REATA PHARMACEUTICALS

Investor Presentation

June 2020

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

Reata at a Glance

Chronic Kidney Disease Franchise

- Positive pivotal data for Bard[1] in Alport syndrome[2]
- NDA planned for 2020[3]
- Pipeline in rare forms of CKD[1]
  - Pivotal ADPKD[1] study ongoing
  - Positive proof-of-concept data in FSGS[1], IgAN[1], and T1D CKD[1]

Neurology Franchise

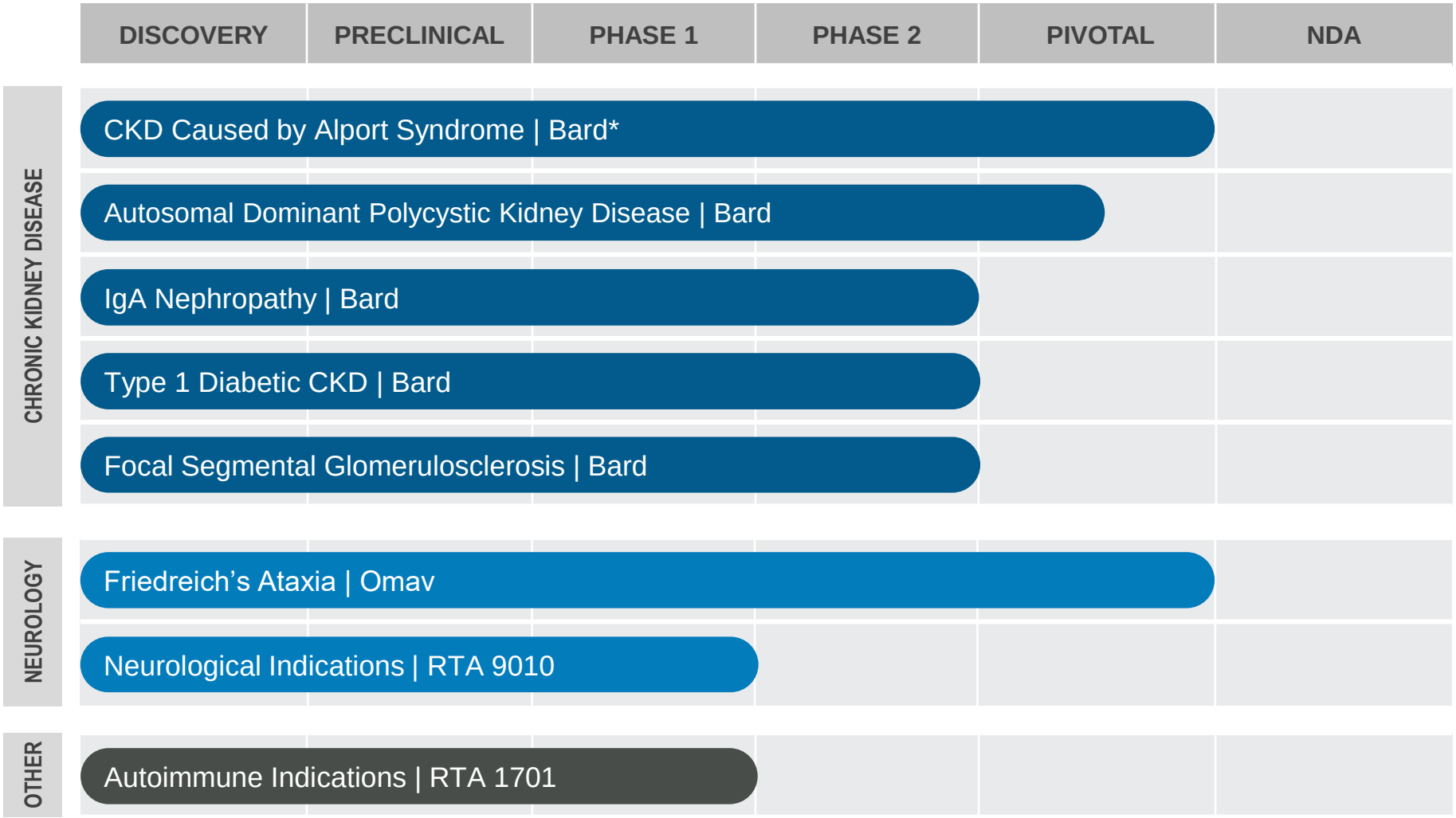
- Positive pivotal data for Omav[1] in Friedreich's ataxia
- NDA planned for 2020[3]
- Plan to study Omav and RTA 901 in additional indications in neurological disease

Global Opportunity

- Few or no effective therapies currently approved for lead indications
- Reata possesses worldwide commercial rights to all pipeline assets[4]
- Commercial leadership in place for global commercial launches
- Robust intellectual property protection for Bard and Omav

[1]See slide 23 for a list of abbreviations; [2]The CARDINAL study reported one-year data in November 2019, and is an ongoing two-year study; [3]Subject to discussion with regulatory authorities; [4]Ex-Asia for Bard

Deep Pipeline With Two Pre-Registration Programs and Two Pivotal Studies Ongoing



**The CARDINAL study reported one-year data in November 2019, and is an ongoing two-year study*

Chronic Kidney Disease

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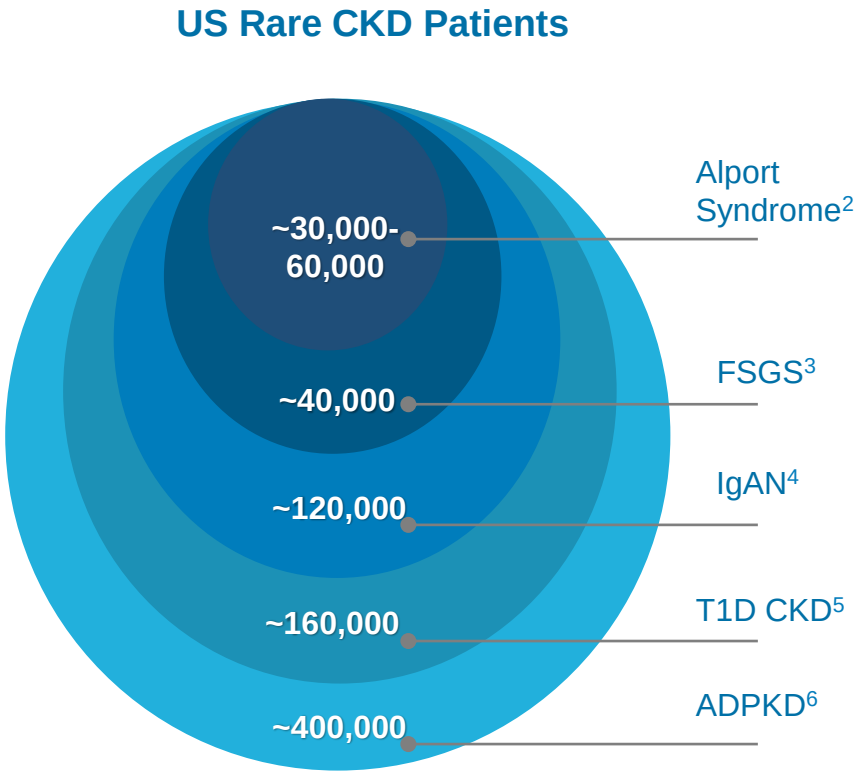
Bard Development for Rare Forms of CKD

Significant opportunity in rare forms of CKD
- We estimate aggregate prevalence exceeds 700,000 patients in the US
- Few or no effective therapies currently approved

CARDINAL Phase 3 study in Alport syndrome met its primary and key secondary endpoints[1]

FALCON Phase 3 study in ADPKD
- FALCON design and endpoints similar to CARDINAL Phase 3 study
- Enrollment temporarily paused in March due to COVID-19, plan to reinitiate this trial

Positive data from PHOENIX in IgAN, FSGS, and T1D CKD; planning to pursue commercially



[1]The CARDINAL study reported one-year data in November 2019, and is an ongoing two-year study; [2]Alport Syndrome Foundation; Estimated based on Persson Clin Nephrol (2005); USRDS Report; Hasstedt Am J Hum Genet (1983); Temme, Kidney Int (2012); [3]Wetmore (2016), Sim (2016); [4]Berthoux (2011), Wetmore (2016), Sim (2016); [5]American Diabetes Association, Garofolo (2018), Ohta (2010); [6]PKD Foundation

Alport Syndrome is a Severe, Inherited Form of CKD

- Alport syndrome is the second most common inherited cause of kidney failure[1,2]
- Collagen mutations drive mitochondrial dysfunction, inflammation, and fibrosis[3,4]
- Progressive loss of kidney function leads to need for dialysis or kidney transplant
- In most severe forms of disease, median age for kidney failure is 25 years[1]
- Very severe form of CKD with no approved therapies

Estimated Prevalence[5]

[1]Jais et al., JASN (2000); [2]Kashtan et al., Kidney Int (2018); [3]Suh et al., Nat Rev Nephrol (2013); [4]Gomez et al., JCI (2015); [5]Alport Syndrome Foundation; Estimated based on Persson Clin Nephrol (2005); USRDS Report; Hasstedt Am J Hum Genet (1983); Temme, Kidney Int (2012)

Reata is Sponsoring KIDNEYCODE, a Genetic Testing Program for Chronic Kidney Disease

Recent literature suggests a large number of Alport syndrome patients are currently undiagnosed or misdiagnosed[1]

KidneyCode is a genetic test that includes a panel of 17 genes, including those that cause Alport syndrome

KidneyCode is helping to identify prevalence of Alport syndrome

- Approximately half of patients with available results tested positive for mutations in COL4A genes
- Approximately half of patients with mutations in COL4A genes were misdiagnosed with another form of CKD

Three straightforward clinical risk factors able to identify patients with Alport Syndrome

- Risk factors include 1) CKD, 2) family history of CKD, and 3) hematuria
- 78% of patients with all three risk factors tested positive for mutations in COL4A genes

Misdiagnoses for Patients who have Alport Syndrome
ADPKD
Benign Familial Hematuria
Benign Hereditary Nephritis
Familial Hematuria
FSGS
Hypertensive CKD
IgA Nephropathy
Thin Basement Membrane Disease
Congenital Familial Hematuria

[1]*Groopman 2019; Bullich 2018; Malone 2014; Papazachariou 2014*

CARDINAL Phase 3: Pivotal Study Design and Status

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

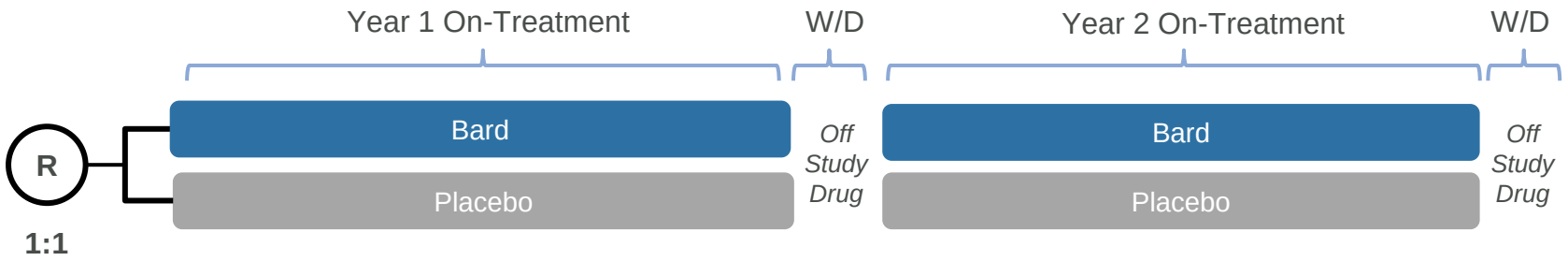
Largest interventional study in patients with AS

Enrolled a wide and representative range of patients with AS
- eGFR: 30 to 90 mL/min
- Age: 12 to 70 years

Endpoints: eGFR change from baseline on-treatment at week 48 and off-treatment at week 52

FDA provided guidance that off-treatment eGFR benefit versus placebo at one year may support accelerated approval and at two years may support full approval

Integrity of year two is intact during the COVID-19 pandemic
- Blood-based endpoints enable data collection for safety and efficacy through at-home visits
- Shipping drug supply directly to patients' homes
- Over half of enrolled patients have completed the study

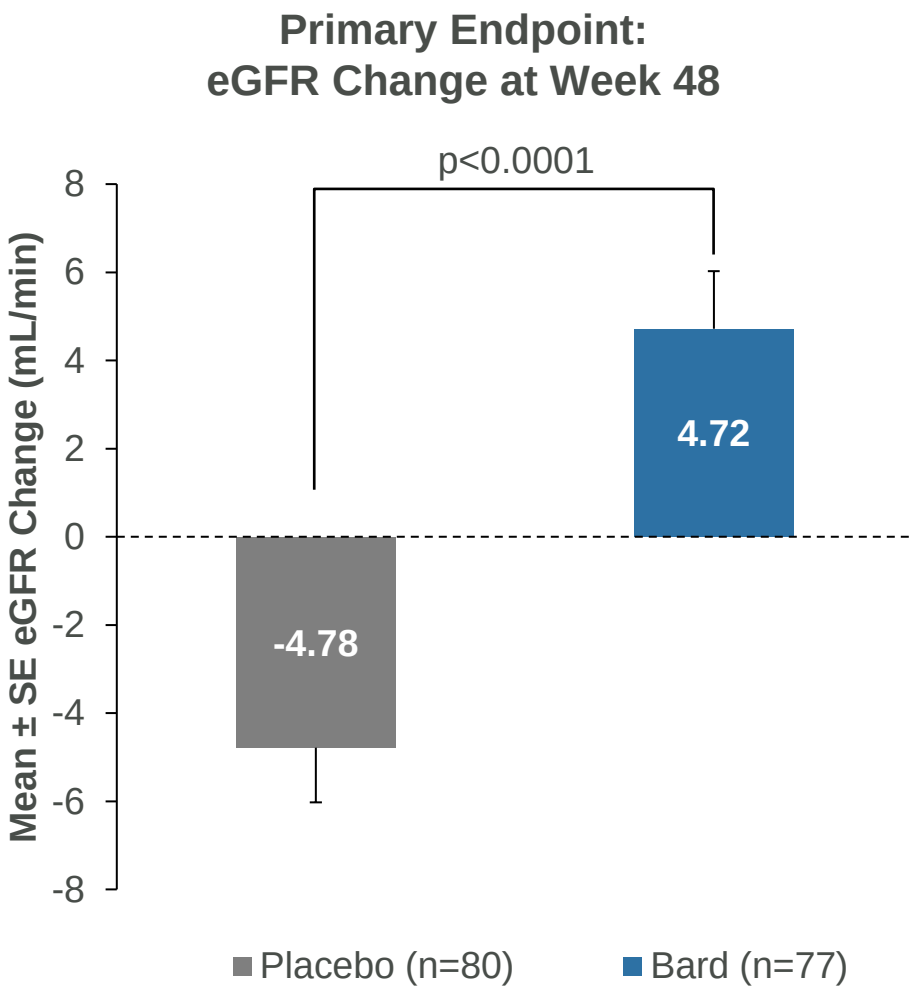


CARDINAL Met the Primary Endpoint

Bard treatment significantly improved on-treatment eGFR at Week 48 by 9.50 mL/min relative to placebo (p<0.0001)

Placebo patients had significant kidney function loss versus baseline (p=0.0002)*

Bard significantly improved eGFR (p=0.0004)* versus baseline



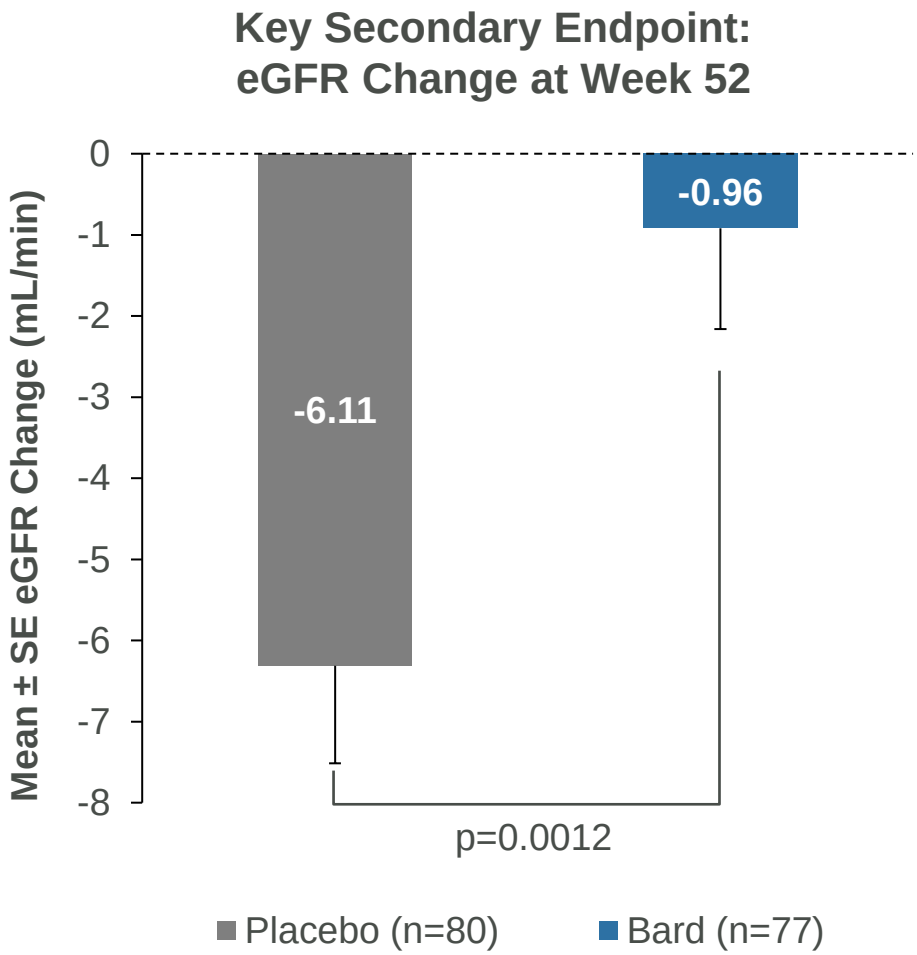
*p-value estimated comparing the mean changes to zero

CARDINAL Met the Key Secondary Endpoint

Bard treatment significantly improved off-treatment eGFR at Week 52 by 5.14 mL/min relative to placebo (p=0.0012)

Placebo patients had significant kidney function loss relative to baseline (p<0.0001)*

Bard patients had a slight, non-significant decline relative to baseline (p=0.45)*



ANCOVA analyses include off-treatment eGFR values from patients that discontinued treatment early

*p-value estimated comparing the mean changes to zero

Bard Treatment Produced Benefit Across All Quartiles of Response

	Week 48 eGFR Change* Mean ± SD		Week 52 eGFR Change* Mean ± SD	
ΔeGFR Quartile	Placebo	Bard	Placebo	Bard
1	4.2 ± 2.4	20.9 ± 11.5	5.0 ± 4.2	12.9 ± 8.1
2	-1.3 ± 1.4	9.2 ± 2.4	-2.5 ± 1.4	2.5 ± 1.6
3	-5.7 ± 1.5	1.7 ± 2.2	-7.4 ± 2.3	-3.2 ± 1.7
4	-16.1 ± 6.4	-10.5 ± 6.4	-17.3 ± 5.0	-12.7 ± 5.6

**eGFR change measured as mL/min*

Phase 3 CARDINAL Summary of Safety at Year One

Fewer SAEs reported in Bard patients

No fluid overload or major adverse cardiac events in patients treated with Bard

Most common AEs included muscle spasms and increased ALT or AST

Albuminuria increased with Bard at Week 48 but was unchanged when adjusted for eGFR and unchanged from baseline at Week 52

No individual AE contributed to more than two discontinuations in either group

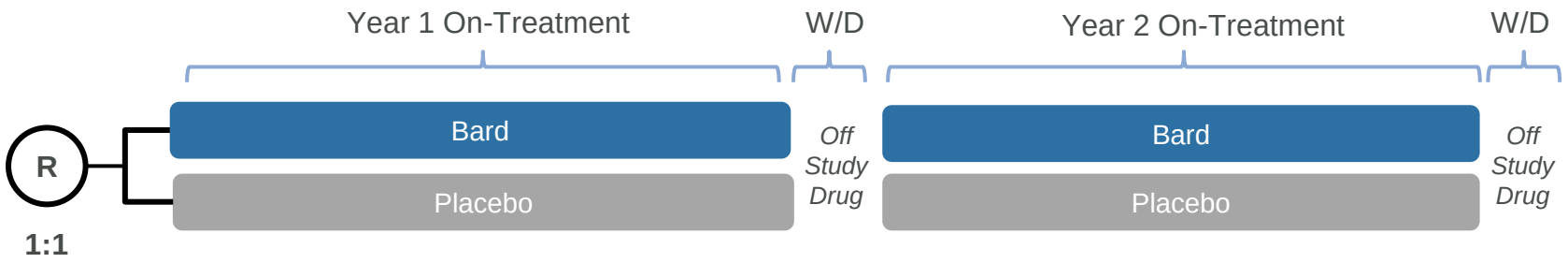
	Placebo (n=80)	Bard (n=77)
Number of Patients with AE	73 (91%)	75 (97%)
Number of Patients with SAE	10 (13%)	4 (5%)
Number of Patients with AE Leading to Permanent Treatment Discontinuation	4 (5%)	9 (12%)

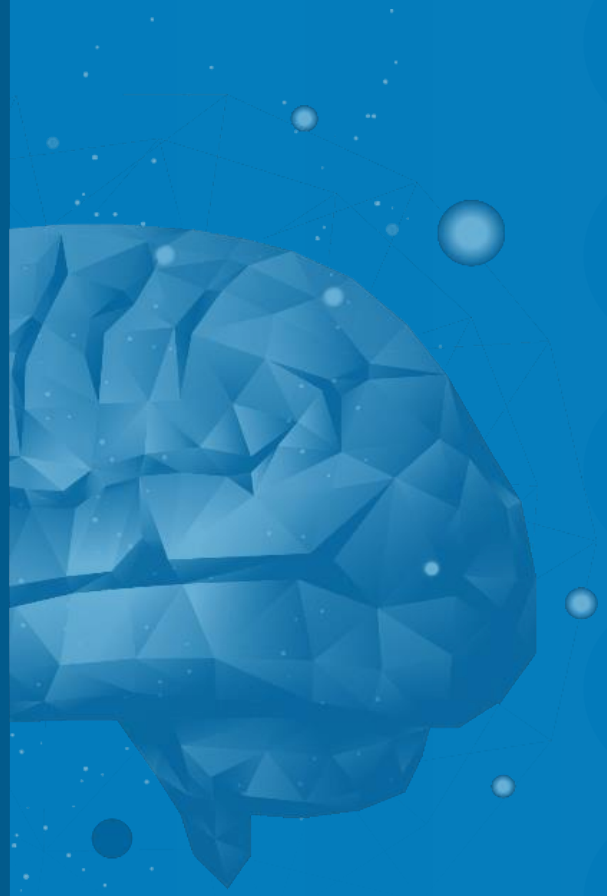
FALCON Phase 3 Trial of Bard for ADPKD

Pivotal Phase 3 similar in design to CARDINAL
- Two-year total treatment duration
- Planning to enroll patients across approximately 60 sites in the US, Europe, and Australia
- eGFR 30-90 mL/min
- Age 18-70 years old
- On- and off-treatment eGFR change endpoints support approval

Trial enrollment paused in March 2020; screening may restart in current quarter

Integrity of study is intact during the COVID-19 pandemic
- Blood-based endpoints enable data collection for safety and efficacy through at-home visits
- Shipping drug supply directly to patients' homes





Neurology

Omaveloxolone

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Overview of Friedreich's Ataxia

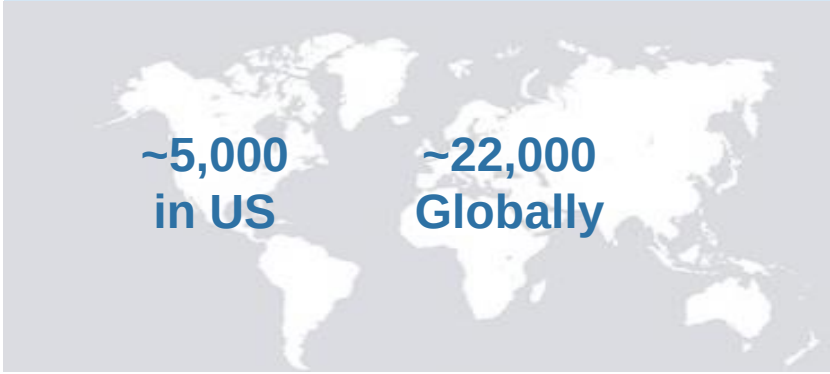
Friedreich's ataxia is a rare, debilitating, life-shortening, neuromuscular disorder

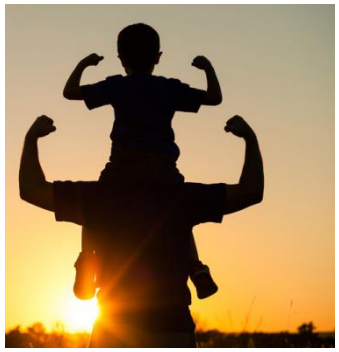
Patients typically become wheelchair-dependent 10 to 15 years after diagnosis and eventually lose independence

Most patients diagnosed as children and survive into their mid-30s[1-3]

Numerous failed trials and no approved therapies

Estimated Prevalence[4-9]





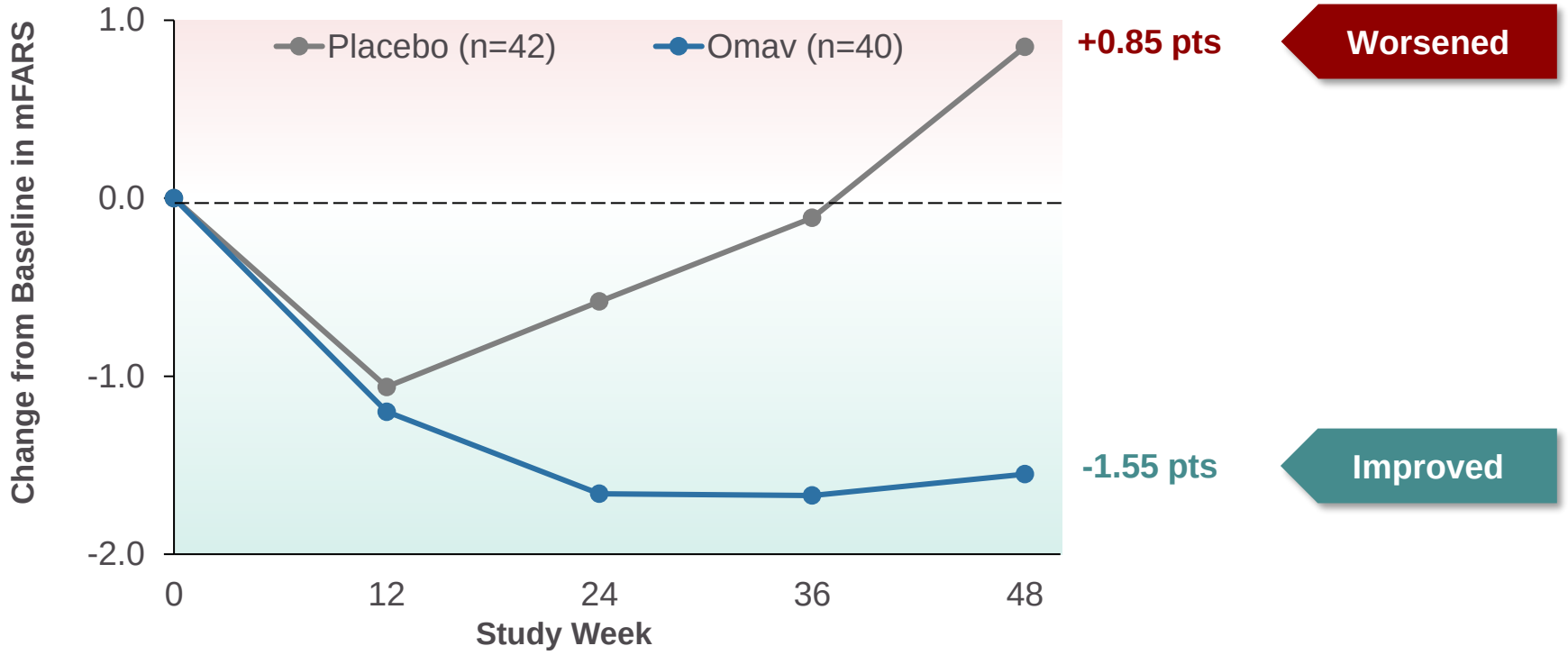
[1] Parkinson MH, et al., J Neurochem 2013; [2]Santos R, et al., Antioxid Redox Signal 2010; [3]Klockgether T, et al., Brain. 1998; ; [4]Friedreich's Ataxia Research Alliance; [5]Vankan P, J Neurochem 2013; [6]Zheng J, et. al. J Neurol Sci 2015, [7]Sasaki H, et. al. J Neurol Sci 2000 ; [8]Mariño TC, et. al. Clin Genet 2010; [9]Fussiger H, et. al. Cerebellum 2019

MOXIe Pivotal Study: Design and Results

International, double-blind, placebo-controlled, randomized, registrational Phase 2 trial

- Largest global interventional study in patients with FA
- Enrolled a wide and representative range of patients with FA
- Patients randomized 1:1 to receive 150 mg Omav or placebo for 48 weeks

Achieved primary endpoint of change in the mFARS relative to placebo after 48 weeks of treatment (2.4 points, p=0.014)



MOXIe Met Important Secondary Endpoints

Improvements observed in Patient Global Impression of Change (PGIC)

- Primary analysis population (n=82; p=0.125)
- All randomized population (n=103; p=0.028)
- Patient-reported PGIC correlated with physician-assessed changes in mFARS (p<0.001)

Activities of Daily Living score achieved nominal significance versus placebo (n=82; p=0.042)

65% reduction in frequency of falls

Activities of Daily Living

Section	Improved with Omav[1]
Speech	✓
Swallowing	✓
Cutting Food and Handling Utensils	✓
Dressing	✓
Personal Hygiene	✓
Falling	✓
Walking	✓
Quality of Sitting Position	✓
Bladder Function	✓
Total	✓ **(p=0.042)**

[1] All sections demonstrated numerical improvements relative to placebo

MOXIe Summary of Safety

AEs generally mild to moderate in intensity
- 4 (8%) Omav patients and 2 (4%) placebo patients discontinued study due to AEs
- ALT and AST increases are a pharmacological effect of Omav[1]
  - Not associated with liver injury
  - Coincide with decreases in total bilirubin
  - May reflect improvements in mitochondrial metabolism

Low rate of serious AEs (SAEs)
- SAEs reported in 3 (6%) Omav patients and 3 (6%) placebo patients while receiving study drug
- Two additional Omav patients reported SAEs approximately 2 weeks after receiving final dose

Ninety-eight percent of patients chose to enroll in the long-term extension study

Summary of Adverse Events*

Preferred Term	Placebo (n=52)	Omav (n=51)
Contusion	19 (37%)	17 (33%)
Headache	13 (25%)	19 (37%)
Upper respiratory tract infection	15 (29%)	14 (28%)
Excoriation	12 (23%)	13 (26%)
Nausea	7 (14%)	17 (33%)
ALT increased	1 (2%)	19 (37%)
Fatigue	7 (14%)	11 (22%)
Abdominal pain	3 (6%)	11 (22%)
AST increased	1 (2%)	11 (22%)

*AEs reported in >20% of patients

[1]*Miller et al., ASN 2010*

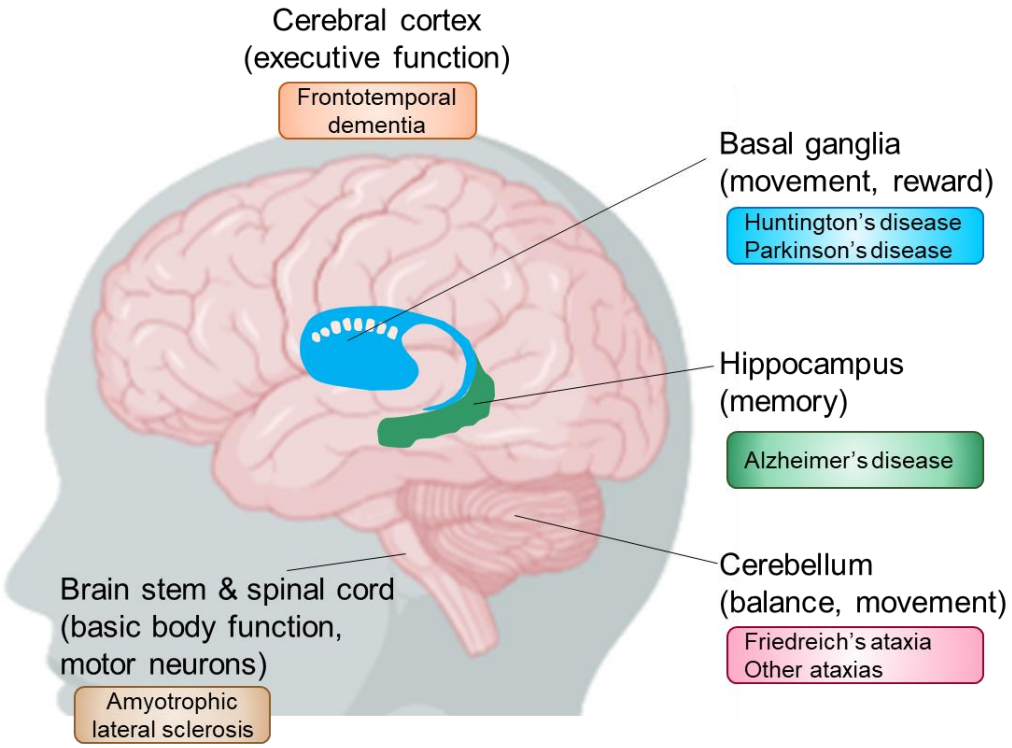
Omav Pharmacology May Be Applicable to a Broad Set of Neurological Diseases

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

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

Omav and analogs have demonstrated broad activity in neurological models

- Parkinson's disease
- Dementia
- Epilepsy
- Huntington's disease
- Amyotrophic lateral sclerosis (ALS)
- Alzheimer's disease



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)

Appendix

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Reata Patent Portfolio

BARDOXOLONE METHYL

- Morphic form patent (US 8,088,824) claims commercial (amorphous) forms of Bard
- Claims granted in US, EU, Japan, Canada, China, Mexico, Eurasia, and 10 other territories, with applications pending in 7 other countries
- Anticipated protection to 2034 in US and 2033 in rest of world with term extensions

OMAVELOXOLONE

- Composition of matter patent (US 8,993,640) claims Omav and several solid forms
- Granted in US, Europe, Japan, China, Eurasia, 14 other territories, and pending in 13 other countries
- Anticipated protection to 2035 in US and 2036 in EU and Japan with term extensions
- Genus patent (US 8,124,799) claims granted in 17 foreign territories, and pending in 1 more

List of Abbreviations

6MWD	Six-minute walk distance
ACR	Albumin to creatinine ratio
ADPKD	Autosomal dominant polycystic kidney disease
AE	Adverse Event
ALS	Amyotrophic lateral sclerosis
ALT	Alanine aminotransferase
AS	Alport syndrome
AST	Aspartate transaminase
Bard	Bardoxolone methyl
CKD	Chronic kidney disease
eGFR	Estimated glomerular filtration rate
ESKD	End-stage kidney disease
FA	Friedreich's ataxia
FDA	US Food and Drug Administration
FSGS	Focal segmental glomerulosclerosis
GFR	Glomerular filtration rate
HSP90	Heat shock protein 90
IgAN	IgA nephropathy
mFARS	Modified Friedreich's ataxia rating scale
mL/min	mL/min/1.73 m^2
NDA	New drug application
Nrf2	Nuclear factor (erythroid-derived 2)-related factor 2
Omav	Omaveloxolone
PK/PD	Pharmacokinetics/pharmacodynamics
SAE	Serious Adverse Event
T1D CKD	Type 1 diabetic chronic kidney disease
T2D CKD	Type 2 diabetic chronic kidney disease
TBMN/TBMD	Thin basement membrane nephropathy/disease
UACR	Urinary albumin to creatinine ratio
W/D	Withdrawal


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