REATA PHARMACEUTICALS

# MANAGEMENT CALL TO DISCUSS PHASE 2 PHOENIX UPDATES

**September 25, 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.

# Bardoxolone is Currently in Development for Five Rare Forms of CKD

CARDINAL study in Alport syndrome

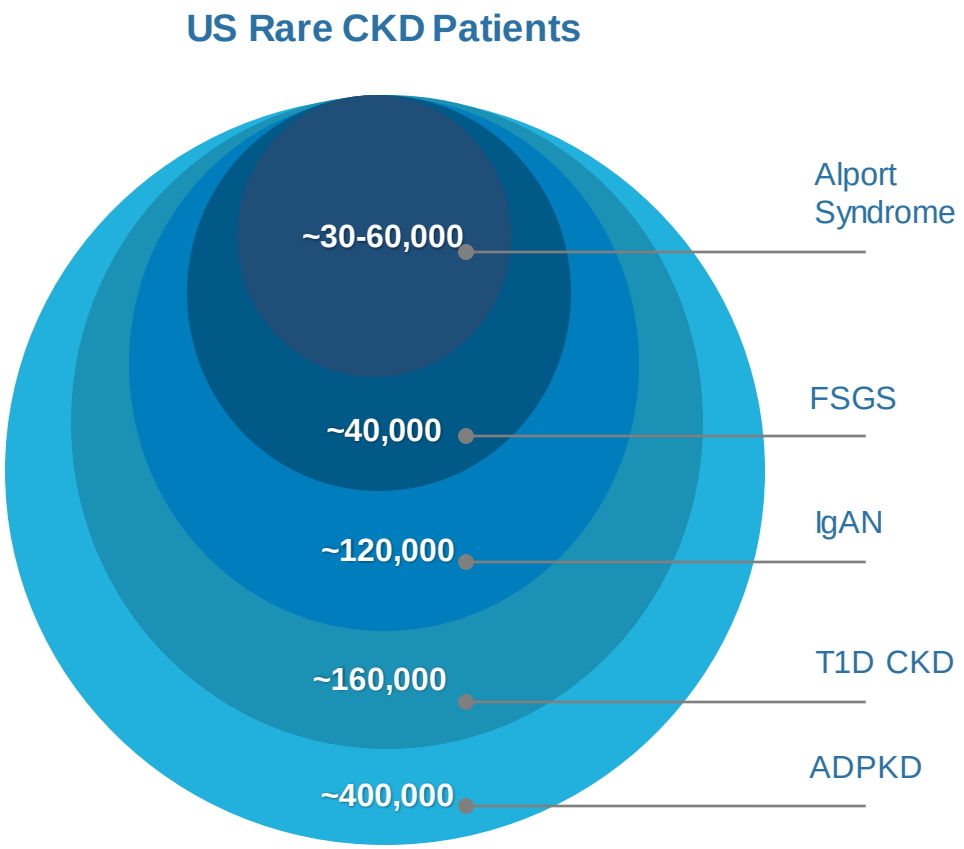
- Phase 2 portion demonstrated significant retained benefit at one year
- Pivotal Phase 3 portion is ongoing with data expected in 2H19

PHOENIX study designed to determine efficacy of bardoxolone (Bard) in other rare forms of CKD:

- ADPKD: full 12-week data reported in July 2018
- IgAN and T1D-CKD: full 12-week data being reported today
- FSGS: full 12-week data expected 1H19

Significant market opportunity in rare forms of CKD

AYAME study being conducted by KHK in T1D/T2D-CKD with data expected 1H22



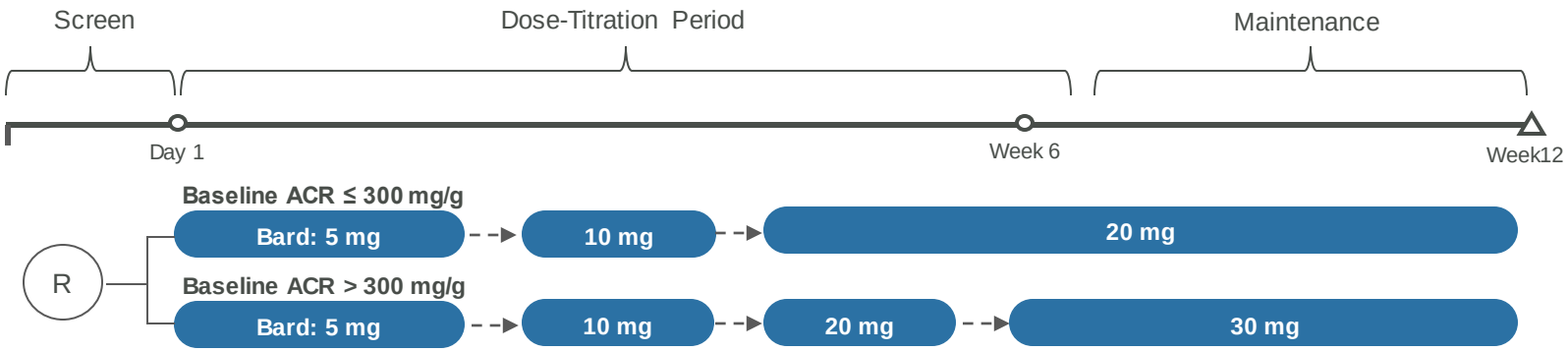
# PHOENIX Trial Design

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[1]) and age (18-65 years old)

Today reporting full primary endpoint data for IgAN and T1D CKD cohorts




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

# IgA Nephropathy Overview

Most prevalent primary chronic glomerular disease worldwide

Annual incidence of 1 case per 100,000 persons and affects roughly 120,000 patients in US

Deposition of immunoglobulin complexes in the glomerulus causes inflammation, fibrosis and progressive loss of kidney function

Clinical course of disease

- Patients typically present at age 20 to 30 with hematuria
- Annual average decline of 1.7 mL/min
- ESRD develops in 20 to 40% of patients within 20 years

No approved therapies for IgAN and typical treatment includes[1]:

- Antihypertensive agents (ACEi/ARB)
- Corticosteroids (6 months) for patients with persistent proteinuria (≥ 1 g/d) despite optimized RAAS blockade and GFR > 50 mL/min

[1]*2012 KDIGO Clinical Practice Guideline for Glomerulonephritis*

# PHOENIX IgAN: Baseline Characteristics and Historical eGFR Decline Data

PHOENIX IgAN cohort enrolled 26 patients

Average eGFR upon study entry 46 mL/min despite 96% of patients receiving standard of care ACEi/ARB

Historical eGFR data from 3 years prior to enrollment collected for 23/26 patients

Average annual loss of eGFR of 1.2 mL/min prior to study entry

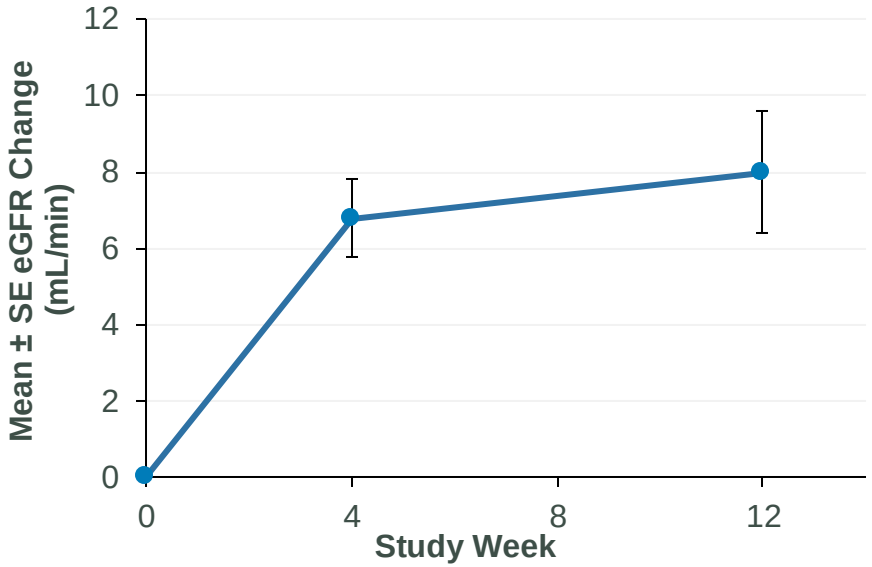
Patients had lost ~ one-half of their kidney function prior to study entry despite low proteinuria

| Characteristic | Total (N=26) |
|---|---|
| Age, years (mean ± SD) | 49 ± 10 |
| Baseline eGFR, mL/min (mean ± SD) | 46 ± 13 |
| Baseline ACR, mg/g (geometric mean) | 104.0 |
| Receiving ACEi or ARB (n,%) | 25 (96%) |
| Average yearly historical eGFR decline (mL/min, n=23) | 1.2 |

# PHOENIX IgAN: Bard Significantly Improved eGFR

Bard produced statistically significant increase in eGFR of 8.0 mL/min at Week 12

Increase represents recovery of ~ six prior years of loss based on historical data

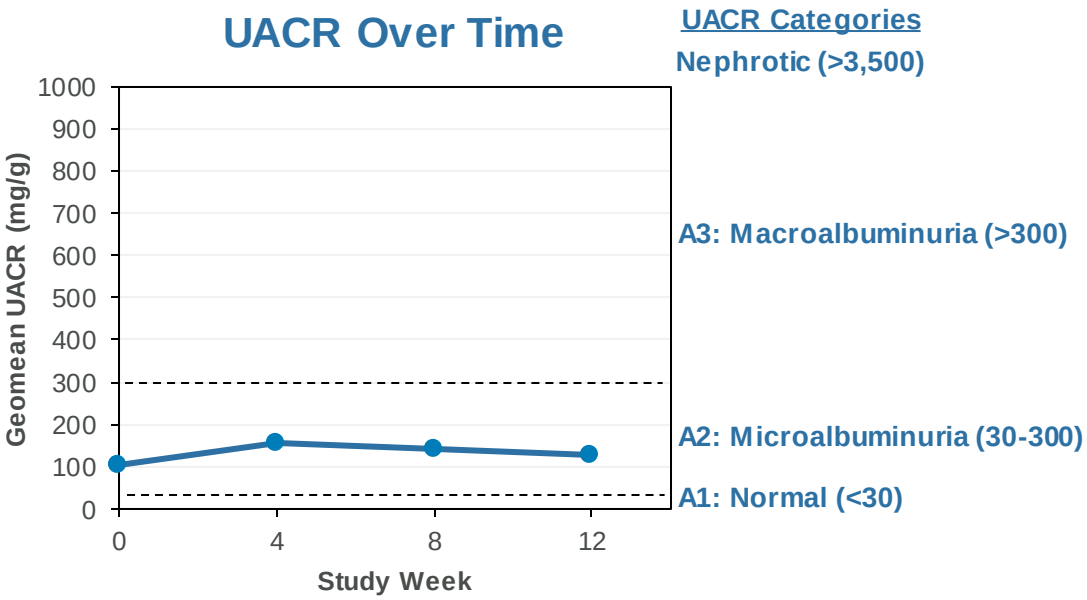



| | BL eGFR | Change from Baseline in eGFR (n=26) | |
|---|---|---|---|
| | | WK4 | WK12 |
| Mean ± SE | 46.2 ± 2.5 | 6.8 ± 1.0 | 8.0 ± 1.6 |
| p-value | - | p<0.0001 | p<0.0001 |

# PHOENIX IgAN: Urinary Protein Unchanged

Patients optimized on available therapies upon study entry as evidenced by low urinary protein within international treatment guideline goals (<1,000 mg/g)[1]

Bard treatment resulted in no significant change in urinary albumin excretion




[1]2012 KDIGO Clinical Practice Guideline for Glomerulonephritis

# Overview of CKD Associated with Type 1 Diabetes

Type 1 diabetes (T1D) affects an estimated 1.25 million patients in the US
- CKD affects approximately 10-20% of patients with T1D or roughly 160,000 patients in the US
- Average annual decline of 1.4 mL/min

In patients with T1D CKD, hyperglycemia due to poor glycemic control in some initiates pathological pathways that lead to fibrosis and loss of kidney function

No approved therapies for improving kidney function in T1D patients

Various therapeutic approaches used to attempt to slow disease progression
- RAAS blockade
- Glycemic control

# PHOENIX T1D: Baseline Characteristics and Historical eGFR Decline Data

PHOENIX T1D CKD cohort enrolled 28 patients

Historical eGFR data from 3 years prior to enrollment collected for 22/28 patients

Average annual loss of eGFR of 1.9 mL/min prior to study entry

Patients had lost ~ one-third of their kidney function prior to study entry

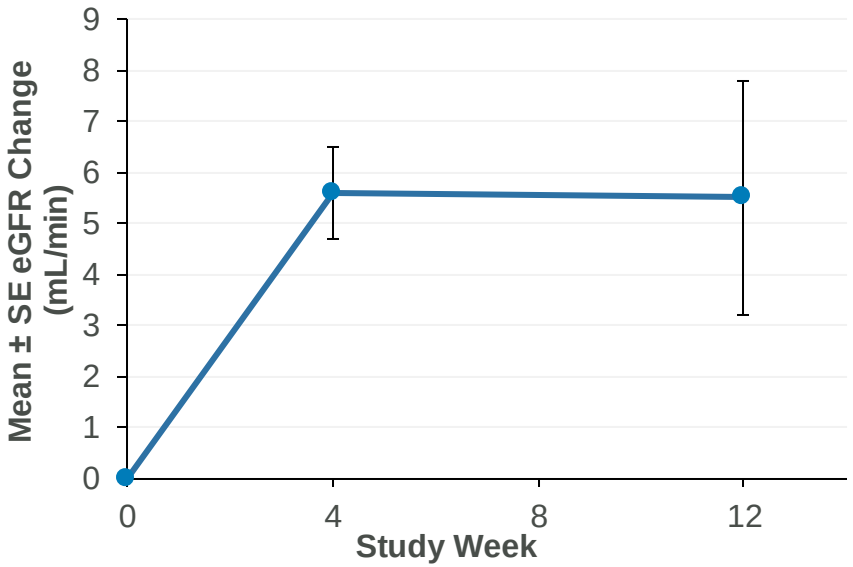
| Characteristic | Total (N=28) |
|---|---|
| Age, years (mean ± SD) | 49 ± 10 |
| Baseline eGFR, mL/min (mean ± SD) | 68 ± 17 |
| Baseline ACR, mg/g (geometric mean) | 30.9 |
| Receiving ACEi or ARB (n,%) | 19 (68%) |
| Average yearly historical eGFR decline (mL/min n=21) | 1.9 |

# PHOENIX T1D: Bard Significantly Improved eGFR in T1D Patients

Bard produced statistically significant increase in eGFR of 5.5 mL/min at Week 12

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

Lower treatment effect and higher variability in subset of patients with near-normal eGFR at baseline (≥80 mL/min; n=8/28)

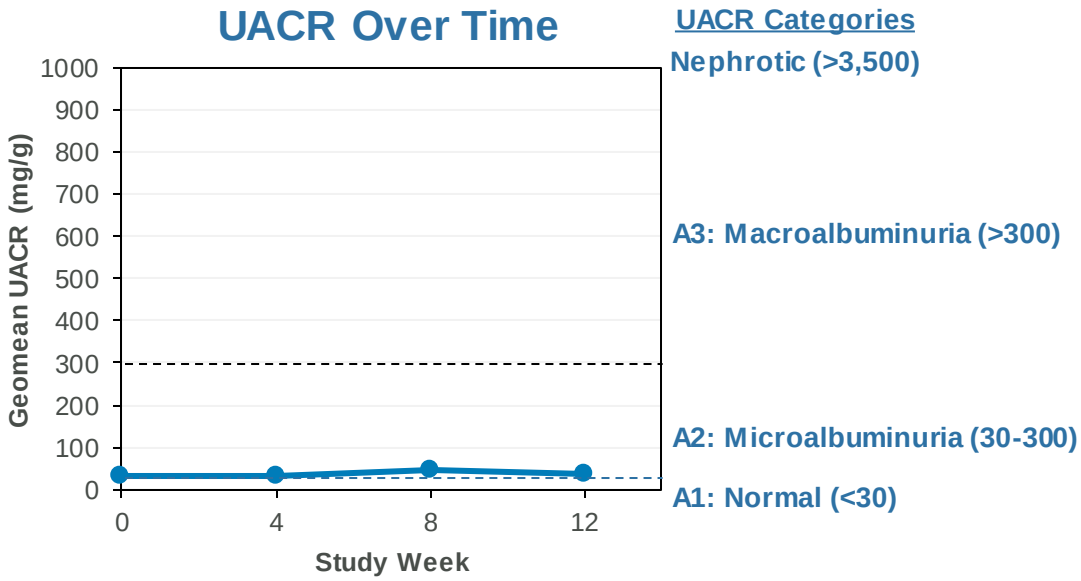



| | BL eGFR | Change from Baseline in eGFR (n=28) | |
|---|---|---|---|
| | | WK4 | WK12 |
| Mean ± SE | 67.5 ± 3.2 | 5.6 ± 0.9 | 5.5 ± 2.3 |
| p-value | - | p<0.001 | p=0.02 |

# PHOENIX T1D: Urinary Protein Unchanged in T1D Patients

Patients had normal to near-normal levels of urinary protein at baseline

Bard treatment did not change urinary albumin excretion despite the increase in eGFR



# Patient Disposition and Summary of Safety

Bard was well-tolerated

- 23/26 (88%) IgAN patients completed treatment
- 24/28 (86%) T1D patients completed treatment

No treatment-related serious adverse events in IgAN or T1D patients

AE profile similar to other CKD populations that have been studied

- AEs were generally mild to moderate in intensity
- Most commonly reported AE is muscle spasms (35% IgAN and 32% T1D)
- Associated with reductions in CK

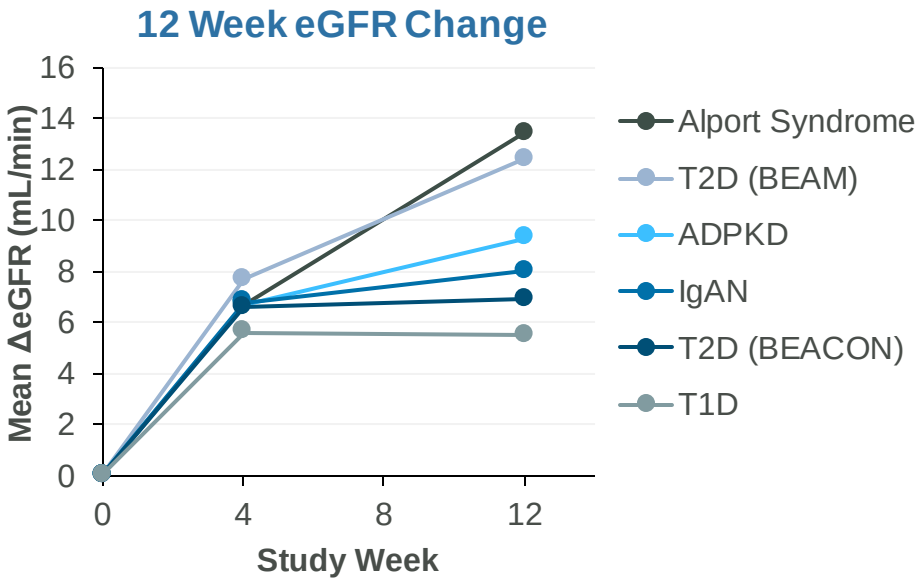
No changes in blood pressure or fluid-overload events

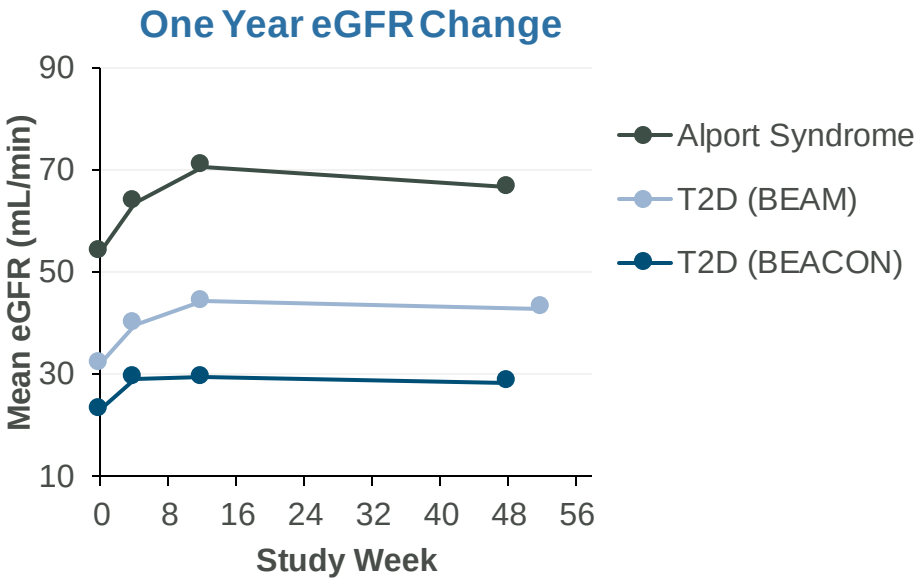
# Bard Improved eGFR Across Multiple Types of CKD

Increases in eGFR observed in six distinct patient populations

- Long-term eGFR increases of one to two years observed in three patient populations
- eGFR improvement post-withdrawal observed in two patient populations
- Acute changes in eGFR correlate with durable response and associated with retained eGFR benefit

Long-term eGFR improvements and retained eGFR benefit observed in other forms of CKD may translate to patients with ADPKD, IgAN and T1D



# Bard in CKD: Recent Highlights and Key Upcoming Milestones

**CARDINAL trial in Alport syndrome**

- Released one year Phase 2 data
- Phase 3 portion of CARDINAL underway
- Next milestone: One year, pivotal Phase 3 data in 2H19

**PHOENIX trial in rare forms of CKD**

- Released complete ADPKD, IgAN, and T1D CKD data
- Next milestone: FSGS data in 1H19

**Future development in ADPKD, IgAN, and T1D CKD**

- Developing plans to initiate pivotal, Phase 3 ADPKD trial in 2019
- Planning to pursue IgAN and T1D CKD as commercial indications

**Phase 3 trial in diabetic CKD**

- Phase 3 AYAME trial underway
- Next milestone: Data in 1H22

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# Q&A