Allena Pharmaceuticals, Inc.

(Exact name of registrant as specified in its charter)

DELAWARE

(State or other jurisdiction
of incorporation) 001-38268

(Commission
File Number) 45-2729920

(I.R.S. Employer
Identification No.)

One Newton Executive Park, Suite 202
Newton, Massachusetts
(Address of principal executive offices)

02462

(Zip Code)

Registrant’s telephone number, including area code (617) 467-4577

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 or Rule 12b-2 of the Securities Exchange Act of 1934.

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒
Item 7.01 Regulation FD Disclosure.

Allena Pharmaceuticals, Inc. (the “Company”) will be presenting a corporate update at the Ladenburg Thalmann 2018 Healthcare Conference on October 2, 2018 and the 2018 Cantor Global Healthcare Conference on October 3, 2018. As part of these presentations, the Company will deliver the slide presentation furnished to this report as Exhibit 99.1 and which is incorporated herein by reference.

The information in this report furnished pursuant to Item 7.01 shall not be deemed “filed” for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to the liabilities of that section. It may only be incorporated by reference in another filing under the Exchange Act or the Securities Act of 1933, as amended, if such subsequent filing specifically references the information furnished pursuant to Item 7.01 of this report.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

<table>
<thead>
<tr>
<th>Exhibit No.</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>99.1</td>
<td>Corporate presentation furnished by Allena Pharmaceuticals, Inc. on October 2, 2018</td>
</tr>
</tbody>
</table>
SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: October 2, 2018

Allena Pharmaceuticals, Inc.

By: /s/ Edward Wholihan
Edward Wholihan
Chief Financial Officer
Bringing First-in-Class Oral Enzyme Therapeutics to Patients with Rare and Severe Metabolic and Kidney Disorders

October 2018
Allena Pharmaceuticals, Inc.

These slides contain forward-looking statements and information. The use of words such as “may,” “might,” “will,” “should,” “expect,” “plan,” “anticipate,” “believe,” “estimate,” “project,” “intend,” “future,” “potential,” or “continue,” and other similar expressions are intended to identify forward-looking statements. All forward-looking statements are based on estimates and assumptions by our management that, although we believe them to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that we expected. These statements are also subject to a number of material risks and uncertainties that are described under the heading “Risk Factors” in our Annual Report on Form 10-K for the year ended December 31, 2017 filed with the Securities and Exchange Commission on March 27, 2018 and in our Quarterly Report on Form 10-Q for the quarter ended June 30, 2018 filed with the Securities and Exchange Commission on August 7, 2018, as well as discussions of potential risks, uncertainties and other important factors in our subsequent filings with the Securities and Exchange Commission. Any forward-looking statement speaks only as of the date on which it was made. We undertake no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.
Investment Highlights

**Significant Unmet Need in Oxalate and Urate Disorders**
- Focused on rare and severe metabolic disorders that can cause kidney stones, damage the kidney, and potentially lead to CKD and ESRD
- No approved oxalate therapies; potential untapped multi-billion dollar market

**Late-Stage Development Candidate: Reloxaliase**
- First-in-class, oral therapy for severe hyperoxaluria
- Enrolling Phase 3 study, URIROX-1, in enteric hyperoxaluria, topline data expected 2H 2019
- Enrolling Phase 2 basket study in other severe and orphan indications

**Pioneering Expertise in Oral Enzyme Therapeutics**
- Approach enables treatment of metabolic diseases with oral, non-absorbed enzyme therapeutics
- GI MOA reduces subsequent metabolic burden on the kidney

**Strong Support from Leading Biotechnology Investors**
- Raised $75M in successful initial public offering in 4Q 2017
**Allena’s Pipeline: First-in-Class Therapeutic Strategy for Oxalate and Urate Disorders**

<table>
<thead>
<tr>
<th>Product</th>
<th>Indication</th>
<th>Discovery</th>
<th>Pre-Clinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Next Milestone</th>
<th>Commercial Rights</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reloxalase</strong></td>
<td>Enteric hyperoxaluria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2H ’18: Initiate URIROX-2</td>
<td>Worldwide</td>
</tr>
<tr>
<td></td>
<td>Systemic oxalosis*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2H ’18: Interim data</td>
<td>Worldwide</td>
</tr>
<tr>
<td></td>
<td>Primary hyperoxaluria* (Orphan Designation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2H ’18: Interim data</td>
<td>Worldwide</td>
</tr>
<tr>
<td></td>
<td>Pediatric hyperoxaluria* (Orphan Designation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2H ’18: Interim data</td>
<td>Worldwide</td>
</tr>
<tr>
<td><strong>ALLN-346</strong></td>
<td>Hyperuricemia and CKD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1H ’19: IND filing</td>
<td>Worldwide</td>
</tr>
</tbody>
</table>

*To be evaluated in a single Phase 2 clinical trial with a basket design that will enroll subsets of patients suffering from complications of severe hyperoxaluria, including adolescents and adults with primary or enteric hyperoxaluria with advanced CKD, both of which can lead to systemic oxalosis.
Hyperoxaluria is characterized by markedly elevated urinary oxalate levels.

Primary Hyperoxaluria:
- Orphan genetic disorder caused by endogenous excess production of oxalate in the liver

Secondary Hyperoxaluria:
- Disorder caused by excess absorption of oxalate in the GI tract
  - Enteric: due to underlying GI disorders
  - Idiopathic: due to an unknown cause

Fecal Excretion
- Oxalate is absorbed and secreted along the GI tract

Gastrointestinal Tract
- Oxalate is absorbed and secreted along the GI tract

Kidney Damage and Inflammation
- Crystal deposition in parenchyma
- Kidney Impairment
  - Kidney is unable to filter (declining eGFR)
  - Oxalate is measured in plasma

Kidney Stones
- Excreted or removed
- Oxalate is measured in the urine

Oxalate is measured in plasma.

Kidney Stones Excreted or removed
Oxalate Rich Foods
- Oxalate is absorbed in the GI tract

Degrades Oxalate along GI tract
- Reloxaliase

Liver blood

Expertise and Proprietary Technological Approach in Enzyme Therapeutics Enables First-in-Class Therapeutic Strategy for Oxalate and Urate Disorders

Oral enzymes designed to **rapidly degrade a specific metabolite** within the gut, **reducing GI absorption and accumulation** in the kidney, blood and other organ systems

**Reloxaliase**
- **Recombinant Protein**
  - Oral, non-absorbed, highly specific
- **Optimized Highly Active Form of the Enzyme for GI Tract**
  - Stabilize enzyme without compromising activity
  - Convenient, 1-2 capsules per meal
  - RT-stable formulation
- **Streamlined, Robust Manufacturing**
  - COGS in the range of oral small molecules
There are no FDA approved pharmacological therapies to treat any form of hyperoxaluria.

Enteric Hyperoxaluria (EH) Urinary Oxalate (UOx) Study 713 Patient Examples

- Whipple (Pancreatic Insufficiency): 14 stones in last 5 years (16 stones visible by CT)
- Celiac disease: 3 stones in last 2 years (4 stones visible by CT)
- Gastric Bypass: 8 stones in the last 5 years (3 stones visible by CT)

EH patients in Allena’s Phase 2 clinical program:
- Very high baseline UOx
- >94% of EH subjects in Study 713 had experienced at least one kidney stone with an average of >3 kidney stones visible by routine CT scan

Study 713 Patient Examples

- Celiac disease: 3 stones in last 2 years (6 stones visible by CT)
- Gastric Bypass: 8 stones in the last 5 years (3 stones visible by CT)
- Whipple (Pancreatic Insufficiency): 14 stones in last 5 years (16 stones visible by CT)

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Enteric Hyperoxaluria (EH) Urinary Oxalate (UOx)

Kidney Stone Disease ~5M Enteric ~200K–250K ~5K

Primary Enteric Hyperoxaluria Patients are a High Risk Patient Population who are Identifiable by Physicians and Need Treatment.

- Direct expenditures 4 years post GI malabsorptive procedure or disease diagnosis
- 18K average annual direct expenditures
- $66K average annual direct expenditures

High unmet need: more frequent and more complex stones, fail standard of care (i.e., hydration, dietary modifications)

Stones and CKD burden: 66K average annual direct expenditures 4 years post GI malabsorptive procedure or disease diagnosis

- Enteric GI malabsorptive conditions include: gastric bypass surgery, Crohn’s disease, ulcerative colitis, pancreatic insufficiency, celiac disease, and liver disease

~5K
Reloxaliase Initially Targets Enteric Hyperoxaluria Patients with Underlying Malabsorptive GI Diseases and Kidney Stones

Kidney stones, often the first clinical manifestation of hyperoxaluria, allow patients to be identified

4.4 Million
Estimated Patients with GI Malabsorptive Conditions with no Prior Kidney Stones or CKD

Patients potentially at risk for enteric hyperoxaluria

400-250K
Estimated Patients with Enteric Hyperoxaluria and Kidney Stones

Patients developed Kidney Stone Disease of which 40% developed CKD

~2017 US population impacted by stones and enteric hyperoxaluria

4 year progression of disease

- Analysis tracked patients for whom an enteric disease diagnosis or procedure code was entered between 7/1/10-6/30/12 who did not have a claim with an KSD, CKD/ESRD/Dialysis diagnosis or procedure code from 1/1/10-6/30/10. Analysis showed that approximately 5% of patients subsequently developed one or more kidney stones ("Kidney Stone Disease"), of which 40% also developed CKD over the subsequent four years to determine prevalence in 2012. 2017 figures apply 3.6% CAGR to 2012 population figures.

- Approximately 9% of patients develop CKD without kidney stone disease within 4 years

1. Truven Health Analytics, part of the IBM Watson Health business longitudinal claims analysis, August 2017.
Risk of Renal Complications Rises with Higher Urinary Oxalate Levels: Reduction of UOx Improves Renal Outcomes

Key Peer Review Studies:
- Higher baseline UOx predicts future stone events in enteric hyperoxaluria patients (Leske ASN 2017)
- 50% reduction in risk of kidney stone recurrence associated with ≥20% decrease in UOx (Borghéi 2002)
- 25-50% reduction in kidney stone recurrence rate associated with ≥20% decrease in UOx (Curhan and Taylor 2008)
- Preservation of renal function associated with ~10% reduction in UOx (Milliner 1994)

Literature and KOL Input:
*20% reduction in UOx would be clinically meaningful

Kidney Damage

Kidney Failure

Kidney Stones

CaOx Crystal Formation

Normal Kidney Function

Risk of Renal Complications*

Increasing Severity

Systemic Crystal Deposition

Primary Hyperoxaluria

Enteric Hyperoxaluria

Normal

Idiopathic Hyperoxaluria

Idiopathic Hyperoxaluria

Enteric Hyperoxaluria

Preservation of renal function associated with ~10% reduction in UOx (Milliner 1994)

* the complications noted in the figure represent a general progression of kidney harm and disease associated with increasing urinary oxalate excretion levels, but it is important to understand that there is considerable variability among individuals between urinary oxalate excretion levels and kidney function and disease.
The Patient Journey for Severe Hyperoxaluria Patients can Include Complex Specialty Care with Progressive Disease

Recurrent Kidney Stones
Stone Clinic
Kidney Stone Disease Management
Nephrologist/Urologist

Metabolic Management
Complex Specialty Care
Monitoring Kidney Function
Nephrologist

Renal Damage and ESRD
Dialysis Center
Routine Dialysis

Strong Patient Advocacy:
The Kidney Health Initiative project brings together patients, clinicians, industry and the FDA to evaluate potential endpoints for future clinical trials in enteric and primary hyperoxaluria

Clinical and Regulatory Progression of Reloxaliase

### Pre clinical
- Progressive Increase in Enzyme Activity
- Porcine Rhubarb Model Presented at AUA 2016
- Porcine Western Diet Model Presented at ASN 2016

<table>
<thead>
<tr>
<th>2° Hyperoxaluria</th>
<th>Pre clinical</th>
<th>Orphan Designation</th>
<th>Initiated Ph 2 Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGTKO Mouse Model</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Porcine Dietary Hydroxyproline and Porcine Sodium ox-IV injection model</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1° Hyperoxaluria</th>
<th>Pre clinical</th>
<th>Orphan Designation</th>
<th>Initiated Ph 2 Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>FDA grants Orphan Disease Designation for Reloxaliase in both PH and Pediatric Hyperoxaluria (Primary and Secondary)</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>EC grants Orphan Disease Designation for Reloxaliase in PH</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

### Ph 1 Healthy Volunteers
- n=30

### Ph 2 Open Label
- n=16
- Multicenter, Open Label, Single Arm Outpatient Study in Enteric and Idiopathic Hyperoxaluria
- Presented at ASN 2014

### Ph 2 Randomized Controlled
- n=30
- 648: Multi-Center, Randomized, Double-Blind, Placebo-Controlled, Crossover in Enteric and Idiopathic Hyperoxaluria
- n=67
- 712: Multi-Center, Randomized, Double-Blind, Placebo-Controlled in Enteric and Idiopathic Hyperoxaluria
- Presented at ASN 2015

### Ph 3 Randomized Controlled
- n=124
- URIROX-1: Multi-Center, Global, Randomized, Double-Blind, Placebo-Controlled Study in Enteric Hyperoxaluria
- CT.gov: NCT03456830
- n=400
- Proposed URIROX-2: Multi-Center, Global, Randomized, Double-Blind, Placebo-Controlled Study in Enteric Hyperoxaluria

<table>
<thead>
<tr>
<th>Ph 3 Randomized Controlled</th>
<th>Initiated 1Q 2018 and 2H 2018</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓</td>
<td></td>
</tr>
</tbody>
</table>

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Phase 3 Program Incorporates Key Learnings from Phase 2

**Phase 2 Program**

- **Identified Proposed Phase 3 Patient Population**
  Enteric population demonstrated consistent, superior, clinically and statistically significant response

- **Identified Proposed Pivotal Endpoint**
  Percent change from baseline to average UOx excretion/24h: we believe most appropriate endpoint because it reflects benefit of metabolic control of UOx excretion over time

- **Identified Proposed Phase 3 Trial Design**
  Parallel, RCT vs placebo optimal design, dosing up to 5x per day per meal or snack

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**713 Results**

<table>
<thead>
<tr>
<th>Key Endpoints</th>
<th>Overall (n=67)</th>
<th>Enteric (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reloxaliase vs placebo (Δ)</td>
<td>p-value</td>
</tr>
<tr>
<td>Change in UOx (mg/24h) from baseline to Week 4</td>
<td>-6.35 mg/24h</td>
<td>0.160</td>
</tr>
<tr>
<td>Change in UOx (mg/24h) from baseline to TWA across 4 weeks†</td>
<td>-8.13 mg/24h</td>
<td>0.016</td>
</tr>
<tr>
<td>Percent change in UOx from baseline to TWA across 4 weeks†</td>
<td>-14.23%</td>
<td>0.015</td>
</tr>
</tbody>
</table>

†. Beyond the primary endpoint analysis, all P-values are descriptive.
Study 713: Substantially Greater Reloxaliase Treatment Response in Enteric Population

<table>
<thead>
<tr>
<th>Reduction in TWA UOx (%)</th>
<th>-10</th>
<th>-20</th>
<th>-30</th>
<th>-40</th>
<th>-50</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enteric Reloxaliase</td>
<td>73</td>
<td>64</td>
<td>36</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>Enteric Placebo</td>
<td>29</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Beyond the primary endpoint analysis, all P-values are descriptive.*
Reloxaliase Generally Well-Tolerated in Clinical Trials to Date

**Rare disease and demonstrated safety profile of reloxaliase lead to reduced requirement for Phase 3 trial size**

<table>
<thead>
<tr>
<th>Study 396</th>
<th>Study 649</th>
<th>Study 713</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>All (n=16)</strong></td>
<td><strong>Reloxaliase¹ (n=30)</strong></td>
<td><strong>Placebo (n=24)</strong></td>
</tr>
<tr>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>TEAE²</td>
<td>9 (56.3%)</td>
<td>13 (43.3%)</td>
</tr>
<tr>
<td>Severe TEAE</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Related TEAE</td>
<td>2 (12.5%)</td>
<td>5 (16.7%)</td>
</tr>
<tr>
<td>Serious AE (SAE)</td>
<td>0</td>
<td>1 (3.3%)³</td>
</tr>
<tr>
<td>Related SAEs</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>AEs Leading to Study Drug Withdrawal</td>
<td>0</td>
<td>1 (3.3%)³</td>
</tr>
<tr>
<td>AEs Leading to Death</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

1. 1,500 units, 3,000 units, and 7,500 units
2. TEAE = Treatment-emergent adverse events defined as AEs with onset at or following the first dose of treatment, with study drug through 7 days after the last dose of study medication, or AEs starting before the start of treatment but occurring in severity or relationship to the time of or following the start of treatment through 7 days after the last dose of study medication.
3. One subject-reported congestive heart failure of moderate severity, considered unrelated to study drug, but secondary to a recent coronary stent for atrial fibrillation. This resulted in hospitalization and withdrawal from the study. Same subject in both trials.
4. Two placebo-treated subjects withdrew from study drug due to nausea, considered not related, and another due to hives/dermatitis with onset 5 days after starting placebo, considered possibly related.

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Reloxaliase: Proposed Pivotal Phase 3 Program in Enteric Hyperoxaluria

<table>
<thead>
<tr>
<th>1H 2017</th>
<th>2H 2017</th>
<th>1H 2018</th>
<th>2H 2018</th>
<th>1H 2019</th>
<th>2H 2019</th>
<th>1H 2020</th>
<th>2H 2020</th>
<th>1H 2021</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</table>

URIROX-1 in Enteric HO
RCT x 4 weeks n=124

URIROX-2 in Enteric HO, RCT = 24 weeks n=400
Follow-up (in discussion) 2-3.5 years

Proposed Phase 3 Program – in Discussions with the FDA, URIROX-1 Enrolling

- **Patient Population:** EH subjects with severe Hyperoxaluria (>50mg/24h), normal to moderate CKD (eGFR ≥30)
- **URIROX-1:** Primary endpoint – UOx averaged during weeks 1-4
- **URIROX-2:** Primary endpoint – UOx averaged during weeks 1-4, 24 weeks plus follow-up
- **Pivotal Program:** ~500 patients
URIROX-1: Evaluate the Safety and Efficacy of Reloxaliase in Patients with Enteric Hyperoxaluria

**Study Aim:** Determine the Safety and Efficacy of Reloxaliase in Reducing UOx in Subjects with Enteric Hyperoxaluria

<table>
<thead>
<tr>
<th>Screening</th>
<th>Randomization</th>
<th>Follow up 4 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>UOx≥50mg/24h Normal to Stage 3 CKD (eGFR ≥30)</td>
<td>1:1</td>
<td>1X 24h 2X 24h 2X 24h 2X 24h 2X 24h</td>
</tr>
<tr>
<td>n=124</td>
<td>2EP: Proportion of subjects with a ≥20% reduction from baseline in 24-h UOx excretion averaged during weeks 1-4 vs placebo</td>
<td></td>
</tr>
</tbody>
</table>

**Key Features:**

- Tailored dose regimen: dose with each meal/snack 3-5x per day to maximize degradation of oxalate ingested or secreted
- **1st EP:** Percent change from baseline in 24-h UOx excretion averaged during weeks 1-4, with demonstration of a mean reduction in UOx with reloxaliase vs placebo
- **2nd EP:** Proportion of subjects with a ≥20% reduction from baseline in 24-h UOx excretion averaged during weeks 1-4

**Safety**
Regulatory Status Overview: Hyperoxaluria is a High Unmet Need with No Approved Therapeutic

<table>
<thead>
<tr>
<th>Opportunities</th>
<th>Regulatory Challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Enteric Hyperoxaluria is a rare disease with a high burden of kidney stones and kidney damage</td>
<td>• With no approved drugs:</td>
</tr>
<tr>
<td>• High unmet need due to inadequate existing therapies</td>
<td>- No established regulatory pathway</td>
</tr>
<tr>
<td></td>
<td>- Limited data sets correlating baseline UOx and the change in clinical outcomes</td>
</tr>
</tbody>
</table>

**General FDA Alignment:**

**URIROX-1 trial design:**
- Enteric hyperoxaluria is a rare disease without available treatment
- Four week study in 124 subjects with Enteric Hyperoxaluria
- Primary endpoint – Percent change from baseline in 24-h UOx averaged during weeks 1-4, with demonstration of a mean reduction in UOx with reluxalase vs placebo
- Secondary endpoint – Proportion of subjects with a ≥ 20% reduction in UOx averaged during weeks 1-4
- Dosing up to 5x a day with meals and snacks

**Points of Active Discussion:**

**URIROX-2 trial design,** including the use of data from this trial to validate both UOx as a surrogate marker endpoint and to confirm clinical benefit post-approval, consistent with FDA guidance on the accelerated approval pathway:
- Remain in active dialogue with FDA on the protocol and statistical plan
- Subject to final written confirmation, we believe we have achieved alignment on the pre-approval surrogate marker phase of the trial
- Received guidance on two remaining elements for the post-marketing outcome phase of the trial
Reloxaliase Additional Indications
Elevated Plasma Oxalate Increases Risk for CaOx Crystal Deposition in the Kidney and Other Organ Systems

**Study Population:**
- Enteric Hyperoxaluria
- Primary and Enteric Hyperoxaluria with Hyperoxalemia

**Systemic Oxalosis**

<table>
<thead>
<tr>
<th>CKD 1</th>
<th>CKD 2</th>
<th>CKD 3</th>
<th>CKD 4</th>
<th>ESRD</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>15</td>
<td>30</td>
<td>45</td>
<td>60</td>
</tr>
</tbody>
</table>

**Time and progression of disease**

- 0 Normal range 2-5 µmol/L
- 15 Oxalate crystals are associated with renal inflammation, fibrosis and progressive renal failure
- 30 Patients with hyperoxalemia or systemic oxalosis can accumulate oxalate in the blood and other tissues, creating "oxalate stores" in the body
- 45 Hyperoxalemia or systemic oxalosis can prohibit patients from getting a transplant or jeopardize the transplanted kidney
- 60 Growing awareness of association between oxalate crystal deposition and poor long-term graft survival, declining kidney function and return to dialysis

**Unmet Need: Reduce Risk of Oxalate Damage to the Kidney**

- Oxalate crystals are associated with renal inflammation, fibrosis and progressive renal failure
- Patients with hyperoxalemia or systemic oxalosis can accumulate oxalate in the blood and other tissues, creating "oxalate stores" in the body
- Hyperoxalemia or systemic oxalosis can prohibit patients from getting a transplant or jeopardize the transplanted kidney
- Growing awareness of association between oxalate crystal deposition and poor long-term graft survival, declining kidney function and return to dialysis

Reloxaliase has the Potential to Reduce Oxalate Burden in Patients with Primary Hyperoxaluria and Enteric Hyperoxaluria with Advanced CKD

Prevented Nephrocalcinosis and Increased Survival in the AGXT KO Mouse Model of Primary Hyperoxaluria

<table>
<thead>
<tr>
<th>Nephrocalcinosis (% of animals)</th>
<th>Control</th>
<th>25 mg</th>
<th>80 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100%</td>
<td>63%</td>
<td>0%</td>
</tr>
</tbody>
</table>

In porcine model, reloxaliase reduced plasma 30% and urinary oxalate 35% after 7 days of treatment with reloxaliase. Oral therapy of 22,500 u/day normalized both plasma and urine oxalate.*

Degraded Oxalate in the GI Tract and Reduced Plasma Oxalate in Porcine Model of Secondary Hyperoxaluria

<table>
<thead>
<tr>
<th>Urine Oxalate (mg/gCr/24h)</th>
<th>Plasma Oxalate (µmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High oxalate diet</td>
<td>10</td>
</tr>
<tr>
<td>High oxalate diet + ALLN-177</td>
<td>8</td>
</tr>
</tbody>
</table>

*As not colonized with oxalobacter formigenes.

Sources: Grujic et al, Am J Nephrol 2009; 29:86-93 and company data
Study 206: Reloxaliase Treatment of Adult and Pediatric Patients with Primary or Enteric Hyperoxaluria and Advanced CKD ('Basket' Study)

**Study Aim:** Evaluate effect of Reloxaliase in reducing plasma and UOx in patients with primary or enteric hyperoxaluria and hyperoxalemia.

<table>
<thead>
<tr>
<th>Screening and Baseline 28 d</th>
<th>Treatment x 12 Weeks</th>
<th>Follow up 4 Weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n ≥ up to 20</strong></td>
<td><strong>ALLN 2 caps per meal/snack up to 5 x/day [max 10/d]</strong></td>
<td><strong>Week 4</strong></td>
</tr>
<tr>
<td>≥12 yrs PH or EH</td>
<td></td>
<td>1 x POx</td>
</tr>
<tr>
<td>UOx ≥ 40mg/24hr</td>
<td>2 x POx</td>
<td>2 x 24h</td>
</tr>
<tr>
<td><strong>≥ POx &gt; 5 µmol/L</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>eGFR &lt; 45 mL/minute/1.73 m²</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>** Required for EH**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- **Study Initiation:** 1Q18 (CT.GOV: NCT03391804)

- **Hypothesis:**
  - By degrading oxalate within the GI tract, reloxaliase is designed to reduce plasma and urinary oxalate levels in patients with PH or EH and hyperoxalemia. Reduction in plasma oxalate levels (POx) should lead to decreased systemic oxalate deposition (systemic oxalosis).

- **Study Design:**
  - Uncontrolled, Open-label
  - Enroll subjects ≥12 yrs, body weight ≥ 35 kg, in PH or EH with hyperoxalemia
  - Dose regimen as 5 x/day to maximize the degradation of oxalate ingested or secreted in the GI tract
  - Key Endpoints: Change from baseline in POx and UOx

- Regulatory: Reloxaliase has been granted separate orphan designations for primary hyperoxaluria and pediatric hyperoxaluria

**Allena Pharmaceuticals**

- Primary Hyperoxaluria (PH)
- Enteric Hyperoxaluria (EH)
- Urinary Oxalate (UOx)
- Plasma Oxalate (POx)
ALLN-346: Significant Opportunity in Gout Patients with Moderate-to-Severe CKD

The Gout Market is Incompletely Served by Existing Therapies

- ~375,000 gout patients with moderate to severe CKD who have uncontrolled gout on urate lowering therapy (ULT)
- Gout patients with renal impairment are not well managed due to safety concerns for existing therapies
  - Gout patients with kidney and liver problems are contraindicated for allopurinol, Uloric, and Zurampic
  - Current ULT’s may interact with other medications
  - Co-morbidities (e.g. cardiovascular) may also limit ULT options
- Significant unmet need for safe and effective therapy that can be used in patients with renal impairment

ALLN-346 Therapeutic Strategy:

- Novel urate degrading enzyme optimized for stability in the GI tract
- MOA: orally administered, gut restricted enzyme therapeutic
- Animal POC: demonstrated a robust reduction in urine and plasma uric acid levels in a severe animal model of hyperuricemia with advanced CKD
  - Accepted for poster presentation at American College of Rheumatology meeting October 22, 2018

*Abstract number: 1299.

## Clinical and Regulatory Milestones

<table>
<thead>
<tr>
<th>YEAR</th>
<th>TARGET</th>
<th>MILESTONE</th>
<th>STATUS (October 2018)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2017</td>
<td>4Q17</td>
<td>Initiate ALLN-346 Animal Studies</td>
<td>✔</td>
</tr>
<tr>
<td>2018</td>
<td>1Q18</td>
<td>Initiate Study 301</td>
<td>✔</td>
</tr>
<tr>
<td></td>
<td>1Q18</td>
<td>Initiate Study 206, Phase 2 Study in PH and EH with Hyperoxalemia</td>
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<tr>
<td></td>
<td>2H18</td>
<td>Initiate Study 302</td>
<td>On Track</td>
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<tr>
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<td>2H18</td>
<td>Study 206 Interim Data</td>
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</tr>
<tr>
<td>2019</td>
<td>1H19</td>
<td>File IND ALLN-346</td>
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<tr>
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<td>1H19</td>
<td>Study 206 Additional Interim Data</td>
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<td>2H19</td>
<td>Study 301 Topline Data</td>
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<tr>
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<td>Study 206 Topline Data</td>
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<tr>
<td>2020</td>
<td>2H20</td>
<td>Phase 3 Results</td>
<td>On Track</td>
</tr>
</tbody>
</table>
Investment Highlights

Significant Unmet Need in Oxalate and Urate Disorders
- Focused on rare and severe metabolic disorders that can cause kidney stones, damage the kidney, and potentially lead to CKD and ESRD
- No approved oxalate therapies; potential untapped multi-billion dollar market

Late-Stage Development Candidate: Reloxaliase
- First-in-class, oral therapy for severe hyperoxaluria
- Enrolling Phase 3 study, URIROX-1, in enteric hyperoxaluria, topline data expected 2H 2019
- Enrolling Phase 2 basket study in other severe and orphan indications

Pioneering Expertise in Oral Enzyme Therapeutics
- Approach enables treatment of metabolic diseases with oral, non-absorbed enzyme therapeutics
- GI MOA reduces subsequent metabolic burden on the kidney

Strong Support from Leading Biotechnology Investors
- Raised $75M in successful initial public offering in 4Q 2017