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 at a Glance

- **Two pivotal trials fully enrolled and reading out this year**
  - Alport syndrome
  - Friedreich’s ataxia

- **Addressing unmet needs**
  - Deadly rare diseases
  - No approved therapy
  - Little to no competition

- **Pathway to potential approval**
  - Strong Phase 2 outcomes
  - Conservative Phase 3 clinical trial designs
  - Clear formal FDA guidance on the path to approval

- **Franchise building**
  - Expansion into other rare forms of CKD and neurological diseases

- **Strong financial position**
  - Cash through three pivotal readouts and into 2021
### Deep Pipeline With Three Pivotal Studies and Many Expansion Opportunities

<table>
<thead>
<tr>
<th>DISCOVERY</th>
<th>PRECLINICAL</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PIVOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKD Caused by Alport Syndrome</td>
<td>Bard</td>
<td></td>
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<tr>
<td>Friedreich’s Ataxia</td>
<td>Omav</td>
<td></td>
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<tr>
<td>Connective Tissue Disease Associated Pulmonary Arterial Hypertension</td>
<td>Bard</td>
<td></td>
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<tr>
<td>Autosomal Dominant Polycystic Kidney Disease</td>
<td>Bard</td>
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<tr>
<td>IgA Nephropathy</td>
<td>Bard</td>
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<tr>
<td>Type 1 Diabetic CKD</td>
<td>Bard</td>
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<tr>
<td>Focal Segmental Glomerulosclerosis</td>
<td>Bard</td>
<td></td>
<td></td>
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<tr>
<td>Neurological Indications</td>
<td>RTA 901</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autoimmune Indications</td>
<td>RTA 1701</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Bardoxolone (Bard)
Chronic Kidney Disease
A New Paradigm For Treating Chronic Kidney Disease

Bard increases kidney function (GFR) by resolving inflammation

In preclinical models, Bard:
- Acutely increases glomerular filtration surface area
- Reduces fibrosis and remodeling long-term, leading to structural improvement of the kidney

Healthy Nephron

Inflamed Nephron

Non-functional, Fibroosed Nephron

Can be rescued

Insult: genetic mutation, hypertension, diabetes, etc.

Chronic inflammation, increased pressure, fibrosis, & remodeling progress to ESKD

1Ding et al. 2013; 2Aminzadeh et al. 2014
Bard has demonstrated significant improvements in eGFR in over 10 separate CKD studies. Studies enrolled diverse etiologies of CKD. Improvements in eGFR demonstrated across a broad range of kidney function.

<table>
<thead>
<tr>
<th>Study</th>
<th>Patient Population</th>
<th>eGFR Range (ml/min)</th>
<th>N</th>
<th>∆eGFR (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereditary</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDINAL</td>
<td>Alport syndrome</td>
<td>30 – 90</td>
<td>30</td>
<td>13.4 (p&lt;0.001)</td>
</tr>
<tr>
<td>PHOENIX</td>
<td>ADPKD</td>
<td>30 – 90</td>
<td>31</td>
<td>9.3 (p&lt;0.001)</td>
</tr>
<tr>
<td>Autoimmune</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHOENIX</td>
<td>IgAN</td>
<td>30 – 90</td>
<td>26</td>
<td>8.0 (p&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td>T1D CKD</td>
<td>30 – 90</td>
<td>28</td>
<td>5.5 (p=0.02)</td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSUBAKI</td>
<td>T2D CKD</td>
<td>15 – 59</td>
<td>124</td>
<td>6.6 (p=0.008 vs PBO)</td>
</tr>
<tr>
<td>BEACON</td>
<td>T2D CKD</td>
<td>15 – 29</td>
<td>2185</td>
<td>6.4 (p&lt;0.001 vs PBO)</td>
</tr>
<tr>
<td>BEAM</td>
<td>T2D CKD</td>
<td>20 – 45</td>
<td>227</td>
<td>8.6 (p&lt;0.001 vs PBO)</td>
</tr>
<tr>
<td>402-C-0902</td>
<td>T2D CKD</td>
<td>15 – 45</td>
<td>131</td>
<td>6.5 (p&lt;0.001)</td>
</tr>
</tbody>
</table>

Selected trials listed; ¹Change in eGFR vs. baseline; ²Change in eGFR vs. placebo
Bard is Currently in Development for Five Rare Forms of CKD

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

FALCON Phase 3 study in ADPKD
- PHOENIX Phase 2 demonstrated significant eGFR improvement at 12 weeks
- Study design and endpoints similar to CARDINAL Phase 3 study
- Pivotal Phase 3 planned to start mid-2019

PHOENIX Phase 2 study in other forms of CKD
- Positive data reported for ADPKD, IgAN, and T1D-CKD
- FSGS data expected 1H19

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

US Rare CKD Patients

- ~30,000-60,000
- ~40,000
- ~120,000
- ~160,000
- ~400,000

ADPKD
FSGS
T1D CKD
IgAN
Alport Syndrome
CARDINAL is an International Phase 3 Trial of Bard for the Treatment of Alport Syndrome

Pivotal Phase 3 fully enrolled with 157 patients
- Randomized, double-blind, placebo-controlled international study
- Two-year total treatment duration
- Enrolled patients across approximately 50 sites in the US, Europe, Japan, and Australia

Broad eligibility criteria
- eGFR 30-90 ml/min
- Age 12-70 years old
- Patients required to be on standard of care unless contraindicated

Retained eGFR benefit endpoints support approval
- Potential accelerated approval on retained eGFR after one year of treatment and drug withdrawal
- Potential full approval on retained eGFR after two years of treatment and drug withdrawal
- Conservatively powered to detect improvement in retained eGFR

One-year data expected 2H19

[Diagram of study timeline and treatment regimens]
CARDINAL Phase 2 Results: Sustained eGFR Improvement in Patients Actively Declining on SOC

Enrolled 30 patients and treated for 48 weeks

Baseline characteristics:
- Mean age of 45 years old
- Mean eGFR of 56 ml/min
- 84% of patients were on ACEi or ARB
- Representative of AS population

Average annual eGFR loss prior to study of ~4.2 ml/min

Large, statistically significant increase in eGFR after one year
- Change durable from Week 12 to Week 48
- Week 12 change correlates with Week 48 change

CARDINAL Historical Average eGFR Decline
-4.2 ml/min per year (n=22)

Week 48 ΔeGFR
+10.4 ml/min
n=25, p<0.0001
CARDINAL Phase 2 Results: Significant Retained eGFR Benefit Observed After One Year of Treatment

Observed significant 4.1 ml/min retained eGFR benefit vs. baseline after one year

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

Potential placebo-corrected retained benefit of 8.3 ml/min

At 150 patients, the Phase 3 is powered to detect a placebo-corrected retained benefit of 2.2 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.
FALCON is an International Phase 3 Trial of Bard for the Treatment of ADPKD

Pivotal Phase 3 will enroll approximately 300 patients
- Randomized, double-blind, placebo-controlled international study
- Two-year total treatment duration
- Planning to enroll patients across approximately 60 sites in the US, Europe, and Australia

Broad eligibility criteria
- eGFR 30-90 ml/min
- Age 18-70 years old
- Tolvaptan use is allowed but not required

Retained eGFR benefit endpoints support approval
- Potential accelerated approval on retained eGFR after one year of treatment and drug withdrawal
- Potential full approval on retained eGFR after two years of treatment and drug withdrawal
- Conservatively powered to detect improvement in retained eGFR

Trial initiation expected mid-2019
Enrolled 31 patients with ADPKD and treated for 12 weeks

Baseline characteristics:
- Mean age of 47 years old
- Mean eGFR of 48 ml/min
- 81% of patients were on ACEi or ARB
- Representative of ADPKD population

Average annual eGFR loss prior to study of ~4.8 ml/min

Large, statistically significant increase in eGFR after 12 weeks
- High response rate with all but one patient showing improvement at Week 12
- No change in urinary albumin excretion

**Week 12 ΔeGFR**
+9.3 ml/min
p<0.0001
Available Data Support FALCON Retained Benefit Power Calculations

Observed significant 9.3 ml/min on-treatment eGFR change vs. baseline at Week 12

Prior one-year duration trials demonstrated:
- Correlation between Week 12 and Week 48 eGFR change
- Significant retained eGFR benefit after one year of treatment and four-week washout

Phase 3 modeling\(^1\) assumes:
- Phase 2 retained eGFR benefit of 9.3 ml/min is sustained through Week 48
- 30% (2.8 ml/min) of on-treatment eGFR change is retained after withdrawal
- Placebo loss of 3.4 ml/min based on several recently published ADPKD trials

Potential placebo-corrected retained benefit of 6.2 ml/min

At 300 patients, the Phase 3 is powered to detect a placebo-corrected retained benefit of 1.6 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.
PHOENIX Phase 2 Study of Bard for the Treatment of Rare Forms of CKD

Phase 2, open-label, multi-center, US-only 12-week trial
- Large range of eGFR (30-90 ml/min) and age (18-65 years old)
- Four separate cohorts of patients with ADPKD, IgAN, T1D CKD, and FSGS

Baseline characteristics
- Mean age of 49 in both IgAN and T1D CKD cohorts
- Mean eGFR of 46 ml/min (IgAN) and 68 ml/min (T1D CKD)
- Most patients were receiving standard of care ACEi or ARB

Primary endpoint is change in eGFR from baseline at Week 12
- Statistically significant improvements in eGFR observed to date in ADPKD, IgAN, and T1D CKD
- FSGS cohort ongoing with data expected 1H19

![Graph showing mean ± SE eGFR change over study weeks for IgAN and T1D CKD cohorts.](image)

- IgAN (n=26)
  - Week 12 ΔeGFR: +8.0 ml/min
  - p<0.0001

- T1D CKD (n=28)
  - Week 12 ΔeGFR: +5.5 ml/min
  - p=0.02
Clear Regulatory Guidance from FDA for Alport Syndrome and ADPKD

Completed EOP2 interaction with FDA regarding ADPKD during 4Q 2018

Agreed to same eGFR-based retained benefit approval endpoints for ADPKD as previously recommended for Alport syndrome
- One-year, placebo-corrected retained eGFR data could support accelerated approval
- Two-year, placebo-corrected retained eGFR data could support full approval

FDA again confirmed 4-week withdrawal period is appropriate for Bard
- Bard’s maximum pharmacodynamic effect on eGFR occurs in two to four weeks
- Bard reaches sub-therapeutic concentrations within 4 weeks after withdrawal of drug
- After withdrawal of drug, retained eGFR stabilizes by week 4 in T2D CKD patients

FDA indicated that the sample size for FALCON will be sufficient to assess safety of Bard
- Large safety database with over 2,000 people having been exposed to Bard
- Bard well-tolerated without major safety signals in patient populations under study

FDA has indicated that Reata has conducted all preclinical toxicology studies and clinical pharmacology studies required for NDA submission

Consistent guidance from FDA on endpoints provides confidence that CARDINAL and FALCON trial designs will support NDA submission and approval if successful
**Bard Has the Potential to be the First Therapy for the Treatment of CKD Caused by Alport Syndrome**

<table>
<thead>
<tr>
<th>Today: Unmet Need, Limited Awareness, Misdiagnosis</th>
<th>Tomorrow: Identification, Awareness, Novel Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Deadly Disease</strong></td>
<td><strong>Improvement or Maintenance</strong></td>
</tr>
<tr>
<td>“Classic” AS patients may reach ESKD by their late 30's and are likely to receive dialysis or a kidney transplant</td>
<td>Improvement or maintenance of kidney function in a high proportion of patients declining on SOC</td>
</tr>
<tr>
<td><strong>Prevalence</strong></td>
<td><strong>Patient Identification</strong></td>
</tr>
<tr>
<td>It is not well-known that AS is the 2nd most common form of inherited CKD. Women and men are equally impacted</td>
<td>Patients with CKD, family history, and hematuria are routinely screened and AS identified</td>
</tr>
<tr>
<td><strong>SOC is Insufficient</strong></td>
<td><strong>A Novel Therapeutic Option</strong></td>
</tr>
<tr>
<td>There is no specific treatment for Alport syndrome. SOC includes the use of ACEi/ARB therapy</td>
<td>Potential for Bard to be 1st approved treatment for CKD caused by AS</td>
</tr>
<tr>
<td><strong>Misdiagnosed</strong></td>
<td><strong>Accurate Diagnosis</strong></td>
</tr>
<tr>
<td>Genetic tests are rarely ordered and misdiagnosis is common</td>
<td>Genetic testing to confirm type IV collagen mutations</td>
</tr>
</tbody>
</table>

**Alport Syndrome US Patient Opportunity**

- **~11K** Diagnosed Patients
- **30-60K** “Classic” AS Patients
- **Stage 2-3 CKD with Collagen 4A 3, 4, or 5 Mutations**
The marketing, operations, market access, and sales commercial leadership team is onboard.

A specialty pharmacy distribution model will support product availability to patients.

A patient-centered HUB model is under development to support on-label utilization, ease of access, and patient compliance.

Preliminary field force sizing and structure is complete for the sales and access teams.

Trade naming process is underway.

Disease awareness campaigns have launched, designed to educate physicians about Alport syndrome.

A genetic testing program is under development to improve the accuracy of diagnosis in the US.
Supply chain readiness on track for planned NDA and commercial launch

Low cost of goods with 5-step synthesis from regulatory starting material to drug substance

Established supply chain with adequate capacity for near-term clinical and commercial demand

Manufacturing and Quality teams in place

Process validation batches completed for drug substance; registration batches completed for drug product

3-year, room temperature, shelf life established for Bard capsules at 5, 10, 20, and 30 mg dose
Omaveloxolone
Friedreich’s Ataxia
Omav is in Development for Friedreich’s Ataxia with Broadly Applicable Mechanism of Action

MOXIe pivotal study in FA
- Fully enrolled at 103 patients
- Data expected 2H19

Mechanism may be broadly applicable to neuromuscular diseases
- Mitochondrial dysfunction is a feature of many neuromuscular diseases
- In preclinical models, Omav improves mitochondrial function by activating Nrf2 and increasing ATP production

Demonstrated activity in preclinical and clinical settings
- Omav reduced seizure frequency in refractory, progressive preclinical epilepsy models
- Omav restored mitochondrial function in biopsy samples from FA, ALS, and familial Parkinson’s disease patients
- Omav improved mitochondrial function as demonstrated by reductions in blood lactate and heart rate in patients with primary mitochondrial disease (MOTOR study)
- Omav improved neuromuscular function in FA patients as assessed by the mFARS rating scale (MOXIe Part 1)

If pivotal MOXIe data are positive, we are poised to advance Omav into a series of indication expansion opportunities in settings with few or no effective therapies
MOXIle is an International Pivotal Trial of Omav for the Treatment of Friedreich’s Ataxia

Pivotal portion fully enrolled with 103 patients
- Randomized, double-blind, placebo-controlled international study
- 48-week total treatment duration
- Enrolled patients across 11 sites in the US, Europe, and Australia

Broad eligibility criteria
- mFARS 20-80
- Age 16-40 years old
- Up to 20% of patients can have *pes cavus* foot deformity

mFARS endpoint supports approval
- Potential full approval on mFARS after 48 weeks of treatment
- Conservatively powered

Data expected 2H19
Enrolled 69 patients and treated for 12 weeks

Dose ranging, double-blind, placebo-controlled trial that tested 7 dose levels

Baseline characteristics:
- Mean age of 26 years old
- Mean duration since diagnosis of 10 years
- 90% of patients ambulatory
- Mean mFARS of 41

Omav significantly improved mFARS from baseline across all doses (p<0.0001)

Optimal dose identified as 160 mg
- Placebo-corrected change at 160 mg (-2.3) neared statistical significance (p=0.06)
- Two-thirds of patients at 160 mg dose had *pes cavus*, a foot deformity that influences measurement of treatment response
- Placebo-corrected mFARS improvement of -4.4 points (p=0.01) at 160 mg in patients without *pes cavus*

*P-values are change from baseline as compared to zero*
Pivotal Portion of MOXIe Trial Optimized Based on Part 1 Study Results

Based on data from dose-ranging portion of trial, we have optimized the pivotal portion of the trial
- Extended treatment duration to 48 weeks
- Increased the sample size to 103 patients
- Excluded patients who have large differences between two independent mFARS screening scores
- Limited patients with pes cavus to 20%

Phase 3 modeling\(^1\) assumes:
- Improvement of mFARS in Omav-treated patients of -3.5, of which almost all is derived from patients without pes cavus
- No change in placebo-treated patients

Potential placebo-corrected improvement of -3.5

At 100 patients, the study is powered to detect a placebo-corrected difference in mFARS of -1.2 (p<0.05) to -1.7 (p<0.01)

\(^1\)Modeled changes are not intended as a forecast of probable results. No assurance is given about the results that will be obtained.
Clear Regulatory Guidance from FDA for Friedreich’s Ataxia

FDA has provided written guidance that mFARS is an acceptable endpoint for approval for Omav in FA

No major safety signals detected in completed trials

Clinical pharmacology studies required for NDA submission are underway

Nonclinical toxicology studies required for NDA submission are underway

Anticipate positive MOXIe trial demonstrating improvement in mFARS equivalent to at least one year of progression with acceptable safety profile would provide strong rationale for Omav to be the first approved treatment for FA
# Omav Has the Potential to be the First Therapy for the Treatment of Friedreich’s Ataxia

## Today: Deadly Disease with No Approved Therapy

<table>
<thead>
<tr>
<th>Deadly Disease</th>
<th>FA impacts children and young adults, leading to wheelchair use and shortened life expectancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Specific Treatment</td>
<td>Vitamin supplements are used but there is no approved treatment</td>
</tr>
<tr>
<td>Misdiagnosed</td>
<td>Often misunderstood for clumsiness or other ataxias, FA may take years to diagnose</td>
</tr>
<tr>
<td>FA Treatment</td>
<td>Concentrated in neurology ataxia centers and 8 US academic FA treatment centers</td>
</tr>
</tbody>
</table>

## Tomorrow: 1<sup>st</sup> Approved FA Therapy

<table>
<thead>
<tr>
<th>Measured Improvement</th>
<th>May preserve or improve function measured through change in mFARS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potential 1&lt;sup&gt;st&lt;/sup&gt; Approved Treatment</td>
<td>For patients with Friedreich’s ataxia</td>
</tr>
<tr>
<td>Patient Identification</td>
<td>Will aid earlier, accurate diagnosis of Friedreich’s ataxia and a therapeutic option earlier</td>
</tr>
<tr>
<td>Broadened Awareness</td>
<td>Through FA centers, ataxia centers, and large neurology practices</td>
</tr>
</tbody>
</table>

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**Friedreich’s Ataxia US Patient Opportunity & Treatment Landscape**

- **~6,000** Friedreich’s Ataxia Patients (US ICD-9 2015)
- **8** FA Centers of Excellence
- **~1,700** Treating Neurologists
- **21** Ataxia Centers
The marketing, operations, market access, and sales commercial leadership team is onboard.

A specialty pharmacy distribution model will support product availability to FA patients.

A patient-centered HUB model is under development to support on-label utilization, ease of access, and patient compliance.

Preliminary field force sizing and structure is complete for the FA sales and access teams.

Trade naming process is underway.

Disease awareness campaigns have launched designed to educate about FA.
Bardoxolone
CTD-PAH
Bard in Development for Connective Tissue Disease Associated Pulmonary Arterial Hypertension

CATALYST Phase 3 study in CTD-PAH
- Phase 3 enrolling 200 patients
- Data expected 1H20

CTD-PAH is an autoimmune form of PAH
- Affects 10% to 15% of scleroderma and lupus patients and is their leading cause of death
- Estimated prevalence: 12,000 in US; 50,000 worldwide; 30% of all PAH

Despite available therapies, large unmet need
- Vasodilators are the only current treatment option and have lower benefit in CTD-PAH versus idiopathic PAH
- Vasodilators 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
CATALYST is a Global Pivotal Trial of Bard for Treatment of CTD-PAH

Phase 3 enrolling 200 patients
- Randomized, double-blind, placebo-controlled international study
- 24-week treatment duration
- Enrolling patients across approximately 100 sites in North America, Europe, Australia, South America, and Asia

Broad eligibility criteria
- 6MWD ≥ 150 meters
- Age 18-75 years old
- WHO Functional Class II and III on up to two background therapies

6MWT endpoint supports approval
- Potential full approval on 6MWT after 24 weeks of treatment
- Conservatively powered

Data expected 1H20
LARIAT Phase 2 Results: Large Increase in 6MWD

Enrolled idiopathic PAH and CTD-PAH patients and treated for 16 weeks
- PAH patients required to be on 1 to 2 background therapies
- Assessed safety and change in 6MWD

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

Subset of patients with CTD-PAH demonstrated largest responses
- Clinically meaningful improvement of 28.4 m observed across all patients
- Patients without anemia demonstrated a larger placebo-corrected difference of 48.5 m
- Patients with anemia at baseline demonstrated larger variability and are excluded in CATALYST

<table>
<thead>
<tr>
<th>Dataset</th>
<th>Treatment</th>
<th>N</th>
<th>Week 16 Δ6MWD (m)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Change from Baseline</td>
</tr>
<tr>
<td>All</td>
<td>Placebo</td>
<td>7</td>
<td>9.8 p=0.44</td>
</tr>
<tr>
<td></td>
<td>Bard</td>
<td>15</td>
<td>38.2 p&lt;0.001</td>
</tr>
<tr>
<td>Without Anemia</td>
<td>Placebo</td>
<td>5</td>
<td>-5.8 p=0.68</td>
</tr>
<tr>
<td></td>
<td>Bard</td>
<td>14</td>
<td>42.7 p&lt;0.001</td>
</tr>
</tbody>
</table>
Recent Highlights and Key Upcoming Milestones

**Bard in Alport syndrome**
Pivotal Phase 3 fully enrolled with data available in 2H19

**Bard in rare forms of CKD**
- 12-week data from ADPKD, IgAN, and T1D CKD reported in 2018
- 12-week data from FSGS in 1H19

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

**Omav in Friedreich’s ataxia**
Pivotal Phase 2, part 2 fully enrolled with data available in 2H19

**Bard in CTD-PAH**
Phase 3 CATALYST pivotal data available in 1H20

**Partner Program: Bard in diabetic CKD**
Phase 3 AYAME trial underway, data available in 1H22
Sponsored by KHK, Reata’s licensee in Asia
# Reata Patent Portfolio

## BARDOXOLONE METHYL

- Morphic form patents (US 8,088,824 and US 8,309,601) claim all known pure forms of Bard and the solid dispersion (SD) used in the commercial form.
- Claims to amorphous Bard and SD granted in US, EU, Japan, Canada, China, Mexico, Eurasia, and 10 other territories with applications pending in 10 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, and 11 other territories; pending in 20 other territories.
- Anticipated protection to 2038 worldwide with term extensions.
- Genus patent (US 8,124,799) claims granted in 15 foreign territories & pending in 3.
Injury due to hyperfiltration would cause UACR to increase over time

UACR not significantly different from baseline at Weeks 48 or 52
- Initial increase in UACR explained by increase in GFR
- After initial eGFR-based increase, UACR trends down through Week 48
- Mean changes clinically insignificant

Patients with baseline UACR > 300 mg/g (n=9) had 10% reduction at Week 48 and 28% reduction at Week 52

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
Retained eGFR Benefit is an Accepted Approvable Endpoint in Rare CKD Trials

Withdrawal of drug after long-term treatment provides evidence whether an intervention protected or harmed the kidney during treatment

For pivotal trials, FDA has accepted or required retained benefit analyses in ADPKD, Alport syndrome, IgAN, and FSGS

<table>
<thead>
<tr>
<th>Trial</th>
<th>Company</th>
<th>Drug</th>
<th>Disease</th>
<th>Phase</th>
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<tbody>
<tr>
<td>REPRISE</td>
<td>Otsuka</td>
<td>Tolvaptan</td>
<td>ADPKD</td>
<td>Approved</td>
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<tr>
<td>FALCON</td>
<td>Reata</td>
<td>Bardoxolone</td>
<td>ADPKD</td>
<td>Phase 3</td>
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<td>CARDINAL</td>
<td>Reata</td>
<td>Bardoxolone</td>
<td>Alport syndrome</td>
<td>Phase 3</td>
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<tr>
<td>PROTECT</td>
<td>Retrophin</td>
<td>Sparsentan</td>
<td>IgA Nephropathy</td>
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<td>ARTEMIS-IGAN</td>
<td>Omeros</td>
<td>OMS721</td>
<td>IgA Nephropathy</td>
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<td>DUPLEX</td>
<td>Retrophin</td>
<td>Sparsentan</td>
<td>FSGS</td>
<td>Phase 3</td>
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</tbody>
</table>

The duration of withdrawal does vary by drug and is based on its PK/PD profile
- Time required for the drug to reach sub-therapeutic concentrations
- Time needed for reversal of PD markers
- Dosing duration required for maximal effect
Four Week Off-Treatment Duration is Sufficient to Assess Retained Benefit with Bard

Bard’s maximal effect on eGFR after initiation of dosing occurs within 2-4 weeks after the last increase in dose.

Bard is eliminated within 2 weeks after cessation of dosing:
- 4-week withdrawal represents approximately 17 half-lives of drug.
- Sub-therapeutic concentrations are achieved with no active metabolites.
- Pharmacodynamic markers of Nrf2 activation, which are monitored in all patients, are not significantly different from baseline after 4-week withdrawal.

Post-treatment eGFR values stabilize by Week 4 in T2D CKD patients.

**BEACON:** eGFR Changes are Maximal 4 Weeks After the Last Increase in Dose

**BEACON:** Post-treatment eGFR Relatively Stable through 8 Weeks Following Cessation of Bard Treatment

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Bard</th>
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<tbody>
<tr>
<td>Baseline</td>
<td>22.5</td>
<td>22.4</td>
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<tr>
<td>Change 4 weeks post-last dose</td>
<td>2.1</td>
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<tr>
<td>Change 8 weeks post-last dose</td>
<td>2.4</td>
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## List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>6MWD</td>
<td>Six-minute walk distance</td>
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<tr>
<td>ADPKD</td>
<td>Autosomal dominant polycystic kidney disease</td>
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<tr>
<td>ACEi</td>
<td>Angiotensin-converting enzyme inhibitor</td>
</tr>
<tr>
<td>ALS</td>
<td>Amyotrophic lateral sclerosis</td>
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<tr>
<td>ARB</td>
<td>Angiotensin receptor blocker</td>
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<td>AS</td>
<td>Alport syndrome</td>
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<tr>
<td>ATP</td>
<td>Adenosine triphosphate</td>
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<td>Bard</td>
<td>Bardoxolone methyl</td>
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<td>CKD</td>
<td>Chronic kidney disease</td>
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<td>CTD-PAH</td>
<td>Connective tissue disease-associated pulmonary arterial hypertension</td>
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<tr>
<td>eGFR</td>
<td>Estimated glomerular filtration rate</td>
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<td>EOP2</td>
<td>End-of-Phase 2 meeting</td>
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<td>ESKD</td>
<td>End-stage kidney disease</td>
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<td>FA</td>
<td>Friedreich’s Ataxia</td>
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<td>FDA</td>
<td>US Food and Drug Administration</td>
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<td>FSGS</td>
<td>Focal segmental glomerulosclerosis</td>
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<td>GBM</td>
<td>Glomerular basement membrane</td>
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<td>GFR</td>
<td>Glomerular filtration rate</td>
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<td>IgAN</td>
<td>IgA nephropathy</td>
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<td>KHK</td>
<td>Kyowa Hakko Kirin Co. Ltd.</td>
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<td>mFARS</td>
<td>Modified Friedreich’s ataxia rating scale</td>
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<tr>
<td>ml/min</td>
<td>mL/min/1.73 m²</td>
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<tr>
<td>NDA</td>
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<tr>
<td>Nrf2</td>
<td>Nuclear factor (erythroid-derived 2)-related factor 2</td>
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<td>Omav</td>
<td>Omaveloxolone</td>
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<td>PAH</td>
<td>Pulmonary arterial hypertension</td>
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<tr>
<td>PBO</td>
<td>Placebo</td>
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<tr>
<td>PK/PD</td>
<td>Pharmacokinetics/pharmacodynamics</td>
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<td>SOC</td>
<td>Standard of Care</td>
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<td>T1D CKD</td>
<td>Type 1 diabetic chronic kidney disease</td>
</tr>
<tr>
<td>T2D CKD</td>
<td>Type 2 diabetic chronic kidney disease</td>
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<td>UACR</td>
<td>Urinary albumin to creatinine ratio</td>
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<tr>
<td>W/D</td>
<td>Withdrawal</td>
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