REATA PHARMACEUTICALS

MANAGEMENT CALL TO DISCUSS PHASE 2 PHOENIX RESULTS AND CKD PROGRAM UPDATES

February 20, 2019

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.

Bard Activates Nrf2, a Central Player in Many Forms of Progressive CKD

Through Nrf2, Bard[1] promotes the resolution of inflammation: restoring mitochondrial function, reducing oxidative stress, and inhibiting NF-κB and inflammatory signaling

- Our Nrf2 activators are the subject of over 400 peer-reviewed publications and have been studied in over 50 preclinical animal models
- They have demonstrated anti-inflammatory, tissue protective, or anti-fibrotic effects in the kidney, heart, brain, liver, lungs, vasculature, fat tissue, pancreas, bone marrow, intestines, eyes, spinal cord, prostate, inner ear, and skin

Inflammatory processes initiated by a variety of pathogenic stimuli, including genetic mutations, diabetes, hypertension, and IgA deposition, drive loss of kidney function

A recent human genomic study identified molecular pathways associated with GFR[2] decline in patients with nine different forms of CKD[3]. The identified pathways clustered into networks that were either metabolism- or inflammation-related with Nrf2 serving as the hub between the networks[4]

In preclinical studies, Bard and our other Nrf2 activators have been shown to improve kidney function, reduce inflammation, and prevent injury, remodeling, and fibrosis in a number of animal models of kidney disease

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[1]Bardoxolone methyl; [2]Glomerular filtration rate; [3]Chronic kidney disease; [4]Martini et al. 2014

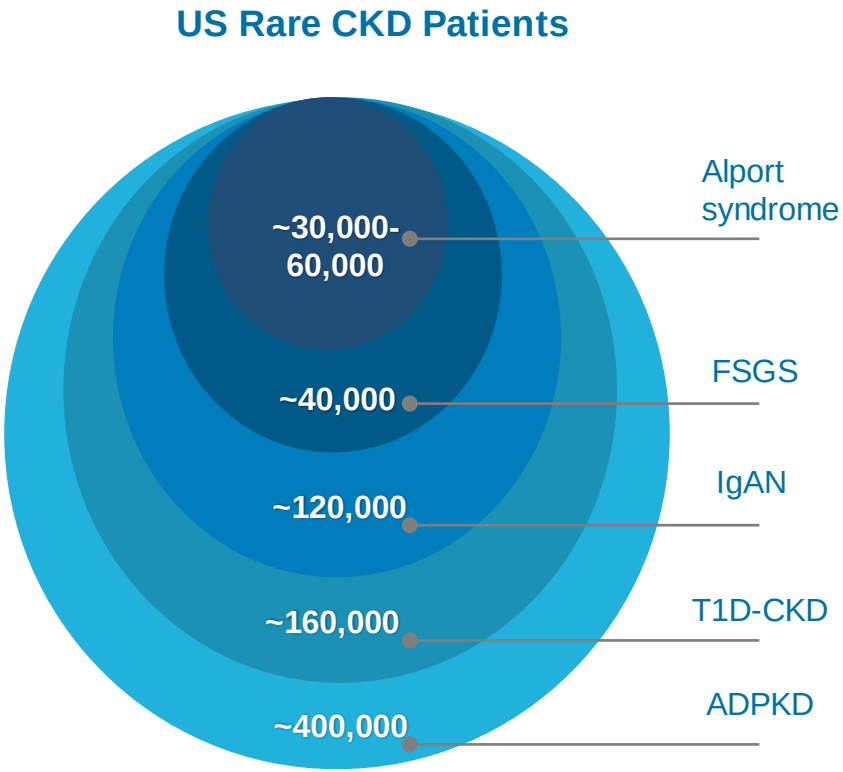
Bard is Currently in Development for Five Rare Forms of CKD

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

CARDINAL Phase 3 study in Alport syndrome
- Fully enrolled at 157 patients
- Data expected 2H19

FALCON Phase 3 study in ADPKD[1]
- Study design and endpoints similar to CARDINAL Phase 3 study
- Planned to start mid-2019

PHOENIX Phase 2 study in rare forms of CKD
- Positive data reported for ADPKD, IgAN[2], and T1D-CKD[3]
- Positive FSGS[4] data reported today



[1]Autosomal dominant polycystic kidney disease; [2]IgA nephropathy; [3]Type 1 diabetic CKD; [4]Focal segmental glomerulosclerosis

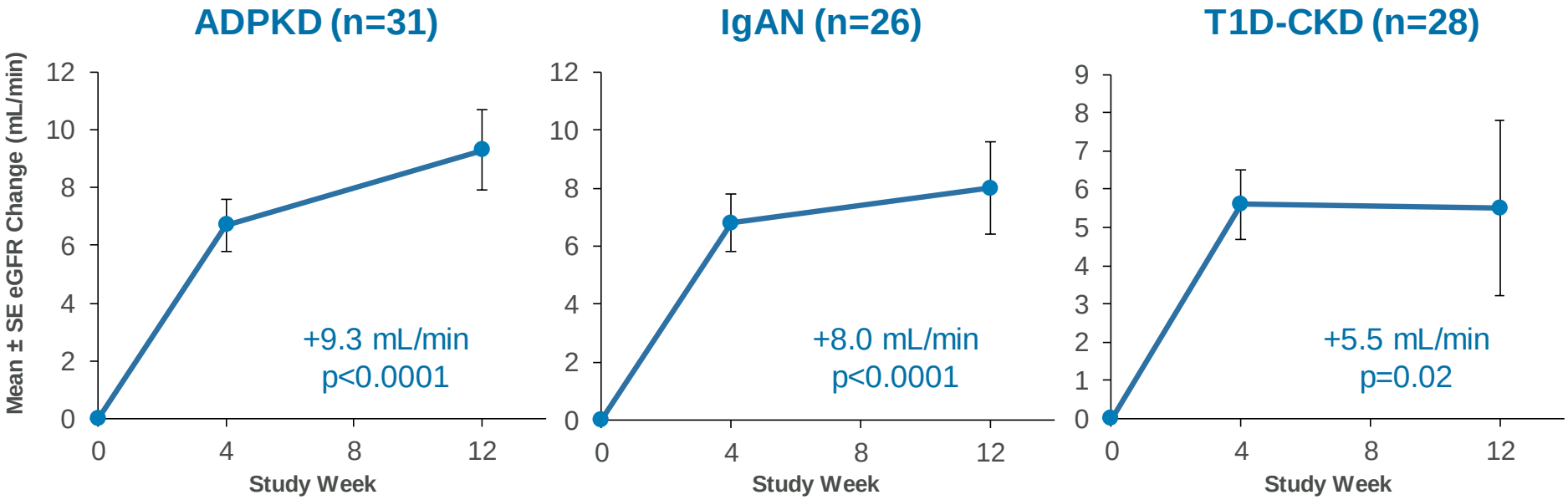
PHOENIX: Trial Design and Results to Date

Phase 2, open-label, multi-center, US-only trial

- Primary endpoint was change from baseline in eGFR at Week 12
- Enrolled large range of eGFR (30-90 mL/min[1]) and age (18-65 years old)

Patients had progressively declining eGFR[2] at study entry despite optimized ACEi/ARB therapy

In each cohort, Bard reversed multiple years of annual kidney function loss



[1]*Units of mL/min/1.73 m² are represented as mL/min throughout this presentation;* [2]*Estimated GFR*

FSGS Overview

Etiology of primary FSGS is either unknown or caused by mutations that result in podocyte injury and subsequent inflammation, fibrosis, and progressive loss of kidney function

One of the most common forms of chronic glomerular disease with approximately 40,000 patients in US

Clinical course of disease

- Patients typically present at age 20 to 30 with hypertension, proteinuria, low blood albumin, and loss of kidney function
- Annual average decline of -2.4 mL/min[1]
- Increasingly common cause of ESKD[2]

No approved therapies for FSGS

- 80% of patients take antihypertensive agents (ACEi/ARB)[1]
- Corticosteroids or immunosuppressants prescribed in certain cases

[1]Sim et al. 2018; [2]End-stage kidney disease

PHOENIX FSGS: Baseline Characteristics and Historical eGFR Decline Data

PHOENIX FSGS cohort enrolled 18 patients with primary FSGS

Average annual loss of eGFR of -2.6 mL/min prior to study entry (n=17)

Patients had lost roughly one-half of their kidney function and were actively progressing upon study entry despite optimized ACEi/ARB therapy and low UACR[1]

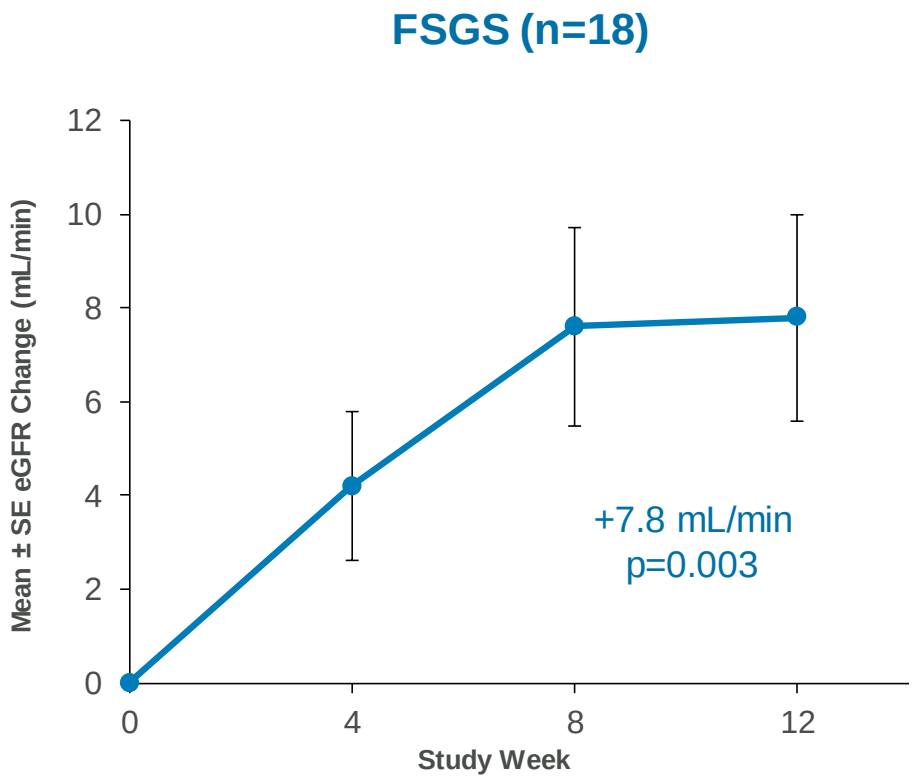
Characteristic	Total (n=18)
Age, years (mean ± SD)	49 ± 13
Baseline eGFR, mL/min (mean ± SD)	52 ± 18
Baseline UACR, mg/g (geometric mean)	184
Receiving ACEi or ARB (n,%)	17 (94%)
Average yearly historical eGFR loss (mL/min, n=17)	-2.6

[1]*Urine albumin to creatinine ratio*

PHOENIX FSGS: Bard Significantly Improved eGFR

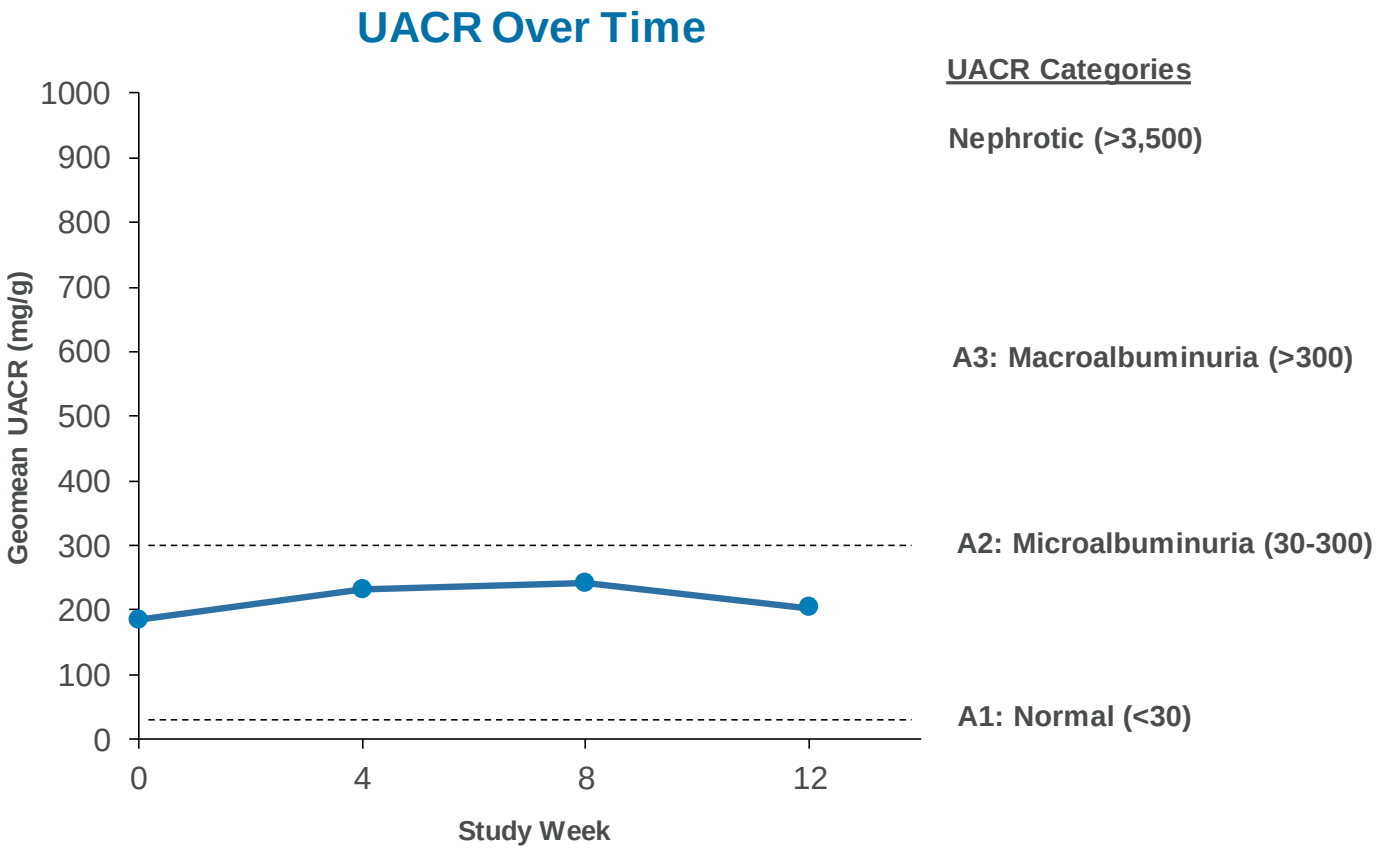
Bard significantly increased eGFR by 7.8 mL/min at Week 12

Increase represents recovery of three prior years of loss based on historical data



PHOENIX FSGS: Urinary Albumin Unchanged and Consistent with Other PHOENIX Cohorts

Bard treatment resulted in no change in UACR



All PHOENIX Cohorts: Baseline Characteristics and Historical eGFR Decline Data

PHOENIX enrolled 103 patients who had lost roughly one-half of their kidney function upon study entry

Patients had progressive loss of eGFR despite optimized ACEi/ARB therapy and low UACR

Broad inclusion/exclusion criteria enabled enrollment of cohorts that were representative of the general patient population receiving standard of care

Characteristic	All (n=103)	ADPKD (n=31)	IgAN (n=26)	T1D-CKD (n=28)	FSGS (n=18)
Age, years (mean ± SD)	48 ± 10	47 ± 9	49 ± 10	49 ± 10	49 ± 13
Baseline eGFR, mL/min (mean ± SE)	53 ± 2	48 ± 2	46 ± 2	68 ± 3	52 ± 4
Baseline UACR, mg/g (geometric mean)	63.9	44.4	104.0	30.9	184.3
Receiving ACEi or ARB (n,%)	85 (83%)	24 (77%)	25 (96%)	19 (68%)	17 (94%)
Average yearly historical eGFR loss (mL/min)	-2.8	-4.5	-1.2	-1.9	-2.6

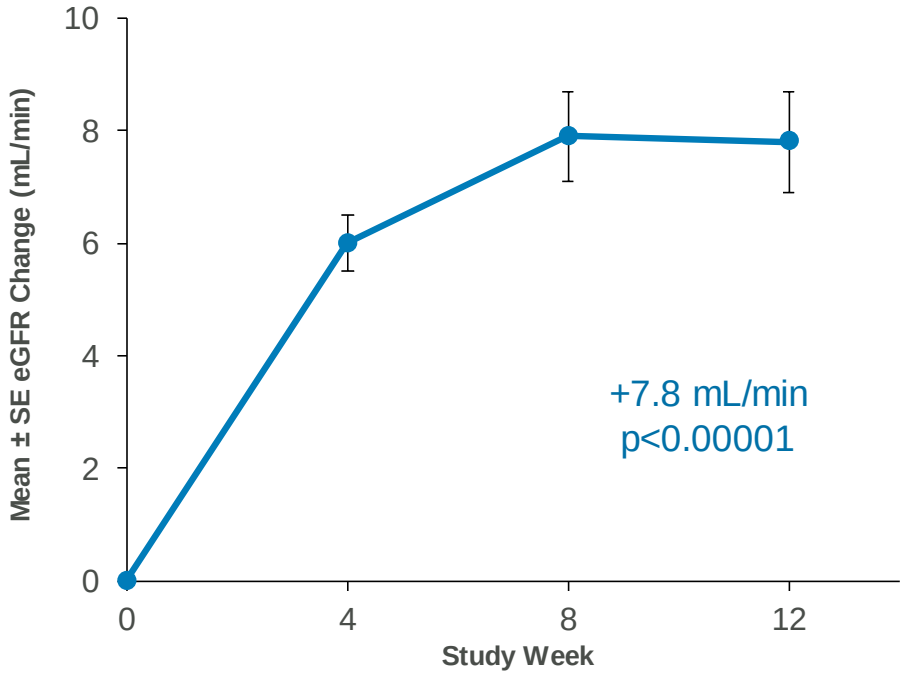
All PHOENIX Cohorts: Bard Significantly Improved eGFR

Bard significantly increased eGFR by 7.8 mL/min at Week 12

High consistency of response – eGFR increased in 88% of patients at Week 12

Improvements across multiple forms of CKD suggest that Bard is addressing a common, final pathway of progression in CKD

All PHOENIX Cohorts (n=103)



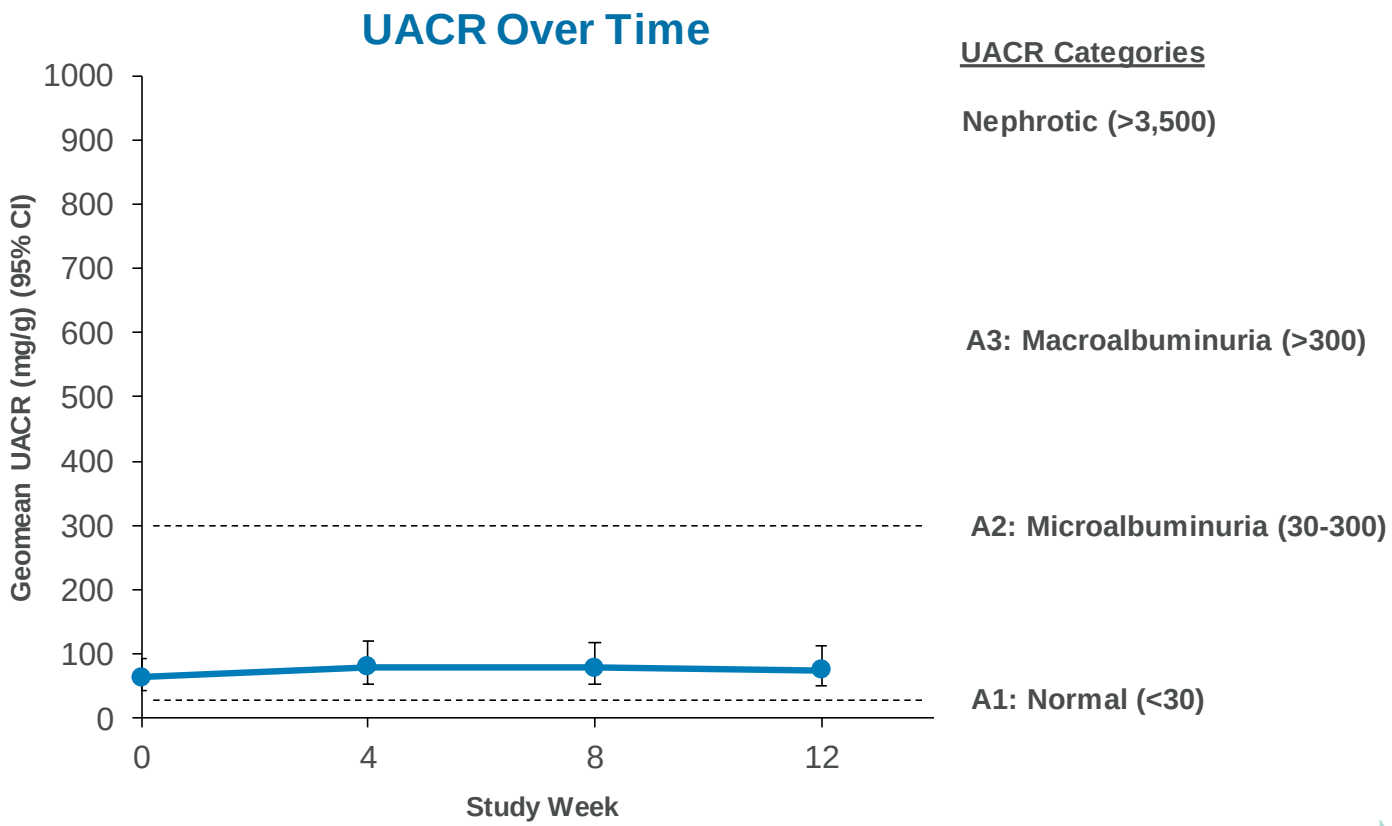
Cohort	Week 12 Response Rate[1]
ADPKD	96%
IgAN	91%
T1D-CKD	75%
FSGS	88%
All	88%

[1]*Defined as proportion of patients reaching Week 12 with eGFR values above baseline at Week 12*

All PHOENIX Cohorts: Urinary Albumin Unchanged

PHOENIX generally enrolled patients without long-standing hereditary or acquired defects to the kidney's filtration barrier

UACR low upon study entry and did not change with treatment (p=0.6)



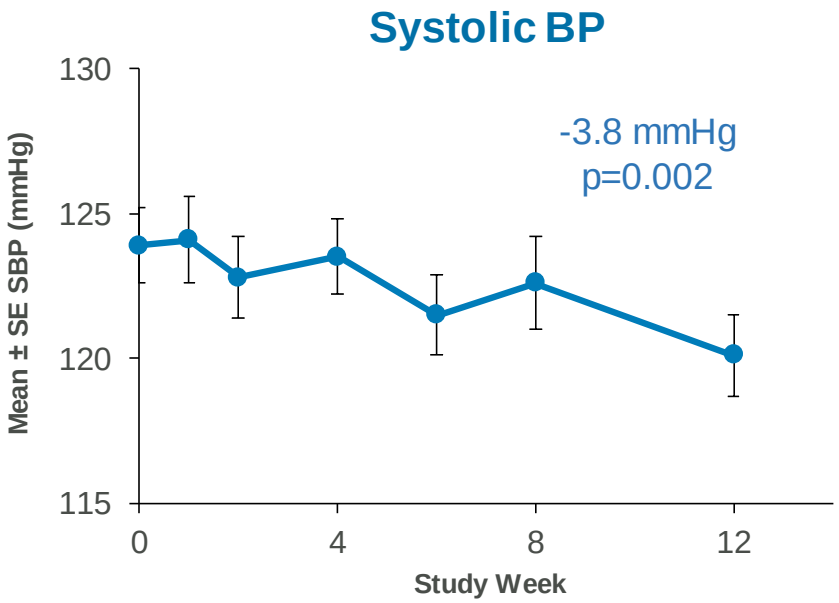
All PHOENIX Cohorts: Significant Reduction in Blood Pressure

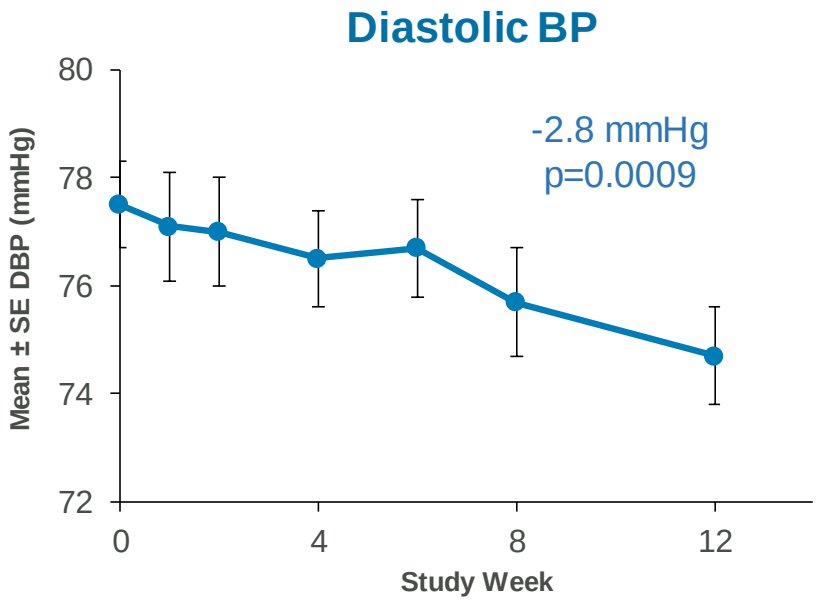
Bard significantly reduced systolic and diastolic blood pressure at Week 12 with no changes in diuretic usage

Demonstrates that the risk mitigation strategy for fluid retention has been effective

Profile inconsistent with mechanism of pressure-mediated effects

- Reduced blood pressure
- No change in UACR
- No correlation between changes in blood pressure and eGFR





All PHOENIX Cohorts: Summary of Tolerability and Adverse Events

92/103 (89%) completed treatment through Week 12

Bard generally well-tolerated with only 7 (6.8%) patients permanently discontinuing due to AEs[1]

- AE profile consistent with prior trials
- Most common AE reported was muscle spasms
- No Bard or fluid-related SAEs[2]

Summary of Adverse Events*

Preferred Term	Total (n=103)
Muscle spasms	44 (43%)
Headache	14 (14%)
Fatigue	12 (12%)

*AEs reported in >10% patients

[1]Adverse events; [2]Serious adverse events

PHOENIX Summary

PHOENIX enrolled 103 patients who had lost roughly one-half of their kidney function upon study entry and had clear evidence of progressive CKD despite receiving SOC[1]

Improvements were observed in large majority of patients across all four forms of CKD

Data support the hypothesis that Bard activation of Nrf2 affects a final common pathway of progression in CKD

Bard generally well-tolerated in PHOENIX

- Low discontinuation rate due to AEs
- No Bard-related SAEs

Fluid retention and evidence of pressure-mediated effects not observed in PHOENIX

- Significant reduction in blood pressure
- Proteinuria unchanged from baseline
- Risk mitigation strategy for fluid retention has been effective

Provides proof-of-concept in all four diseases; planning to pursue development in all four diseases

[1]*Standard of care*

Next Steps

Pivotal Phase 3 CARDINAL study in Alport syndrome fully enrolled with 157 patients

- If approved, Bard would be the first therapy approved for Alport syndrome
- One-year data required for accelerated approval expected 2H19

NDA preparations are underway

- Large safety database with over 2,000 people having been exposed to Bard
- FDA indicated that Reata has conducted all preclinical toxicology studies and clinical pharmacology studies required for NDA submission for Alport syndrome

Commercial preparations are underway

- Supply chain readiness on track for planned NDA[1] and product launch
- Marketing, operations, and sales commercial leadership team is onboard
- Disease awareness campaigns have launched to educate physicians about Alport syndrome

Expansion opportunities in process

- Launch of pivotal study in ADPKD (FALCON) in mid-2019
- Pivotal study in diabetic kidney disease (AYAME) ongoing in Japan and being conducted by KHK, Reata's licensee

[1]New drug application

Key Upcoming Milestones for Pivotal CKD Programs

Bard in Alport syndrome

Pivotal Phase 3 fully enrolled with data available in 2H19

Bard in ADPKD

Initiating a pivotal Phase 3 trial in ADPKD in mid-2019

Partner Program: Bard in diabetic kidney disease

Phase 3 AYAME trial underway, data available in 1H22

Sponsored by KHK, Reata's licensee in Asia

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Q&A