Jefferies Virtual Healthcare Conference

June 2020

Forward Looking Statements

This presentation contains forward-looking statements that are based on the Company's beliefs and assumptions and on information currently available to management. All statements contained in this presentation other than statements of historical fact are forward-looking statements, including but not limited to statements regarding the potential benefits of the merger, the use of proceeds of the private placement, Company's ability to develop and commercialize CTI-1601 and other planned product candidates, Company's planned research and development efforts, and other matters regarding Company's business strategies, use of capital, results of operations and financial position, and plans and objectives for future operations.

In some cases, you can identify forward-looking statements by the words "may," "will," "could," "would," "should," "expect," "intend," "plan," "anticipate," "believe," "estimate," "predict," "project," "potential," "continue," "ongoing" or the negative of these terms or other comparable terminology, although not all forward-looking statements contain these words. These statements involve risks, uncertainties and other factors that may cause actual results, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. These risks, uncertainties and other factors include, among others, the Company's ability to achieve the anticipated benefits of the merger; the success, cost and timing of the Company's product development activities, studies and clinical trials; the ongoing impact of the COVID-19 pandemic on the Company's clinical trial timelines, ability to raise additional capital and general economic conditions; the Company's ability to optimize and scale CTI-1601's manufacturing process; the Company's ability to obtain regulatory approval for CTI-1601 and future product candidates; the Company's ability to develop sales and marketing capabilities, whether alone or with potential future collaborators, and successfully commercialize any approved product candidates; the Company's ability to raise the necessary capital to conduct its product development activities; and other risks described in the filings made by Zafgen with the Securities and Exchange Commission (SEC), including but not limited to the Definitive Proxy Statement relating to the merger filed on April 29, 2020, and the Company's subsequent periodic reports, including the annual report on Form 10-K, quarterly reports on Form 10-Q and current reports on Form 8-K, filed with or furnished to the Securities and Exchange Commission and available at www.sec.gov. These forward-looking statements are based on a combination of facts and factors currently known by the Company and its projections of the future, about which it cannot be certain. As a result, the forward-looking statements may not prove to be accurate. These forward-looking statements are based on information currently available to us, and we assume no obligation to update any forward-looking statements, except as required by law.

Larimar Therapeutics

Larimar Therapeutics Introduction

- **Created by merger of Zafgen and Chondrial Therapeutics**
- **Traded under ticker "LRMR"**
- **Merger accompanied by $80 million private placement – World class life science investor shareholder base**
  - Financing led by Cowen Healthcare Investments with Acuta, Janus, Logos, OrbiMed, RA Capital and Vivo
  - Continued shareholder support of Deerfield Management and Atlas Ventures
  - Company has ~ $116 million in cash from merger and financing
- **Leadership team bolstered**
  - Joseph Truitt – Board chair
  - Nancy Ruiz, MD, FACP, FIDSA – CMO
  - Michael Celano - CFO

Chondrial Therapeutics, Inc.

Zafgen™

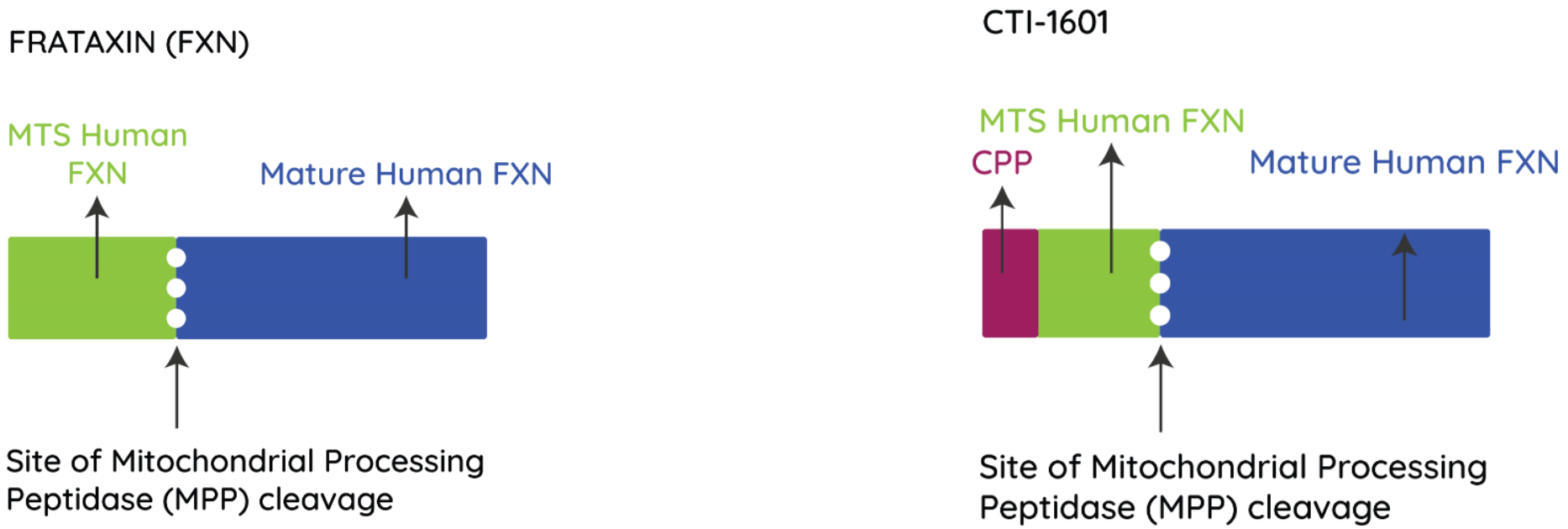
Investment Highlights

- Clinical-stage biotech with a **novel protein replacement therapy platform** to address untreated, serious and complex rare diseases
- Lead candidate, **CTI-1601**, in **Phase 1 clinical development** for treatment of **Friedreich's ataxia** (FA)
  - **We believe frataxin (FXN) protein replacement therapy** is the only protein replacement therapy in clinical development
  - Nonclinical studies have shown **promising results in several models of FA**, including heart, brain and muscle function, and overall survival
  - Multiple FDA designations: **Orphan Drug, Rare Pediatric Disease (Voucher), Fast Track**
  - **Topline Phase 1 data expected in 1H 2021**
- **Experienced** leadership team
- **Extensive IP** with 12 years market exclusivity expected if approved; patents pending around efficacy biomarkers
- **Strong balance sheet** with ~ $116M in cash as of merger close; based on current estimates of funding needs, cash expected to last for ~ 2 years into first half of 2022

Friedreich's ataxia (FA): Rare and Progressive Disease

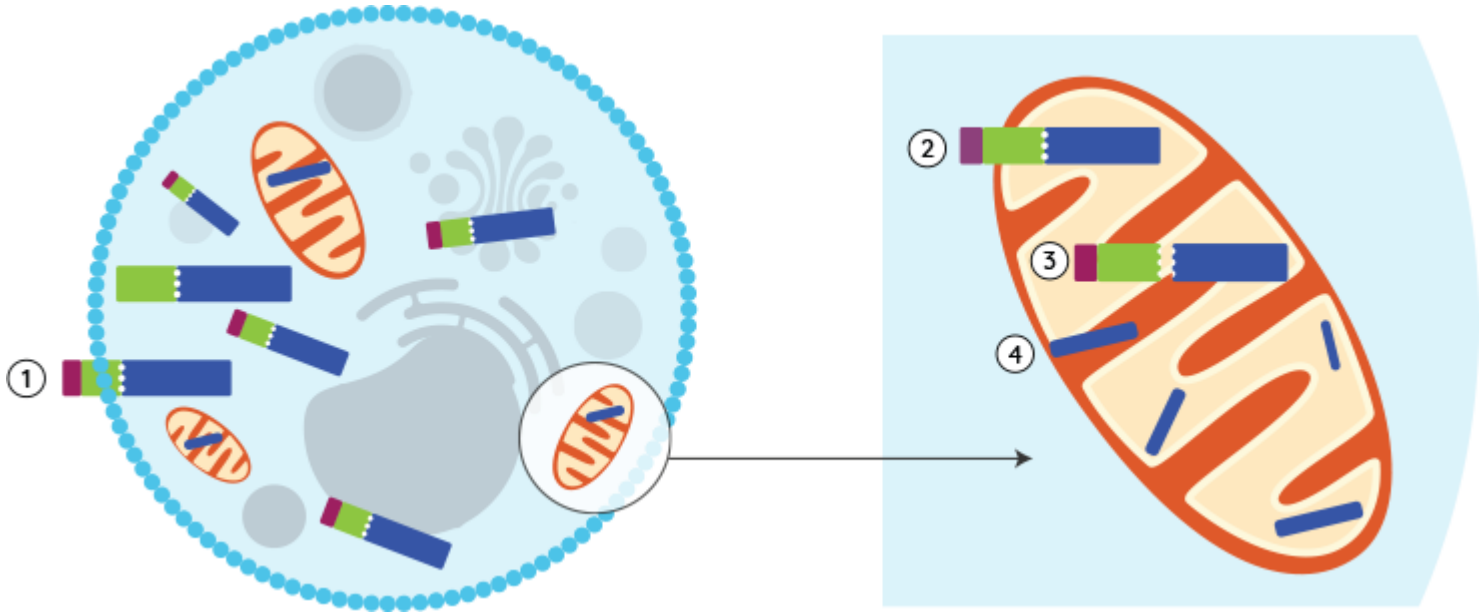
- **Rare disease** caused by genetic defect resulting in **abnormally low levels of frataxin (FXN)**
  - Affects ~5,000 patients in U.S. and ~20,000 patients in EU
- Disease affects **multiple body systems**, particularly the brain and heart
- **Onset:** Age of **onset correlated with severity and speed of progression** (earlier onset correlated with more drastic progression)
  - >70% of patients **present before age 14**
  - Initial symptoms may include **unsteady posture, frequent falling and progressive difficulty in walking**
  - By the time symptoms occur, **heart damage has already occurred**
- **Progression of disease:** Symptoms worsen and patients are eventually confined to wheelchair
- **Life expectancy of 30-50 years**, early death usually caused by **heart disease**
- **Treatment limited to symptom management; currently no approved therapies**

CTI-1601 – Designed to Deliver Frataxin (FXN)



CTI-1601 similar to frataxin except it has a CPP attached on end to allow it to move into cell and mitochondria

CTI-1601 – Delivering Frataxin to the Mitochondria



1. CTI-1601 crosses the cell membrane into cytoplasm
2. CTI-1601 crosses the mitochondrial membrane
3. Mitochondrial Processing Peptide (MPP) cleaves CTI-1601. MTS and CPP will leave cell mitochondria
4. Mature human frataxin remains within the mitochondria to function

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CTI-1601 – POC Achieved Through Multiple Non-Clinical Studies

Nonclinical efficacy and PD data provides proof of concept in support of continued development to potentially replace FXN in patients with FA

- ✓ **Extended survival** in a well-characterized nonclinical model of FA
- ✓ **Prevented ataxic gait** in another nonclinical model of FA
- ✓ Demonstrated **capability of delivering sufficient amounts of FXN** to mitochondria
- ✓ Prevented left **ventricle dilation and** maintained **function**
- ✓ **Safe and well tolerated** in multiple species

CTI-1601 Extends Survival in FXN-deficient KO Mice

Initial Proof of Concept for FXN Replacement Therapy in FA

TAT-FXN was administered 10 mg/kg SC every other day

✓CTI-1601 extended survival in a well-characterized cardiac mouse model of FRDA

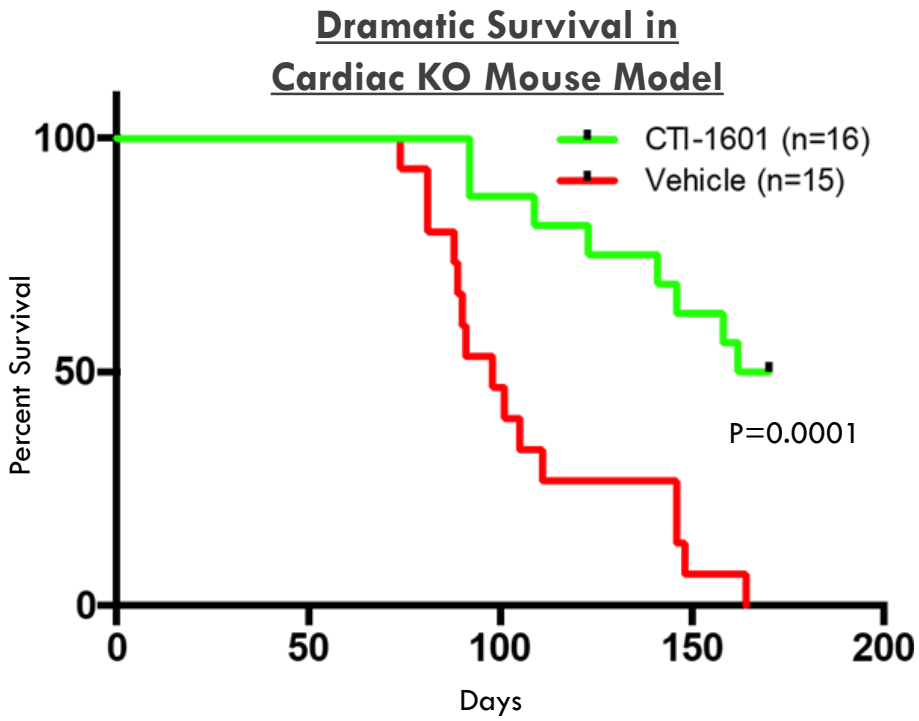
Median Survival of MCK-Cre FXN-KO Mice

- 166 days (CTI-1601) vs 98.0 days (Vehicle)

Survival beyond Vehicle mean (107.5 days)

- 87.5% (CTI-1601) vs. 33% (Vehicle)

✓Confirms that CTI-1601 is capable of delivering sufficient amounts of FXN to mitochondria, rescuing a severe disease phenotype



MCK = Muscle creatine kinase

CTI-1601 Prevents Neurological Phenotype and Ataxic Gait in KO mice

In-Vivo Efficacy Data in Neurologic KO Mouse Model

Pvalb-Cre FXN-KO mouse

- Single dose level: 10 mg/kg CTI-1601 or Vehicle given intraperitoneally (IP) three times per week

✓ hFXN replacement with CTI-1601 prevents development of ataxic gait

✓ Treated mice survive longer than untreated mice

✓ Human frataxin present in brain, dorsal root ganglia and spinal cord

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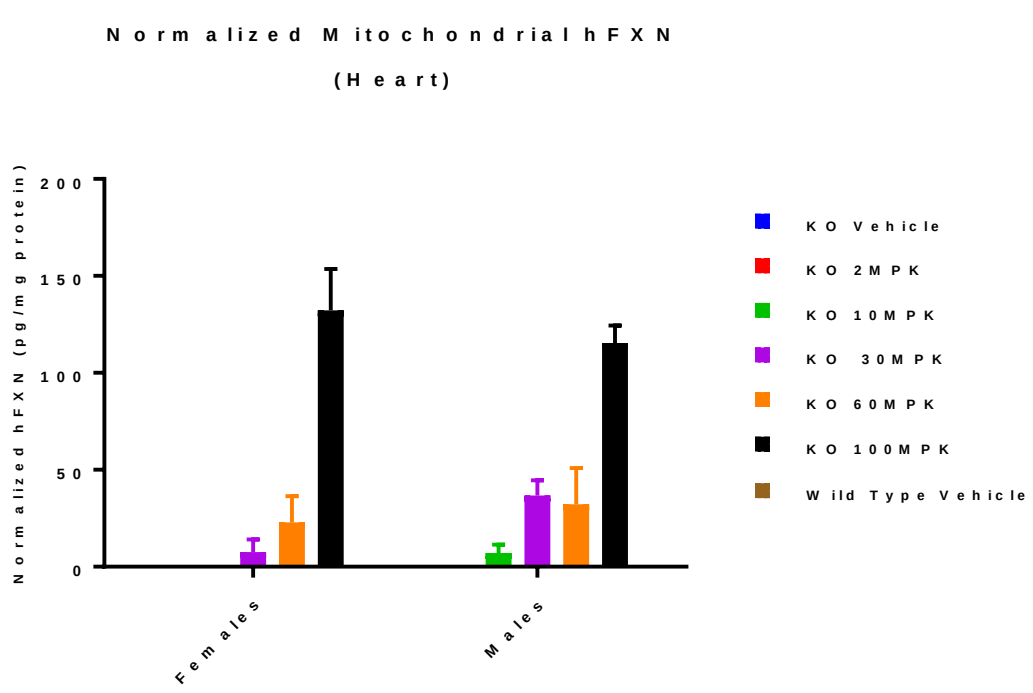
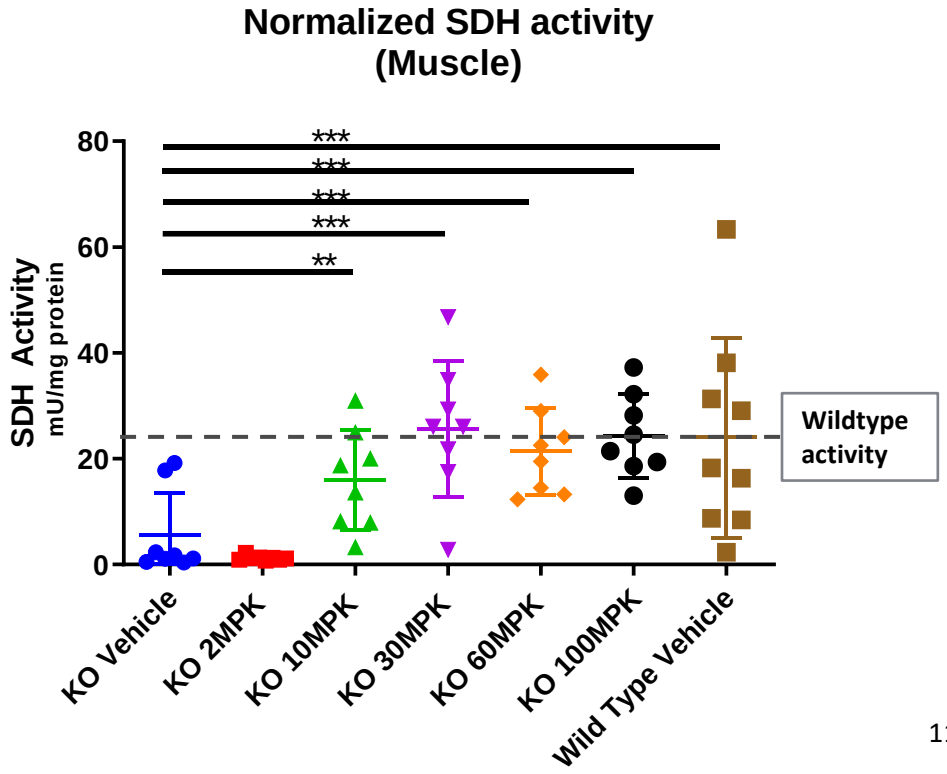
CTI-1601 Effectively Traffics to Mitochondria; Delivers hFXN

- ✓ hFXN concentration within mitochondria increases in a dose-dependent manner
- ✓ Given subcutaneously, CTI-1601 functionally replaces hFXN in mitochondria of KO mice
- ✓ SDH* activity increases in a dose-dependent manner after administration of CTI-1601; activity plateaus at 30 mg/kg and is equivalent to activity in wildtype animals
- ✓ Demonstrated normalization of gene expression in cardiac tissue

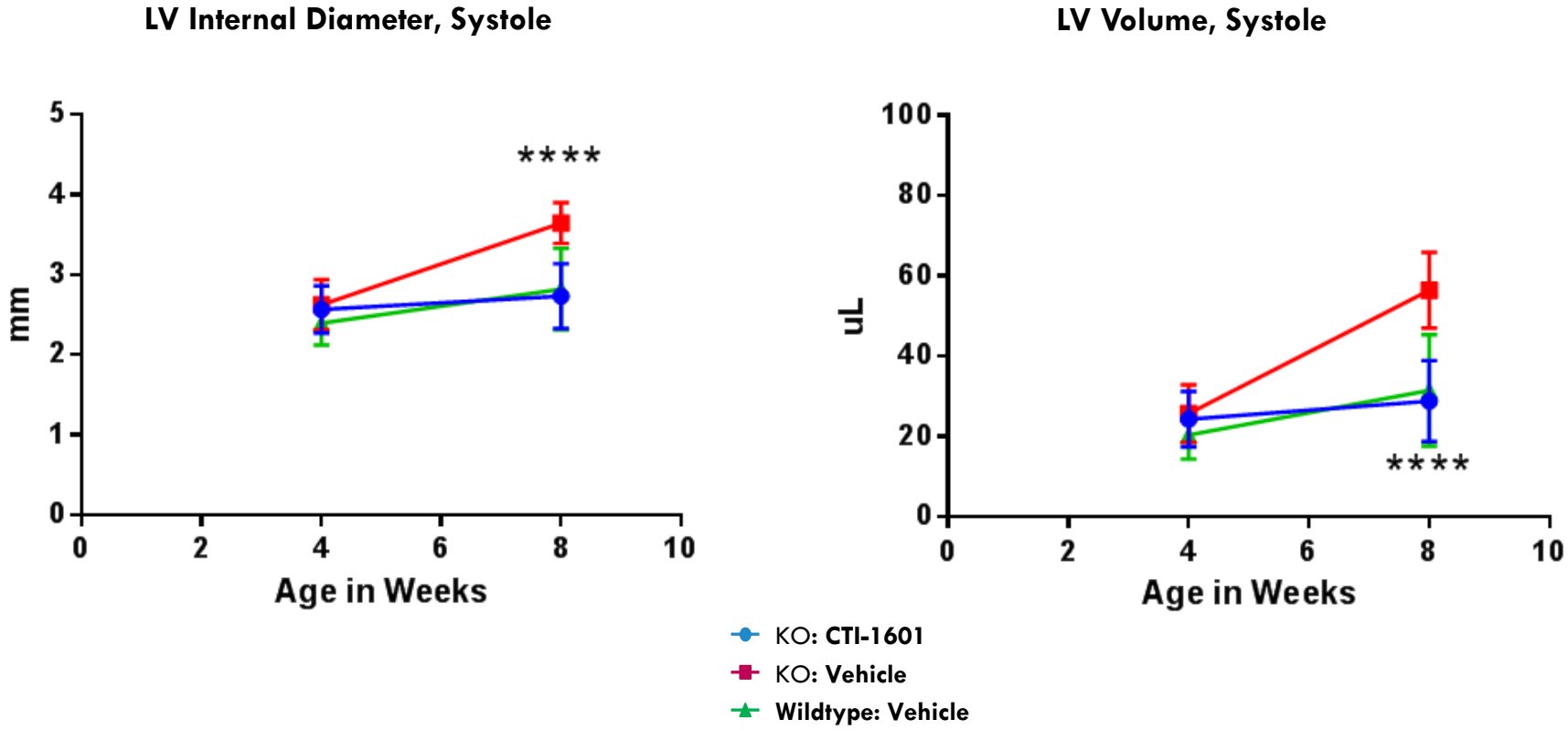




*Succinate dehydrogenase

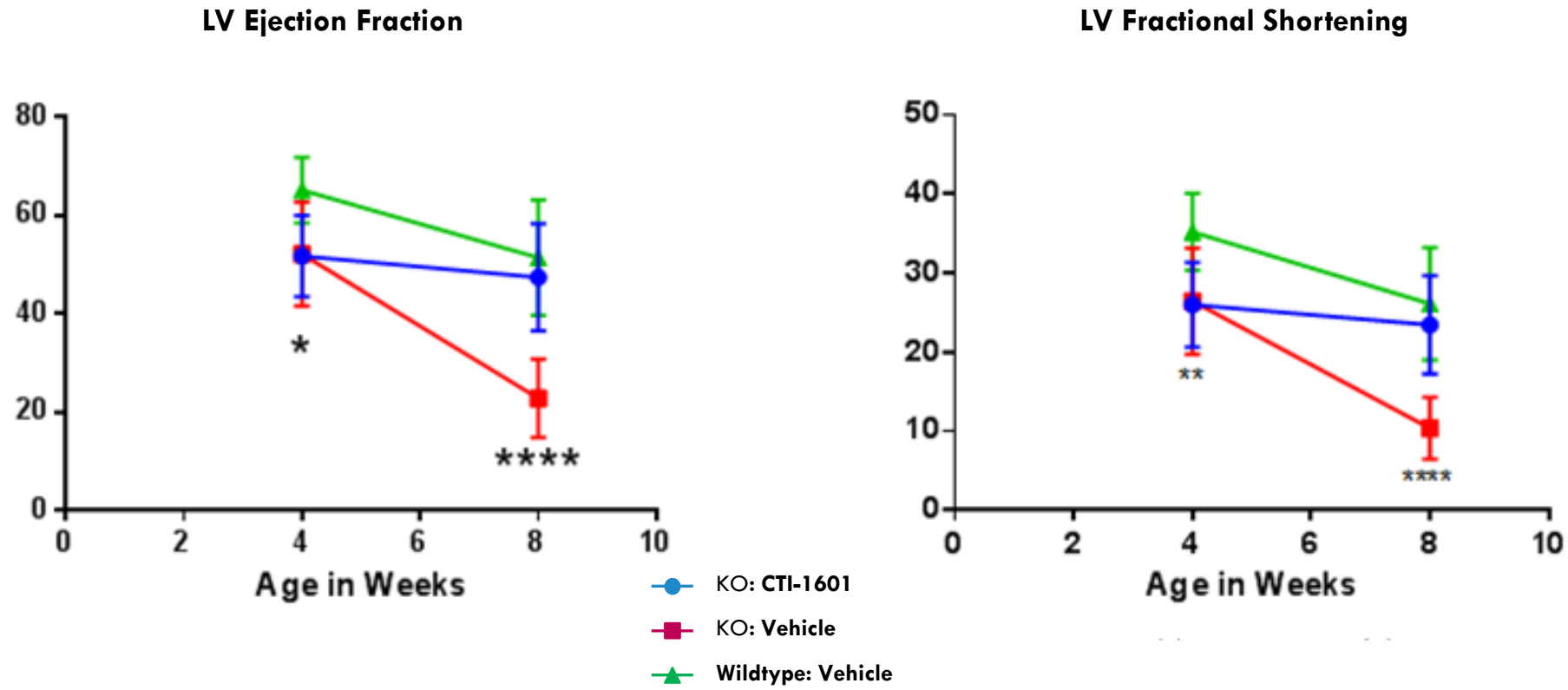
CTI-1601 Prevents Left Ventricle (LV) *Dilation*

- ✓ Left ventricular volume increases in systole in untreated mice by 8 weeks (after 4 weeks of dosing with vehicle), but remains similar to wildtype when treated with CTI-1601
- ✓ **CTI-1601-treated mice similar to controls**; echocardiogram shows significant differences between KO:Vehicle and KO:CTI-1601 treated mice



CTI-1601 Preserves Left Ventricle (LV) *Function*

- ✓ LV function drops significantly in untreated mice by week 8
- ✓ **CTI-1601-treated mice similar to controls**; echocardiogram again shows significant differences between KO:Vehicle and KO:CTI-1601 treated mice



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Human Frataxin Distributed Into All Tissues Tested

Observed hFXN across all tissue and cell types tested:

- ✓ Brain
- ✓ Heart
- ✓ Liver
- ✓ Dorsal Root Ganglia
- ✓ Spinal Cord
- ✓ Cardiac Mitochondria
- ✓ CSF (Cerebrospinal Fluid)
- ✓ Skeletal Muscle
- ✓ Skin
- ✓ Buccal Cells
- ✓ Platelets

Tissues Examined, By Study	
Study Vehicle	**Human Frataxin Distribution**
Rats	Brain, Heart, Liver
Neuro KO Mice	Brain, Dorsal Root Ganglia, Spinal Cord
Cardiac KO Mice	Mitochondria of Skeletal Muscle and Cardiomyocytes
Cynomolgus Monkey	CSF, Skin, Buccal Cells, Platelets

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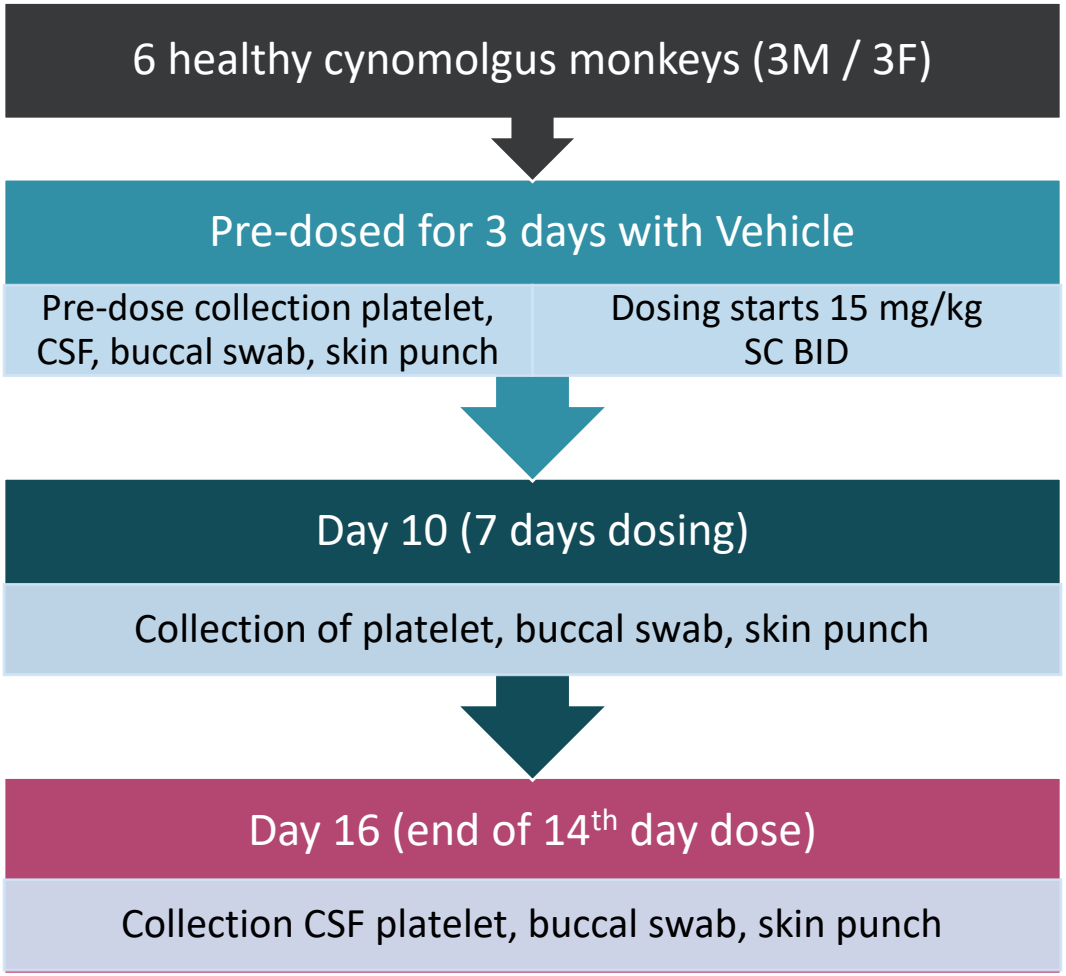
PK/PD Study in Healthy Cynomolgus Monkeys

Study Design



- ✓ CTI-1601 is **bioavailable** when given subcutaneously
- ✓ **Sustained levels of processed hFXN** are found after **14 days** in the cerebrospinal fluid of monkeys, suggesting **CNS penetration**
- ✓ **Sustained levels of hFXN** are found in **blood cells (platelets) and peripheral tissues** (buccal cells, skin) as **early as the 7th** day and still present **after 14 days**
- ✓ **Preliminary results from the 90 Day GLP Toxicology support these findings**

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CTI-1601: Safe and Well Tolerated in Multiple Animal Models

Cynomolgus Monkey

Injection Site Observations

- Some injection sites raised and firm; increased injection site pathology at higher doses most likely due to local irritation

Systemic Toxicity Analysis

- No other clinical observations or treatment-related changes in food consumption, body weight or organ weight
- No systemic histopathological findings

Sprague Dawley Rat

Injection Site Observations

- Some injection sites showed irritation, firmness, inflammation at higher doses

Systemic Toxicity Analysis

- No significant clinical observations or clinical pathology results
- No systemic histopathological findings

No systemic clinical or pathological observations related to CTI-1601 in early GLP studies

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CTI-1601: Ongoing Phase 1 Clinical Program in FA Patients

Double-blind, placebo-controlled SAD/MAD clinical trials in FRDA patients 18-years-old or older

- Dosing regimen: Single Ascending Doses given SC (SAD); Multiple Ascending Doses given SC (MAD)
- Number of subjects:
  - SAD: approximately 32-34 subjects (currently 4 cohorts planned)
  - MAD: 24-30 subjects (currently 3 cohorts planned)
- Outcome measures:
  - Primary: Safety and tolerability
  - Secondary: PK; PD (hFXN, gene expression in buccal swab and blood); multiple exploratory
- Patient dosing began December 2019
  - Two cohorts have completed SAD Phase 1 – all data collected successfully
  - 3rd cohort delayed given COVID-19; company actively working to initiate
- Sufficient drug supply for Phase 1 clinical program (5 GMP batches)
- **Topline results from Phase 1 clinical program expected in 1H 2021**

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Marketing Exclusivity and Accelerated Programs

FDA has granted CTI-1601:

- ✓ Orphan Drug Status
  - Granted July 2017
  - Eligible for 7 years market exclusivity upon approval
- ✓ Fast Track Designation
  - Granted November 2019
  - Actions to expedite development and review
- ✓ Rare Pediatric Disease Designation
  - Granted December 2019
  - Eligible for voucher upon BLA approval

Company recently applied for European Orphan Drug designation

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Strong Relationship with FARA

- Company has strong relationship with Friedreich's Ataxia Research Alliance (FARA)
  - National, non-profit organization dedicated to the pursuit of scientific research leading to treatments and a cure for FA
- FARA provides industry with several key items
  - Assists with patient recruitment and education
  - Access to Global Patient Registry with demographic and clinical information on more than 1,000 FA patients
  - Sponsored a Patient-Focused Drug Development Meeting in 2017 resulting in a publication called The Voice of the Patient

FARA Friedreich's Ataxia Research Alliance

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Summary

- Clinical-stage biotech with a **novel protein replacement therapy platform**
- **CTI-1601** in **Phase 1 clinical development** for treatment of **Friedreich's ataxia** (FA)
  - **We believe frataxin (FXN) protein replacement therapy is the only protein replacement therapy currently in clinical development**
  - Nonclinical studies have shown **promising results in several models of FA**
  - Multiple FDA designations: **Orphan Drug, Rare Pediatric Disease (Voucher), Fast Track**
  - **Topline Phase 1 data expected in 1H 2021**
- **Experienced** leadership team
- **Extensive IP**
- **Strong balance sheet** of ~ $116M in cash from merger and financing*

*as of 6/1/2020, 15,319,075 shares of common stock are outstanding, and 628,403 pre-funded warrants to purchase common stock are outstanding

Larimar Therapeutics

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