in these forward-looking statements are reasonable, we cannot assure you that our expectations will prove to be correct. Therefore, actual outcomes and results could materially differ from what is expressed, implied, or forecasted in these statements. Any differences could be caused by a number of factors including but not limited to: our expectations regarding the timing, costs, conduct, and outcome of our clinical trials, including statements regarding the timing of the initiation and availability of data from such trials; the timing and likelihood of regulatory filings and approvals for our product candidates; whether regulatory authorities determine that additional trials or data are necessary in order to obtain approval; our ability to obtain funding for our operations, including funding necessary to complete further development and commercialization of our product candidates; our plans to research, develop, and commercialize our product candidates; the commercialization of our product candidates, if approved; the rate and degree of market acceptance of our product candidates; our expectations regarding the potential market size and the size of the patient populations for our product candidates, if approved for commercial use, and the potential market opportunities for commercializing our product candidates; the success of competing therapies that are or may become available; our expectations regarding our ability to obtain and maintain intellectual property protection for our product candidates; the ability to license additional intellectual property relating to our product candidates and to comply with our existing license agreements; our ability to maintain and establish relationships with third parties, such as contract research organizations, suppliers, and distributors; our ability to maintain and establish collaborators with development, regulatory, and commercialization expertise; our ability to attract and retain key scientific or management personnel; our ability to grow our organization and increase the size of our facilities to meet our anticipated growth; the accuracy of our estimates regarding expenses, future revenue, capital requirements, and needs for additional financing; our expectations related to the use of our available cash; our ability to develop, acquire, and advance product candidates into, and successfully complete, clinical trials; the initiation, timing, progress, and results of future preclinical studies and developments and projections relating to our competitors and our industry.

Additional factors that could cause actual results to differ materially from our expectations can be found in our Securities and Exchange Commission filings. Moreover, we operate in a very competitive and rapidly changing environment. New risk factors emerge from time to time, and it is not possible for our management to predict all risk factors, nor can we assess the effects of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. All forward-looking statements included in this presentation are expressly qualified in their entirety by these cautionary statements. The forward-looking statements speak only as of the date made and, other than as required by law, we undertake no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events, or otherwise.

Bardoxolone methyl, omaveloxolone, and RTA 901 are investigational drugs, and their safety and efficacy have not been established by any agency.



Today's Agenda

Opening Remarks | Warren Huff, CEO

Program Updates | Colin Meyer, MD, Chief R&D Officer

- Chronic Kidney Disease Pipeline
  - CARDINAL & EAGLE Update
  - FALCON Update
  - MERLIN Update
- Neurology Pipeline
  - MOXIe Update
  - RTA 901 Update

Financial & Operational Update | Manmeet Soni, COO & CFO

Concluding Remarks | Warren Huff, CEO

Reata's First NDA Submitted

Submitted NDA in the U.S. for bardoxolone for treatment of patients with CKD caused by Alport syndrome

- Includes a request for Priority Review
- Expect PDUFA date in 4Q21 pending Priority Review
- Preparing for potential Advisory Committee meeting

FDA

✓ **NDA SUBMITTED**

CARDINAL
FOR PATIENTS WITH ALPORT SYNDROME

FDA: U.S. Food and Drug Administration; NDA: New Drug Application; PDUFA: Prescription Drug User Fee Act; Bardoxolone: bardoxolone methyl

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Reata at a Glance

CKD[1] Pipeline

- Submitted Reata's First NDA for bardoxolone in Alport syndrome
- Enrolled >220 patients in FALCON, expected enrollment completion in 4Q21
- Initiated MERLIN study for patients with CKD at risk of fast progression, data expected 2H21

Neurology Pipeline

- Submitted Type C meeting request with the FDA to discuss the Delayed-Start Analyses and the FA[1] development program
- Plan to initiate Phase 2 study for RTA 901 in DPNP[1] in 4Q21

Global Opportunity

- Few or no effective therapies approved for lead indications
- Worldwide commercial rights to all pipeline assets[2]
- Robust IP[1] protection for bardoxolone, omaveloxolone, and RTA 901

[1]CKD: Chronic kidney disease, FA: Friedreich's ataxia, DPNP: diabetic peripheral neuropathic pain, IP: Intellectual Property; [2]Ex-Asia for bardoxolone


Chronic Kidney
Disease Pipeline
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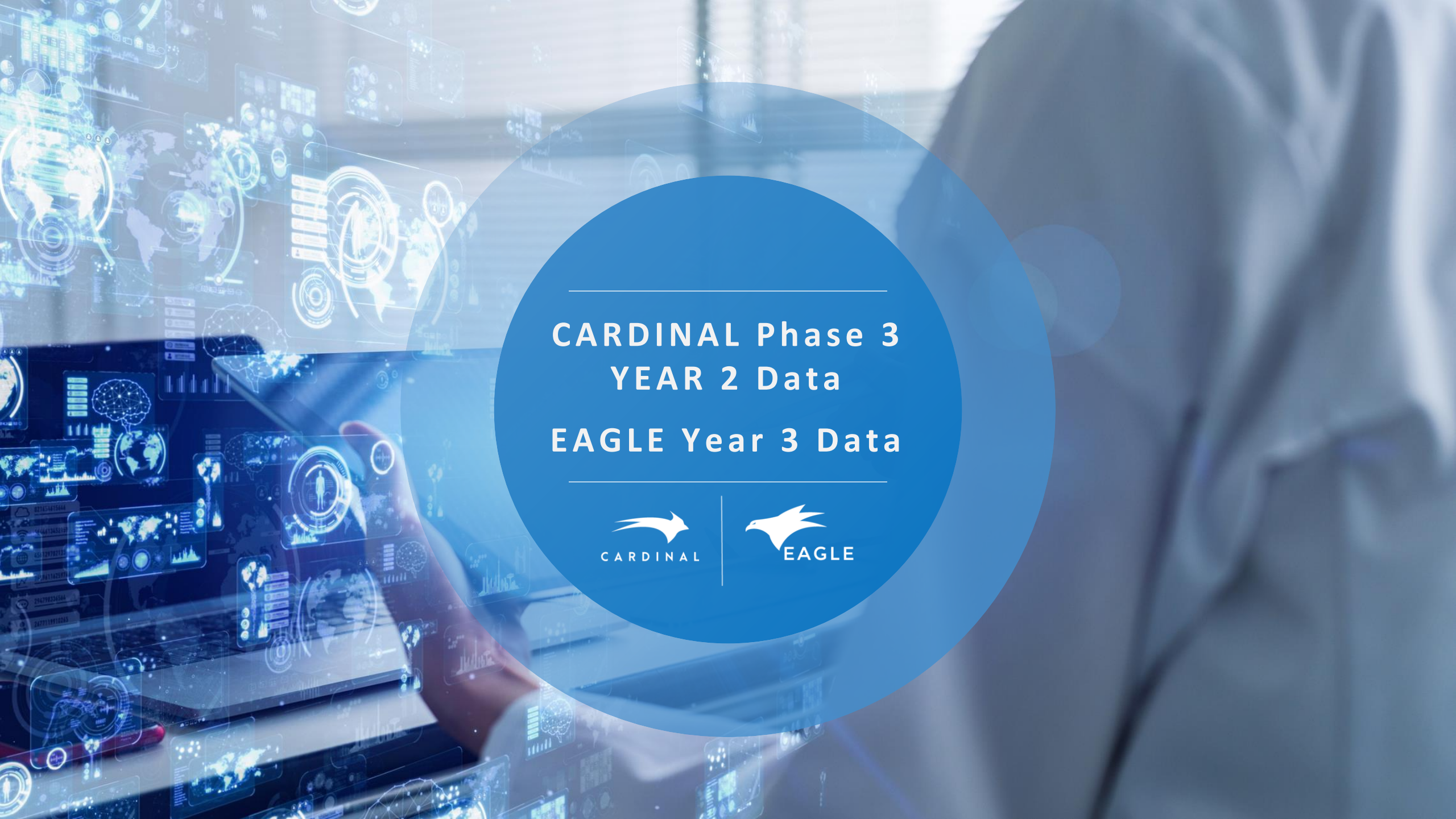

CARDINAL Phase 3
YEAR 2 Data
EAGLE Year 3 Data
CARDINAL
EAGLE

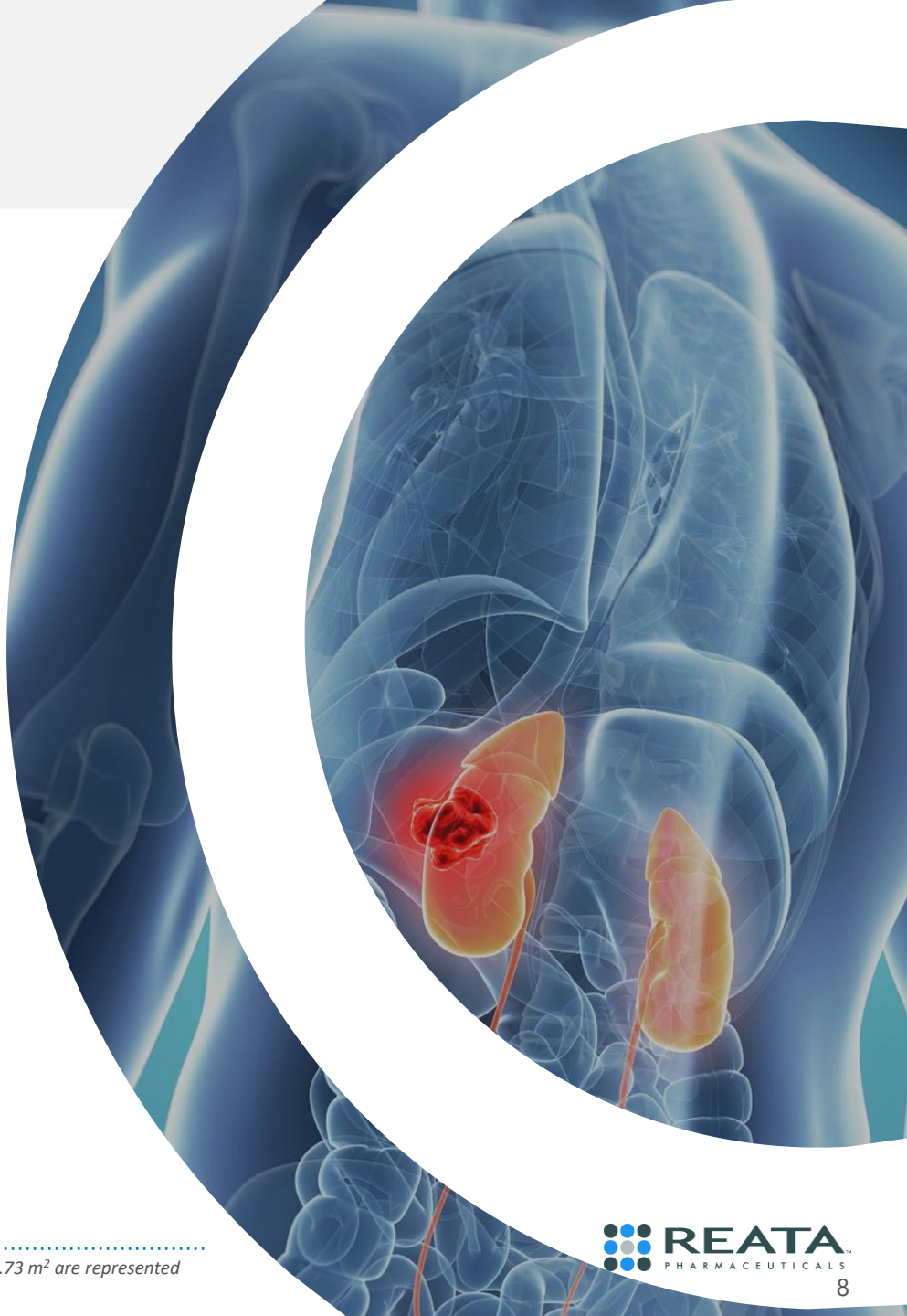
Alport Syndrome: Second Most Common Hereditary Form of CKD

Collagen mutations drive glomerular membrane dysfunction causing **inflammation** and **fibrosis**[1]

Misdiagnosis rate reported to be as high as **62%**[2]

-5.1 mL/min per year
Rate of kidney function loss despite standard of care[3]

Up to **100%** lifetime risk for **ESKD**[4]
25 years is median age at ESKD in most severe patients[5]



[1]Kruegel et al., Nat Rev Nephrol (2013), Gomez et al., JCI (2015); [2]Groopman et al. N Engl J Med. 2019; [3]CARDINAL Phase 3 historical eGFR decline rate, units of mL/min/1.73 m² are represented as mL/min throughout this presentation; [4]Kashtan et al., Kidney Int (2018), ESKD: end-stage kidney disease; [5]Jais et al., JASN (2000)

Data Supporting Bardoxolone Clinical Efficacy and Safety in Alport Syndrome

	CARDINAL Phase 3 N=157	CARDINAL Phase 2 N=30	EAGLE Extension N=100* (Enrollment Ongoing)
Patients (n)	77 Bardoxolone/80 placebo	30 Bardoxolone	100 Bardoxolone
Design	Randomized, double-blind, placebo-controlled	Open-label	Open-label safety extension
Duration	2 years	2 years	Ongoing

*As of February 25, 2021

Clinical Trials Enrolling >3,000 Patients Support Bardoxolone Alport Syndrome NDA

Selected Studies	Patient Population	N[1]
CARDINAL	Alport syndrome	187
PHOENIX	ADPKD[2]	31
PHOENIX	IgAN[2]	26
PHOENIX	T1D CKD[2]	28
PHOENIX	FSGS[2]	18
TSUBAKI	T2D CKD[2]	124
BEACON	T2D CKD	2185
BEAM	T2D CKD	227
402-C-0902	T2D CKD	131
402-C-1102	T2D CKD	24
402-C-0801	T2D CKD	80

Improvements in eGFR, measured GFR (inulin clearance), creatinine clearance

Reductions in inflammatory biomarkers

Sustained eGFR improvements for up to three years

Significant increase in off-treatment eGFR after withdrawal of drug

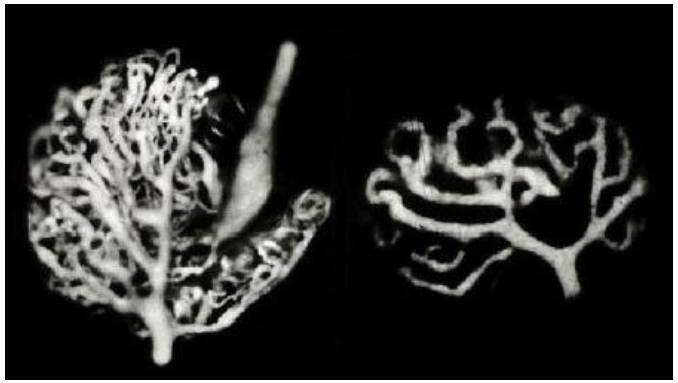
Significant reductions in the risk of kidney failure outcomes

[1]Includes patients treated with placebo; [2]ADPKD: autosomal dominant polycystic kidney disease, IgAN: IgA nephropathy, FSGS: focal segmental glomerulosclerosis, T1D CKD: type 1 diabetic CKD, T2D CKD: type 2 diabetic CKD

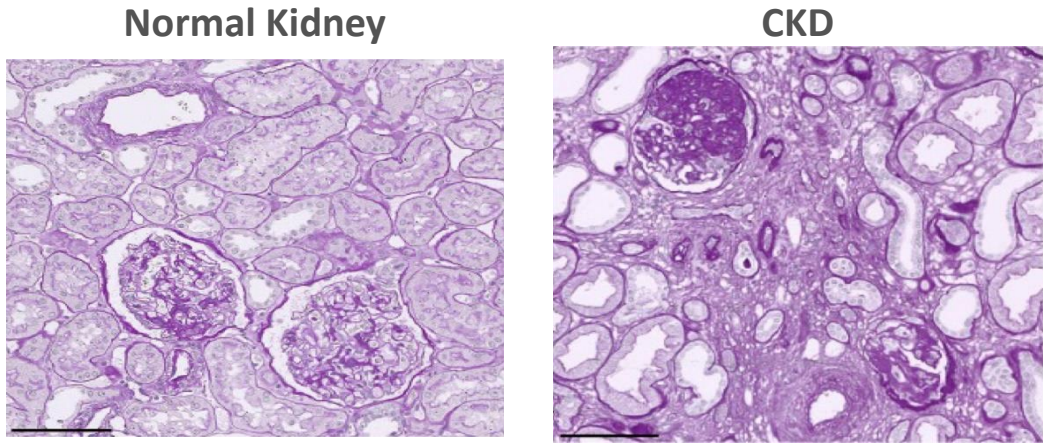
Chronic Inflammation Drives CKD Progression

Chronic Inflammation[1-2]
(Metabolic reprogramming, ROS[3] production, NF-κB and TGFβ activation)

Impairs SNGFR[3] by Reducing Glomerular Surface Area (K_f)[4-7]



Fibrosis and Nephron Loss[8-9]



Nrf2 promotes resolution of inflammation and is suppressed in **many forms of CKD**

Genome-wide analyses validate association between **impaired Nrf2 activity and reduced kidney function** across nine forms of CKD, including Alport syndrome[10]

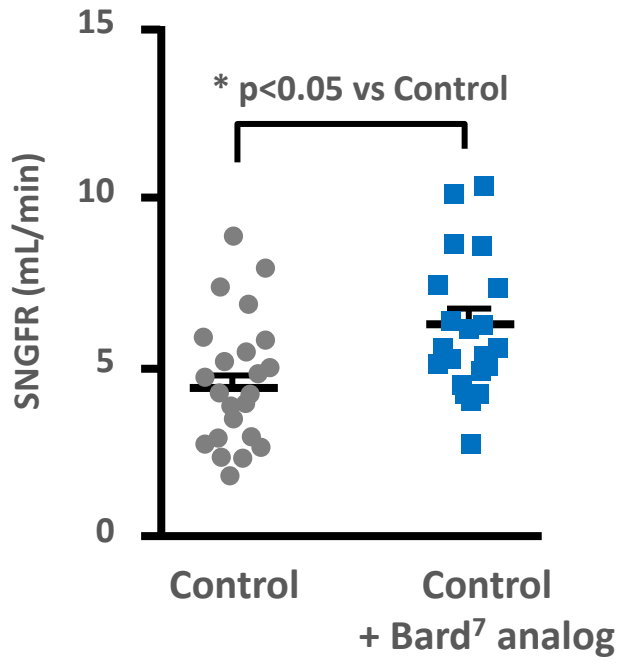
[1]Cuadrado et al., Nat Rev Drug Discov (2019); [2]Rimessi et al., Int J Biochem Cell Biol (2016); [3]ROS: reactive oxigen species, SNGFR: single nephron glomerular filtration rate; [4]Barger, NEJM (1971); [5]Mihai et al., J Immunol Res (2018); [6]Ding et al., Kidney Int (2013); [7]L'Azou et al., Arch Toxicol (1999); [8]Ryu et al., J Pathol (2012); [9]Raptis et al., Diabetes (2001); [10]Martini et al., JASN (2014)

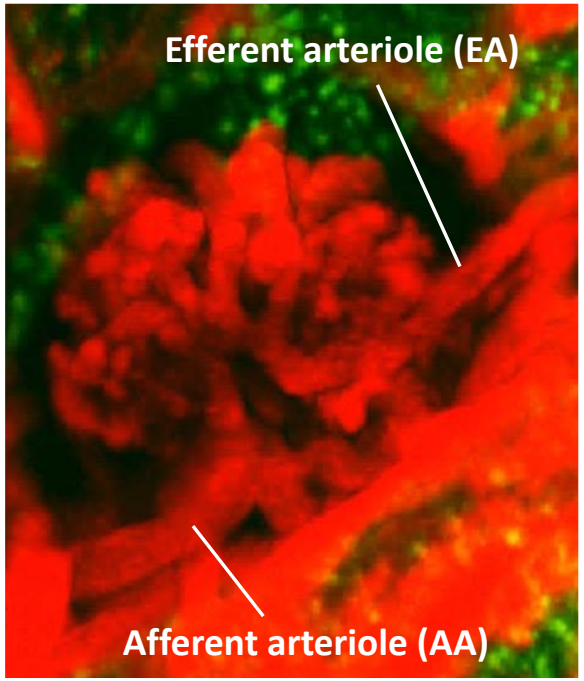
Through Activation of Nrf2 Bardoxolone Suppresses Inflammation and Fibrosis and Restores SNGFR

Increase SNGFR by Restoring Glomerular Surface Area[1,2]

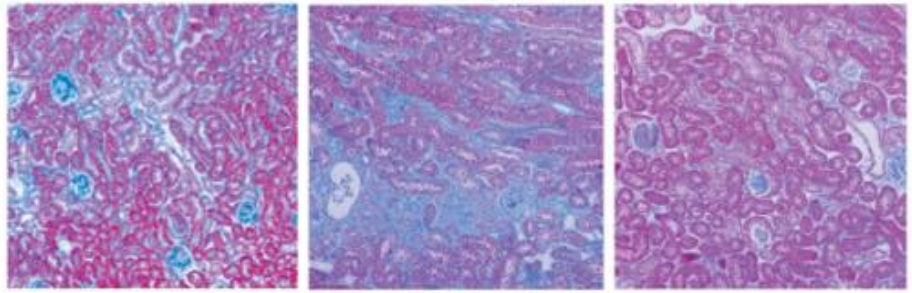
No Effects on Afferent or Efferent Vascular Tone[1]

Reduces Glomerulosclerosis and Fibrosis in Models of Hyperfiltration and CKD[3-6]



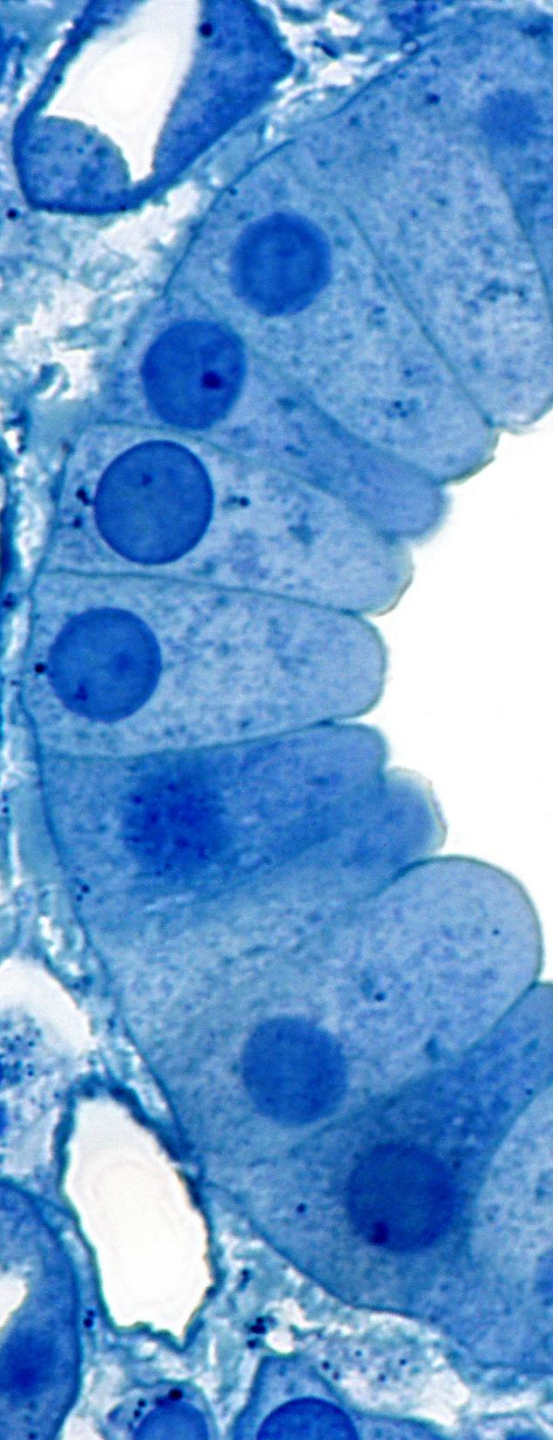


Hypertension-Induced Tubulointerstitial Fibrosis



Control | Ald[8] | Ald + Bard

[1]Kidokoro et al., ASN (2019); [2]Ding et al., Kindey Intertional (2013); [3]Aminzadeh et al., Redox Biol (2013); [4]Hisamichi et al., Hypertension Res (2018); [5]Zoja et al., ASN (2010); [6]Nagasu et al., FASEB (2019)
[7]Bard: bardoxolone mnethyl; [8]Ald: Aldosterone



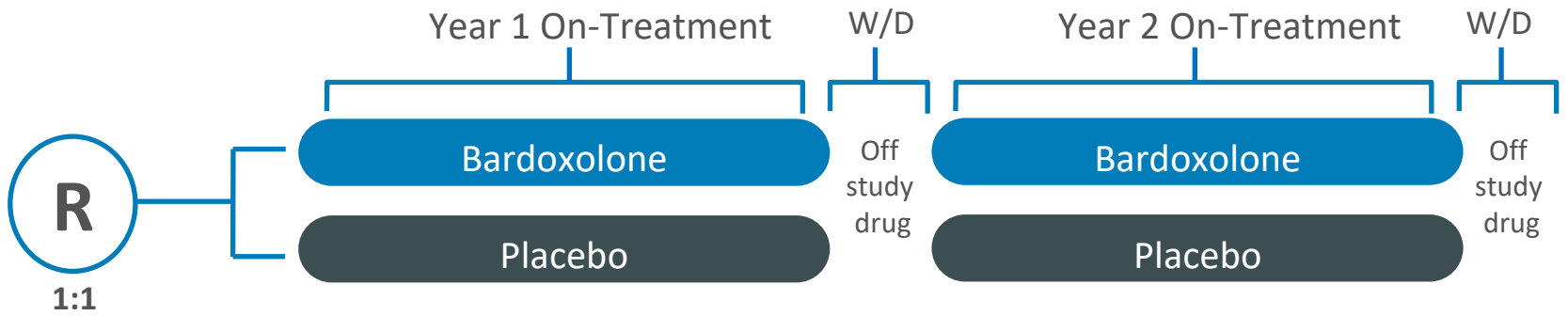
CARDINAL Phase 3: Pivotal Study Design

International, randomized, double-blind, placebo-controlled, registrational, two-year trial

Largest and longest interventional study in patients with AS

Enrolled a wide and representative range of patients with AS

- eGFR: 30 to 90 mL/min
- Age: 12 to 70 years



Primary Endpoints: on-treatment change from baseline in eGFR vs placebo at Week 48 and Week 100

Key Secondary: off-treatment change from baseline in eGFR vs placebo at Week 52 and Week 104 (4 weeks after withdrawal of drug)

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FOR PATIENTS WITH ALPORT SYNDROME

AS: Alport syndrome; eGFR: estimated Glomerular Filtration Rate; R: randomization; W/D: withdrawal or off-treatment

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CARDINAL Phase 3 Met Year 1 and Year 2 Primary Endpoints

Sustained increases in eGFR vs. placebo observed **through 2 years**

ITT population **includes all patients**, including those who discontinued

mITT population only **includes eGFR values for patients who were receiving bardoxolone**

Primary Endpoint	Mean Change from Baseline in eGFR vs. Placebo	p-value
Week 48 eGFR (ITT)	9.49 mL/min	<0.0001
Week 100 eGFR (ITT)	7.65 mL/min	0.0005
Week 100 eGFR (mITT)	11.3 mL/min	<0.0001

CARDINAL
FOR PATIENTS WITH ALPORT SYNDROME

ITT: intend to treat; mITT: modified ITT

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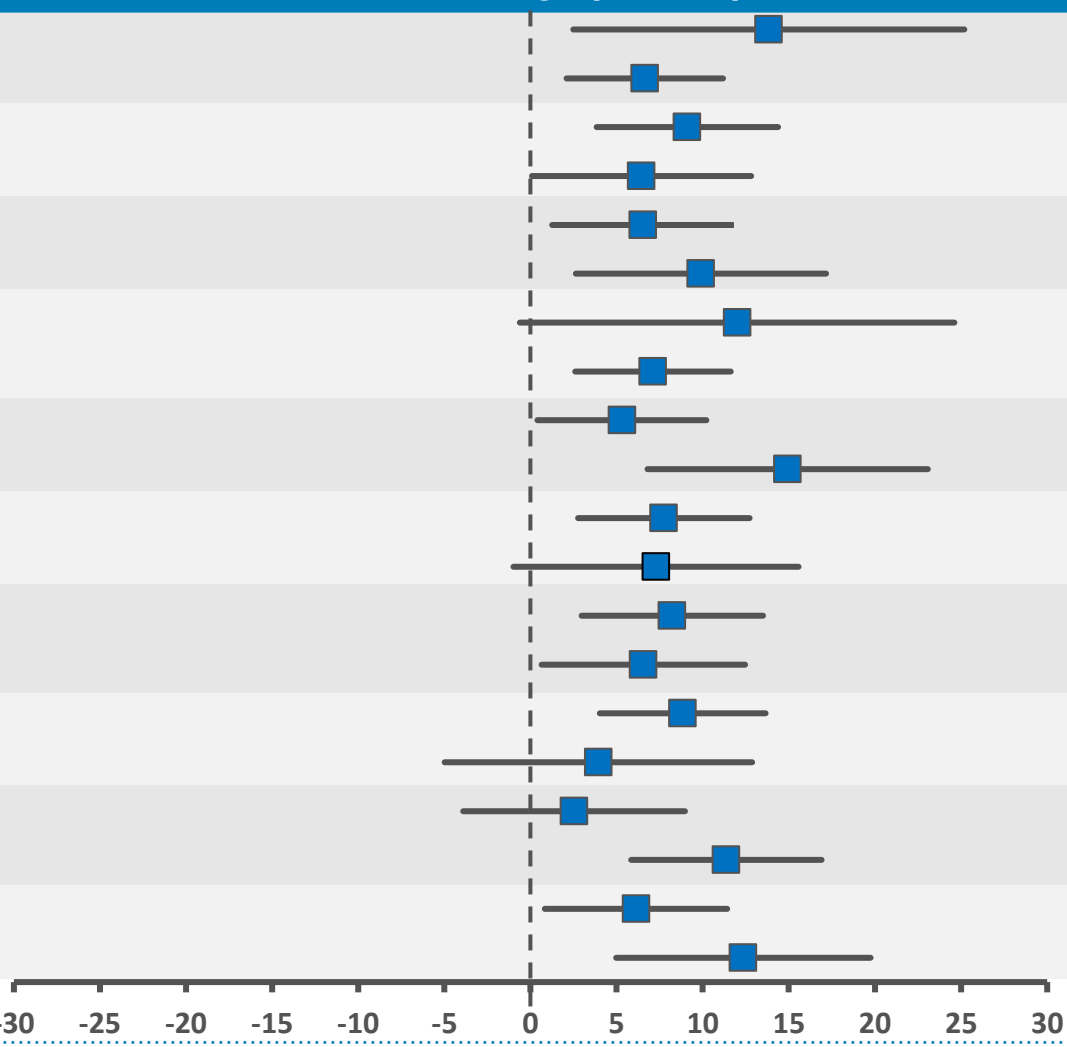
On-Treatment eGFR Benefit Observed Across All Pre-specified Subgroups

Subgroup		N	Difference	95% CI
Age	<18	17	13.84	(2.48, 25.19)
	≥18	121	6.64	(2.10, 11.18)
Gender	Female	85	9.10	(3.84, 14.37)
	Male	53	6.44	(0.08, 12.81)
Geographic Location	U.S.	90	6.54	(1.26, 11.82)
	Non-U.S.	48	9.90	(2.64, 17.17)
Ethnicity	Hispanic or Latino	15	12.01	(-0.61, 24.62)
	Not Hispanic or Latino	123	7.11	(2.59, 11.62)
Race	White	101	5.32	(0.41, 10.22)
	Non-White	37	14.93	(6.80, 23.07)
Body Mass Index	<30 kg/m^2	100	7.75	(2.76, 12.73)
	≥30 kg/m^2	38	7.29	(-0.98, 15.56)
Albumin to Creatinine Ratio	≤300 mg/g	78	8.23	(2.97, 13.50)
	>300 mg/g	60	6.55	(0.64, 12.46)
Use of ACE Inhibitor or ARB	Yes	107	8.84	(4.02, 13.66)
	No	31	3.95	(-4.99, 12.88)
Baseline eGFR	≤60 mL/min/1.73 m^2	58	2.54	(-3.90, 8.98)
	>60 mL/min/1.73 m^2	80	11.37	(5.84, 16.90)
Genetic Subtype	XLAS	86	6.14	(0.84, 11.43)
	Non-XLAS	44	12.35	(4.97, 19.74)



ACE inhibitor: Angiotensin-converting enzyme inhibitor; ARB: Angiotesin II receptor blocker; XLAS: X-linked Alport syndrome

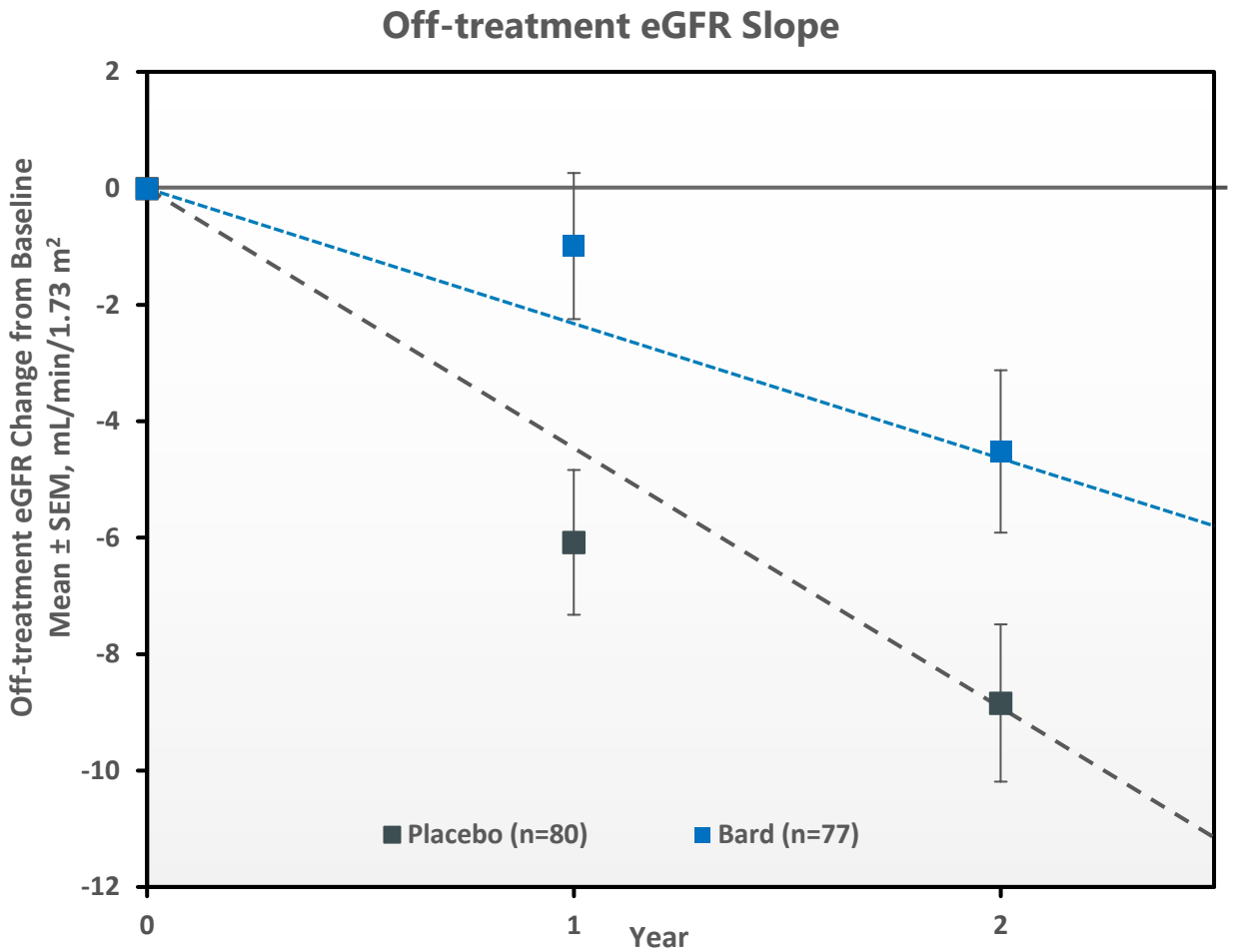
CARDINAL Phase 3 Met Year 1 and Year 2 Key Secondary Endpoints

Bardoxolone **slowed rate of irreversible loss of kidney function** compared to placebo

Validate on-treatment change as beneficial and not harmful

Key Secondary Endpoints	Mean Change from Baseline in eGFR vs. Placebo	p-value
Week 52 eGFR	5.09 mL/min	0.0021
Week 104 eGFR	4.26 mL/min	0.023

Off-Treatment eGFR Increase Confirmed in Year 2 and Slopes Demonstrate Slowing of Disease Progression



Evidence of **disease modifying** effects

Preservation of off-treatment eGFR after 2 years

Slowed disease progression **by 50%**

Based on longitudinal estimates of off-treatment slope

The key secondary, off-treatment eGFR endpoint was analyzed using analysis of covariance (ANCOVA). The point estimate for each treatment group was based on a model that used two UACR categories (≤300; >300). Off-treatment values at Year 1 and 2 were included for those who completed treatment. Patients who discontinued and had an off-treatment value following discontinuation were also included. Patients who discontinued treatment in the first year and did not dose in the second year did not have a Year 2 Week 104 value. Treatment-based multiple imputation was used for patients with missing data.

Early Bardoxolone eGFR Increases in CARDINAL Predictive of Year 2 Response

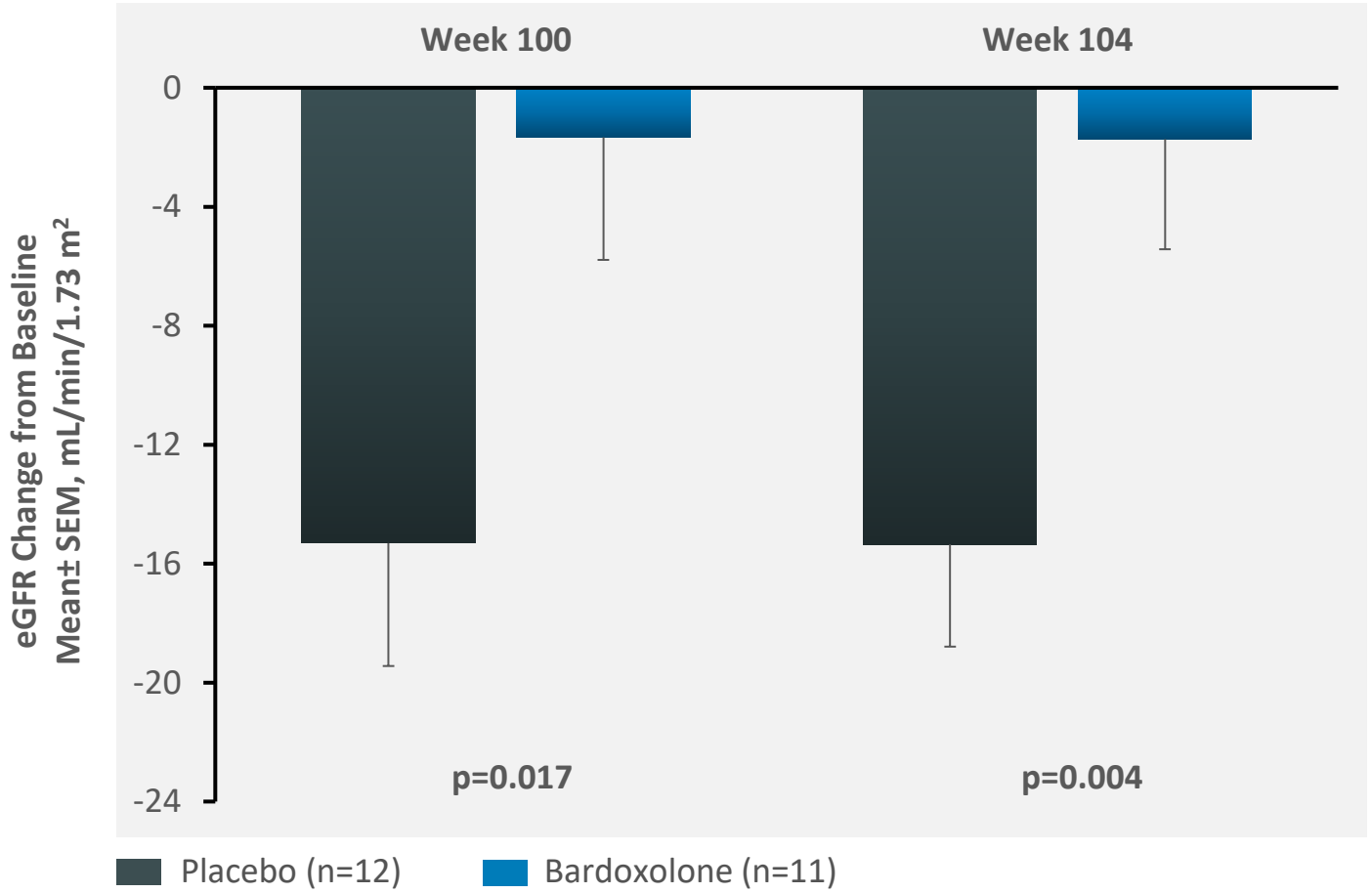
	Quartile	N	Mean ± SD eGFR Change (mL/min)		
			Week 12	Week 100	Week 104
Placebo	1	15	-10.3 ± 4.2	-9.9 ± 13.9	-11.3 ± 15.8
	2	15	-1.7 ± 0.9	-8.9 ± 8.2	-9.1 ± 10.3
	3	15	0.9 ± 1.4	-9 ± 7	-6.3 ± 6.1
	4	15	7.1 ± 3.6	-5.3 ± 6.6	-4.6 ± 6.0
Bard	1	11	2.0 ± 2.7	-4.9 ± 15.3	-9.8 ± 12.9
	2	12	8.8 ± 1.6	4.8 ± 13.1	-5.6 ± 8.1
	3	12	17.3 ± 2.6	2.7 ± 15.1	-2.0 ± 14.1
	4	12	27.0 ± 6.9	12.4 ± 14.8	2.6 ± 13.3

Quartile Analysis by Week 12 eGFR Change:

- Highest responders at Week 12 have highest Week 100 and Week 104 changes in eGFR
- Not associated with accelerated progression of kidney disease
- **Opposite pattern than hyperfiltration**

SD: standard deviation

Largest Treatment Effect Observed in Pediatric Patients



- Pediatric patients have most severe mutations and fastest rate of progression
- Two-year duration represents ~30% of remaining dialysis-free time

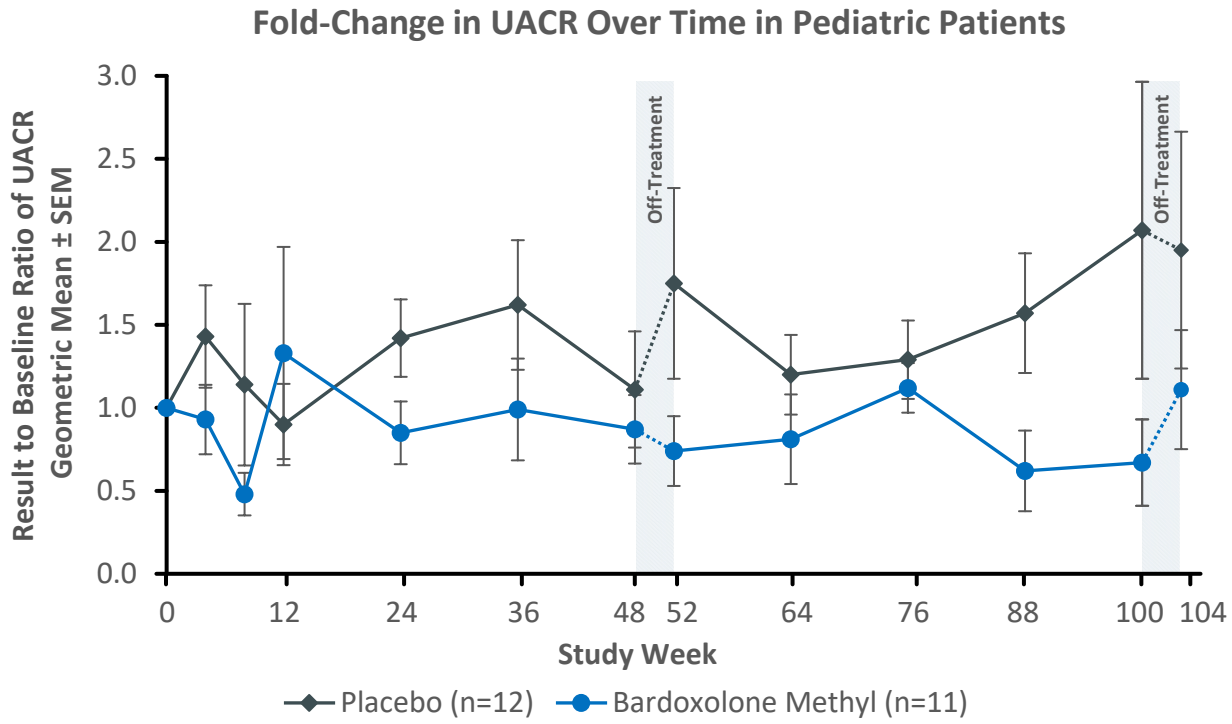
Mean ± SEM eGFR Change (mL/min)

	Difference (Bardoxolone — Placebo)
Week 100	13.8 ± 5.7 (p=0.017)
Week 104	14.6 ± 5.0 (p=0.004)

Week 100 and Week 104 eGFR changes for pediatric subpopulation analyzed using the same methodologies described for overall ITT population
SEM: standard error of the mean

Pediatric Data Demonstrate eGFR Increases with Bardoxolone Not Associated with Glomerular Injury

- Across all patients, UACR not significantly different than placebo at Weeks 100 and 104
- In pediatric placebo patients, progression of UACR observed over two years
- UACR not worsened by bardoxolone and decreased relative to placebo



UACR: urine albumin-to-creatinine ratio

Bardoxolone Profile is Novel and Differs from Hyperfiltration

	Profile	Hyperfiltration[1]	Bardoxolone
Nonclinical Evidence	Mechanism of SNGFR increase	Increased intraglomerular pressure	Increased glomerular surface area[2]
	Glomerular permeability	Increased due to injury	Unchanged[2]
	Effects in model of hyperfiltration	Worsens fibrosis	Reduces fibrosis[3]
Clinical eGFR Profile	Magnitude of increase	Small (~4 mL/min)	Large (~12 mL/min)[4]
	Time profile over 3 years	Transient increase followed by accelerated decline after 6 months	Sustained increases[5]
	Off-treatment change vs. placebo	Worsens	Improves[4]
	Events in kidney failure composite	Increased	Reduced[4]
Clinical UACR Profile	Magnitude	Out of proportion to changes in eGFR	Accounted for by increases in eGFR[4]
	Time profile	Values continue to increase over time	Values plateau after initial increase[4]
	Off-treatment change vs. placebo	Worsens	Returns to baseline[4]

[1]Wright et. Al., JAMA (2002); [2]Kidokoro et al., ASN (2019); [3]Aminzadeh et al., Xenobiotica (2014); [4]CARDINAL Phase 3 data; [5]EAGLE data

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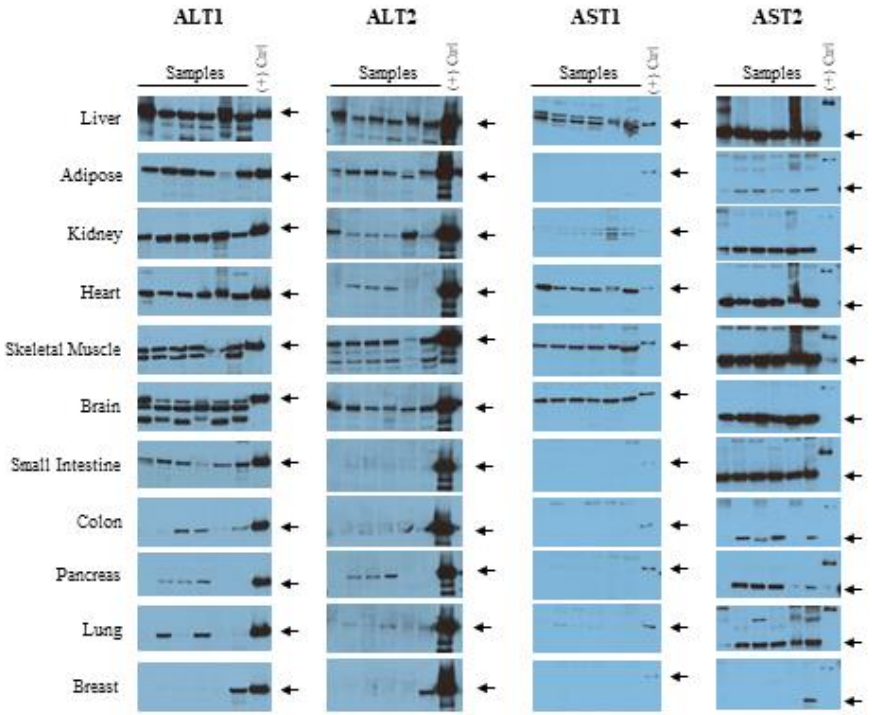
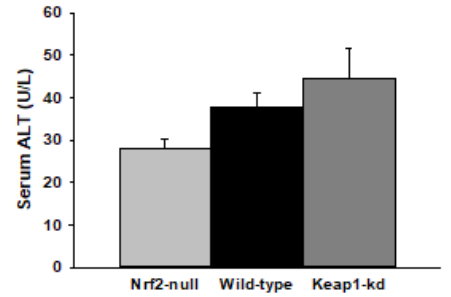
Nonclinical Data Supporting Aminotransferase Increases Are Pharmacological Effect Due to Nrf2 Activation

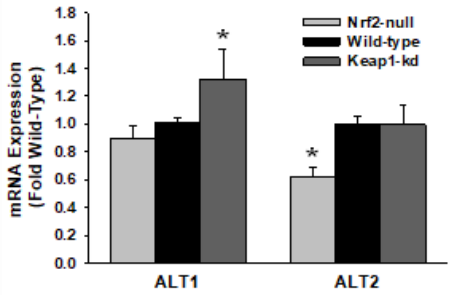
Aminotransferases widely expressed in multiple tissues



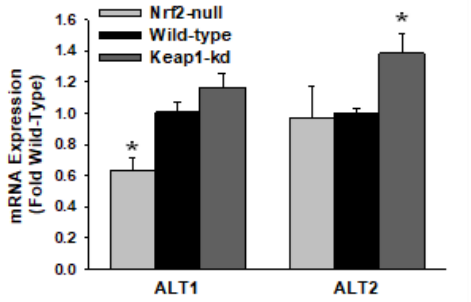
Aminotransferase activity influenced by Nrf2 status



Serum ALT Activity

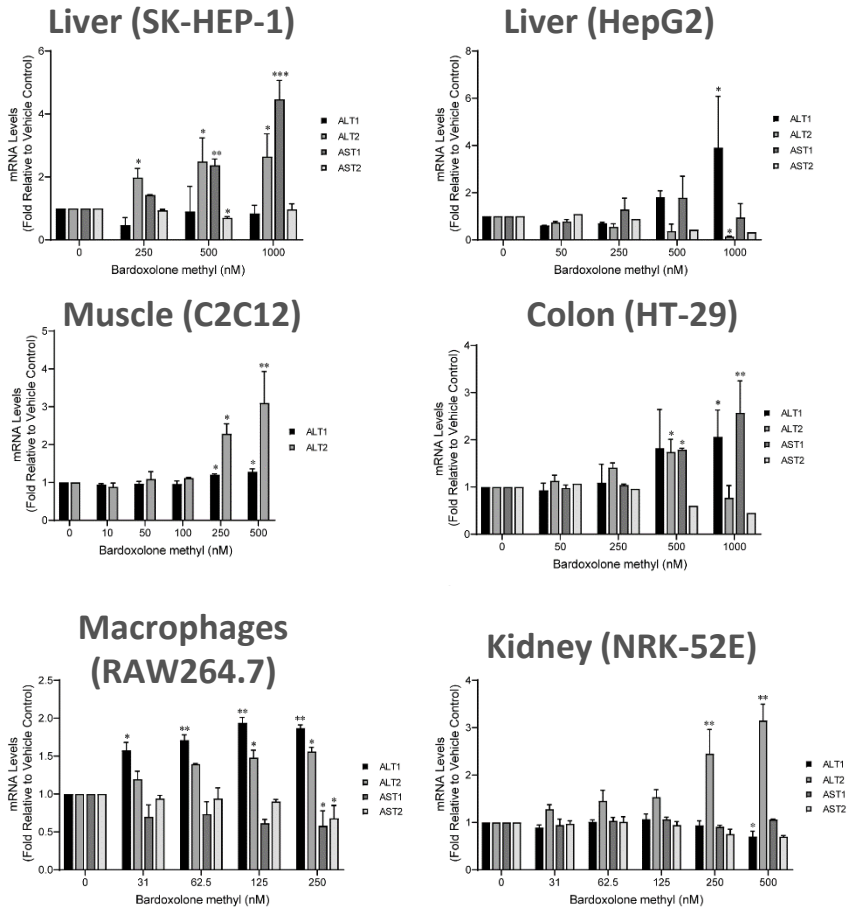


Hepatic mRNA



Kidney mRNA

Bardoxolone increases aminotransferase expression in multiple tissue types



Lewis et al., Clin Transl Sci (2021)

Aminotransferase Increases Associated with Reductions in Total Bilirubin

Profile of transient, reversible aminotransferase increases consistent with prior trials, including no cases of Hy's Law

Elevations believed to be related to pharmacology of drug and not consistent with clinical evidence of liver injury

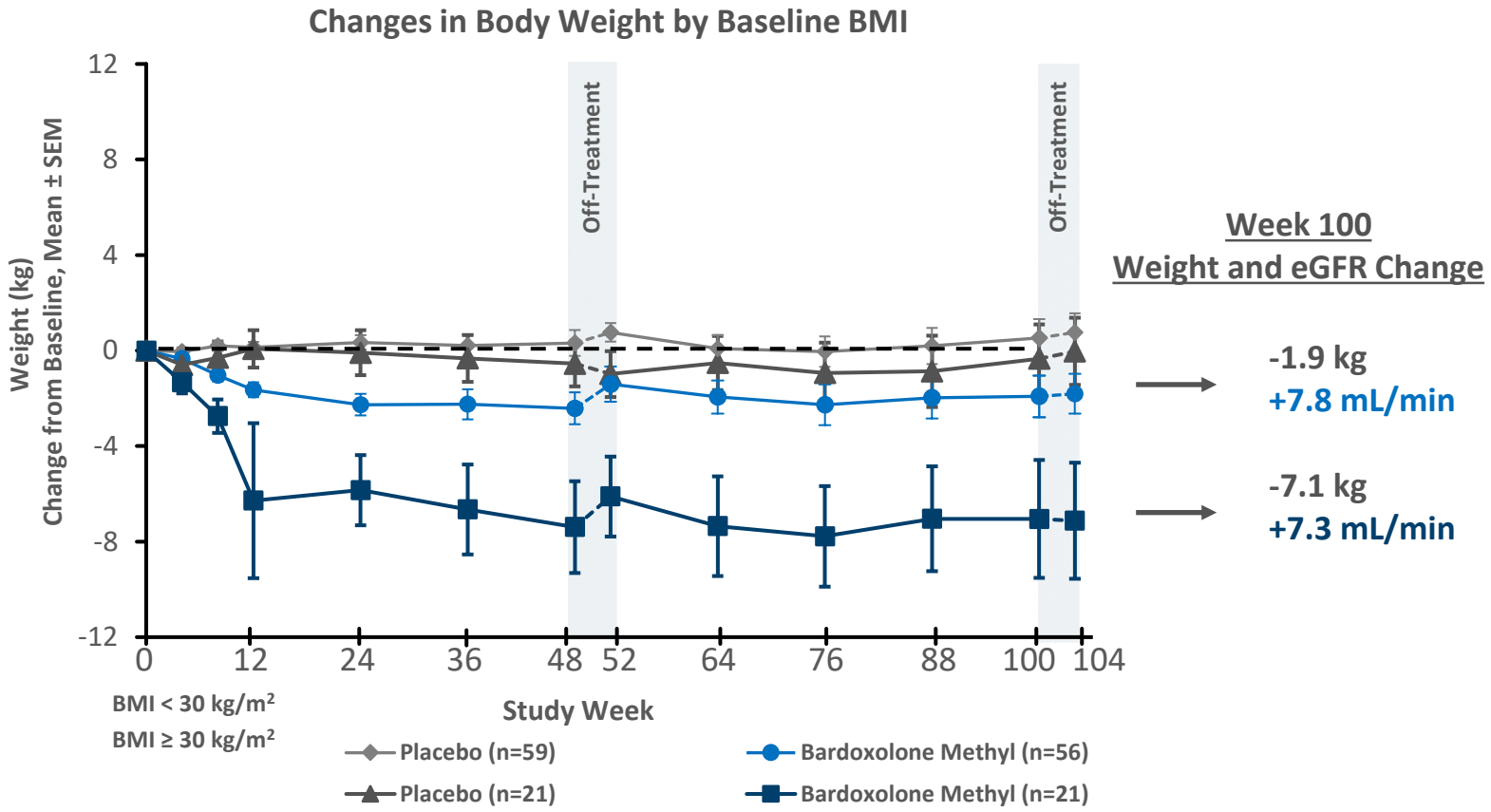
Protocol-required discontinuations (n=6) due to conservative management and not associated with any evidence of liver injury

Proposed USPI provides guidance for appropriate monitoring and management of ALT/AST increases to minimize discontinuations

FALCON ADPKD protocol does not require permanent discontinuations for patients who have profile consistent with pharmacological regulation of ALT/AST

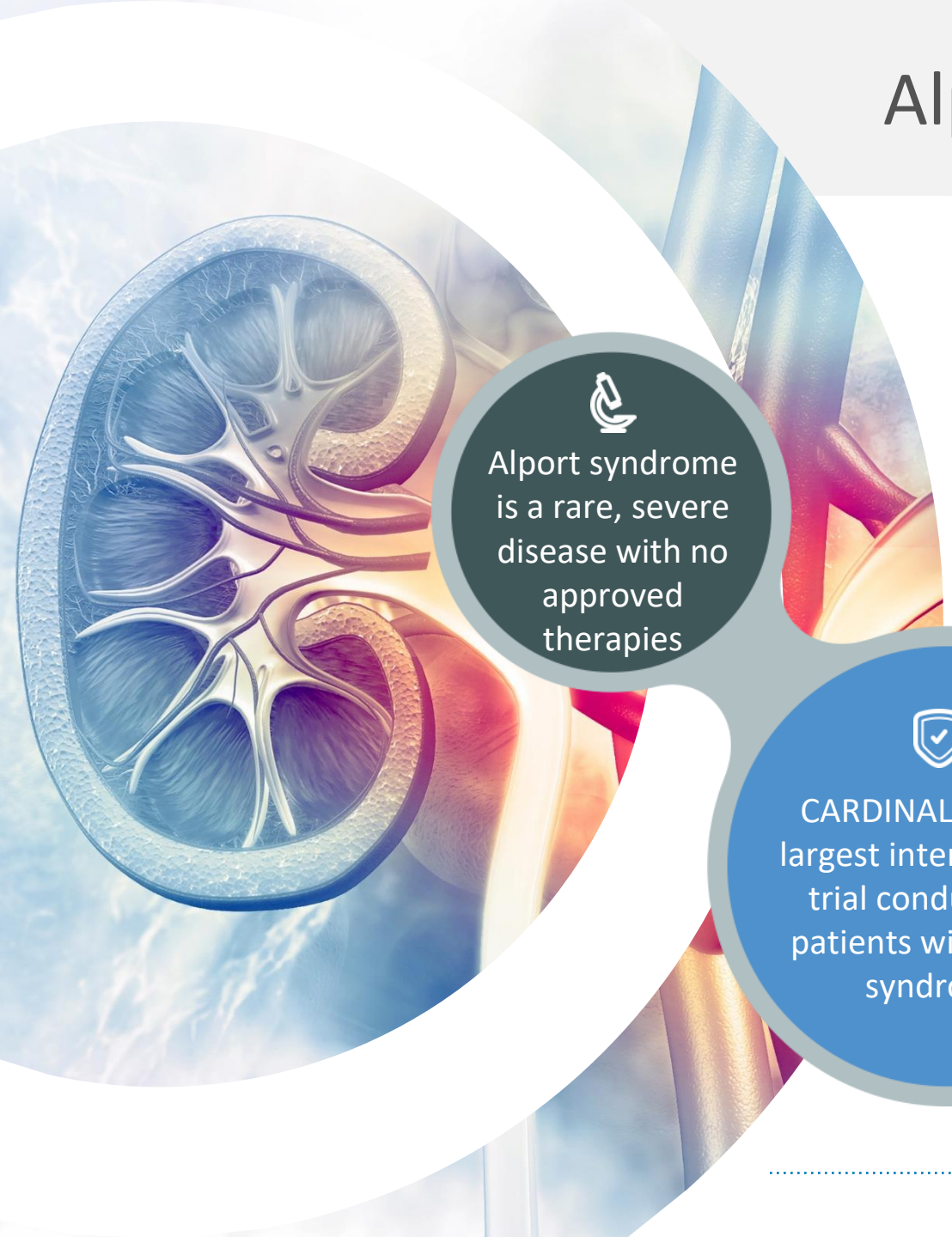
Decreases in Body Weight not Associated with Changes in eGFR

- Decreases in weight observed with bardoxolone that were more pronounced in patients with higher baseline BMI
- Changes in weight not correlated with changes in eGFR with bardoxolone (r = -0.07; p = 0.7)
- eGFR increases with bardoxolone not due to loss of muscle mass but reflect true increases in measured GFR



BMI: body mass index

Alport Syndrome Program Conclusions



Alport syndrome is a rare, severe disease with no approved therapies

CARDINAL was the largest interventional trial conducted in patients with Alport syndrome

Primary and key secondary efficacy endpoints were met at Year 1 and Year 2

Disease modifying profile

Comprehensive safety database in >3,000 patients supports NDA

If approved, bardoxolone may be the first therapy to slow the progression of kidney disease in patients with Alport syndrome

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Bardoxolone
in ADPKD
FALCON
a study in polycystic kidney disease
PHOENIX
A Study For Rare Kidney Disease

ADPKD: Most Common Hereditary Form of CKD

Progressive growth of fluid-filled cysts

Decreased mitochondrial function and chronic inflammation are key drivers of cyst growth and kidney function loss[1,2]

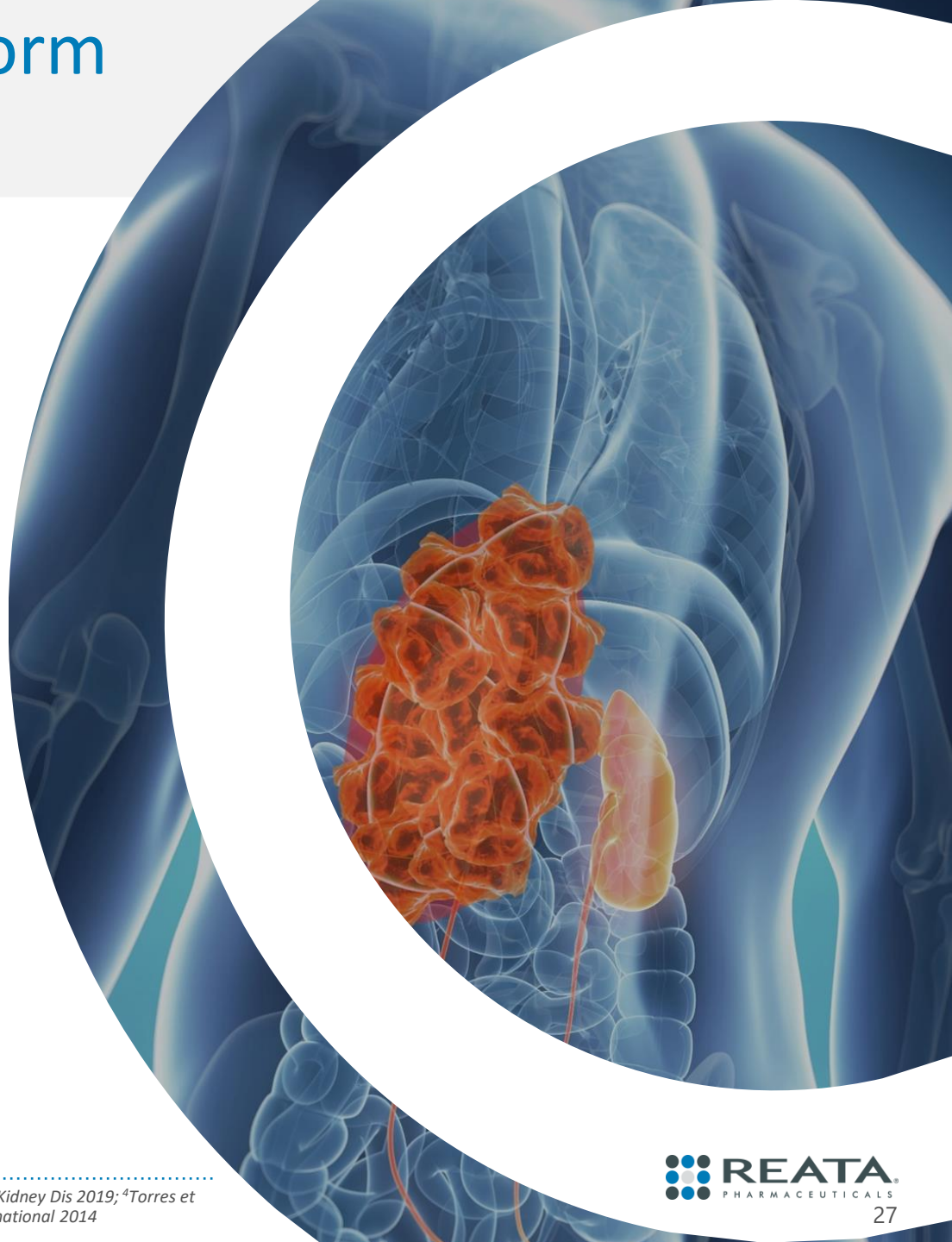
140,000 patients currently diagnosed in the U.S.[3]

-3 to -4 mL/min per year

Rate of kidney function loss despite standard of care[4]

50% of patients 60 years old have **ESKD**

75% of patients 70 years old have ESKD[1,5]



[1]Bergmann et al. Nat Rev Dis Primers. 2018; [2]Podrini et al. Cell Signal. 2020; [3]NIH – About Autosomal Dominant Polycystic Kidney Disease (link), Willey et.al Kidney Dis 2019; [4]Torres et al. N Engl J Med. 2017, Rastogi et al. Ther Clin Risk Manag. 2019; [5]What is ADPKD? Polycystic Kidney Disease Foundation (link), Spithoven et al. Kidney International 2014

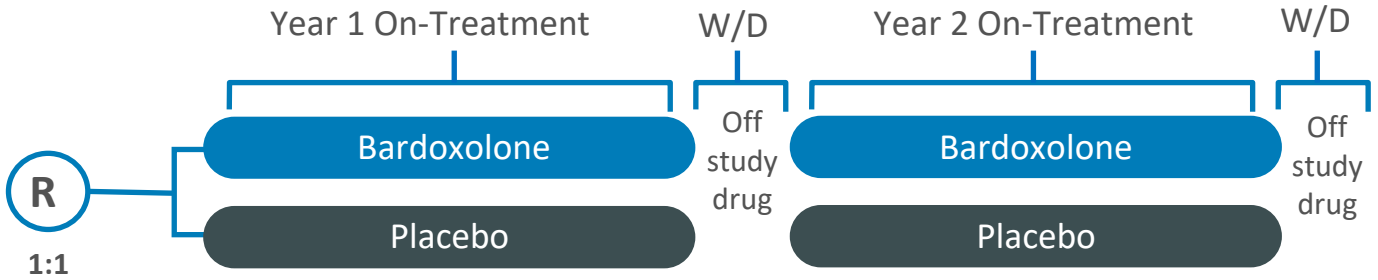
FALCON Phase 3 Study of Bardoxolone in ADPKD

Phase 3 similar in design to CARDINAL with two-year treatment duration

- Planning to enroll 550 patients across approximately 100 sites in the U.S., Europe, Australia, and Japan
- eGFR 30-90 mL/min
- Age 18-70 years old
- **Key primary endpoint**: off-treatment change from baseline in eGFR at Week 52 (4 weeks after withdrawal of drug at Week 48)
- **Secondary endpoint**: off-treatment change from baseline in eGFR at Week 104 (4 weeks after withdrawal of drug at Week 100)

>220 enrolled patients

Planning to complete enrollment by end of 2021

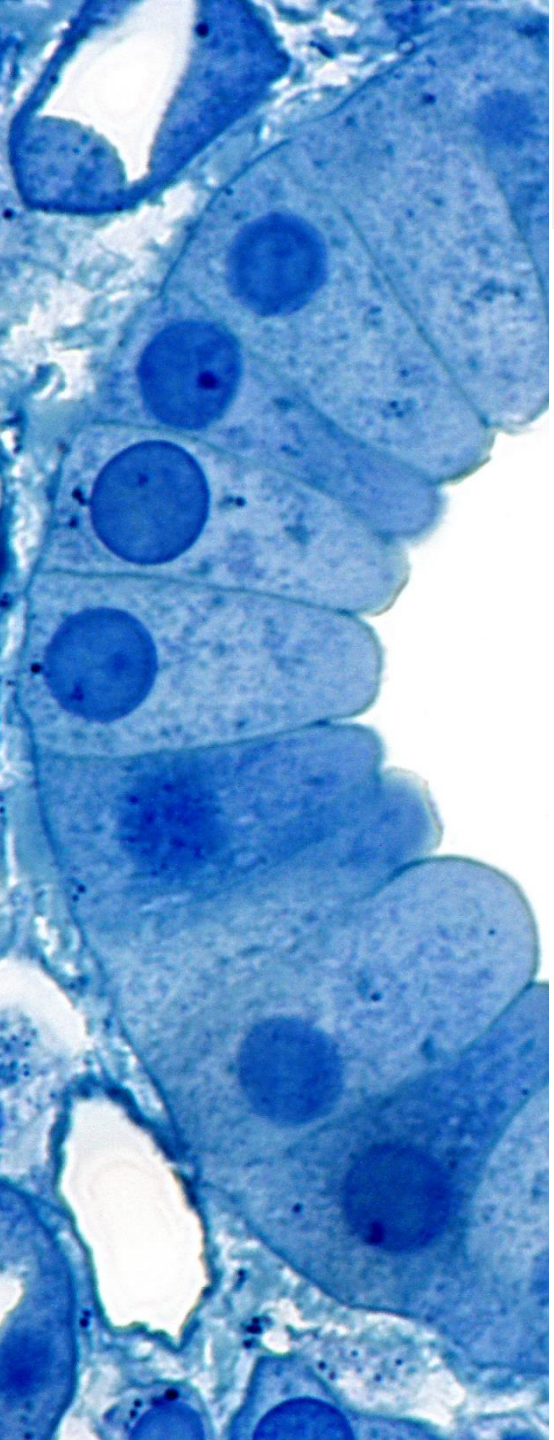


FALCON

REATA PHARMACEUTICALS

Bardoxolone in Patients with CKD at Risk of Rapid Progression

REATA PHARMACEUTICALS | merlin



MERLIN Phase 2 Study of Bardoxolone in Patients with CKD at Risk for Rapid Progression

Proof of concept, Phase 2 trial to evaluate the safety and efficacy of bardoxolone in patients at risk of rapidly progressing CKD due to multiple etiologies

- Multi-center, double-blind, placebo-controlled
- Planning to enroll approximately 70 patients
- eGFR 20-60 mL/min
- One of the following
  - UACR ≥ 300 mg/g
  - Annual eGFR decline rate ≥ 4 mL/min
  - Hematuria
- Age 18-75 years old

Primary endpoint: eGFR change from baseline at Week 12

Secondary endpoint: eGFR change from baseline at Week 12 by CKD etiology

Enrollment began February 2021

Data expected in the second half of 2021

merlin

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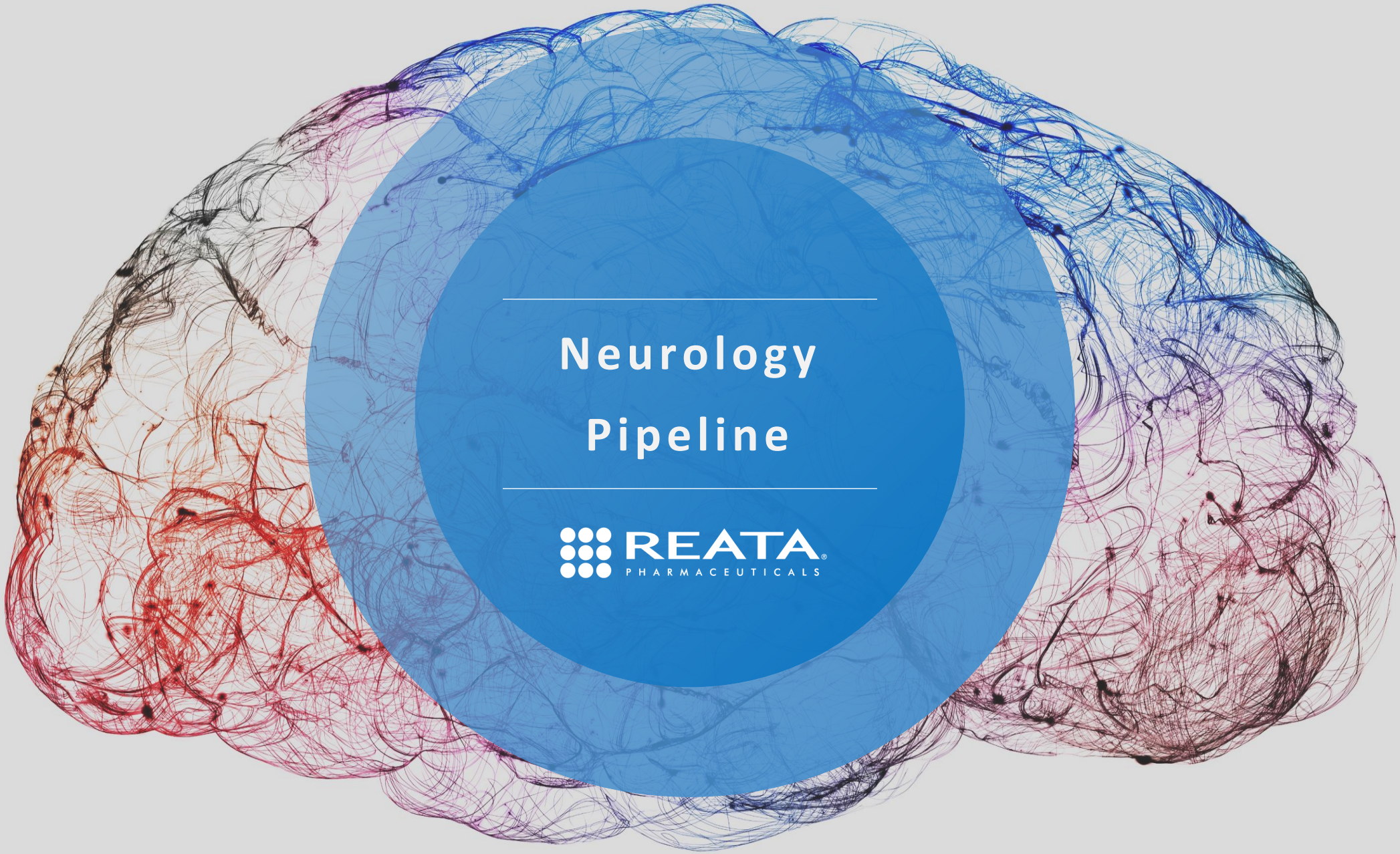
Neurology
Pipeline
REATA
PHARMACEUTICALS


Omaveloxolone in
Friedreich's Ataxia
MOXIe
a study in Friedreich's ataxia
REATA
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Friedreich's Ataxia: No Approved Therapy

Patients progressively lose motor function

Typically diagnosed in teens, wheelchair-bound in 20s[1]

35 Years

Median survival for patients with FA[1]

1-2 points of progression per year

On the modified Friedreich's Ataxia Rating Scale (mFARS)[2]

Most common recessive form of ataxia

In U.S., an estimated 4,000 patients are diagnosed with FA out of 5,000 total

[1]Cook and Giunti, Br. Med. Bull. 2017; [2]Patel et. al. Ann. Clin. Transl. Neurol. 2016

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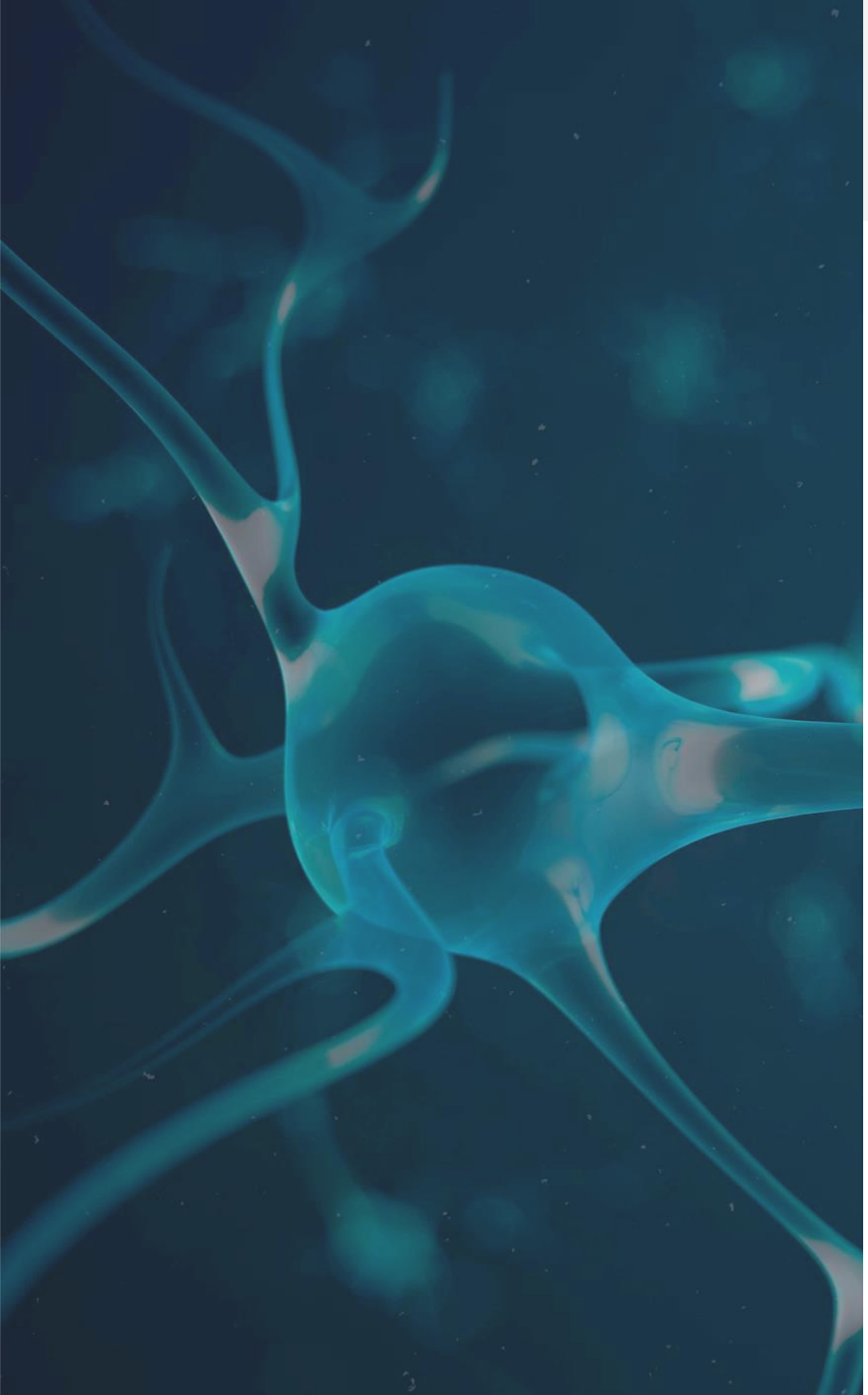


Omaveloxolone Regulatory Update

After review of the MOXIe Part 2 data, FDA recommended additional analyses to increase the persuasiveness of the findings

Treatment-naïve patients from MOXIe Part 1 and Part 2 served as their own controls to assess changes in mFARS in MOXIe Extension in the Baseline-Controlled Study

In response to internal review of the Baseline-Controlled Study analyses, the FDA concluded that they do not believe the results strengthen the results of Part 2 of the MOXIe study

FDA proposed additional analyses comparing patients initially randomized to placebo and omaveloxolone to each other in the extension (the Delayed-Started Analyses)

- We believe these analyses are intended to determine if omaveloxolone's profile is consistent with disease-modification
- FDA stated that the potential for these analyses to sufficiently strengthen the study results was questionable due to the small number of patients available for analysis

Delayed-Start Analyses Support Disease-Modifying Profile

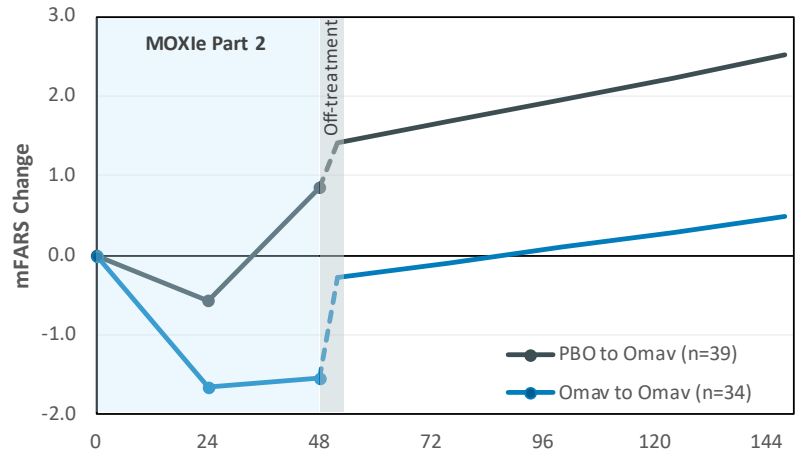
Data from the MOXIe extension study were analyzed as "Delayed-Start" analyses

- Comparison of mFARS during the open-label MOXIe extension for patients randomized to omaveloxlone or placebo during MOXIe Part 2
- Annualized slopes using all data from the MOXIe extension study showed similar slopes in mFARS for both groups
- 89% of MOXIe Part 2 patients enrolled in the extension and were included in the analysis

Parallel trajectories in annualized slopes between both treatment groups is consistent with disease-modifying activity

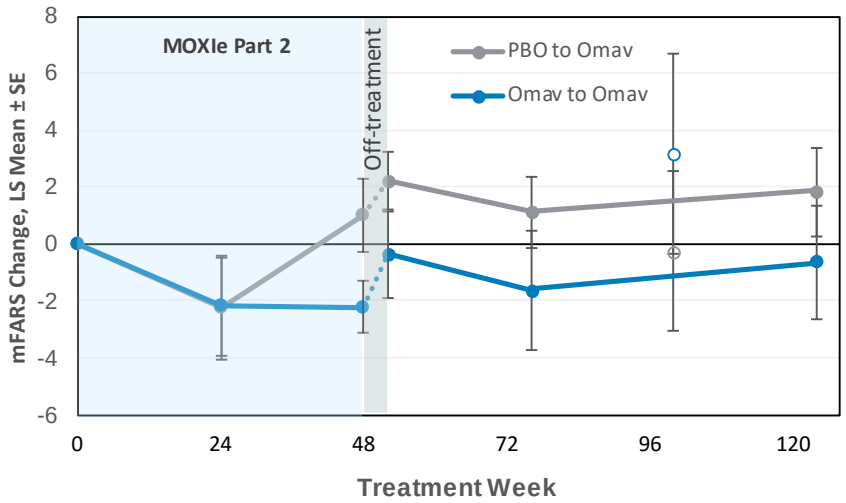
Omaveloxolone prevented worsening of neurological function in 11 patients who have completed 2.5 years of total treatment

All Extension Patients (Annualized Slope)



PBO-Omav (n)	42	41	41	27	22	9	3
Omav-Omav (n)	41	36	34	28	18	11	6

Week 124 Completers (Mean Change from Baseline)



PBO-Omav (n)	9	9	9	9	9	5	9
Omav-Omav (n)	11	11	11	11	11	3	11

mFARS: modified Friedreich's ataxia rating scale; PBO: placebo; Omav: omaveloxolone

Omaveloxolone Next Steps

Requested Type C meeting with FDA to discuss

- Delayed-Start Analyses
- Friedreich's ataxia development program

Plan to initiate a second pivotal study in 2H21, following discussions with FDA and EMA

MOXIe
a study in Friedreich's ataxia

EMA: European Medicines Agency

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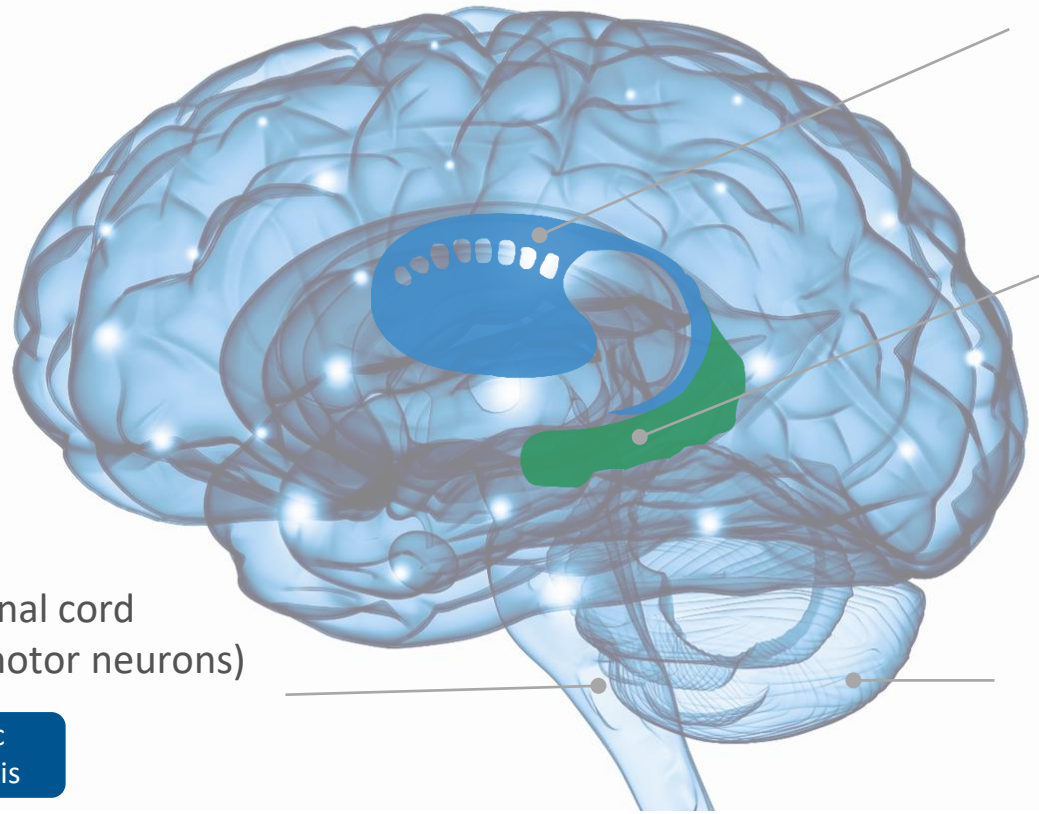
Omaveloxolone Pharmacology May Be Applicable to a Broad Set of Neurological Diseases

MOXIe results provide proof of concept for use of omaveloxolone in other neurological diseases

Mitochondrial dysfunction and neuroinflammation are common features of FA and other neurological diseases

Omaveloxolone and analogs may be applicable to the diseases listed below, and we have observed promising activity in preclinical models of many of these diseases

- Progressive supranuclear palsy (PSP)
- Parkinson's disease
- Frontotemporal dementia
- Huntington's disease
- Amyotrophic lateral sclerosis (ALS)
- Alzheimer's disease
- Epilepsy



Yang et al., PLoS One (2009); Neymotin et al., Free Rad Bio (2011); Dinkova-Kostova et al., (2015); Stack et al., Free Rad Biol Med 49 (2010); Dumont et al., J Neurochem (2009); Kim et al., Cells (2019); Shekh-Ahmad, et al., Brain (2018); Wei et al., Sci Rep (2017)

REATA PHARMACEUTICALS

RTA 901
Hsp90 Modulator for Diabetic Neuropathy

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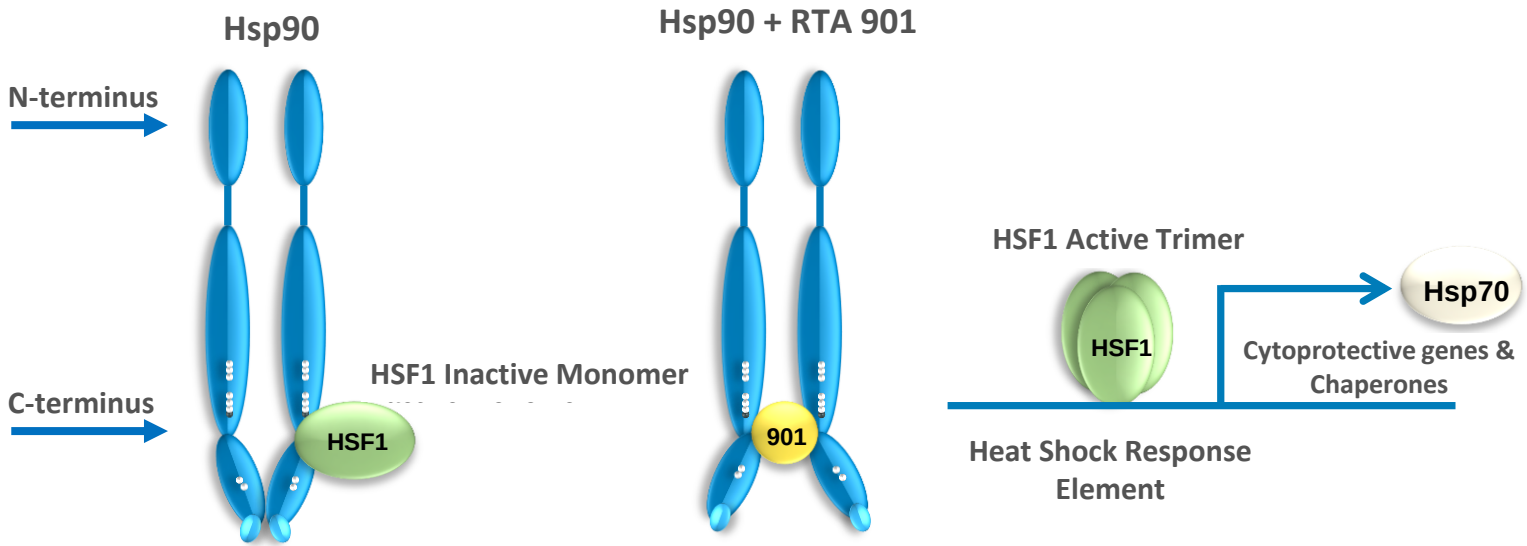
RTA 901: Hsp90 Modulator for Diabetic Peripheral Neuropathic Pain

RTA 901 is a highly potent and selective, oral, small molecule C-terminal modulator of Hsp90

Binding at the C-terminus of Hsp90 disrupts Hsp90-HSF1 complex and leads to increased transcription of Hsp70, a cytoprotective and molecular chaperone gene

- Induction of Hsp70 and cytoprotective response facilitates cell survival in response to cellular stress
- C-terminal Hsp90 inhibition is not associated with cytotoxicity as seen with N-terminal Hsp90 inhibitors previously developed as anti-cancer therapeutics

Reata holds worldwide development and commercial rights to RTA 901



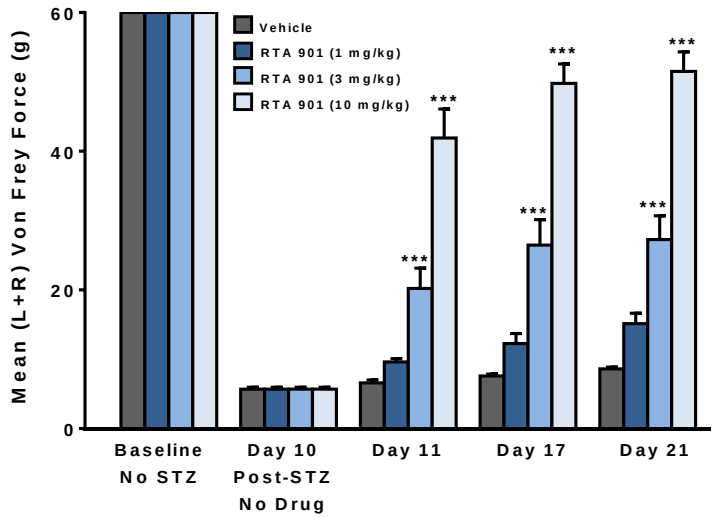
RTA 901 Rapidly Reverses Hyperalgesia and Insensate Neuropathy in Animal Models

RTA 901 treatment in rat model of type 1 diabetic neuropathic pain demonstrated dose-dependent, sustained suppression of hypersensitivity a day after initiation of treatment

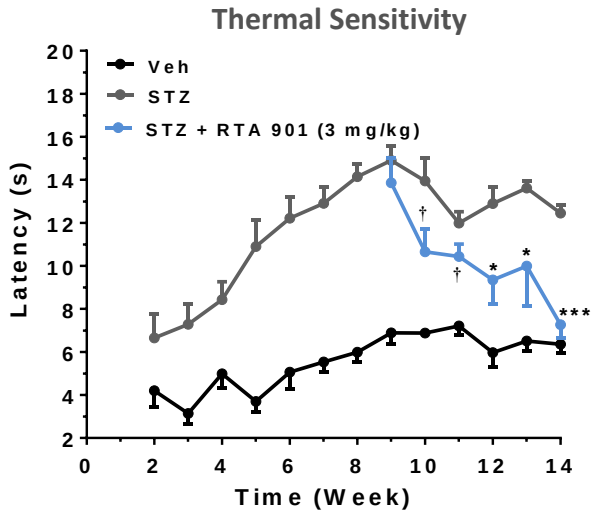
RTA 901 treatment reverses loss of sensation in diabetic mice beginning 2-3 weeks after treatment initiation

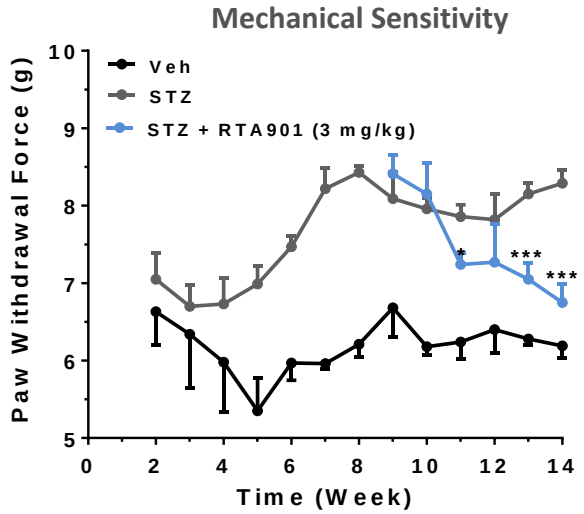
- Significant restoration of mechanical and thermal sensation within 4 weeks
- Effect is dose-dependent, reversible, and Hsp70 dependent
- Nerve conduction velocities and mitochondrial function also significantly improved

Rat Model of Painful Diabetic Neuropathy



Mouse Model of Insensate Diabetic Neuropathy





Phase 2 Study in Diabetic Peripheral Neuropathic Pain Planned for 4Q21

Approximately 4 million patients in the U.S. are affected with moderate or severe diabetic peripheral neuropathic pain (DPNP)[1-3]

- Estimated 2 million adult patients diagnosed with DPNP seek treatment annually[2]
- Approximately half of patients with DPNP do not achieve adequate reduction in pain with approved medications[1,4]
- Approved therapies associated with tolerability issues such as somnolence and dizziness

Phase 1 SAD/MAD study in healthy adult volunteers demonstrated an acceptable PK profile with no safety or tolerability concerns

- Oral, once-daily administration of RTA 901 resulted in exposures approximately 10-fold higher than required for efficacy in preclinical models of DPNP
- RTA 901 was well tolerated across all dose groups with no safety signals, drug discontinuations, or SAEs reported

DPNP development path

- Additional Phase 1 clinical pharmacology studies expected to begin in the second quarter of 2021
- Launch of a randomized, placebo-controlled Phase 2 study in DPNP expected in the fourth quarter 2021

[1]American Diabetes Association; [2]GlobalData Jan 2018 GDHC164PIDR PharmaPoint: Painful Diabetic Neuropathy Global Drug Forecast and Market Analysis to 2026; [3]Yang et.al. Front Endocrinol (Lausanne). 2020, Hicks et.al. Curr Diab Rep. 2019; [4]FDA product labels for Lyrica, Lyrica CR, Cymbalta, Nucynta ER, and Qutenza, Tesfaye et.al. Pain (2013)

Operations, Financials, Intellectual Property

REATA PHARMACEUTICALS

Financial Summary

	Q4 2020	FY 2020
Total Collaboration Revenue	**$ 3.2M**	**$ 9.0M**
Total GAAP Operating Expenses	**$ 57.2M**	**$ 235.3M**
Research and development	$ 37.5M	$ 159.1M
General and administrative	$ 19.4M	$ 75.1M
Depreciation	$ 0.3M	$ 1.1M
Non-GAAP Operating Expenses		
Non-GAAP Research and development[1]	$ 32.7M	$ 131.0M
Non-GAAP General and administrative[1]	$ 12.3M	$ 45.6M
GAAP Net Loss	**$ 65.8M**	**$ 247.8M**
Non-GAAP Net Loss[2]	**$ 43.5M**	**$ 158.3M**

2020 Year End Cash & Shares

- Cash $818.2M
- Total Shares Outstanding 36.1M
  - 31.1M class A shares outstanding
  - 5.0M class B shares outstanding

2021 Financial Guidance

- Based on our operational plans, we anticipate cash runway through mid-2024

[1]Non-GAAP operating expenses exclude stock-based compensation expenses; [2]Non-GAAP net loss excludes stock-based compensation expenses, non-cash interest expense from liability related to sale of future royalties, loss on extinguishment of debt, and gain on lease termination. See the next slide for a reconciliation between GAAP and non-GAAP measures.

Reconciliation of GAAP to Non-GAAP Financial Measures

	Three Months Ended December 31		Twelve Months Ended December 31	
	(unaudited, in thousands, except per share data)			
	2020	2019	2020	2019
Reconciliation of GAAP to Non-GAAP Research and development:				
GAAP Research and development	$ 37,461	$ 40,161	$ 159,080	$ 128,109
Less: Stock-based compensation expense	(4,792)	(3,458)	(28,114)	(8,692)
Non-GAAP Research and development	**$ 32,669**	**$ 36,703**	**$ 130,966**	**$ 119,417**
Reconciliation of GAAP to Non-GAAP General and administrative:				
GAAP General and administrative	$ 19,427	$ 22,271	$ 75,128	$ 58,298
Less: Stock-based compensation expense	(7,158)	(8,833)	(29,519)	(17,689)
Non-GAAP General and administrative	**$ 12,269**	**$ 13,438**	**$ 45,609**	**$ 40,609**
Reconciliation of GAAP to Non-GAAP Operating expenses:				
GAAP Operating expenses	$ 57,173	$ 187,103	$ 235,344	$ 311,737
Less: Stock-based compensation expense	(11,950)	(12,291)	(57,633)	(26,381)
Less: Reacquired license rights	-	(124,398)	-	(124,398)
Non-GAAP Operating expenses	**$ 45,223**	**$ 50,414**	**$ 177,711**	**$ 160,958**
Reconciliation of GAAP to Non-GAAP Net loss:				
GAAP Net loss	$ (65,776)	$ (186,942)	$ (247,752)	$ (290,170)
Add: Stock-based compensation expense	11,950	12,291	57,633	26,381
Add: Reacquired license rights	-	124,398	-	124,398
Add: Non-cash interest expense from liability related to sale of future royalties	10,807	-	21,884	-
Add: Loss on extinguishment of debt	-	-	11,183	-
Less: Gain on lease termination	(470)	-	(1,286)	-
Non-GAAP Net loss	**$ (43,489)**	**$ (50,253)**	**$ (158,338)**	**$ (139,391)**
Reconciliation of GAAP to Non-GAAP Net loss per common share-basic and diluted:				
GAAP Net loss per common share-basic and diluted	$ (1.90)	$ (5.91)	$ (7.35)	$ (9.54)
Add: Stock-based compensation expense	0.35	0.39	1.71	0.87
Add: Reacquired license rights	-	3.93	-	4.09
Add: Non-cash interest expense from liability related to sale of future royalties	0.31	-	0.65	-
Add: Loss on extinguishment of debt	-	-	0.33	-
Less: Gain on lease termination	(0.01)	-	(0.04)	-
Non-GAAP Net loss per common share-basic and diluted	**$ (1.25)**	**$ (1.59)**	**$ (4.70)**	**$ (4.58)**

Preparation for Launch Success
2021 Commercial Readiness Goals

Market Research
Patient journey and path to diagnosis and treatment

Disease Awareness
Physicians, patients, and payers recognize AS, burden of disease, and accurate diagnosis

Product Access & Health Economics
Patients have seamless access to Reata products at approval

Operational Readiness
Reata has all necessary people, processes and systems in place to support a successful launch

2021 Launch Readiness Goals

Disease Awareness Campaign Execution
Q1-Q3 2021

Payer Field Team Deployment
Q2 2021

Nephrology Sales Force Launch
Q4 2021

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Concluding Remarks

Key 2021 Goals

BARDOXOLONE FOR CHRONIC KIDNEY DISEASE		Q1	Q2	Q3	Q4
CARDINAL FOR PATIENTS WITH ALPORT SYNDROME	**Alport Syndrome**				
	NDA submitted requesting Priority Review	✓			
	NDA accepted and filed by FDA		■		
	Expected PDUFA if designated as Priority Review				■
	Submit MAA to EMA				■
FALCON	**ADPKD**				
	Continued FALCON enrollment	■	■	■	■
	Complete FALCON enrollment				■
merlin	**At Risk of Rapid Progression**				
	Initiated MERLIN Phase 2 trial	✓			
	MERLIN data expected			■	■

NEUROLOGY		Q1	Q2	Q3	Q4
MOXIe a study in Friedreich's ataxia	**Omaveloxolone for Friedreich's ataxia**				
	Requested Type C Meeting to discuss additional analyses	✓			
	Initiate second pivotal study			■	■
REATA	**RTA 901 for DPNP**				
	Initiate additional Phase 1 clinical pharmacology studies		■		
	Initiate Phase 2 study in patients with DPNP				■


Q & A