REATA PHARMACEUTICALS

INVESTOR PRESENTATION

July 2018

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, business strategy, and plans and objectives of management for future operations. In some cases, you can identify forward-looking statements by terminology such as “believe,” “will,” “may,” “estimate,” “continue,” “aim,” “assume,” “anticipate,” “contemplate,” “model,” “intend,” “target,” “project,” “should,” “plan,” “expect,” “predict,” “could,” “possible,” “seek,” “goal”, “potential,” “hypothesize,” “likely” 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, 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 forecast in these statements. Any differences could be caused by a number of factors including but not limited to: the success, cost, and timing of our product development activities and clinical trials; our ability to advance our Nrf2 activators and other technologies; our ability to obtain and maintain regulatory approval of our product candidates, and limitations and warnings in the label of an approved product candidate; 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; the size and growth potential of the markets for our product candidates, and our ability to identify target patient populations and serve those markets, especially for diseases with small patient populations; 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 contract with third-party suppliers and manufacturers and their ability to perform adequately; our ability to attract 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; and regulatory developments in the United States and foreign countries.

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.

Reata Develops Drugs with Profound Biological Activity for the Treatment of Severe and Intractable Diseases

Innovative Science

- Lead the emerging field of immuno-metabolism
- Develop first-in-class modulators of Nrf2, Hsp90, and RORγt
- Lead drugs target Nrf2 to improve metabolism and resolve inflammation
- Target genes that are common pathways of injury in many diseases
- Conduct a battery of POC studies and advance indications with best data

Severe Diseases

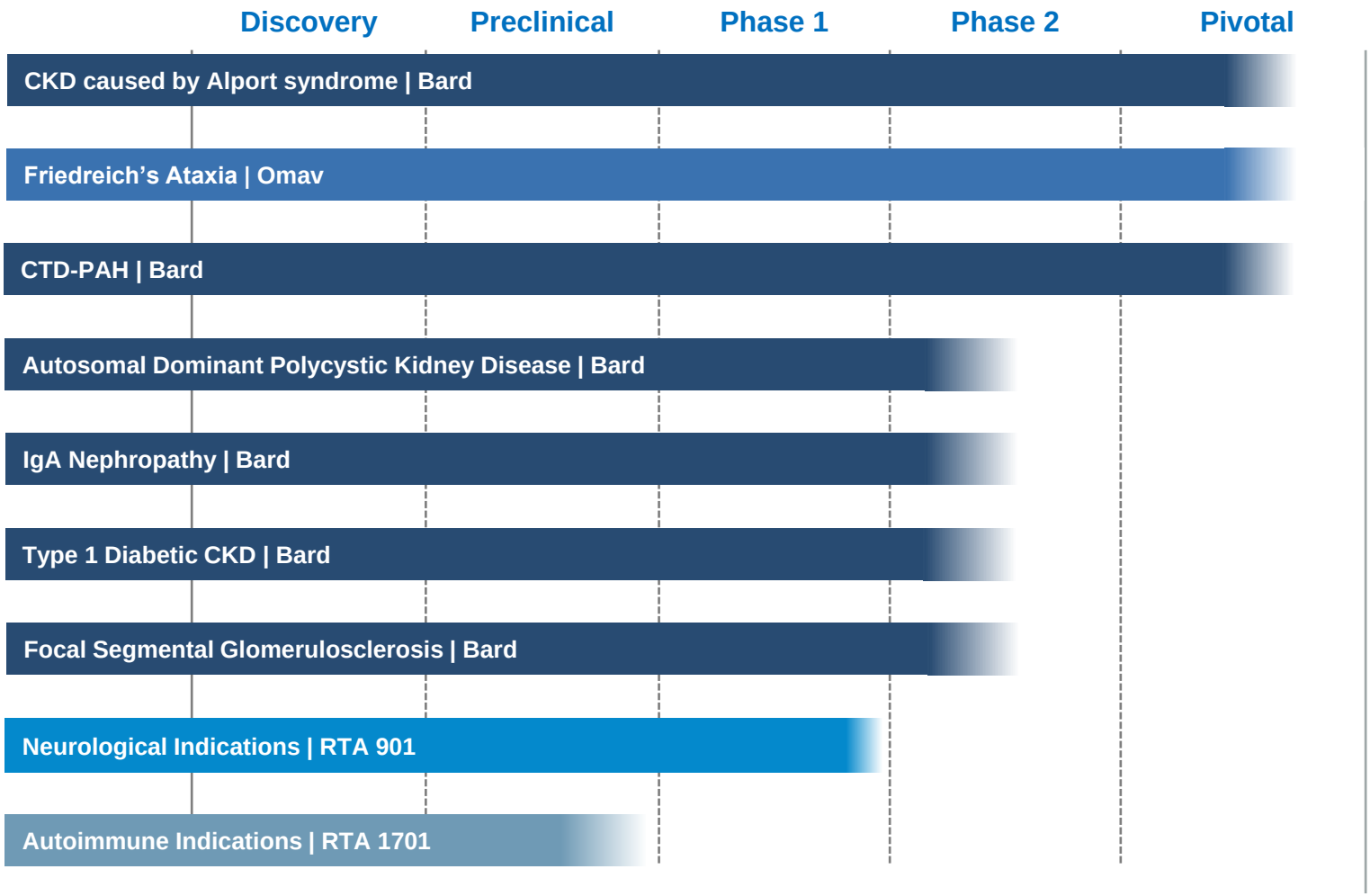
- Select diseases with no approved or effective therapies
- Collaborate actively with patient advocacy groups
- Obtain clear guidance from FDA and regulators on approvable endpoints
- Conducting three pivotal programs in parallel with readouts 2H19 through 1H20

Enterprise Building

- Experienced, long-standing leadership team
- Partnerships for lead programs with global pharma companies
- Strong IP position across all programs
- Strong capital position
- 100 employees located in Dallas/Ft. Worth area

REATA PHARMACEUTICALS

Deep Pipeline with Three Pivotal Studies and Many Expansion Opportunities

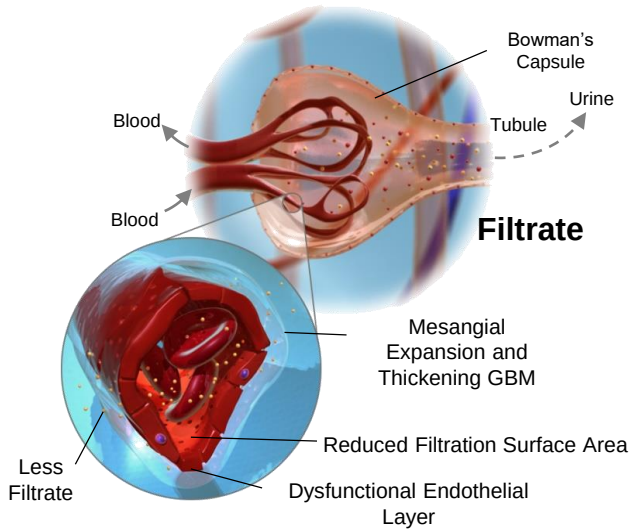


REATA PHARMACEUTICALS

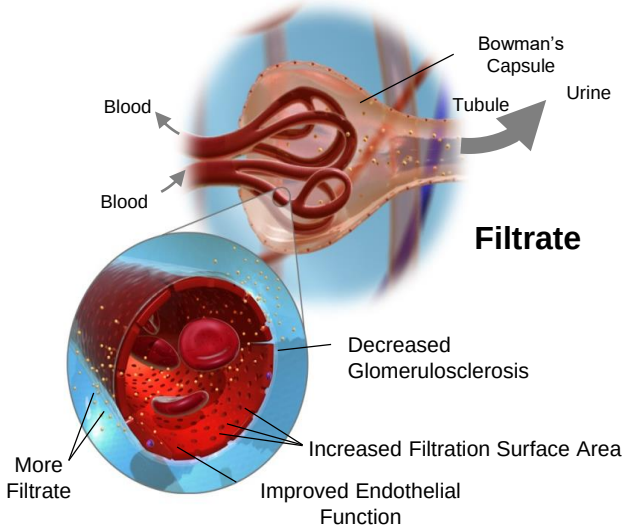
Bardoxolone MOA Addresses Final Common Pathway of Progression in CKD

- In CKD patients, the kidney's filtration rate (eGFR) chronically declines until it reaches ~ 15 ml/min[1] when dialysis or a transplant is required for survival
- Bard acts to increase GFR by reducing inflammation and restoring glomerular function[2]
  - In 11 clinical trials, observed increased eGFR in Bard-treated patients compared to placebo
  - eGFR increases verified as true improvement by "gold standard" methods
  - eGFR improvements durable for two years and partially retained after drug withdrawal
  - Reduced risk of kidney failure observed in diabetic CKD patients treated with Bard in BEACON

Inflamed



Bardoxolone Methyl

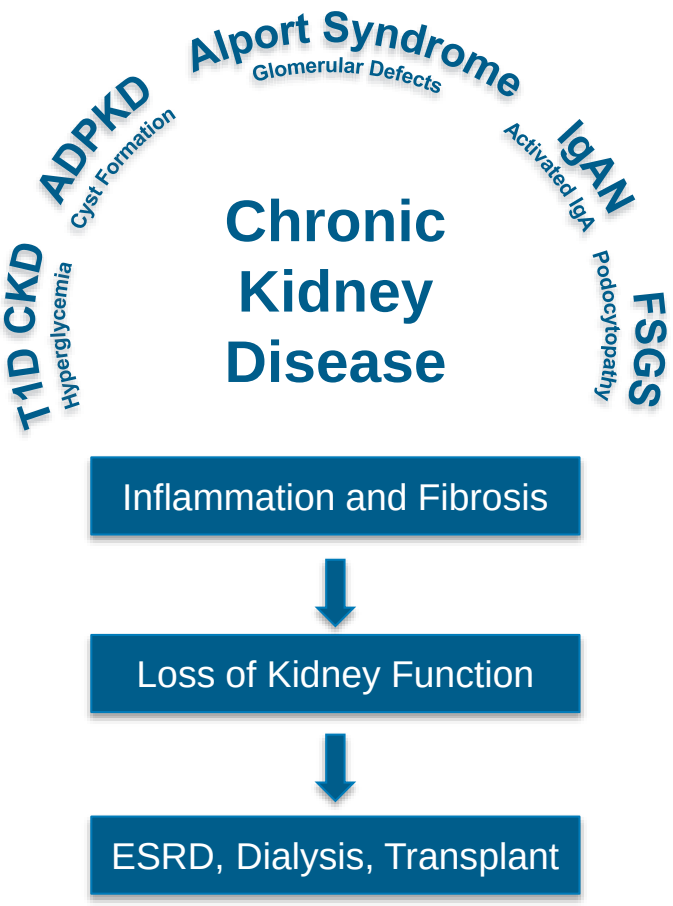


REATA PHARMACEUTICALS

[1]Units of ml/min/1.73 m[2] are represented as ml/min throughout this presentation; [2]Demonstrated in animal models of CKD

Reata is Developing Bardoxolone in Five Rare Forms of CKD

- Conducting pivotal Phase 2/3 CARDINAL study in Alport syndrome (AS)
- Conducting Phase 2 PHOENIX study in ADPKD, IgAN, T1D CKD, and FSGS
- Collectively impact more than 700,000 patients in US
- One-third of kidney failure patients in the US have a rare form of CKD
- Have reported data from both CARDINAL and PHOENIX Phase 2 studies
  - CARDINAL one year on-treatment and retained eGFR benefit in AS
  - PHOENIX full Week 12 eGFR data in ADPKD



FDA Has Accepted Retained eGFR Benefit for Approval in Rare Forms of CKD

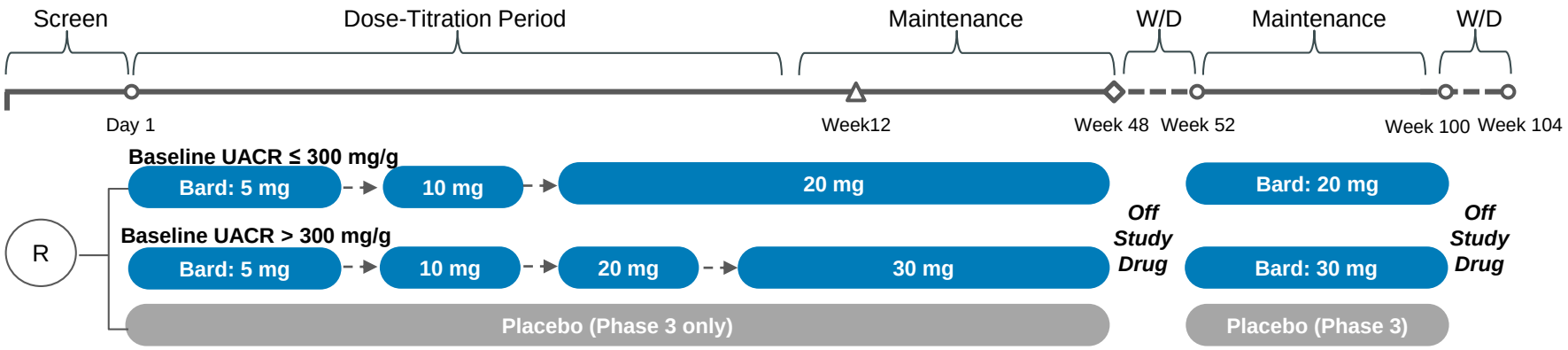
- The “on-treatment” eGFR improvement is the full clinical benefit to the patient, but FDA requires additional evidence that it will likely delay kidney failure
- Withdrawal of drug after long-term treatment provides evidence whether an intervention protected or harmed the kidney during treatment
  - If retained eGFR is higher than placebo, the treatment protected kidney function
  - If retained eGFR is lower than placebo, the treatment harmed kidney function
- A positive retained eGFR benefit:
  - Provides evidence that drug treatment may delay or prevent kidney failure
  - Suggests drug treatment did not improve kidney function through a damaging mechanism
- In rare forms of CKD, FDA has accepted for approval the placebo-corrected “retained eGFR benefit” after withdrawal of drug
- FDA approved tolvaptan for ADPKD on a placebo-corrected (but below baseline) retained eGFR benefit of 1.27 ml/min[1,2]

REATA PHARMACEUTICALS

[1]Units of ml/min/1.73 m^2 are represented as ml/min throughout this presentation; [2]Torres 2017

CARDINAL: Phase 2/3 Trial Design

- CARDINAL Phase 2 is open-label and enrolled 30 patients
  - Reported at ASN 2017 that study achieved primary endpoint of statistically significant eGFR improvement from baseline after 12 weeks of treatment
  - Patients to be followed for two years
- CARDINAL pivotal Phase 3 enrolling up to 150 patients
  - Same eligibility criteria and treatment schedule as Phase 2
  - Potential accelerated approval on retained eGFR after one year of treatment and drug withdrawal
  - Potential full approval on retained eGFR after 2 years of treatment and drug withdrawal
  - Primary data expected 2H19



Baseline Characteristics and Historical eGFR Decline Data

- CARDINAL baseline characteristics representative of AS population
- Collected historical eGFR data for 3 years prior to study initiation
  - 22 of 25 Phase 2 patients
  - Average annual eGFR loss prior to study of ~4.2 ml/min
  - Historical eGFR trend consistent with AS natural history study demonstrating average annual loss of 4 ml/min[1]
- CARDINAL Phase 2 patients' kidney function was, on average, actively declining prior to study entry despite receiving standard of care

CARDINAL Phase 2 Baseline Characteristics

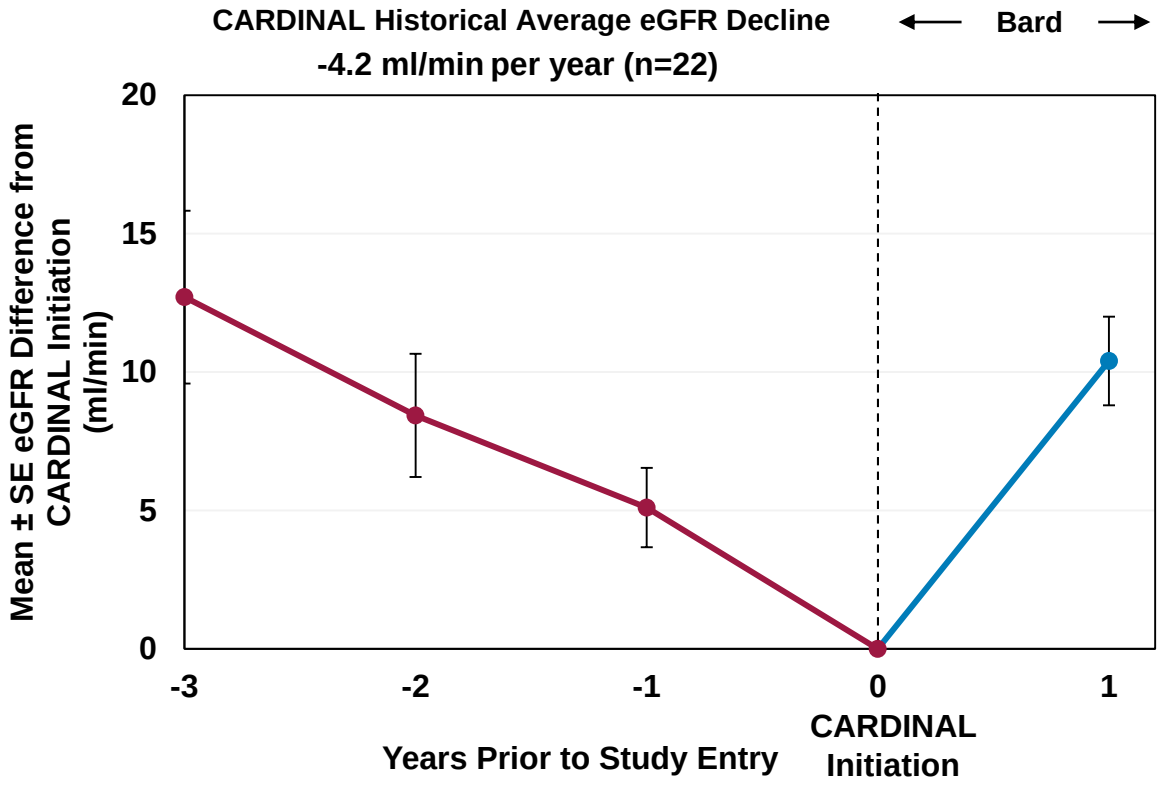
Characteristic	Total (N=25)
Age, years (mean ± SD)	45 ± 12
Baseline eGFR, ml/min (mean ± SD)	56 ± 24
Baseline UACR, mg/g (geometric mean)	129
Receiving ACEi or ARB (n,%)	21 (84%)

CARDINAL Phase 2 Historical eGFR (n=22)

Years Prior to CARDINAL Entry	-3	-2	-1	Average
Mean eGFR Decline (ml/min)	-4.3	-3.3	-5.4	**-4.2**

[1]*ASN 2016; Poster FR-PO636*

Statistically Significant eGFR Improvement Observed in Bard Treated Patients That Historically Declined ~4.2 ml/min

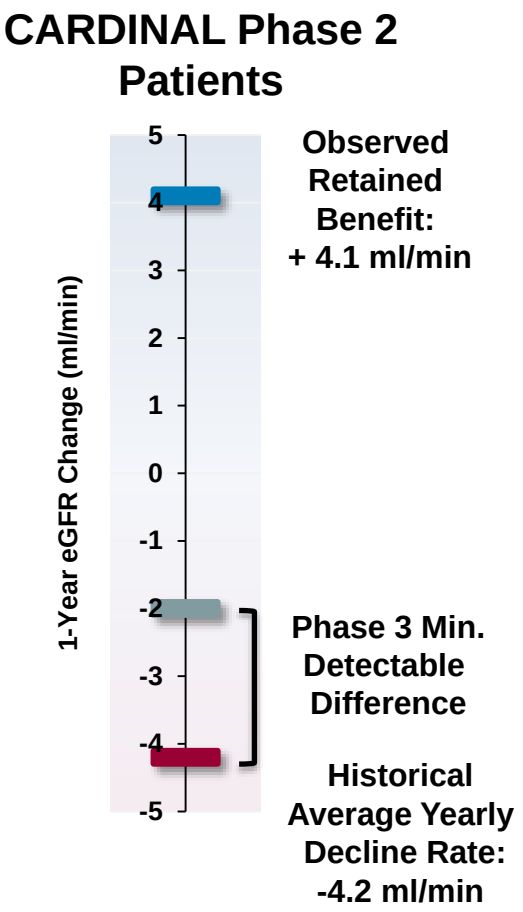


Week 48 ΔeGFR
+10.4 ml/min
n=25, p<0.0001

- Improvement after one year of treatment represents recovery of approximately two years of average loss
- Week 12 eGFR increase positively correlates with Week 48 eGFR increase (r=0.52; p=0.01)

Statistically Significant Retained eGFR Benefit Observed in Bard Treated Patients

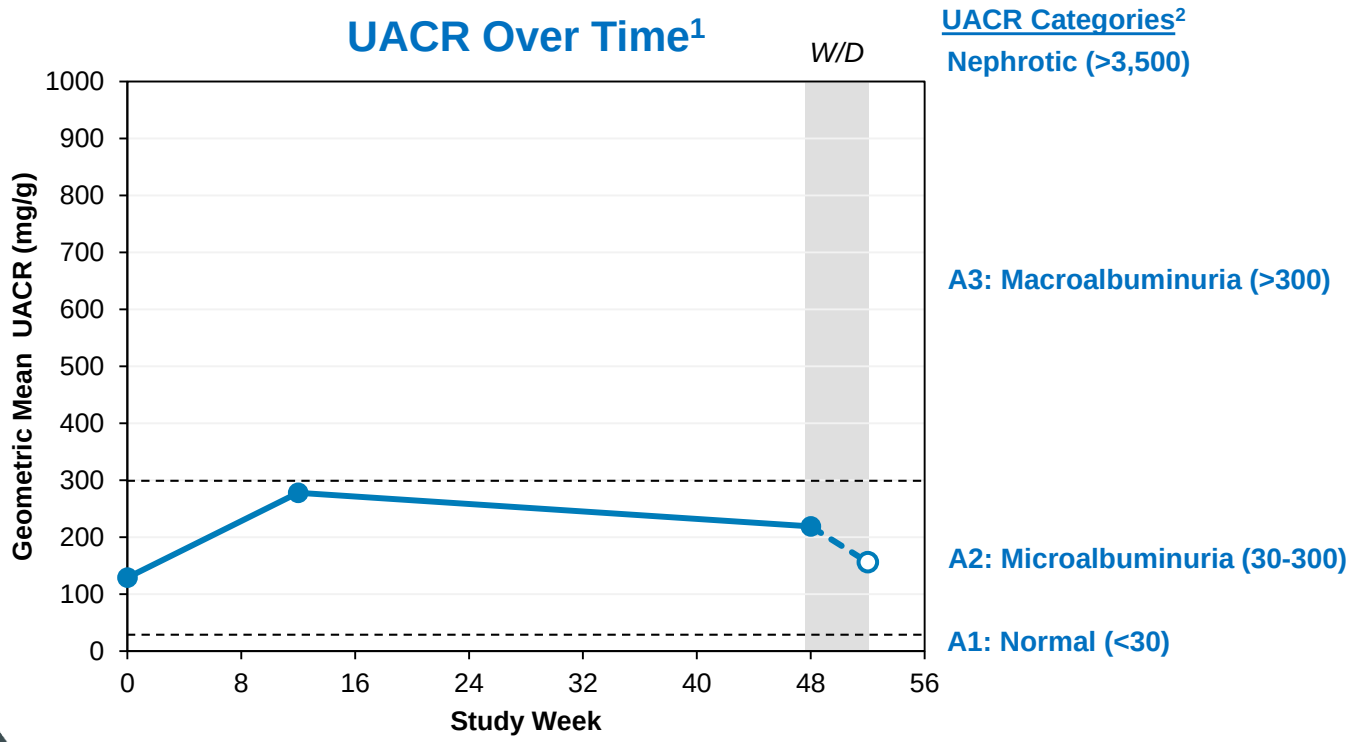
- Mean retained eGFR benefit at Week 52 of 4.1 ml/min ($p<0.05$)
- Bard treatment demonstrated a retained eGFR benefit after withdrawal of drug suggesting that:
  - Bard protected kidney function during treatment
  - Bard did not improve kidney function through a damaging mechanism
- Phase 3 modeling[1] assumes:
  - Phase 2 retained eGFR benefit of 4.1 ml/min will replicate in Phase 3 Bard patients
  - Phase 2 patients' observed mean historical eGFR loss of 4.2 ml/min will replicate in Phase 3 placebo patients
- Phase 3 conservatively powered to detect placebo-corrected retained eGFR benefit of 2.2 ml/min at 150 patients
- FDA approved tolvaptan in rare form of CKD (ADPKD) based on placebo-corrected retained eGFR of 1.27 ml/min



[1]Modeled changes are not intended as a forecast of probable results. No assurance is given about the results that will be obtained.

Urinary Protein Not Significantly Changed at Weeks 48 or 52

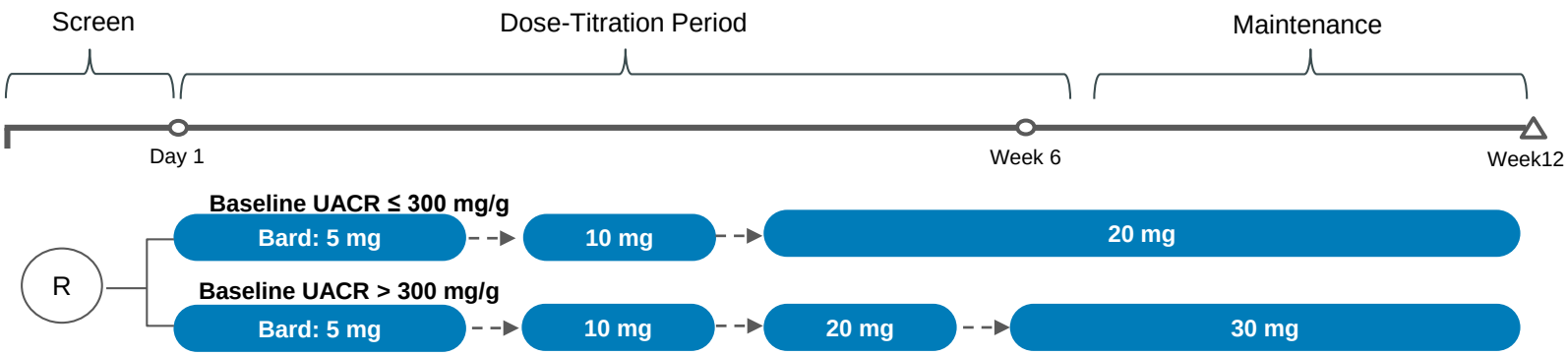
- Urinary protein (UACR) not significantly different from baseline at Weeks 48 or 52
  - Lack of increase in urinary protein when adjusted for eGFR suggests initial increase in UACR explained by increase in GFR
  - After initial eGFR-based increase, UACR trends down through Week 48
  - Mean changes clinically insignificant
- Injury due to hyperfiltration would cause UACR to increase over time



[1]*Uses log-transformation methodology adopted by NKF/FDA/EMA Scientific Working Group to compute changes in albuminuria;*
[2]*KDIGO (Kidney Disease Improving Global Outcomes) 2013 classification of albuminuria*

PHOENIX Trial Design

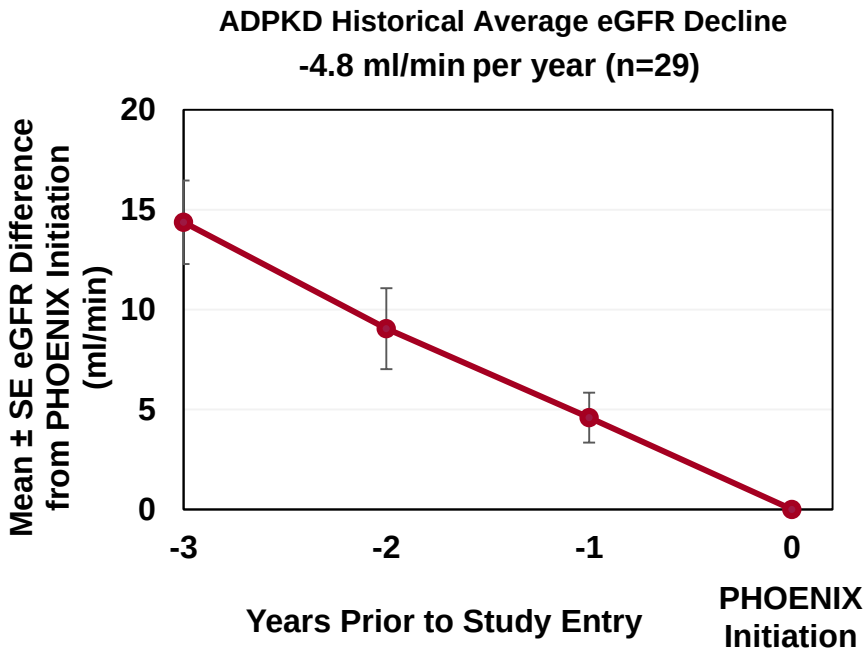
- Phase 2, open-label, multi-center, US-only trial
  - Four separate cohorts of patients with ADPKD, IgAN, T1D CKD, and FSGS
  - Targeting enrollment of 25 to 30 patients per cohort
- Primary endpoint is increase in eGFR from baseline at Week 12
- Enrolling large range of eGFR (30-90 ml/min) and age (18-65 years old)
- Reported full primary endpoint data for ADPKD cohort



Prior to Enrollment, ADPKD Patients had Progressive Loss of Kidney Function

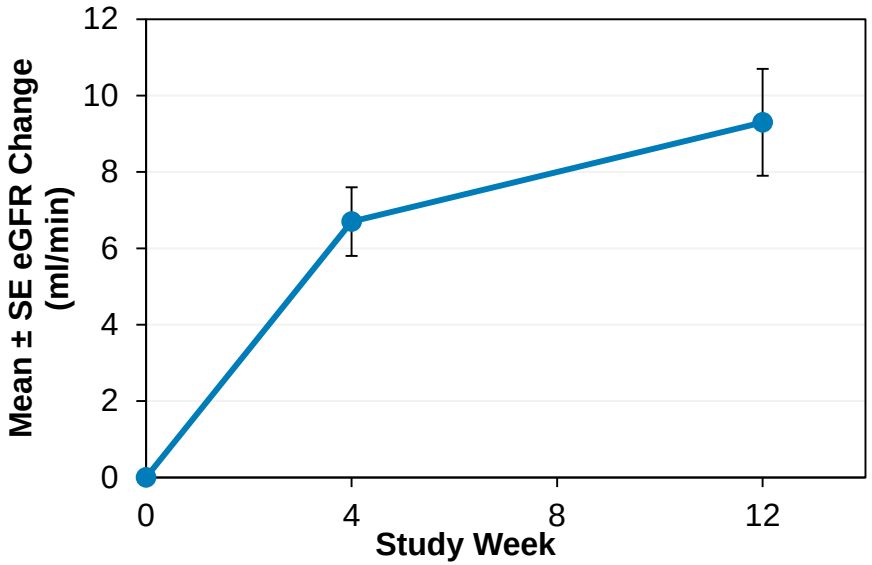
- PHOENIX ADPKD cohort enrolled 31 patients
- Historical eGFR data from 3 years prior to enrollment collected for 29 of 31 patients
- Average annual loss of eGFR of 4.8 ml/min prior to study entry

Characteristic	Total (N=31)
Age, years (mean ± SD)	47 ± 10
Baseline eGFR, ml/min (mean ± SD)	48 ± 14
Baseline UACR, mg/g (geometric mean)	44.4
Receiving ACEi or ARB (n,%)	25 (81%)



Statistically Significant Improvement in eGFR in ADPKD Patients

- Final data demonstrate statistically significant, time-dependent increase in eGFR of 9.3 ml/min
- Increase represents recovery of two prior years of average loss based on historical data



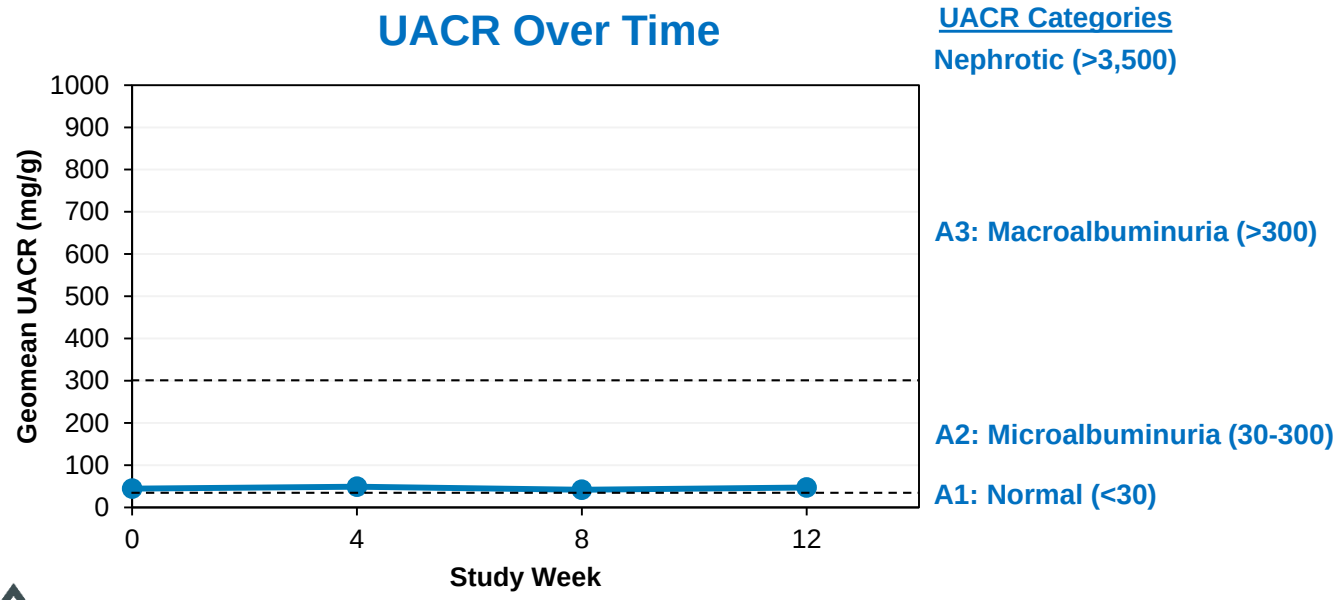
	BL eGFR	Change from Baseline in eGFR (n=31)	
		WK4	WK12
Mean ± SE	47.7 ± 2.4	6.7 ± 0.9	9.3 ± 1.4
p-value	-	p<0.0001	p<0.0001

REATA PHARMACEUTICALS

PHOENIX

Urinary Protein Unchanged in ADPKD Patients

- In ADPKD, the filtration barrier is not damaged and patients had normal to near-normal levels of urinary protein at baseline
  - The filtration barrier remains relatively impermeable to blood protein
  - Changes in GFR would not be expected to meaningfully affect urinary protein unless the filtration barrier was damaged
- No change in urinary protein despite the large increase in eGFR
- Suggests that Bard does not damage the filtration barrier and profile is inconsistent with hyperfiltration

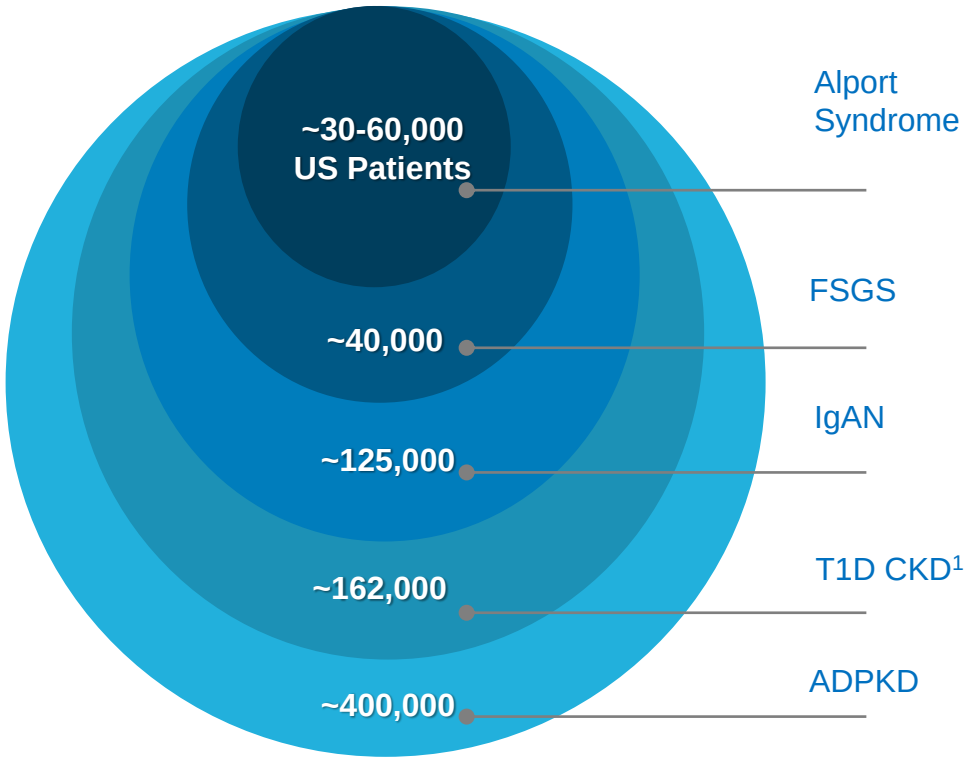


Large Body of Clinical Evidence That Bard Treatment May Delay Kidney Failure in CKD

- One-year CARDINAL data showed a positive on-treatment and retained eGFR benefit in AS patients treated with Bard for one year
  - Provides evidence that Bard treatment may delay or prevent kidney failure
  - Suggests eGFR improvement is not through a damaging mechanism such as hyperfiltration
  - Suggests CARDINAL Phase 3 is conservatively powered for key approval endpoint
- PHOENIX Phase 2 data showed improved eGFR in patients with ADPKD treated with Bard for 12 weeks
  - Adds to evidence that Bard's anti-inflammatory activity targets final common pathway of kidney function loss relevant to many forms of CKD
  - Data suggest that long-term eGFR improvements and retained benefit observed in other forms of CKD may translate to patients with ADPKD

Rare CKD: US Prevalence

US Patients
Rare Kidney Indications



A Therapeutic Area
Prime for Market Expansion

- Reata is studying bardoxolone methyl in 5 rare chronic kidney diseases impacting over 700,000 US patients
- Our first anticipated launch is Alport syndrome, our lead rare kidney program, with Phase 3 data expected in 2H 2019
- Potential consecutive launches in multiple rare kidney disease vastly expands the market opportunity of Bard

REATA PHARMACEUTICALS

[1]Represents stages 2-5 CKD

Overview of Friedreich's Ataxia

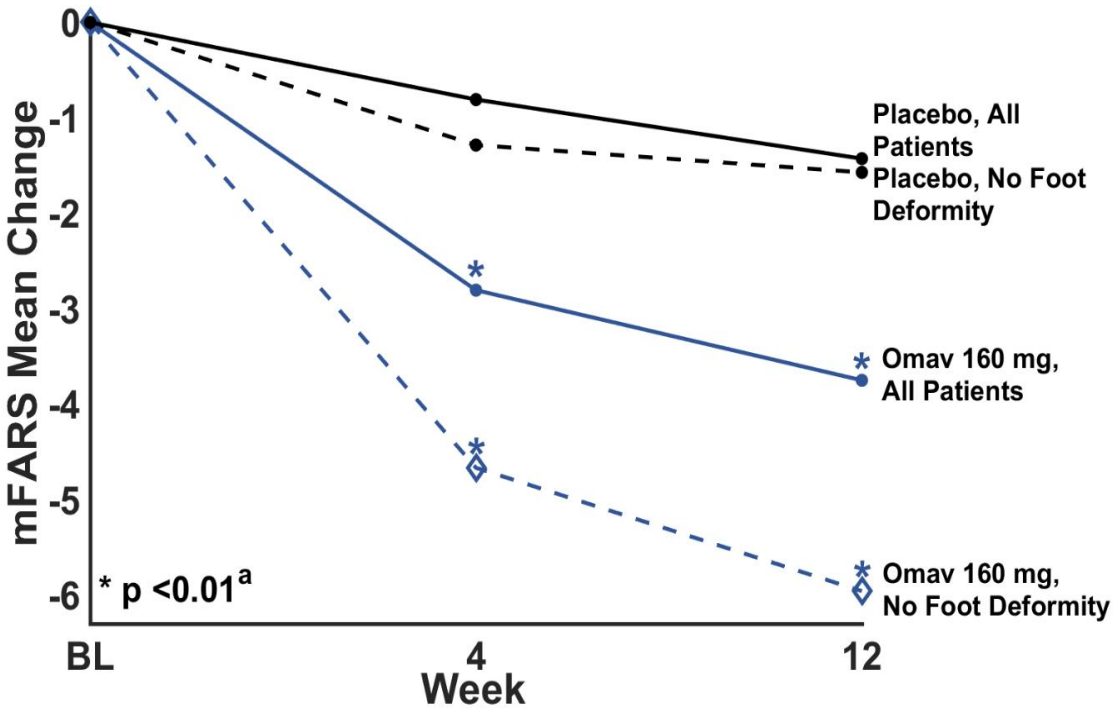
- FA Pathophysiology
  - Caused by mutations that result in hypo-expression of frataxin
  - Frataxin is responsible for biosynthesis of iron-sulfur complexes in the mitochondria used by OXPHOS complexes
  - FA is a multi-system disease that includes neurodegeneration, cardiomyopathy, diabetes, and fatigue
- Patient Statistics
  - On average, patients are diagnosed in their early teens, are wheelchair-bound in their 20s, and die in their 30s[1,2]
  - Cardiomyopathy is the most common cause of death
  - Prevalence is approximately 6,000 in the US and approximately 22,000 globally[1]
  - Patients track neurologic function using mFARS exam scores
  - mFARS score worsens 1 to 2 points per year on average
- Current Disease Management
  - No treatments are currently approved
  - Patients routinely take vitamin cocktails and antioxidants, which have shown no reproducible activity

REATA PHARMACEUTICALS

[1]Friedreich's Ataxia Research Alliance, [2]Parkinson 2010, Klockgether 1998, Santos 2010

MOXIE Part 1: Improved mFARS Observed Across All Omav Doses

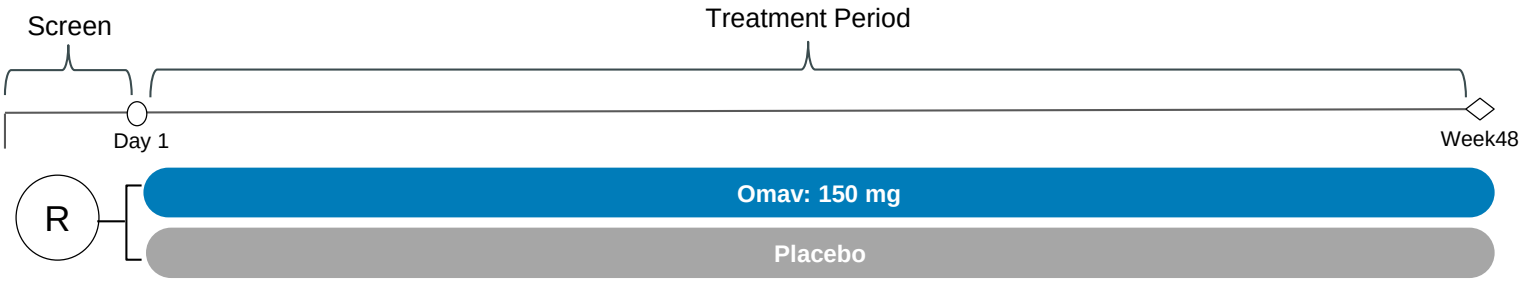
- Significantly improved mFARS from baseline across all Omav doses (p<0.0001)
- Placebo-corrected change at 160 mg (-2.3) neared statistical significance (p=0.06)
- Two-thirds of patients at 160 mg had *pes cavus*, a musculoskeletal foot deformity (MFD) that influences measurement of treatment response
- Placebo-corrected change in mFARS without MFD is -4.4 points (p=0.01) at 160 mg



P-values are change from baseline as compared to zero

MOXIe Part 2: Pivotal Study Design

- MOXIe Part 2 is a pivotal, international, double-blind, placebo-controlled trial targeting enrollment of 100 FA patients (80 patients without MFD)
- Primary endpoint is change in mFARS relative to placebo at Week 48
  - Study is powered to detect a placebo-corrected difference in mFARS of -1.2 ($p<0.05$) to -1.7 ($p<0.01$)
  - Compared to part 1, part 2 design includes larger sample size, longer duration, and limits and stratifies patients with MFD
- Pivotal data expected in 2H 2019



Friedreich's Ataxia: Target Product Profile and Commercial Opportunity

- Prevalence is approximately 6,000 in the US and approximately 22,000 globally[1]
- Significant unmet need with no approved therapy
  - Physically debilitating disease
  - Patients' overall health is currently managed through vitamins, supplements and diet
- Omav could be the first approved therapy for patients with FA, if MOXIe trial is successful:
  - Omav will have demonstrated improved functional capacity as assessed by mFARS scores, a known measure of FA disease progression
  - Omav may be used as first line treatment in patients with FA
- Convenient, oral, once-daily dosing with manageable side effect profile
- We believe orphan drug status and significant unmet need will impact pricing of Omav for FA

REATA PHARMACEUTICALS

[1]*Friedreich's Ataxia Research Alliance*

Overview of CTD-PAH

- CTD-PAH Pathophysiology
  - PAH that occurs in patients with connective tissue diseases such as scleroderma and lupus erythematosus
  - Impaired mitochondrial function, inflammation, fibrosis, and tissue remodeling implicated as prime drivers of PAH
- Patient Statistics
  - 10% to 15% of scleroderma and lupus patients have CTD-PAH[1]
  - CTD-PAH is the leading cause of death for scleroderma and lupus patients
  - 5-year survival of scleroderma patients with PAH is 44% versus 85% without PAH[2]
  - Estimated prevalence: 12,000 in U.S.; 50,000 worldwide; 30% of all PAH
- Disease Management
  - Vasodilators are the only current treatment options: PDE-5 inhibitors, ERAs, and prostacyclins
  - Vasodilators have lower benefit in CTD-PAH versus idiopathic PAH and produce side effects that include syncope, headache, flushing, and jaw pain
  - Poor risk-benefit for vasodilators in CTD-PAH given minimal treatment effect and adverse events resulting from systemic vasodilation

[1]Galie 2005, Hsu 2014, Pereze-Penate 2016; [2]Benza 2012

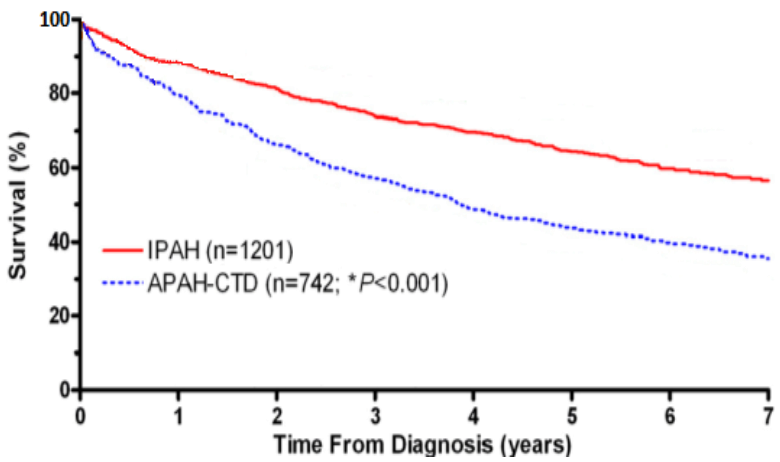
CTD-PAH is Distinct from Idiopathic PAH

- CTD-PAH is a more severe disease than I-PAH
  - Lower baseline 6MWD
  - Median survival in US is 4 years for CTD-PAH versus 7 years for I-PAH patients
- Compared to I-PAH, CTD-PAH is driven more by fibrosis than by impaired hemodynamics, reducing the impact of vasodilators
  - Vasodilators produce one-third the impact on 6MWD in CTD-PAH versus I-PAH
  - Explains lower survival rate in CTD-PAH versus I-PAH

Rhee Meta-Analysis Hemodynamics and 6MWD Change for CTD vs Idiopathic PAH Patients

	CTD-PAH	I-PAH
Baseline Characteristics		
RAP, mmHg	8	9.3
mPAP, mmHg	46	55
PVR, WU	10	13
6MWD (m)	351	365
Response to Vasodilator Therapy		
Δ6MWD (m)	9.6	30.1

Survival in CTD vs Idiopathic PAH Patients



REATA PHARMACEUTICALS

Rhee R, et al; Am J Respir Crit Care Med. (2015); Benza R, et al; Chest. (2012)

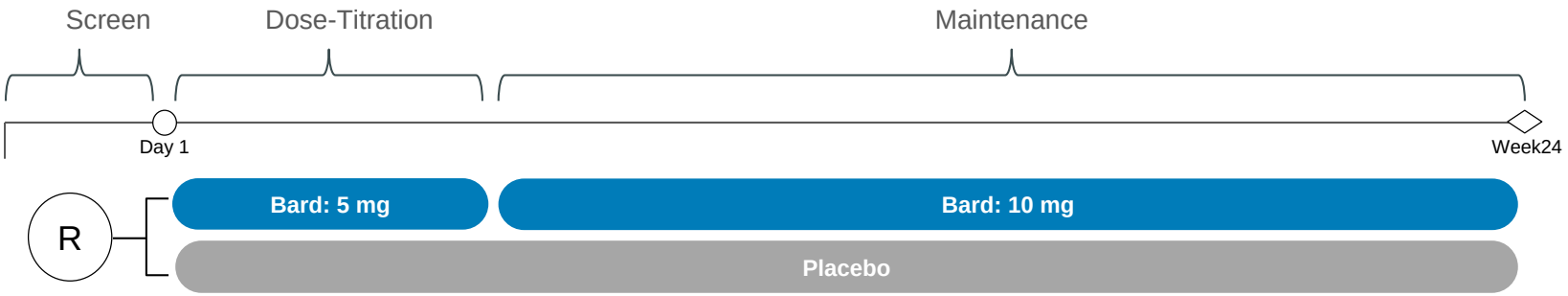
LARIAT: Phase 2 Trial of Bard Combined with Approved Therapies

- US-only, double-blind, randomized, placebo-controlled trial in I-PAH and CTD-PAH
  - PAH patients required to be on 1 to 2 background therapies
  - Assessed safety and change in 6MWD from baseline through 16 weeks
- Primary efficacy analysis of initial cohorts presented at CHEST 2015 showed a placebo-corrected 6MWD of 21 m (p=0.037) at doses of 2.5, 5, and 10 mg
- CTD-PAH patients demonstrated largest responses

Dataset	Treatment	N	Time-Averaged Δ6MWD (m)		Week 16 Δ6MWD (m)	
			Change from Baseline	Placebo-corrected	Change from Baseline	Placebo-corrected
All	Placebo	7	0.6 p=0.96	-	9.8 p=0.44	-
	BARD	15	26.7 p<0.001	26.1 P=0.06	38.2 p<0.001	28.4 p=0.07
Without Anemia	Placebo	5	-10.1 p=0.39	-	-5.8 p=0.68	-
	BARD	14	30.2 p<0.001	40.3 p=0.009	42.7 p<0.001	48.5 p=0.005

CATALYST: Phase 3 Study Design

- CATALYST is a Phase 3, international, double-blind, randomized, placebo-controlled trial targeting enrollment of 200 CTD-PAH patients
- Primary endpoint: 6MWD at Week 24
  - Powered to detect 12.5 m change at 24 weeks with $p<0.05$
  - LARIAT end-of-treatment analysis at Week 16 shows placebo-corrected change in 6MWD of 48.5 m ($p=0.005$)
- Pre-specified sample size recalculation completed
  - Allows power to be maintained if observed variability is higher than modeled
  - Blinded, pooled analysis with no statistical penalty and no predictive value to outcome
  - Set final sample size at 200 subjects
- Pivotal data expected 1H 2020



REATA PHARMACEUTICALS

CATALYST for patients with CTD-PAH

CTD-PAH: Target Product Profile and Commercial Opportunity

- Prevalence is approximately 12,000 patients in the US
- Significant unmet need with limited or no current approved therapies indicated specifically for CTD-PAH
  - CTD-PAH is a more severe form of PAH with high morbidity and mortality
  - Current therapies are vasodilators with limited efficacy in this patient population
- Kidney decline is prevalent among patients with PAH and Bard could be the first therapy to demonstrate an ability to reverse eGFR decline and preserve kidney function
- Convenient, oral, once daily dosing to be used in combination with standard of care
- Orphan drug status and a disease modifying treatment will influence pricing of Bard for CTD-PAH

Territory	Commercial Rights	Royalty
United States	Reata	
Pan-Asia	KHK	Low teens to low 20% royalties to Reata
Rest of World	AbbVie (Option)	15% to high 20% royalties to Reata

Key Upcoming Milestones

CARDINAL trial in Alport syndrome

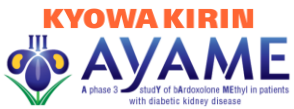
- One year, pivotal Phase 3 data in 2H19

PHOENIX trial in rare forms of CKD

- 12-week data from IgAN and T1D CKD cohorts in 3Q18

Phase 3 trial in ADPKD

- Developing plans to advance the program into a pivotal, Phase 3 trial



Phase 3 trial in diabetic CKD

- Phase 3 AYAME trial underway, data in 1H22

MOXIe trial in Friedreich's ataxia

- Pivotal Phase 2, Part 2 data in 2H19

Bardoxolone in CTD-PAH

- Phase 3 CATALYST pivotal data in 1H20