REATA PHARMACEUTICALS

MANAGEMENT CALL TO DISCUSS POSITIVE TOPLINE PIVOTAL YEAR 1 CARDINAL DATA

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, 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: 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 clinical trials, and our research and development programs; the impact of governmental laws and regulations and regulatory developments in the United States and foreign countries; and developments and projections relating to our competitors and our industry.

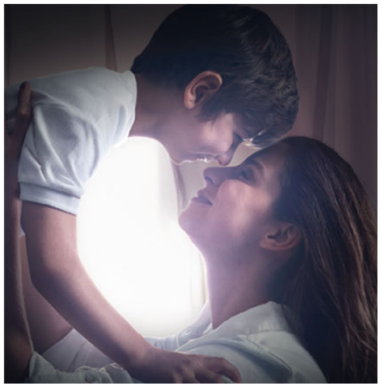
Additional factors that could cause actual results to differ materially from our expectations can be found in our Securities and Exchange Commission filings. Moreover, we operate in a very competitive and rapidly changing environment. New risk factors emerge from time to time, and it is not possible 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.

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

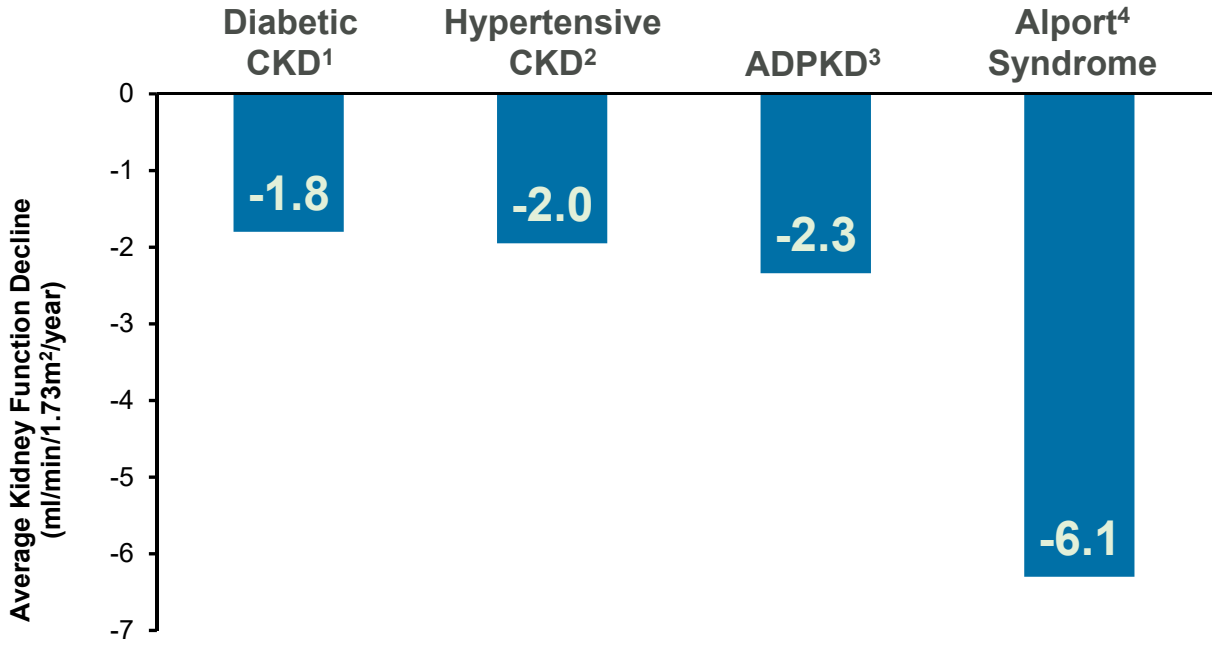
Estimated Prevalence



[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]Estimated based on Persson Clin Nephrol (2005); USRDS Report; Hasstedt Am J Hum Genet. (1983); Temme, Kidney Int. (2012)

Alport Syndrome Patients Rapidly Progress to ESKD

Average Annual Kidney Function Decline on Standard of Care



Estimated Years to ESKD[5]	25	23	19	7

[1]*Perkovic et al. 2018 CANVAS;* [2]*Wright et al. 2002 AASK;* [3]*Torres et al. 2017 REPRISE;* [4]*CARDINAL Phase 3 placebo arm*
[5]*Assuming baseline eGFR=60 mL/min and ESKD occurs at 15 mL/min*

Approved and Widely Used CKD Drugs Slow the Rate of eGFR Decline

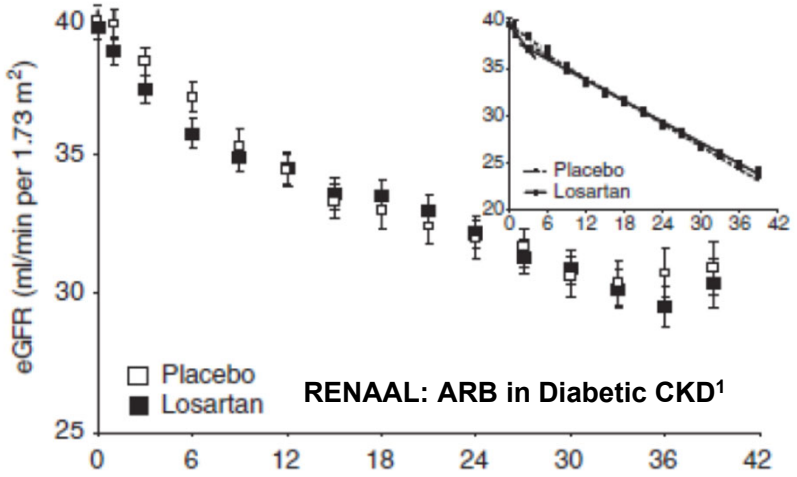
Standard of care treatments for CKD include ACE inhibitors (ACEi), angiotensin receptor blockers (ARBs) and SGLT2 inhibitors

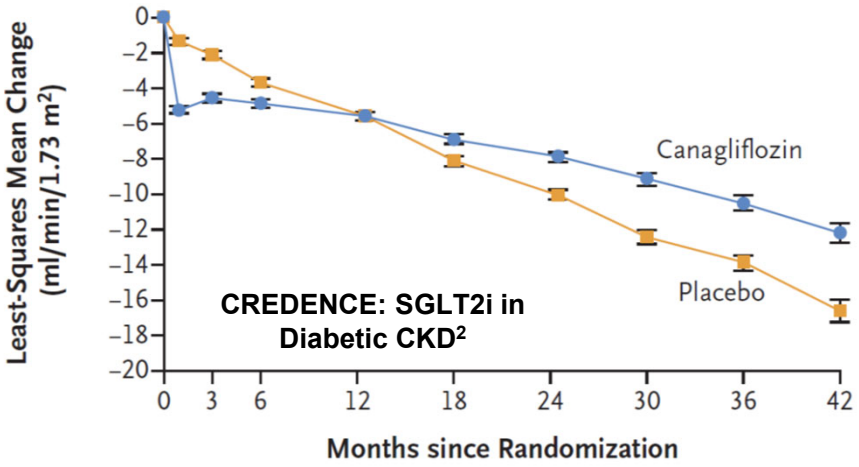
In registrational studies, these drugs slow, but do not stop eGFR decline

- eGFR over time in both the placebo and active drug arms declines
- The treatment effect is measured by comparing the active treatment group to the placebo group

When compared to baseline, both the active and placebo groups' eGFR change is negative

The therapeutic benefit is the delay in the rate of decline which delays the need for dialysis/transplant





[1]Brenner, NEJM (2001), [2]Perkovic (2019)

Tolvaptan Also Only Slows the Rate of eGFR Decline

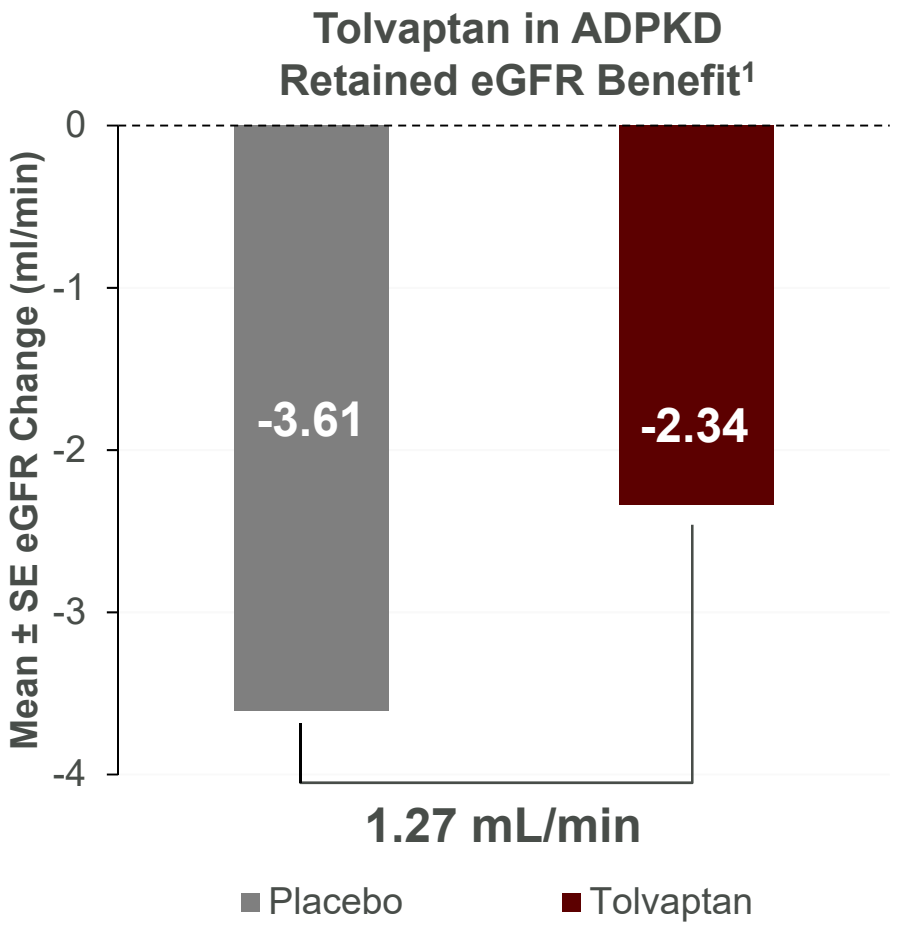
Tolvaptan recently approved for ADPKD using eGFR change after withdrawal of drug, the retained eGFR benefit

Same registrational endpoint as CARDINAL Phase 3 study

In its registrational REPRISE study, tolvaptan slowed, but did not stop eGFR decline

- eGFR in both tolvaptan and placebo groups declined
- The tolvaptan group eGFR declined by 2.34 mL/min
- The placebo group eGFR declined by 3.61 mL/min

Tolvaptan was approved based on the 1.27 mL/min improvement compared to placebo



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[1]Torres, NEJM (2017)

CARDINAL Endpoints: eGFR Improvement On and Off Treatment

FDA guided Reata that retained eGFR benefit versus placebo at one year of treatment may support accelerated approval and after two years may support full approval

- Consistent with tolvaptan precedent
- FDA provided us with similar guidance for our FALCON study in ADPKD
- Has provided this design to other sponsors

CARDINAL Primary: change from baseline in eGFR versus placebo at Week 48

Key Secondary: change from baseline in eGFR versus placebo at Week 52 after withdrawal of drug for 4 weeks

Retained eGFR benefit versus placebo is strong evidence treatment may delay or prevent the need for dialysis or a transplant

CARDINAL Phase 3: Pivotal Study Design

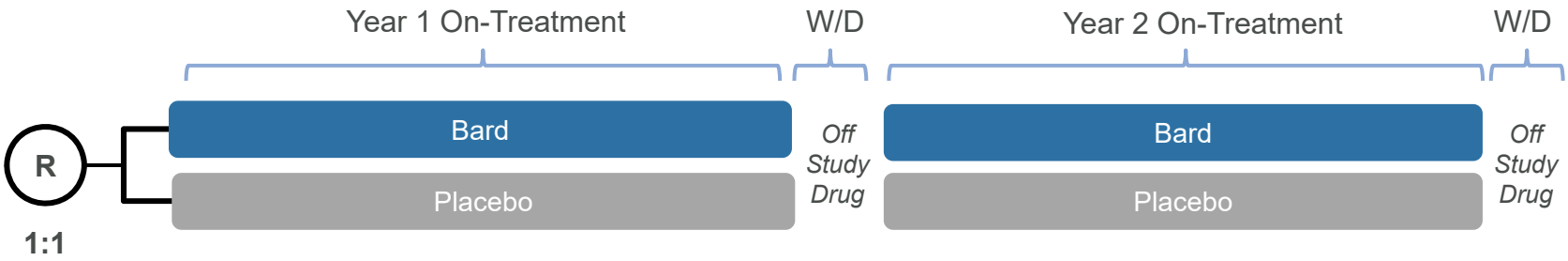
International, double-blind, placebo-controlled, randomized, registrational trial

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

After 52 weeks, patients restart study drug with original treatment assignments and continue for the second year



Demographics and Baseline Disease Characteristics

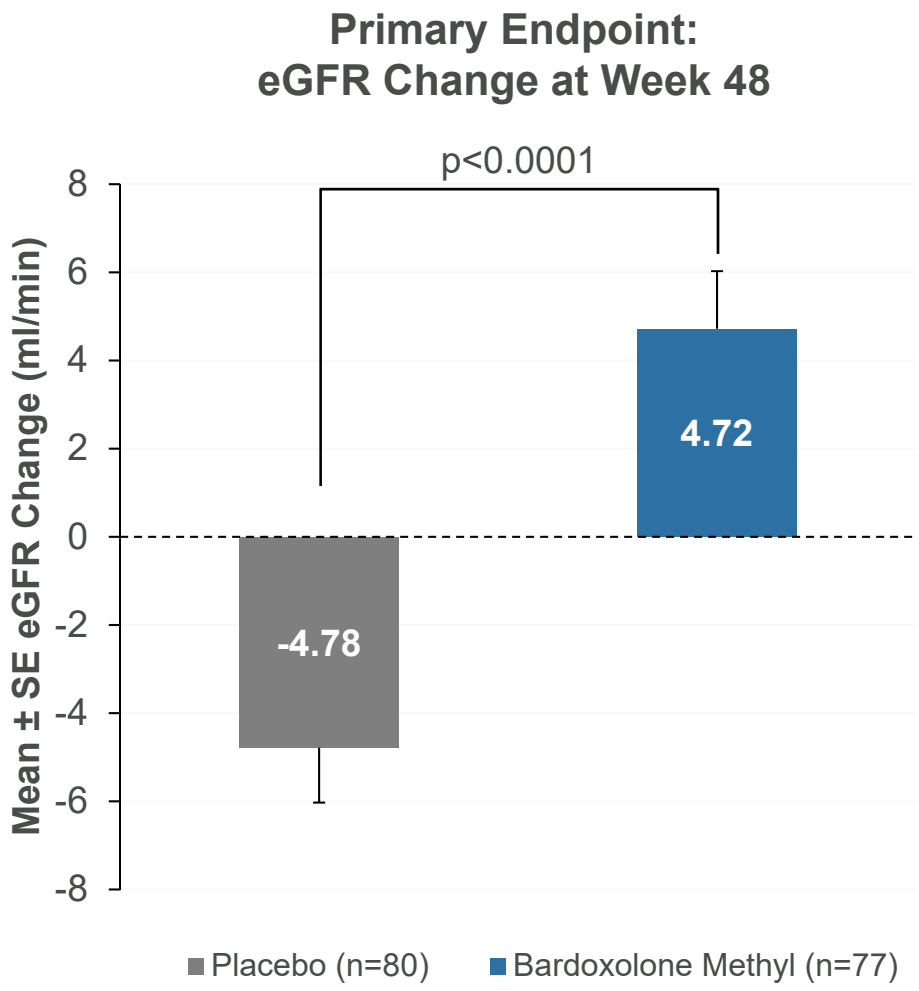
Characteristic (Mean ± SD)	Placebo (n=80)	Bard (n=77)
Male (n,%)	32 (40%)	34 (44%)
Age	39.6 ± 16.0	38.8 ± 14.6
<18 (n,%)	12 (15%)	11 (14%)
eGFR (mL/min/1.73 m^2)	62.6 ± 18.2	62.7 ± 17.7
≤60 (n,%)	33 (41%)	33 (43%)
>60 (n,%)	47 (59%)	44 (57%)
Albumin to Creatinine Ratio (mg/g) (Geometric Mean ± SE)	134.5 ± 33.4	148.1 ± 34.3
≤ 300 mg/g (n,%)	43 (54%)	42 (55%)
> 300 (n,%)	37 (46%)	35 (46%)
SBP (mmHg)	119.6 ± 13.2	119.7 ± 11.9
DBP (mmHg)	72.6 ± 10.5	73.8 ± 9.4
ACEi and/or ARB Use (n, %)	60 (75%)	62 (81%)

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.

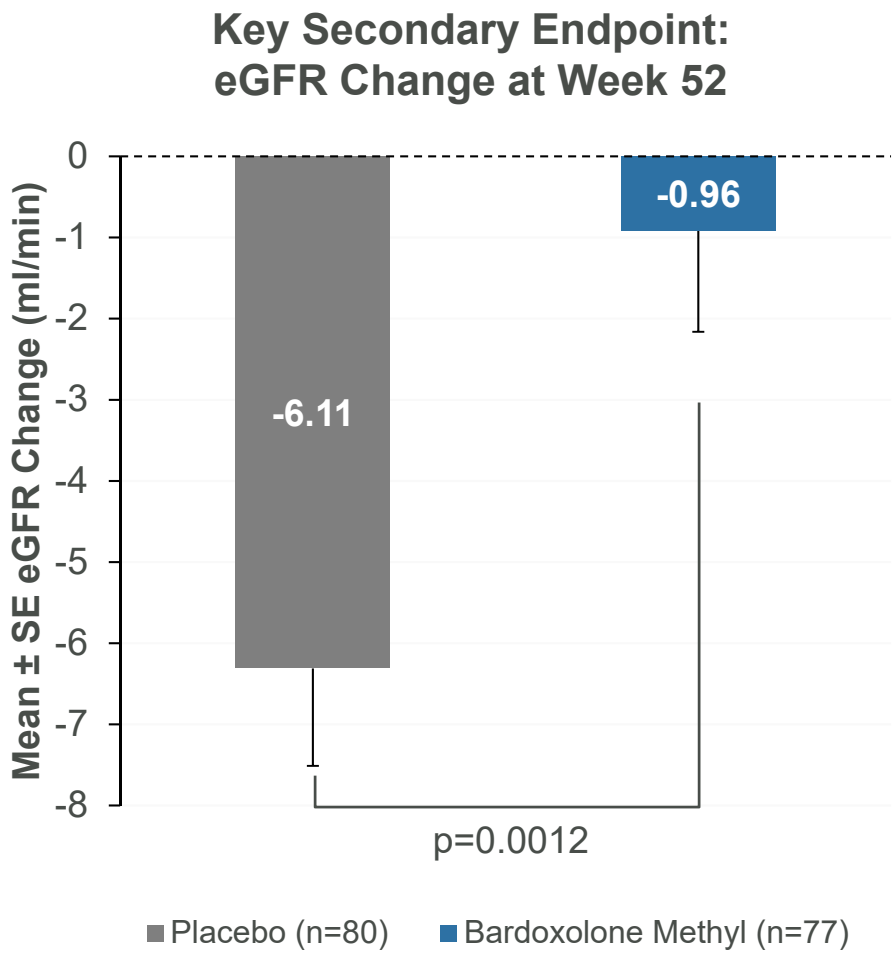
CARDINAL Met Key Secondary Endpoint

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)*

Similar efficacy observed across multiple subgroups, including males versus females, pediatric population, and baseline ACR



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

Summary of Safety

- Fewer SAEs reported in Bard patients
- No fluid overload or major adverse cardiac events in patients treated with Bard
- Blood pressure decreased relative to baseline in the Bard group but was not significantly different between groups
- Overall low rate of cardiac and vascular AEs that was reduced in the Bard arm
- 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

	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%)

Summary and Next Steps

The Phase 3 portion of CARDINAL met its primary endpoint ($p<0.0001$)

The Phase 3 portion of CARDINAL met its precedented, registrational key secondary endpoint ($p=0.0012$)

Bard treatment effectively slowed or halted decline in eGFR in patients with rapidly progressing CKD

- Approximately 75% of bardoxolone treated patients had an improvement in eGFR while on treatment
- Approximately 75% of placebo patients had worsening eGFR

Reported to be well tolerated with numerically fewer SAEs on Bard (5%) versus placebo (13%)

Potential for Bard to be the first approved treatment for AS

Planning to meet with FDA and other regulatory agencies to discuss marketing application submission plans

Beginning to plan for ex-US launch as a result of recent reacquisition of global rights

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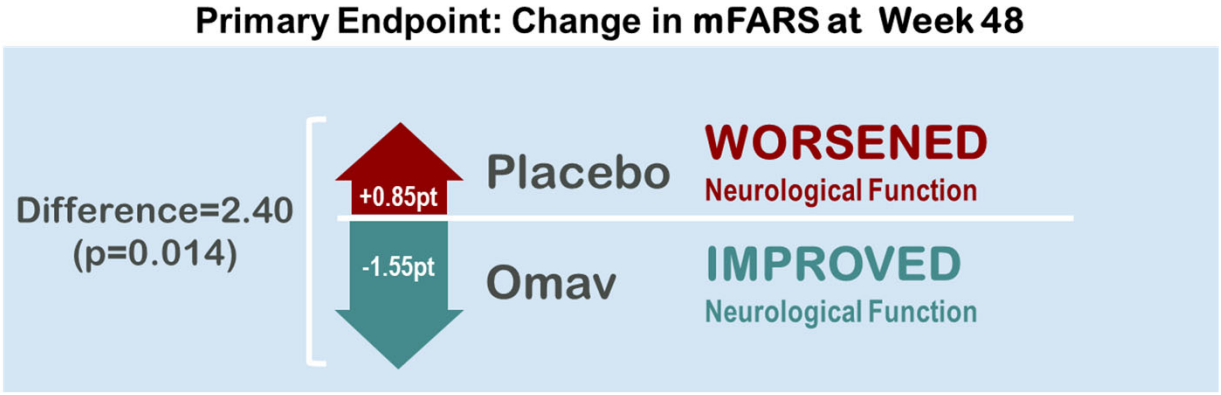
Q&A

An Eventful Q4

On October 10, we announced that we had reacquired the rights to develop Bard, Omav and our other Nrf2 activators

On October 11, we announced the presentation at ASN of several important studies, including the Kashihara work demonstrating the precise mechanism of GFR improvement from Nrf2 activation

On October 14, we announced positive data from the MOXIe study of Omav in patients with Friedreich's ataxia



And yesterday we announced positive data from the CARDINAL Phase 3 study of Bard in patients with Alport syndrome

Poised to Develop Franchises in CKD and Neurological Disease

Bardoxolone in Rare Forms of CKD

- Positive pivotal data in Alport syndrome
- Pipeline in rare forms of CKD
  - Pivotal ADPKD study ongoing
  - Positive proof-of-concept data in FSGS, IgAN, and T1D-CKD

Omaveloxolone in Neurology

- Positive pivotal data in FA
- Proof-of-concept in other neurological diseases
- Plan to study Omav in additional neurological indications

Global Opportunity

- Reata possesses WW commercial rights to all pipeline assets*
- Few or no effective therapies currently approved for lead indications

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*ex-Asia for Bardoxolone