UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): February 12, 2024

Larimar Therapeutics, Inc.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-36510**	**20-3857670**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
Three Bala Plaza East **Bala Cynwyd, Pennsylvania**		**19004**
(Address of Principal Executive Offices)		**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (844) 511-9056

(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))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.001 per share	lrmr	Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

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 2.02 Results of Operations and Financial Condition.

On February 12, 2024, Larimar Therapeutics, Inc. (the "***Company***") announced that, as of December 31, 2023, the Company had approximately $86.8 million of cash, cash equivalents and marketable securities. This unaudited, preliminary amount has been prepared by and is the responsibility of management. This amount is based upon information available to management as of the date of this Current Report on Form 8-K and subject to completion of financial closing procedures that could result in changes to the amount. Furthermore, this amount does not present all information necessary for an understanding of the Company's financial condition as of December 31, 2023. The Company's independent registered public accounting firm, PricewaterhouseCoopers LLP, has not audited, reviewed, compiled or performed any procedures with respect to this preliminary financial data and, accordingly, PricewaterhouseCoopers LLP does not express an opinion or any other form of assurance with respect thereto. The Company's actual results for the year ended December 31, 2023 will be included in the Company's Annual Report on Form 10-K for the year ended December 31, 2023 and may differ materially from the above estimate.

The information furnished pursuant to this Item 2.02 is intended to be furnished and shall not be deemed "filed" for 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, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

Press Release

On February 12, 2024, the Company issued a press release announcing top-line data and successful completion of its four-week, placebo-controlled Phase 2 dose exploration study of nomlabofusp (CTI-1601) in participants with Friedreich's ataxia. A copy of the press release is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

Investor Presentations

On February 12, 2024, the Company posted on its website two slide presentations, which are attached as Exhibit 99.2 and 99.3 to this Current Report on Form 8-K and are incorporated herein by reference. Representatives of the Company will use these presentations in various meetings with investors, analysts and other parties from time to time.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Below is a list of exhibits included with this Current Report on Form 8-K.

Exhibit No.	Document
99.1	Press Release issued by Larimar Therapeutics, Inc. on February 12, 2024*
99.2	Larimar Therapeutics, Inc. Corporate Presentation, dated February 12, 2024*
99.3	Larimar Therapeutics, Inc. Program Update Presentation, dated February 12, 2024*
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

* Filed herewith

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.

Larimar Therapeutics, Inc.

Date: February 12, 2024

By: /s/ Carole S. Ben-Maimon, M.D.

Name: Carole S. Ben-Maimon, M.D.
Title: President and Chief Executive Officer

Larimar Therapeutics Reports Positive Top-line Data from Phase 2 Dose Exploration Study from 25 mg and 50 mg Cohorts of Nomlabofusp in Patients with Friedreich's Ataxia

- *Nomlabofusp was generally well tolerated following repeated subcutaneous injections in patients in the 25 and 50 mg cohorts with no serious adverse events*
- *Dose dependent increases in frataxin levels were observed in skin and buccal cells*
- *Open Label Extension (OLE) study initiated in January 2024 to dose 25 mg daily of nomlabofusp with dosing anticipated to be starting later this quarter; Initial data expected in Q4 2024*
- *Initiated discussions with the FDA on use of tissue frataxin levels as a novel surrogate endpoint to support a potential Biologics License Application ("BLA") submission for accelerated approval targeted for 2H 2025*
- *Company management to host webcast and conference call today at 8:00 a.m. ET*

Bala Cynwyd, PA, February 12, 2024 – Larimar Therapeutics, Inc. ("Larimar") (Nasdaq: LRMR), a clinical-stage biotechnology company focused on developing treatments for complex rare diseases, today announced positive top-line data and successful completion of its four-week, placebo-controlled Phase 2 dose exploration study of nomlabofusp (CTI-1601) in participants with Friedreich's ataxia (FA). Nomlabofusp was generally well tolerated and demonstrated dose dependent increases in frataxin (FXN) levels in all evaluated tissues (skin and buccal cells) after daily dosing of 14 days followed by every other day dosing until day 28 in the 25 mg and 50 mg cohorts. Participants in the 25 mg (n=13) and 50 mg (n=15) cohorts were randomized 2:1 to receive subcutaneous injections of nomlabofusp or placebo.

"We believe the dose-response and increases in FXN levels seen in peripheral tissues further reinforce the therapeutic potential of nomlabofusp to address FXN deficiency, the known root cause of disease in patients with FA," said Carole Ben-Maimon, MD, President, and Chief Executive Officer of Larimar. "Importantly, the patients treated with 50 mg of nomlabofusp presented with individual baseline FXN skin levels less than 17% of the average level found in healthy volunteers, but after 14 days of daily treatment all patients with quantifiable levels at baseline and day 14 achieved FXN levels in skin cells greater than 33% of the average level found in healthy volunteers, and three of the patients achieved levels greater than 50% of average healthy volunteer level. Together with the consistent results seen across our Phase 2 and Phase 1 studies, we believe these findings suggest that nomlabofusp can achieve tissue FXN levels that may have a clinically meaningful effect on disease progression in patients with FA."

Dr. Ben-Maimon continued, "Recently, we had discussions with the FDA regarding the use of tissue FXN levels as a novel surrogate endpoint. The FDA has acknowledged that frataxin deficiency appears to be critical to the pathogenic mechanism of FA, and that there continues to be an unmet need for treatments for FA patients that address the underlying disease pathophysiology. We intend to pursue an accelerated approval using FXN levels, supportive pharmacodynamics and clinical information, and safety data from the OLE study, along with additional nonclinical pharmacology information needed to support the novel surrogate endpoint approach. We are beginning to plan for a confirmatory study and are targeting a BLA submission in the second half of 2025."

Dr. Russell Clayton, Chief Medical Officer of Larimar added, "These promising Phase 2 dose exploration data further expand our nomlabofusp safety database and strengthen clinical support for the generally well tolerated profile and low discontinuation rates seen across studies. In January, we initiated the OLE study that will start with a 25 mg daily dose, and the first subjects will begin self-administration later this quarter. The OLE study will inform on the long-term safety and self-administration of nomlabofusp following daily subcutaneous administration. Further dose expansion in the OLE will be considered based on safety, pharmacokinetics, and tissue FXN levels from the 25 mg dose of nomlabofusp. We expect to provide interim data from the OLE study in the fourth quarter of 2024."

Key Phase 2 Results

- Median **changes in FXN levels** from baseline for the 25 mg and 50 mg cohorts of nomlabofusp
  - Skin cells: 2.81 pg/µg for the 25 mg cohort and 5.57 pg/µg for the 50 mg cohort
  - Buccal cells: 0.56 pg/µg for the 25 mg cohort and 0.72 pg/µg for the 50 mg cohort
- As seen in our multiple ascending dose (MAD) study, when dosing is switched to every other day, FXN levels decline from the levels achieved with daily dosing but remain above baseline.
- All treated patients demonstrated increases in FXN levels in skin cells and the majority of patients also demonstrated increases in FXN levels in buccal cells.
- At Day 14, all patients with quantifiable levels at baseline and Day 14 treated with 50 mg of nomlabofusp achieved FXN levels in skin cells greater than 33% of the average level found in healthy volunteers, and 3 of the patients achieved levels greater than 50% of the average healthy volunteer level.
- While FXN levels measured in buccal cells show a high degree of correlation with FXN levels measured in skin cells, higher variability in FXN levels was seen in buccal cells compared to skin cells in both the multiple ascending dose study and the Phase 2 dose exploration study. Skin cells have a lower turnover rate and skin is a more stable tissue. The collection method for skin cells is also well-established and standardized, which provides a more reliable and reproducible measure of changes in FXN levels with treatment compared to buccal cells.

Median baseline tissue FXN levels in skin cells were 3.70 pg/µg and 2.12 pg/µg for the 25 mg and 50 mg cohorts, respectively, and in buccal cells were 1.78 pg/µg and 1.61 pg/µg for the 25 mg and 50 mg cohorts, respectively. Changes in FXN levels after nomlabofusp administration in the Phase 2 trial at day 14 (QD: once daily for 14 days) and day 28 (QOD: every other day after day 14) are:

Median Change from Baseline in FXN Levels (pg FXN/ µg total protein) *(25th, 75th percentile)*			
	Placebo *N= 9*	**25 mg Nomlabofusp** *N= 9*	**50 mg Nomlabofusp** *N = 10*
Skin Biopsies*			
Day 14 QD	**-0.53** *(-0.96, 0.57)*	**2.81** *(2.16, 3.32)*	**5.57** *(4.25, 6.55)*
Day 28 QOD	**-0.34** *(-0.74, 0.31)*	**2.28** *(-0.03, 2.71)*	**3.14** *(2.50, 3.64)*
Buccal Cells**			
Day 14 QD	**-0.35** *(-0.75, 0.04)*	**0.56** *(-0.28, 0.64)*	**0.72** *(0.05, 1.06)*
Day 28 QOD	**-0.52** *(-1.07, 0.01)*	**0.03** *(-0.66, 0.86)*	**0.48** *(-0.16, 0.76)*

*Subjects *who had one or more FXN measurements below quantifiable levels are excluded from the above analysis.*
For the placebo group, one participant had skin cell FXN levels below quantifiable levels on day 14 and day 28. For the 25 mg group, Day 14 and 28 skin biopsies were not collected from one nomlabofusp treated participant who discontinued treatment and one nomlabofusp treated participant had FXN levels below quantifiable levels at day 14 and day 28. For the 50 mg group, one participant had skin cell FXN levels below quantifiable levels at baseline.

**Subjects *who had one or more FXN measurements below quantifiable levels are excluded from the above analysis*. For the placebo group, one participant had buccal cell FXN levels below quantifiable levels at baseline and four participants had buccal cell FXN levels below quantifiable levels at day 14 and day 28. For 25 mg group, day 28 buccal FXN were not collected from one participant who discontinued treatment and two participants had buccal FXN levels below quantifiable levels at baseline. For the 50 mg, one participant had buccal cell FXN levels below quantifiable levels at baseline, day 14 and day 28, and two participants had buccal FXN levels below quantifiable levels at day 14 and day 28.

Key Phase 2 Pharmacokinetic and Safety Data for 25 mg and 50 mg Cohorts of Nomlabofusp

- Quick absorption after subcutaneous administration of nomlabofusp with dose proportional increases in exposure were observed with increasing doses.
- Generally well tolerated with no serious adverse events reported.
- No severe adverse events in the 50 mg cohort. One severe adverse event for an allergic reaction to the study drug was reported in the 25 mg cohort and was resolved with standard treatment.
- 18 of the 19 participants dosed with nomlabofusp completed the trial, with one participant in the 25 mg cohort withdrawing due to the aforementioned allergic reaction that resolved with standard treatment.
- Most common adverse events were mild and moderate injection site reactions.

Updates on the Nomlabofusp Regulatory Pathway in FA

- FDA acknowledgment that frataxin deficiency appears to be critical to the pathogenic mechanism of FA, and that there continues to be an unmet need for treatments for FA patients that address the underlying disease pathophysiology.
- Ongoing discussions with the FDA on evidence needed to support an accelerated approval application, including supplementary nonclinical pharmacology investigations, FXN, supportive PD and clinical outcomes information from the OLE study, and additional clinical safety data.
- Initiation of a confirmatory study planned prior to BLA submission.
- Potential BLA submission targeted for 2H 2025.

Cash position

As of December 31, 2023, the Company had cash, cash equivalents and marketable securities totaling $86.8 million, which we expect to provide runway into the first quarter of 2025.

Conference Call and Webcast

Larimar will host a conference call and webcast today, February 12, 2024 at 8:00 a.m. ET. To access the webcast, please visit this link to the event. To participate by phone, please dial 1-877-407-9716 (domestic) or 1-201-493-6779 (international) and refer to conference ID 13744180, or click on this link and request a return call. Following the live event, an archived webcast will be available on the “Events & Presentations” page of the Larimar website.

About Nomlabofusp (CTI-1601)

Nomlabofusp is a recombinant fusion protein intended to deliver human frataxin to the mitochondria of patients with Friedreich’s ataxia who are unable to produce enough of this essential protein. Nomlabofusp has been granted Rare Pediatric Disease designation, Fast Track designation and Orphan Drug designation by the U.S. Food and Drug Administration (FDA), Orphan Drug Designation by the European Commission, and a PRIME designation by the European Medicines Agency.

About Larimar Therapeutics

Larimar Therapeutics, Inc. (Nasdaq: LRMR), is a clinical-stage biotechnology company focused on developing treatments for complex rare diseases. Larimar’s lead compound, nomlabofusp (CTI-1601), is being developed as a potential treatment for Friedreich's ataxia. Larimar also plans to use its intracellular delivery platform to design other fusion proteins to target additional rare diseases characterized by deficiencies in intracellular bioactive compounds. For more information, please visit: https://larimartx.com.

Forward-Looking Statements

This press release contains forward-looking statements that are based on Larimar’s management’s beliefs and assumptions and on information currently available to management. All statements contained in this release other than statements of historical fact are forward-looking statements, including but not limited to statements regarding Larimar’s ability to develop and commercialize nomlabofusp (also known as CTI-1601) and other planned product candidates, Larimar’s planned research and development

efforts, including the timing of its nomlabofusp clinical trials, interactions with the FDA and overall development plan and other matters regarding Larimar's business strategies, ability to raise capital, 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 success, cost and timing of Larimar's product development activities, nonclinical studies and clinical trials, including nomlabofusp clinical milestones and continued interactions with the FDA; that preliminary clinical trial results may differ from final clinical trial results, that earlier non-clinical and clinical data and testing of nomlabofusp may not be predictive of the results or success of later clinical trials, and assessments; that the FDA may not ultimately agree with Larimar's nomlabofusp development strategy; the potential impact of public health crises on Larimar's future clinical trials, manufacturing, regulatory, nonclinical study timelines and operations, and general economic conditions; Larimar's ability and the ability of third-party manufacturers Larimar engages, to optimize and scale nomlabofusp's manufacturing process; Larimar's ability to obtain regulatory approvals for nomlabofusp and future product candidates; Larimar's ability to develop sales and marketing capabilities, whether alone or with potential future collaborators, and to successfully commercialize any approved product candidates; Larimar's ability to raise the necessary capital to conduct its product development activities; and other risks described in the filings made by Larimar with the Securities and Exchange Commission (SEC), including but not limited to Larimar's 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 SEC and available at www.sec.gov. These forward-looking statements are based on a combination of facts and factors currently known by Larimar 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. The forward-looking statements in this press release represent Larimar's management's views only as of the date hereof. Larimar undertakes no obligation to update any forward-looking statements for any reason, except as required by law.

Investor Contact:
Joyce Allaire
LifeSci Advisors
jallaire@lifesciadvisors.com
(212) 915-2569

Company Contact:
Michael Celano
Chief Financial Officer
mcelano@larimartx.com
(484) 414-2715

Exhibit 99.2

Larimar Therapeutics

Corporate Deck

February 2024

Forward-Looking Statements

This presentation contains forward-looking statements that are based on the beliefs and assumptions of Larimar Therapeutics, Inc. (“Company”) 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 Larimar’s ability to develop and commercialize nomlabofusp (CTI-1601) and other planned product candidates, Larimar’s planned research and development efforts, including the timing of its nomlabofusp clinical trials and overall development plan and other matters regarding Larimar’s business strategies, ability to raise capital, 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 success, cost and timing of Larimar’s product development activities, nonclinical studies and clinical trials, including nomlabofusp clinical milestones and continued interactions with the FDA; that preliminary clinical trial results may differ from final clinical trial results, that earlier non-clinical and clinical data and testing of nomlabofusp may not be predictive of the results or success of later clinical trials, and assessments; that the FDA may not ultimately agree with Larimar’s nomlabofusp development strategy; the potential impact of public health crises on Larimar’s future clinical trials, manufacturing, regulatory, nonclinical study timelines and operations, and general economic conditions; Larimar’s ability and the ability of third-party manufacturers Larimar engages, to optimize and scale nomlabofusp’s manufacturing process; Larimar’s ability to obtain regulatory approvals for nomlabofusp and future product candidates; Larimar’s ability to develop sales and marketing capabilities, whether alone or with potential future collaborators, and to successfully commercialize any approved product candidates; Larimar’s ability to raise the necessary capital to conduct its product development activities; and other risks described in the filings made by Larimar with the Securities and Exchange Commission (SEC), including but not limited to Larimar’s 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 SEC and available at www.sec.gov. These forward-looking statements are based on a combination of facts and factors currently known by Larimar 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. The forward-looking statements in this presentation represent Larimar’s management’s views only as of the date hereof. Larimar undertakes no obligation to update any forward-looking statements for any reason, except as required by law.

Positive Topline Phase 2 Data for Nomlabofusp (CTI-1601)

Successful completion of 4-week, placebo-controlled dose exploration study (25 mg and 50 mg) in FA

Dose-dependent increases in tissue frataxin (FXN) levels in skin and buccal cells

Nomlabofusp was generally well-tolerated following repeated subcutaneous injections up to 28 days

Participants treated with 50 mg for 14 days and then every other day for an additional 14 days until day 28

- Baseline FXN levels in skin cells < 17% of average FXN levels of healthy volunteers
- After 14 days of daily dosing, FXN levels in skin cells increased to 33% to 59% of average FXN level of healthy volunteers
- After switching to every other day dosing on day 15, continue to observe dose dependent increases in FXN levels with reduced magnitude

All treated patients in the 50 mg dose group had at least a 100% increase over baseline in FXN levels in skin cells at day 14

Across all studies to date, higher variability in FXN levels was observed in buccal vs. skin cells

OLE trial initiated for 25 mg daily dosing

High patient interest in study participation
Initial data expected Q4 2024

Intend to Pursue Accelerated Approval with FDA

Discussions initiated on FXN as surrogate endpoint
Potential **BLA submission targeted for 2H 2025**

Larimar Therapeutics

FA: Friedreich's ataxia; OLE: Open-label extension; BLA: Biologics License Application

Clinical-Stage Novel Protein Replacement Therapy Platform

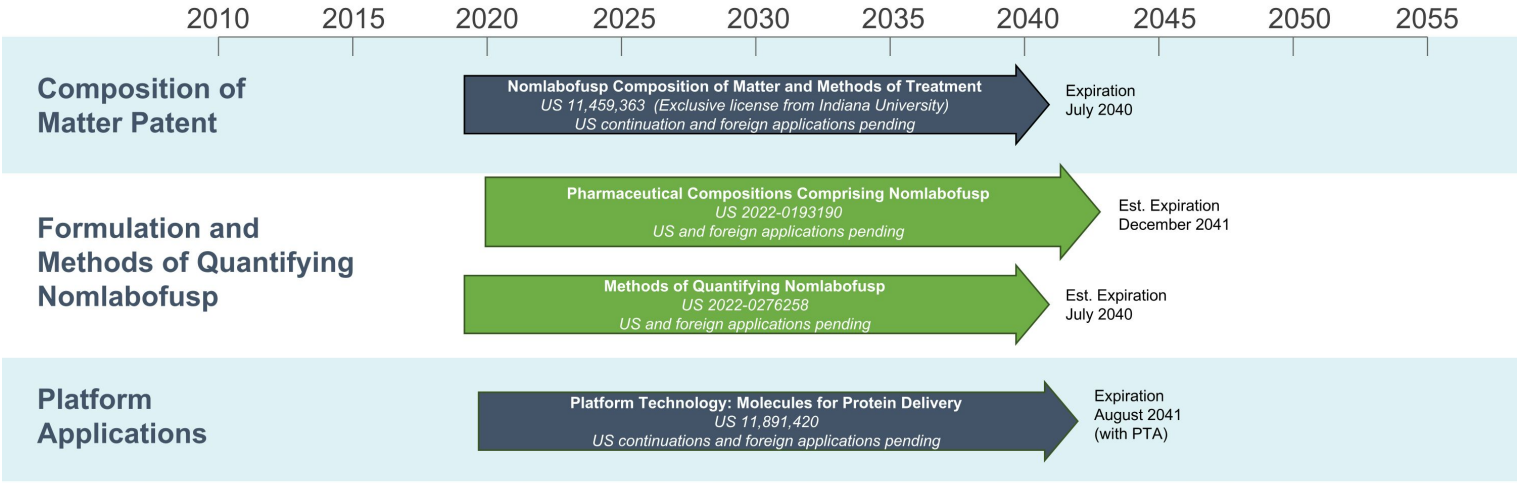
Potential first therapy to increase frataxin levels	Lead candidate nomlabofusp* (CTI-1601) is a recombinant fusion protein designed to directly address frataxin deficiency in patients with Friedreich's ataxia (FA) by delivering the protein to mitochondria. Granted Orphan Drug (US & EU), Rare Pediatric Disease (US), Fast Track (US), & PRIME (EU) designations
Consistent Phase 1 and Phase 2 findings	Nomlabofusp was generally well tolerated and demonstrated dose-dependent increases in frataxin (FXN) levels from baseline in skin and buccal cells in a 4-week placebo-controlled Phase 2 study and a multiple ascending dose Phase 1 study
Intend to pursue accelerated approval with FDA	FDA acknowledgement that FXN deficiency appears to be critical to the pathogenic mechanism of FA, and that there continues to be an unmet need for treatments that address the underlying disease pathophysiology. Discussions to support an accelerated approval are ongoing. BLA submission targeted for 2H 2025
OLE study with near-term catalysts	Initiated OLE study with 25 mg daily dosing in Q1 2024 with **interim data expected in Q4 2024** To potentially escalate dose in the OLE study, 25 mg treatment data will be submitted for FDA review due to continued partial clinical hold
Strong financial foundation	$86.8** million estimated cash balance (December 31, 2023) with projected cash runway into Q1 2025

Larimar Therapeutics

*As of October 2023, nomlabofusp was published as the INN (International Nonproprietary Name) and USAN (United States Adopted Name) for CTI-1601.
**This estimate is unaudited and preliminary and actual results may differ due to the completion of our fiscal 2023 closing procedures. As such, this estimate should not be viewed as a substitute for our full audited financial statements prepared in accordance with U.S. generally accepted accounting principles.

Larimar Technology is Supported by a Strong IP Portfolio

Granted nomlabofusp (CTI-1601) composition of matter patent extends into 2040



Additional nomlabofusp IP protection

- US and foreign pending applications cover key biomarkers, analytical tools and methods of treatment for additional disease indications for nomlabofusp
- Nomlabofusp should be eligible for **12 years of market exclusivity** upon approval in the US (independent of patents) and at least **10 years of market exclusivity** upon approval in EU (independent of patents)

Granted Pending

Friedreich's Ataxia (FA): A rare and progressive disease

Genetic defect on both alleles lowers frataxin levels	Most patients with FA only produce ~20-40% of normal frataxin levels depending on the tissue, sampling technique, and assay considered*
Affects ~20,000 patients globally	~5,000 patients in the U.S., with most remaining patients in the EU ~70% of patients present before age 14
Progressive disease	Initial symptoms include unsteady posture and frequent falling, and patients are eventually confined to a wheelchair Life expectancy of 30-50 years with an early death usually caused by heart disease
No approved therapies increase frataxin levels	Only treatment approved for FA does not address frataxin deficiency

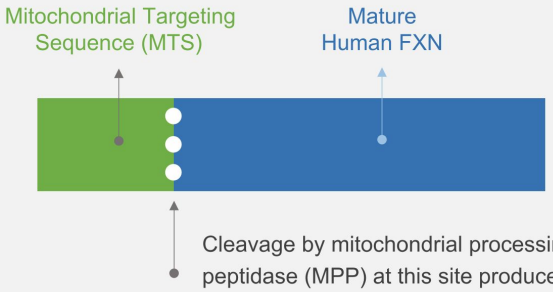
Larimar Therapeutics

* E.C. Deutsch et al. Molecular Genetics and Metabolism 101 (2010) 238–245.

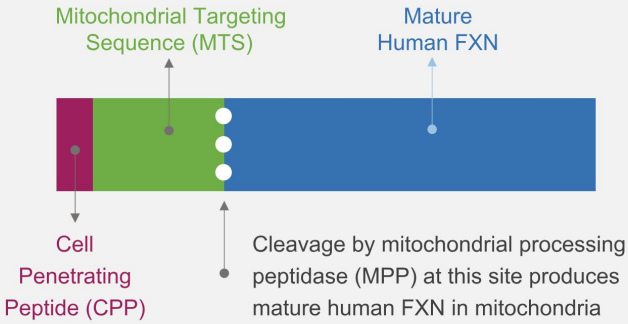
Nomlabofusp is Designed to Deliver Additional Frataxin

Nomlabofusp (CTI-1601) maintains the cleavage site between the MTS and mature human frataxin (FXN)

STRUCTURE OF ENDOGENOUS FXN



STRUCTURE OF NOMLABOFUSP



The presence of the cleavage site allows the CPP and MTS to be removed by mitochondrial processing peptidase to produce mature human FXN in the mitochondria

Larimar Therapeutics

FXN Levels Predict Disease Progression in FA

Lower FXN levels are associated with earlier onset of disease, faster rate of disease progression, and shorter time to loss of ambulation

Median Age of Onset and Rate of Disease Progression in Relation to FXN Levels

FXN Level* (% of Normal Level)	**Age of Onset** (Years)	**FARS**** (Change/Year)
11.2	7	2.9
22.0	11	2.1
31.0	16	2.0
48.7	19	1.6

Adapted from H.L.Plasterer et al. PLoS ONE 2013 8(5):e63958

Median Age of Onset Predicts Time to Loss of Ambulation

Age of Onset (Years)	**Median Time to Loss of Ambulation** (Years)
< 15	11.5
15 to 24	18.3
> 24	23.5

Adapted from C. Rummey et al. EClinicalMedicine. 2020 18:100213

Larimar Therapeutics

*FXN levels measured in peripheral blood mononuclear cells (PBMCs). FXN levels as measured by % of normal demonstrated to be equivalent in PBMCs, buccal cells, and whole blood.
**FARS: Friedreich's ataxia rating score, measures disease progression with a higher score indicating a greater level of disability.

Phase 2 Dose Exploration Study for 25 and 50 mg Cohorts

Goal: Further characterize PK/PD and assess safety to inform long-term dose and dose regimen

Treatment Schedule - nomlabofusp (CTI-1601) or placebo

28-day Treatment Period

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
■	■	■	■	■	■	■	■	■	■	■	■	■	■		■		■		■		■		■		■		■

■ = Subcutaneous administration of nomlabofusp (CTI-1601) or placebo

■ = No Administration

Study Details

Population	Ambulatory and non-ambulatory Friedreich's ataxia patients ≥18 years of age Nomlabofusp (CTI-1601) treatment naïve or participated (if eligible) in a previous Larimar study
Dose	**Cohort 1:** 25 mg **Cohort 2:** 50 mg
Key Endpoints	Frataxin levels in peripheral tissue, PK, safety and tolerability; other exploratory endpoints include lipids and gene expression levels
Number of Patients	**Cohort 1:** Enrolled 13 participants (9 on nomlabofusp; 4 on placebo) **Cohort 2:** Enrolled 15 participants (10 on nomlabofusp; 5 on placebo)

Larimar Therapeutics

Demographics – Phase 2 Trial

	25 mg Cohort			50 mg Cohort		
	Placebo *N = 4*	Nomlabofusp *N = 9*	Overall *N = 13*	Placebo *N = 5*	Nomlabofusp *N = 10*	Overall *N = 15*
Age at Screening (Years)						
Mean (SD)	34.0 (9.20)	37.8 (14.93)	36.6 (13.16)	28.6 (4.67)	28.1 (11.00)	28.3 (9.17)
Median	33	31	31	27	24	26
Q1, Q3	27, 42	27, 42	27, 42	26, 30	21, 32	21, 32
Min, Max	25, 45	25, 69	25, 69	24, 36	19, 54	19, 54
Sex n (%)						
Male	2 (50.0)	5 (55.6)	7 (53.8)	1 (20.0)	4 (40.0)	5 (33.3)
Female	2 (50.0)	4 (44.4)	6 (46.2)	4 (80.0)	6 (60.0)	10 (66.7)
Previously Treated with Nomlabofusp n (%)						
Yes	1 (25.0)	3 (33.3)	4 (30.8)	0	1 (10.0)	1 (6.7)
No	3 (75.0)	6 (66.7)	9 (69.2)	5 (100.0)	9 (90.0)	14 (93.3)

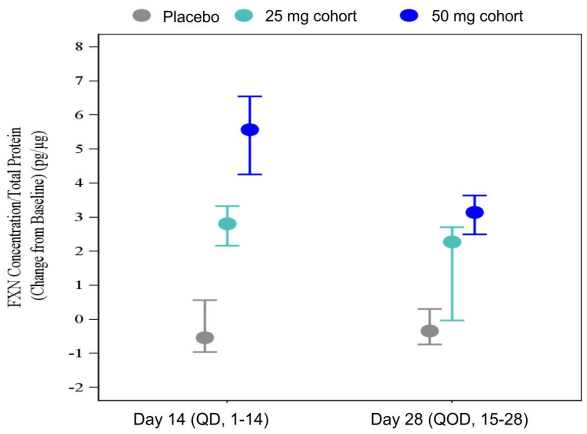
Disease Characteristics – Phase 2 Study

	25 mg Cohort			50 mg Cohort		
	Placebo *N = 4*	Nomlabofusp *N = 9*	Overall *N = 13*	Placebo *N = 5*	Nomlabofusp *N = 10*	Overall *N = 15*
Age at Symptom Onset (Years)						
Mean (SD)	14.5 (4.93)	13.0 (10.47)	13.5 (8.77)	15.2 (7.26)	13.7 (8.37)	14.2 (7.78)
Median	14.5	10	11	14	12.5	14
Q1, Q3	11, 19	8, 13	9, 15	11, 16	7, 18	7, 18
Min, Max	9, 20	5, 38	5, 38	8, 27	5, 30	5, 30
Age at Diagnosis (Years)						
Mean (SD)	17.5 (5.57)	18.6 (11.20)	18.2 (9.58)	18.6 (6.80)	16.6 (8.03)	17.3 (7.46)
Median	16.5	16	16	19	13.5	14
Q1, Q3	14, 22	14, 20	14, 20	13, 20	10, 21	12, 21
Min, Max	12, 25	5, 42	5, 42	12, 29	9, 30	9, 30
Time Since Diagnosis (Years)						
Mean (SD)	16.1 (5.97)	18.5 (11.52)	17.8 (9.94)	9.5 (3.72)	11.9 (7.05)	11.1 (6.10)
Median	13.42	14.32	13.5	11	11.26	11
Q1, Q3	12.9, 19.3	12.8, 21.6	12.8, 21.6	5.8, 11.3	7.4, 15.3	5.8, 15.2
Min, Max	12.5, 25.0	5.4, 45.0	5.4, 45.0	5.6, 14.0	2.3, 25.1	2.3, 25.1

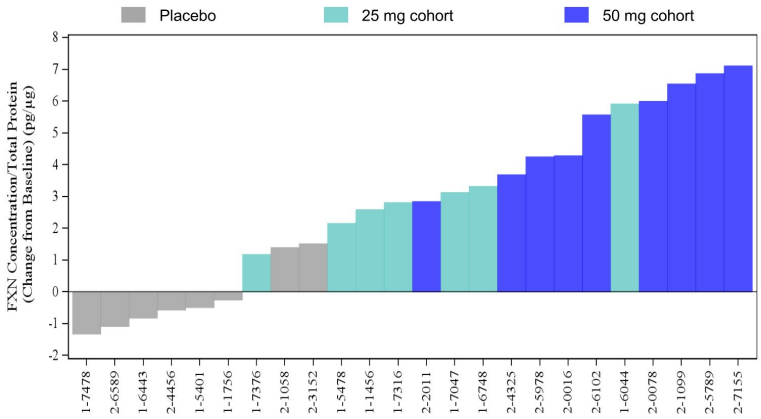
Dose-Dependent Increase in FXN Levels in Skin Cells

Participants dosed daily for 14 days, then every other day until day 28

Skin Cells
FXN Levels* Change from Baseline**



FXN Levels* in Skin Cells
Change from Baseline at Day 14



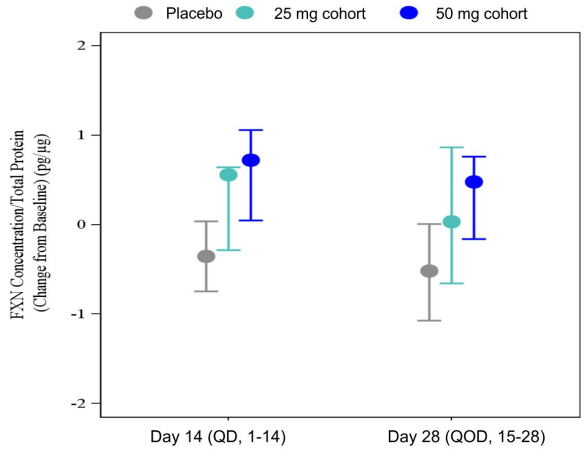
Larimar Therapeutics

*FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations are normalized to total cellular protein content in each sample. Data represent median and 25th and 75th percentiles. Only participants with quantifiable levels at both baseline and Day 14 are included in the figures.
**Median baseline FXN levels in patients were 3.5 pg/µg for the placebo, 3.7 pg/µg for the 25 mg cohort and 2.1 pg/µg for the 50 mg cohort.

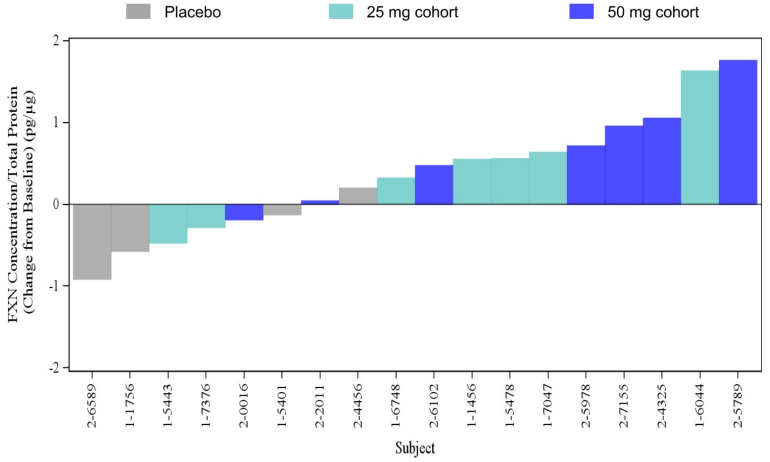
Dose-Dependent Increase in FXN Levels in Buccal Cells

Participants dosed daily for 14 days, then every other day until day 28

Buccal Cells
FXN Levels* Change from Baseline**



FXN Levels* in Buccal Cells
Change from Baseline at Day 14



*FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations are normalized to total cellular protein content in each sample. Data represent median and 25th and 75th percentiles. Only participants with quantifiable levels at both baseline and Day 14 are included in the figures.
**Median baseline FXN level in patients were 2.1 pg/µg for the placebo, 1.8 pg/µg for the 25 mg cohort and 1.6 pg/µg for the 50 mg cohort.

Absolute Increases in Skin FXN Levels

Dose response in tissue FXN concentrations and increases from baseline after dosing

Day 14 Skin FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	3.70	3.38
	Day 14	5.53	6.40
	Change from Baseline	**2.81**	**3.02**
50 mg	Baseline	2.12	2.08
	Day 14	7.40	7.32
	Change from Baseline	**5.57**	**5.24**

Day 28 Skin FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	3.70	3.38
	Day 28	4.39	4.80
	Change from Baseline	**2.28**	**1.41**
50 mg	Baseline	2.12	2.08
	Day 28	5.23	5.24
	Change from Baseline	**3.14**	**3.17**

Larimar Therapeutics

Only participants with quantifiable levels at baseline and day 14 and day 28 are included in the tables.

Absolute Increases in Buccal FXN Levels

Dose response in tissue FXN concentrations and increases from baseline after dosing

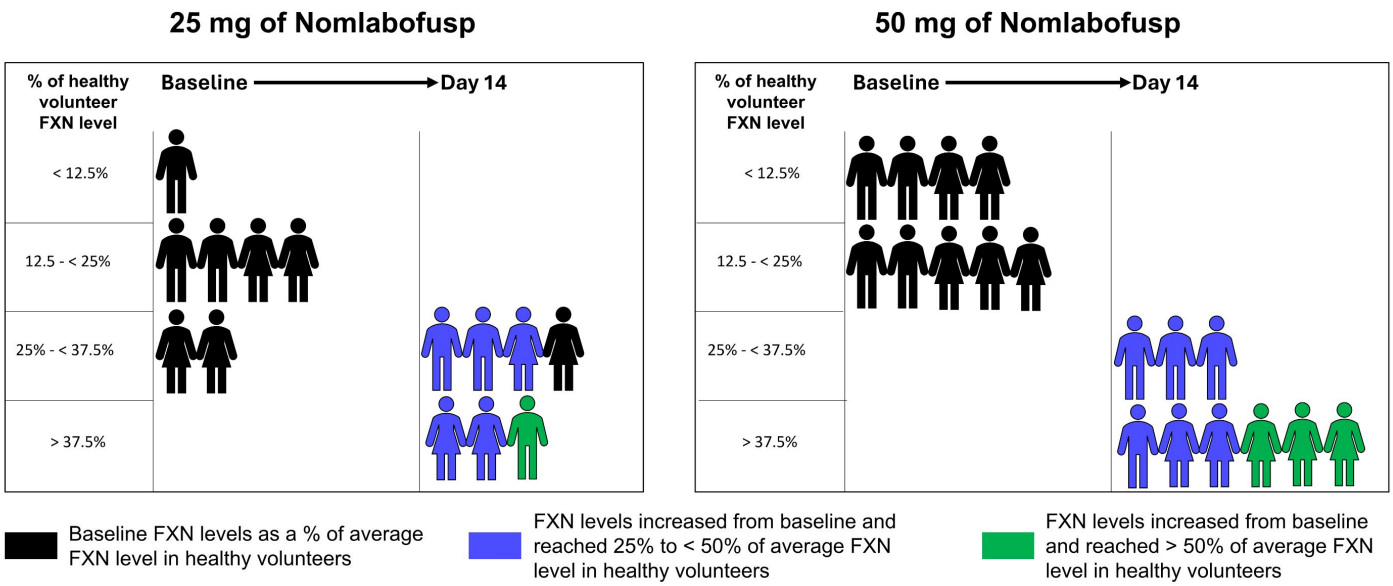
Day 14 Buccal FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	1.78	1.80
	Day 14	2.24	2.22
	Change from Baseline	**0.56**	**0.42**
50 mg	Baseline	1.61	1.69
	Day 14	2.44	2.38
	Change from Baseline	**0.72**	**0.69**

Day 28 Buccal FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	1.70	1.65
	Day 28	1.73	1.76
	Change from Baseline	**0.03**	**0.11**
50 mg	Baseline	1.76	1.77
	Day 28	2.15	2.15
	Change from Baseline	**0.48**	**0.38**

Larimar Therapeutics

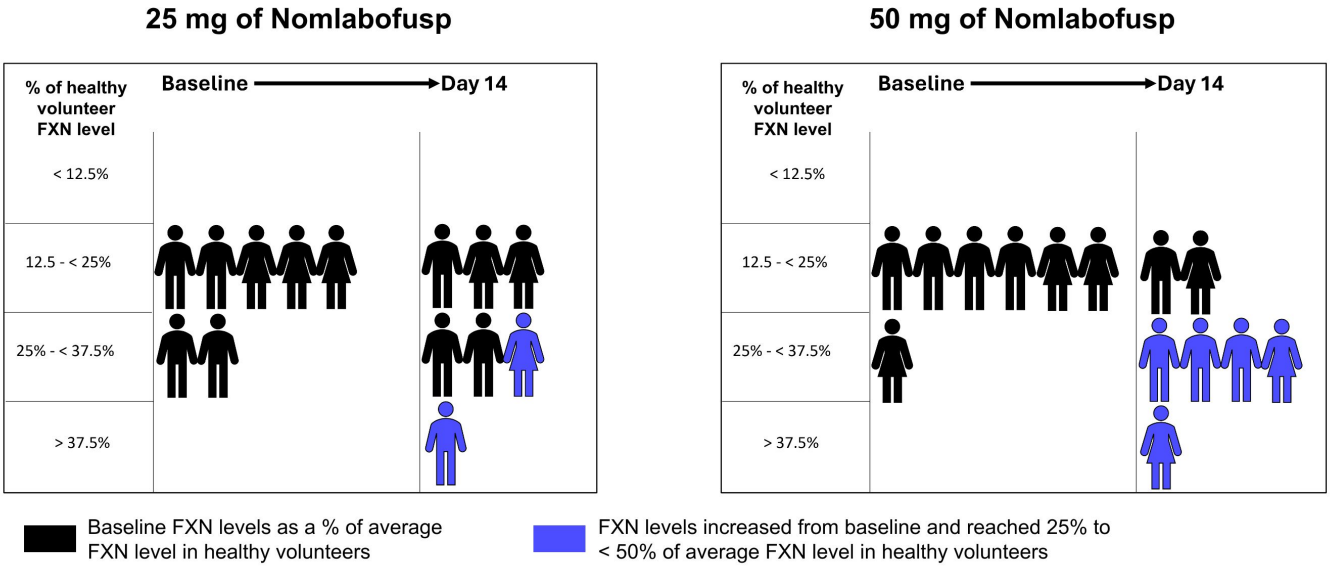
Only participants with quantifiable levels at baseline and day 14 and day 28 are included in the tables.

Skin Cell FXN Levels Achieve Higher % of Healthy Volunteers* Following 14 days of Daily Nomlabofusp



Only participants with quantifiable levels at baseline and day 14 are included in the figures.
*% of healthy volunteer FXN level is calculated by dividing each participant's FXN level by the average FXN level (16.34 pg/µg) from the noninterventional healthy volunteer study (N=60).

Buccal Cell FXN Levels Achieve Higher % of Healthy Volunteers* Following 14 days of Daily Nomlabofusp



Only participants with quantifiable levels at baseline and day 14 are included in the figures.
*% of healthy volunteer FXN level is calculated by dividing each participant's FXN level by the average FXN level (8.24 pg/µg) from Larimar's noninterventional healthy volunteer study (N=60).

Increasing FXN Levels May Slow Disease Progression

Disease Characteristics* Based on Literature**	Nomlabofusp Administration in Phase 2 Study
Patients with FXN levels 11% of average healthy volunteers • Median age of onset at 7 years • Deteriorate by 2.9 points/year as measured by FARS • Lose ambulation at a median of 11.5 years	**25 mg daily for 14 days shifted FXN levels in** • All but one patient to > 25% of average healthy volunteers in skin cells with a median value of 33.9%
Patients with FXN levels > 30% of average healthy volunteers • Median age of onset at 16 years • Deteriorate by 2.0 points/year as measured by FARS • Lose ambulation at a median of 18.3 years	**50 mg daily for 14 days shifted FXN levels in** • All patients from < 25% of average healthy volunteers to 33% to 59% (3 patients > 50%) in skin cells with a median value of 45%

H.L.Plasterer et al. PLoS ONE 2013 8(5):e63958; C. Rummey et al. EClinicalMedicine. 2020 18:100213

Larimar Therapeutics

*FARS: Friedreich's ataxia rating scale, measures disease progression with a higher score indicating a greater level of disability.
**FXN levels measured in peripheral blood mononuclear cells (PBMCs). FXN levels as measured by % of normal demonstrated to be equivalent in PBMCs, buccal cells, and whole blood.

Encouraging Therapeutic Potential for Nomlabofusp

Frataxin deficiency is the root cause of the disease

Lower levels of frataxin correlate with disease burden

Animal models show that increasing frataxin mitigates clinical outcomes

Dose-dependent increases in frataxin levels with nomlabofusp in several studies

Continue nomlabofusp clinical development

Nomlabofusp: Predictable Pharmacokinetics

1. Quick absorption after subcutaneous administration
2. Dose-proportional increases in exposure observed
3. Pharmacokinetic profile consistent with Phase 1 studies

Ph1 & Ph2 Data: Nomlabofusp is Generally Well Tolerated

61 patients have participated in our Phase 1 and Phase 2 studies with no serious adverse events in any nomlabofusp clinical study. One severe adverse event (allergic reaction that resolved with standard treatment referenced below).

44 of 46 clinical trial participants dosed with nomlabofusp completed their respective study
One Phase 2 participant in the 25 mg cohort withdrew due to allergic reaction that resolved with standard treatment
One Phase 1 participant in the 50 mg cohort withdrew due to mild-to-moderate nausea and vomiting

Most common adverse events (AEs) were mild and moderate injection site reactions (ISRs)
No study discontinuations due to ISRs and all resolved

Open-label Extension Study: Initiated Q1 2024

Preliminary interim data expected in Q4 2024

Key Eligibility Criteria

Previous participation in Phase 1 or Phase 2 trials

Daily subcutaneous injection of 25 mg nomlabofusp;
self-administered or by a caregiver

1 site initiated and screening has begun

Key Study Objectives

- Safety and tolerability
- Long-term PK
- Tissue FXN concentrations and potential use as surrogate endpoint to support accelerated approval
- Clinical efficacy measures compared to the matched set of untreated patients from FACOMS* database

Screening Period ≤ 42 days** → Treatment Period Planned for ≥ 1 year → Potential extensions

Larimar Therapeutics

*FACOMS: Friedreich's Ataxia Clinical Outcome Measures Study.
**Estimated screening period may be extended for those study participants who have not been on a stable regimen of omaveloxolone for at least six months.

Nomlabofusp Clinical Development Plan

Intend to pursue accelerated approval pathway with potential BLA submission targeted for 2H 2025

Ongoing open-label extension study with 25 mg daily dosing for eligible patients who participated in SAD, MAD, and/or four-week dose exploration studies

Initial data expected Q4 2024

Planned pediatric MAD trial in patients 2 to 17 years of age*

Participants eligible to screen for OLE trial

Planned global double-blind placebo-controlled registration/confirmatory study**

BLA submission targeted for 2H 2025

*Company is discussing with FDA how to best include patients 2 to 17 years of age in clinical development.
**Company initiated discussions with FDA on the role of FXN levels to support accelerated approval. Also, the Company is planning discussions with regulators and investigators outside the U.S. to expand clinical program to international geographies.
Initiation of additional U.S. clinical trials is contingent on FDA review of clinical data due to partial clinical hold.

Nomlabofusp is a Competitively Differentiated Treatment Approach*

$7.3B Acquisition supports the **robust market potential** for FA treatments

REATA PHARMACEUTICALS Biogen

Nomlabofusp is a potential **first-and-only protein replacement therapy** designed to address the underlying cause of FA

Approach	Product	Company	Mechanism of Action	Clinical Status
Protein replacement	**Nomlabofusp (CTI-1601)**	**Larimar**	**Recombinant frataxin protein**	**Phase II**
Mitochondrial Oxidative Stress Modifier	Omaveloxolone (SKYCLARYS™)	Reata Pharma/Biogen	Nrf2 Activator	Approved (US and EU)
	Vatiquinone	PTC Therapeutics	15-Lipoxygenase Inhibitor	Phase III
Gene Expression Regulator	DT-216	Design Therapeutics	GeneTAC	Phase I
Gene Therapy	LX2006	Lexeo Therapeutics	Frataxin Gene Replacement	Phase I/II

Larimar Therapeutics

*Competitive landscape focuses on clinical-stage, industry-sponsored programs from public companies

Positive Topline 50 mg & 25 mg Ph 2 Data and OLE Initiated in Q1 2024

Consistent Ph 1 and Ph 2 Findings	Nomlabofusp is generally well tolerated at doses tested up to 4 weeks Dose-dependent increases in FXN levels from baseline in evaluated tissues (skin and buccal cells) Baseline FXN levels in skin cells in the 50 mg cohort were < 17% of the average of healthy volunteers. After daily dosing for 14 days, FXN levels increased to 33% to 59%
Regulatory Updates	Initiated discussions with FDA regarding use of FXN as a surrogate endpoint to support accelerated approval Intend to pursue accelerated approval with potential BLA submission for 2H 2025 Beginning preparations to expand nomlabofusp clinical program to ex-U.S. geographies
Expected Milestones	**Q1 2024:** Dosing of first patient in OLE study **Q4 2024:** Initial data from OLE study; initiated in Q1 2024 **2H 2024:** Final Phase 2 data planned to be presented at a conference **2H 2025:** BLA submission

Larimar Therapeutics


Larimar
Therapeutics
Larimar Therapeutics
February 2024 Corporate Deck
THANK YOU

Larimar Therapeutics

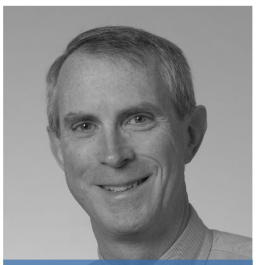
Appendix

Scientific Advisory Board

Giovanni Manfredi, MD, PhD

Finbar and Marianne Kenny Professor in Clinical and Research Neurology at Weill Cornell Medicine.

Professor of Neuroscience at Weill Cornell Medicine.



Mark Payne, MD

Co-founder of Chondrial Therapeutics, which became Larimar Therapeutics, Inc.

Professor of Pediatrics at Indiana University School of Medicine

Marni J. Falk, MD

Executive Director of the Mitochondrial Medicine Frontier Program at The Children's Hospital of Philadelphia (CHOP)

Professor in the Division of Human Genetics, Department of Pediatrics at University of Pennsylvania Perelman School of Medicine

Jill Ostrem, MD

Medical Director and Division Chief of the University of California San Francisco (UCSF) Movement Disorders and Neuromodulation Center.

Carlin and Ellen Wiegner Endowed Professor of Neurology

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

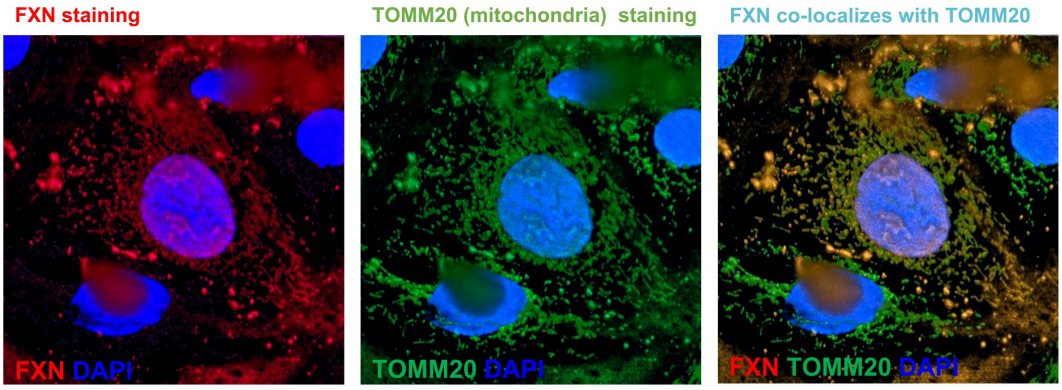
FARA provides industry with several key items

- Assistance 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 titled "The Voice of the Patient"

FARA Friedreich's Ataxia Research Alliance

Mitochondrial Localization and Preclinical Data

Nomlabofusp Transduction of Cells In Vitro Leads to hFXN Located in Mitochondria



- Rat cardiomyocytes (H9C2) were transduced with nomlabofusp
- Cells were fixed and analyzed by immunofluorescence microscopy to detect the presence of human frataxin (hFXN) and TOMM20 (a mitochondrial outer membrane protein)
- Nuclei were stained with DAPI

Larimar Therapeutics

Nomlabofusp Extends Survival in FXN-deficient KO Mice

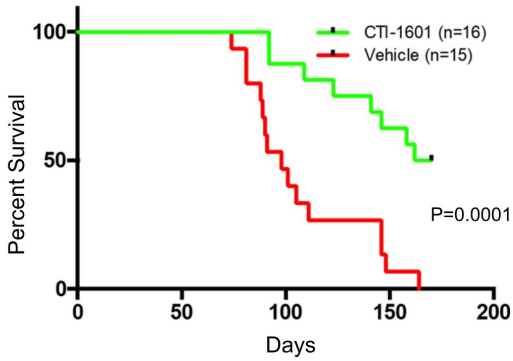
Initial proof-of-concept for FXN replacement therapy in cardiac mouse model of FA

Median survival of MCK-Cre FXN-KO mice

- 166 days (nomlabofusp) vs. 98 days (Vehicle)
- Nomlabofusp administered 10 mg/kg SC every other day

Survival beyond vehicle mean (107.5 days)

- 87.5% (nomlabofusp) vs. 33% (Vehicle)
- Demonstrates that nomlabofusp is capable of delivering sufficient amounts of FXN to mitochondria



Nomlabofusp (CTI-1601) rescues a severe disease phenotype in a well-characterized cardiac mouse model of FA

Nomlabofusp Prevents Development of Ataxic Gait in Neurologic KO Mouse Model

In-Vivo Efficacy Data in Pvalb-Cre FXN-KO Mouse Model

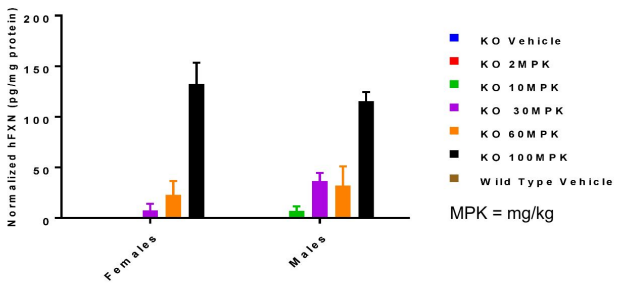
Single dose level: 10 mg/kg nomlabofusp or vehicle given intraperitoneally three times per week

- hFXN replacement with nomlabofusp **prevents development of ataxic gait**
- Nomlabofusp-treated mice **survive longer** than untreated mice
- Human frataxin **present in brain, dorsal root ganglia and spinal cord** demonstrating central nervous system penetration

Nomlabofusp Delivers hFXN to Mitochondria and Restores SDH Activity in KO Mice

Study Design – Cardiac and skeletal muscle FXN knockout mice (MCK-CRE) were treated at varying SQ doses of nomlabofusp every other day for two weeks at Jackson Laboratories (Bar Harbor, ME). After dosing, animals were sacrificed, and heart and skeletal muscle were evaluated for hFXN concentration in mitochondrial extracts and SDH activity was assessed.

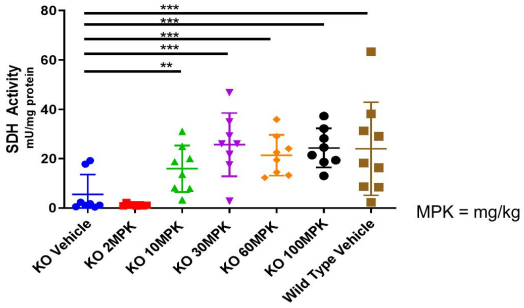
Mitochondrial FXN (Heart)



Mitochondria hFXN concentration increases dose-dependently

Given subcutaneously, nomlabofusp functionally replaces hFXN in mitochondria of KO mice

SDH Activity (Muscle)



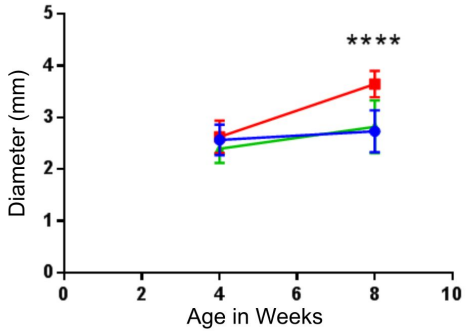
Succinate dehydrogenase (SDH) activity, which is indicative of mitochondrial function, increases in a dose-dependent manner after administration of nomlabofusp; activity plateaus at 30 mg/kg and is equivalent to activity in wild type

Larimar Therapeutics

Nomlabofusp Prevents Left Ventricle Dilation in KO Mice

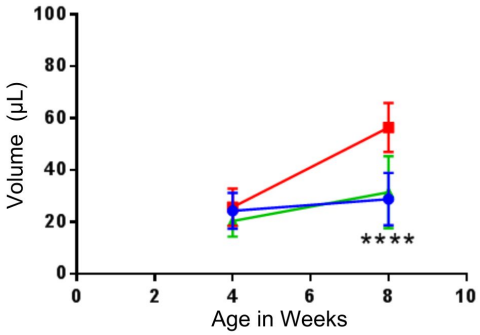
Study Design – Cardiac and skeletal muscle FXN knockout mice (MCK-CRE) were treated at 10 mg/kg every other day at Jackson Laboratories (Bar Harbor, ME). Echocardiograms were performed pre-dose and post dose.

Left Ventricle Internal Diameter (Systole)



Left ventricular (LV) 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 nomlabofusp (10 mg/kg every other day)

Left Ventricle Volume (Systole)



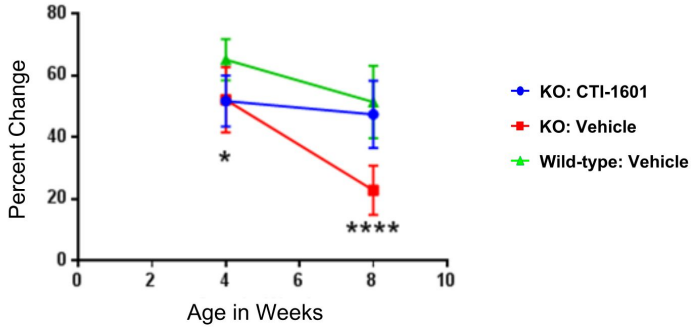
Nomlabofusp-treated mice have similar LV volume as wild type; echocardiogram shows significant differences between vehicle and nomlabofusp treated (10 mg/kg every other day) KO mice

Larimar Therapeutics

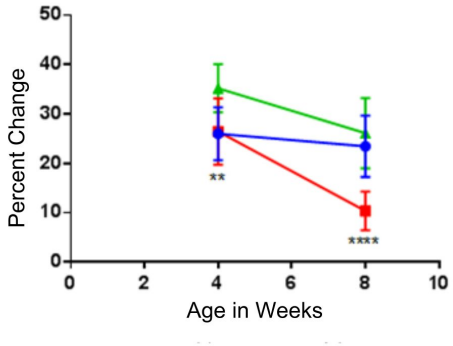
Nomlabofusp Preserves Left Ventricle Function in KO Mice

Study Design – Cardiac and skeletal muscle FXN knockout mice (MCK-CRE) were treated at 10 mg/kg every other day at Jackson Laboratories (Bar Harbor, ME). Echocardiograms were performed pre-dose and post dose.

Left Ventricle Ejection Function



Left Ventricle Fractional Shortening



Left ventricular (LV) function drops significantly in vehicle treated mice by Week 8

Nomlabofusp-treated (10 mg/kg every other day) mice have similar LV function as wildtype; echocardiogram shows significant differences between vehicle and nomlabofusp treated KO mice

Larimar Therapeutics

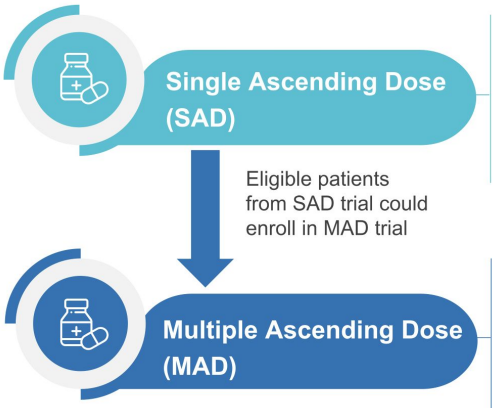
Phase 1 Clinical Data

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

Program consisted of double-blind, placebo controlled single- and multiple-ascending dose trials

Phase 1 Development Plan

- Two double-blind, placebo-controlled dosing trials in patients with FA
- Patient dosing began December 2019
- Safety Review Committee assessed all blinded data between each cohort to ensure patient safety



Number of subjects: 28
Dose levels: 25 mg, 50 mg, 75 mg and 100 mg (subcutaneous administration)
Treatment Duration: 1 day
1º Endpoint: Safety and tolerability
2º Endpoints: PK; PD; FXN levels; multiple exploratory
Status: Complete

Number of Subjects: 27
Dose Range: 25 mg, 50 mg, 100 mg (subcutaneous administration)
Treatment Regimen: Multiple increasing doses administered subcutaneously over 13 days
1º Endpoint: Safety and tolerability
2º Endpoints: PK; PD; FXN levels (buccal cells, platelets, optional skin biopsies); multiple exploratory
Status: Complete

Larimar Therapeutics

Completed Phase 1 Multiple Ascending Dose Study

Treatment Schedules for Each Cohort- nomlabofusp (CTI-1601) or placebo

Cohort 1 (25 mg; n = 8)

13-day Treatment Period

1	2	3	4	5	6	7	8	9	10	11	12	13	14

= Administration of nomlabofusp or placebo

= No Administration

Cohort 2 (50 mg; n = 9)

13-day Treatment Period

1	2	3	4	5	6	7	8	9	10	11	12	13	14

= Administration of nomlabofusp or placebo

= No Administration

Cohort 3 (100 mg n = 10)

13-day Treatment Period

1	2	3	4	5	6	7	8	9	10	11	12	13	14

= Administration of nomlabofusp or placebo

= No Administration

FXN Level Sampling Days Presented for Each Cohort

Cohort 1 Sampling Days	
Buccal Cells	Baseline, Day 4, Day 13
Skin	Baseline, Day 13
Platelets	Baseline, Day 4, Day 13

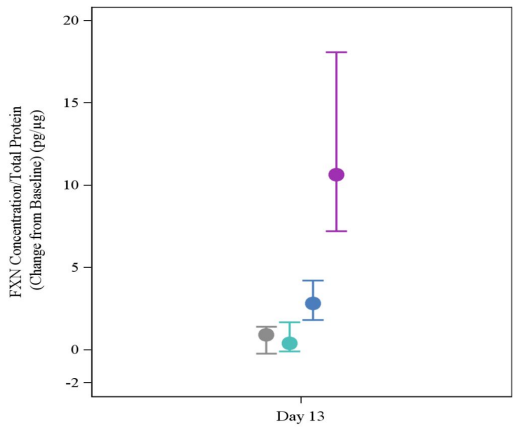
Cohort 2 Sampling Days	
Buccal Cells	Baseline, Day 7, Day 13
Skin	Baseline, Day 13
Platelets	Baseline, Day 7, Day 13

Cohort 3 Sampling Days	
Buccal Cells	Baseline, Day 7, Day 13
Skin	Baseline, Day 13
Platelets	Baseline, Day 7, Day 13

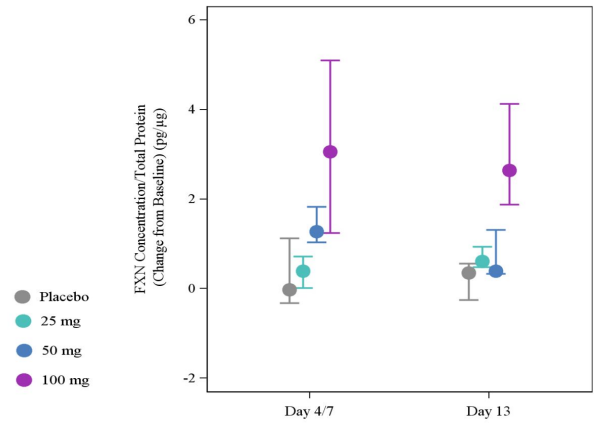
Larimar Therapeutics

Dose Dependent Increases in FXN Levels Observed in Skin and Buccal Cells in Phase 1

FXN* Change from Baseline By Dose Group (Skin Cells)



FXN* Change from Baseline By Dose Group (Buccal Cells)



Placebo: Participants randomized to placebo in each cohort
25 mg: Dosed daily for 4 days, every third day thereafter
50 mg: Dosed daily for 7 days, every other day thereafter
100 mg: Dosed daily for 13 days

*FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations are normalized to total cellular protein content in each sample; Data represent median and 25th and 75th percentiles; FXN levels from Day 4, & Day 13 measurements are shown for data derived from the 25 mg cohort; FXN levels from Day 7 & Day 13 measurements are shown for data derived from the 50 & 100 mg cohorts; Sample collection days varied in each cohort per the trial protocol

MAD Trial Patient Demographics

Parameter	Statistic	All placebo (n=7)	25 mg CTI-1601 (n=6)	50 mg CTI-1601 (n=7)	100 mg CTI-1601 (n=7)	All CTI-1601 (n=20)	Overall (n=27)
Sex							
Male	n (%)	5 (71.4)	3 (50.0)	4 (57.1)	3 (42.9)	10 (50.0)	15 (55.6)
Female	n (%)	2 (28.6)	3 (50.0)	3 (42.9)	4 (57.1)	10 (50.0)	12 (44.4)
Age (years)							
	Mean	25.7	39.7	34.7	28.0	33.9	31.7
	SD	6.37	16.59	9.03	8.96	12.13	11.40
	Median	23	37	36	24	34	28
	Min, Max	20,36	21,65	19,47	20,44	19,65	19,65
Race							
White	n (%)	6 (85.7)	6 (100.0)	6 (85.7)	6 (85.7)	18 (90.0)	24 (88.9)
Asian	n (%)	0	0	1 (14.3)	1 (14.3)	2 (10.0)	2 (7.4)
American Indian	n (%)	1 (14.3)	0	0	0	0	1 (3.7)
Ethnicity							
Hispanic/Latino	n (%)	2 (28.6)	0	0	0	0	2 (7.4)
Not Hispanic/Latino	n (%)	5 (71.4)	6 (100.0)	7 (100.0)	7 (100.0)	20 (100.0)	25 (92.6)

Larimar Therapeutics

SD: Standard deviation

MAD Trial Patient Disease Characteristics

Parameter	Statistic	All placebo (n=7)	25 mg CTI-1601 (n=6)	50 mg CTI-1601 (n=7)	100 mg CTI-1601 (n=7)	All CTI-1601 (n=20)	Overall (n=27)
Age at Symptom Onset							
	Mean	14.1	24.0	19.3	11.9	18.1	17.1
	SD	5.34	14.48	6.21	6.72	10.37	9.39
	Median	15.0	18.0	19.0	10.0	18.0	16.0
	Min, Max	8,23	12,44	8,28	5,22	5,44	5,44
Age at Diagnosis							
	Mean	18.3	31.5	26.4	15.9	24.3	22.7
	SD	7.87	19.88	4.28	8.21	13.24	12.23
	Median	20.0	25.5	28.0	13.0	27.0	21.0
	Min, Max	9,32	14,64	17,30	5,27	5,64	5,64
Assistive Device							
Walker	n (%)	0	2 (33.3)	3 (42.9)	0	5 (25.0)	5 (18.5)
Wheelchair	n (%)	4 (57.1)	3 (50.0)	1 (14.3)	6 (85.7)	10 (50.0)	14 (51.9)
Other	n (%)	1 (14.3)	0	1(14.3)	0	1 (5.0)	2 (7.4)
None	n (%)	2 (28.6)	1 (16.7)	2 (28.6)	1 (14.3)	4 (20.0)	6 (22.2)

SD: Standard deviation

PK analyses support evaluating once-daily and every-other-day dosing regimens for CTI-1601

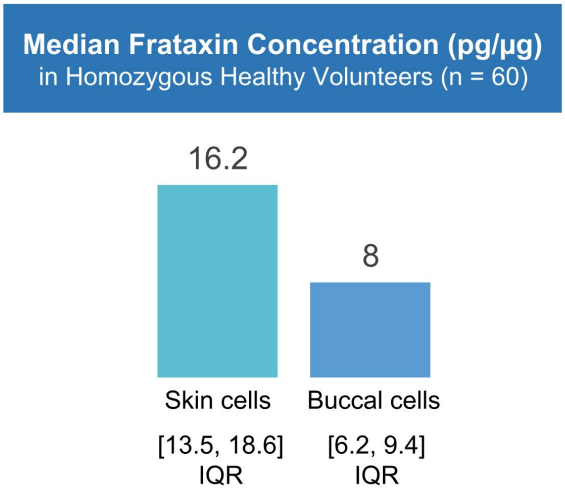
Summary of MAD Trial PK Analyses

- CTI-1601 was quickly absorbed after subcutaneous administration
- Dose-proportional increases in exposure observed with increasing doses of CTI-1601
- Mean half life of CTI-1601 in plasma was approximately 11 hours
- CTI-1601 appeared to be at or close to steady state exposure after 13 days of dosing 100 mg once daily

Non-Interventional Study Data

CLIN-1601-002: Top-line Non-interventional Study Results

Recently completed non-interventional study measured FXN in homozygous healthy volunteers



Most patients with FA only produce ~20-40%[1] of normal frataxin levels depending on the tissue, sampling technique, and assay considered

Lower FXN levels seen with typical onset[2] (5 to 15 years of age)

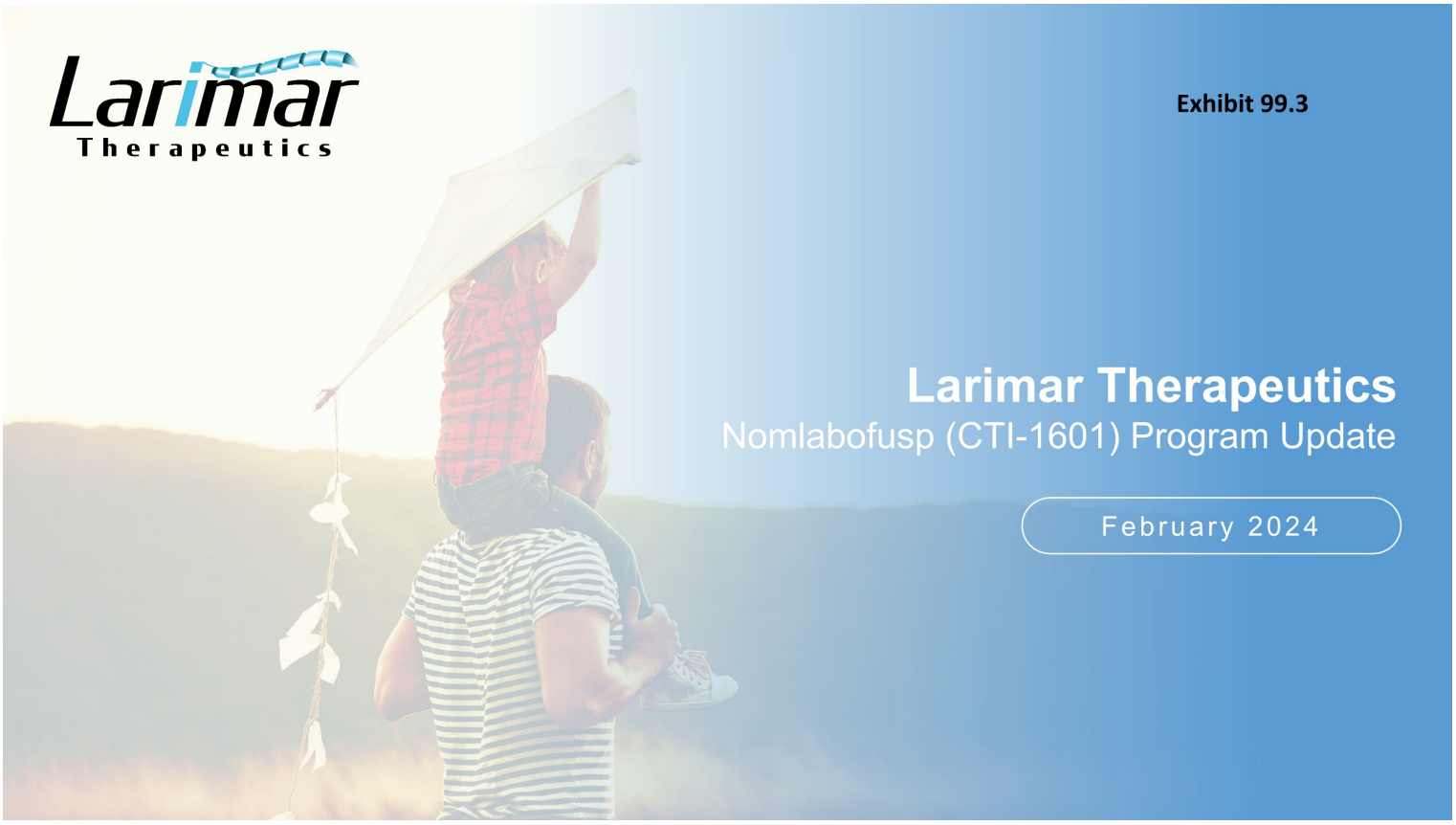
Higher FXN levels seen with late onset[2] (after 25 years of age)

Heterozygous carriers who show no signs of disease have buccal cell FXN levels of ~50% of unaffected healthy persons[1]

Larimar Therapeutics

FXN concentrations were measured in skin and buccal cells from 60 homozygous healthy volunteers utilizing the same sampling technique and assay as clinical trials of nomlabofusp; FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations normalized to total cellular protein content in each sample. 1. E.C. Deutsch et al. Molecular Genetics and Metabolism 101 (2010) 238–245. 2. Friedreich's Ataxia Research Alliance

Exhibit 99.3



Larimar Therapeutics

Nomlabofusp (CTI-1601) Program Update

February 2024

Forward-Looking Statements

This presentation contains forward-looking statements that are based on the beliefs and assumptions of Larimar Therapeutics, Inc. (“Company”) 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 Larimar’s ability to develop and commercialize nomlabofusp (CTI-1601) and other planned product candidates, Larimar’s planned research and development efforts, including the timing of its nomlabofusp clinical trials and overall development plan and other matters regarding Larimar’s business strategies, ability to raise capital, 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 success, cost and timing of Larimar’s product development activities, nonclinical studies and clinical trials, including nomlabofusp clinical milestones and continued interactions with the FDA; that preliminary clinical trial results may differ from final clinical trial results, that earlier non-clinical and clinical data and testing of nomlabofusp may not be predictive of the results or success of later clinical trials, and assessments; that the FDA may not ultimately agree with Larimar’s nomlabofusp development strategy; the potential impact of public health crises on Larimar’s future clinical trials, manufacturing, regulatory, nonclinical study timelines and operations, and general economic conditions; Larimar’s ability and the ability of third-party manufacturers Larimar engages, to optimize and scale nomlabofusp’s manufacturing process; Larimar’s ability to obtain regulatory approvals for nomlabofusp and future product candidates; Larimar’s ability to develop sales and marketing capabilities, whether alone or with potential future collaborators, and to successfully commercialize any approved product candidates; Larimar’s ability to raise the necessary capital to conduct its product development activities; and other risks described in the filings made by Larimar with the Securities and Exchange Commission (SEC), including but not limited to Larimar’s 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 SEC and available at www.sec.gov. These forward-looking statements are based on a combination of facts and factors currently known by Larimar 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. The forward-looking statements in this presentation represent Larimar’s management’s views only as of the date hereof. Larimar undertakes no obligation to update any forward-looking statements for any reason, except as required by law.

Positive Topline Phase 2 Data for Nomlabofusp (CTI-1601)

Successful completion of 4-week, placebo-controlled dose exploration study (25 mg and 50 mg) in FA

Dose-dependent increases in tissue frataxin (FXN) levels in skin and buccal cells

Nomlabofusp was generally well-tolerated following repeated subcutaneous injections up to 28 days

Participants treated with 50 mg for 14 days and then every other day for an additional 14 days until day 28

- Baseline FXN levels in skin cells < 17% of average FXN levels of healthy volunteers
- After 14 days of daily dosing, FXN levels in skin cells increased to 33% to 59% of average FXN level of healthy volunteers
- After switching to every other day dosing on day 15, continue to observe dose dependent increases in FXN levels with reduced magnitude

All treated patients in the 50 mg dose group had at least a 100% increase over baseline in FXN levels in skin cells at day 14

Across all studies to date, higher variability in FXN levels was observed in buccal vs. skin cells

OLE trial initiated for 25 mg daily dosing

High patient interest in study participation
Initial data expected Q4 2024

Intend to Pursue Accelerated Approval with FDA

Discussions initiated on FXN as surrogate endpoint
Potential **BLA submission targeted for 2H 2025**

Larimar Therapeutics

FA: Friedreich's ataxia; OLE: Open-label extension; BLA: Biologics License Application

Clinical-Stage Novel Protein Replacement Therapy Platform

Potential first therapy to increase frataxin levels	Lead candidate nomlabofusp* (CTI-1601) is a recombinant fusion protein designed to directly address frataxin deficiency in patients with Friedreich's ataxia (FA) by delivering the protein to mitochondria. Granted Orphan Drug (US & EU), Rare Pediatric Disease (US), Fast Track (US), & PRIME (EU) designations
Consistent Phase 1 and Phase 2 findings	Nomlabofusp was generally well tolerated and demonstrated dose-dependent increases in frataxin (FXN) levels from baseline in skin and buccal cells in a 4-week placebo-controlled Phase 2 study and a multiple ascending dose Phase 1 study
Intend to pursue accelerated approval with FDA	FDA acknowledgement that FXN deficiency appears to be critical to the pathogenic mechanism of FA, and that there continues to be an unmet need for treatments that address the underlying disease pathophysiology. Discussions to support an accelerated approval are ongoing. BLA submission targeted for 2H 2025
OLE study with near-term catalysts	Initiated OLE study with 25 mg daily dosing in Q1 2024 with **interim data expected in Q4 2024** To potentially escalate dose in the OLE study, 25 mg treatment data will be submitted for FDA review due to continued partial clinical hold
Strong financial foundation	$86.8** million estimated cash balance (December 31, 2023) with projected cash runway into Q1 2025

Larimar Therapeutics

*As of October 2023, nomlabofusp was published as the INN (International Nonproprietary Name) and USAN (United States Adopted Name) for CTI-1601.
**This estimate is unaudited and preliminary and actual results may differ due to the completion of our fiscal 2023 closing procedures. As such, this estimate should not be viewed as a substitute for our full audited financial statements prepared in accordance with U.S. generally accepted accounting principles.

Friedreich's Ataxia (FA): A rare and progressive disease

Genetic defect on both alleles lowers frataxin levels

Most patients with FA only produce ~20-40% of normal frataxin levels depending on the tissue, sampling technique, and assay considered*

Affects ~20,000 patients globally

~5,000 patients in the U.S., with most remaining patients in the EU
~70% of patients present before age 14

Progressive disease

Initial symptoms include unsteady posture and frequent falling, and patients are eventually confined to a wheelchair
Life expectancy of 30-50 years with an early death usually caused by heart disease

No approved therapies increase frataxin levels

Only treatment approved for FA does not address frataxin deficiency

Larimar Therapeutics

* E.C. Deutsch et al. Molecular Genetics and Metabolism 101 (2010) 238–245.

FXN Levels Predict Disease Progression in FA

Lower FXN levels are associated with earlier onset of disease, faster rate of disease progression, and shorter time to loss of ambulation

Median Age of Onset and Rate of Disease Progression in Relation to FXN Levels

FXN Level* (% of Normal Level)	**Age of Onset** (Years)	**FARS**** (Change/Year)
11.2	7	2.9
22.0	11	2.1
31.0	16	2.0
48.7	19	1.6

Adapted from H.L.Plasterer et al. PLoS ONE 2013 8(5):e63958

Median Age of Onset Predicts Time to Loss of Ambulation

Age of Onset (Years)	**Median Time to Loss of Ambulation** (Years)
< 15	11.5
15 to 24	18.3
> 24	23.5

Adapted from C. Rummey et al. EClinicalMedicine. 2020 18:100213

Larimar Therapeutics

*FXN levels measured in peripheral blood mononuclear cells (PBMCs). FXN levels as measured by % of normal demonstrated to be equivalent in PBMCs, buccal cells, and whole blood.
**FARS: Friedreich's ataxia rating score, measures disease progression with a higher score indicating a greater level of disability.

Phase 2 Dose Exploration Study for 25 and 50 mg Cohorts

Goal: Further characterize PK/PD and assess safety to inform long-term dose and dose regimen

Treatment Schedule - nomlabofusp (CTI-1601) or placebo

28-day Treatment Period

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
= Subcutaneous administration of nomlabofusp (CTI-1601) or placebo																											
= No Administration																											

Study Details

Population	Ambulatory and non-ambulatory Friedreich's ataxia patients ≥18 years of age Nomlabofusp (CTI-1601) treatment naïve or participated (if eligible) in a previous Larimar study
Dose	**Cohort 1:** 25 mg **Cohort 2:** 50 mg
Key Endpoints	Frataxin levels in peripheral tissue, PK, safety and tolerability; other exploratory endpoints include lipids and gene expression levels
Number of Patients	**Cohort 1:** Enrolled 13 participants (9 on nomlabofusp; 4 on placebo) **Cohort 2:** Enrolled 15 participants (10 on nomlabofusp; 5 on placebo)

Larimar Therapeutics

Demographics – Phase 2 Trial

	25 mg Cohort			50 mg Cohort		
	Placebo *N = 4*	Nomlabofusp *N = 9*	Overall *N = 13*	Placebo *N = 5*	Nomlabofusp *N = 10*	Overall *N = 15*
Age at Screening (Years)						
Mean (SD)	34.0 (9.20)	37.8 (14.93)	36.6 (13.16)	28.6 (4.67)	28.1 (11.00)	28.3 (9.17)
Median	33	31	31	27	24	26
Q1, Q3	27, 42	27, 42	27, 42	26, 30	21, 32	21, 32
Min, Max	25, 45	25, 69	25, 69	24, 36	19, 54	19, 54
Sex n (%)						
Male	2 (50.0)	5 (55.6)	7 (53.8)	1 (20.0)	4 (40.0)	5 (33.3)
Female	2 (50.0)	4 (44.4)	6 (46.2)	4 (80.0)	6 (60.0)	10 (66.7)
Previously Treated with Nomlabofusp n (%)						
Yes	1 (25.0)	3 (33.3)	4 (30.8)	0	1 (10.0)	1 (6.7)
No	3 (75.0)	6 (66.7)	9 (69.2)	5 (100.0)	9 (90.0)	14 (93.3)

Larimar Therapeutics

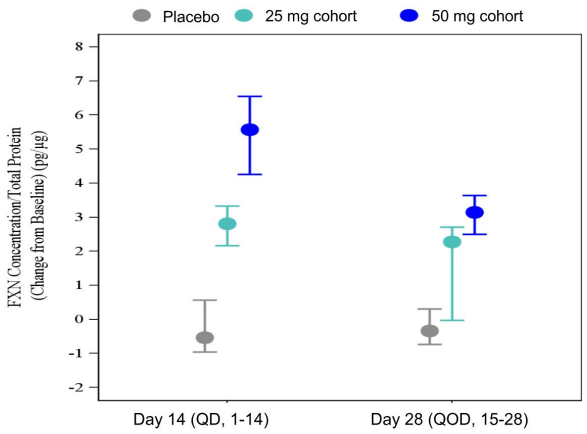
Disease Characteristics – Phase 2 Study

	25 mg Cohort			50 mg Cohort		
	Placebo *N = 4*	Nomlabofusp *N = 9*	Overall *N = 13*	Placebo *N = 5*	Nomlabofusp *N = 10*	Overall *N = 15*
Age at Symptom Onset (Years)						
Mean (SD)	14.5 (4.93)	13.0 (10.47)	13.5 (8.77)	15.2 (7.26)	13.7 (8.37)	14.2 (7.78)
Median	14.5	10	11	14	12.5	14
Q1, Q3	11, 19	8, 13	9, 15	11, 16	7, 18	7, 18
Min, Max	9, 20	5, 38	5, 38	8, 27	5, 30	5, 30
Age at Diagnosis (Years)						
Mean (SD)	17.5 (5.57)	18.6 (11.20)	18.2 (9.58)	18.6 (6.80)	16.6 (8.03)	17.3 (7.46)
Median	16.5	16	16	19	13.5	14
Q1, Q3	14, 22	14, 20	14, 20	13, 20	10, 21	12, 21
Min, Max	12, 25	5, 42	5, 42	12, 29	9, 30	9, 30
Time Since Diagnosis (Years)						
Mean (SD)	16.1 (5.97)	18.5 (11.52)	17.8 (9.94)	9.5 (3.72)	11.9 (7.05)	11.1 (6.10)
Median	13.42	14.32	13.5	11	11.26	11
Q1, Q3	12.9, 19.3	12.8, 21.6	12.8, 21.6	5.8, 11.3	7.4, 15.3	5.8, 15.2
Min, Max	12.5, 25.0	5.4, 45.0	5.4, 45.0	5.6, 14.0	2.3, 25.1	2.3, 25.1

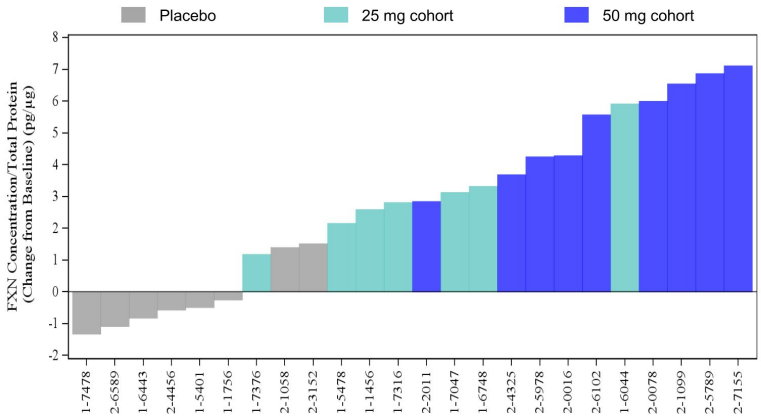
Dose-Dependent Increase in FXN Levels in Skin Cells

Participants dosed daily for 14 days, then every other day until day 28

Skin Cells
FXN Levels* Change from Baseline**



FXN Levels* in Skin Cells
Change from Baseline at Day 14



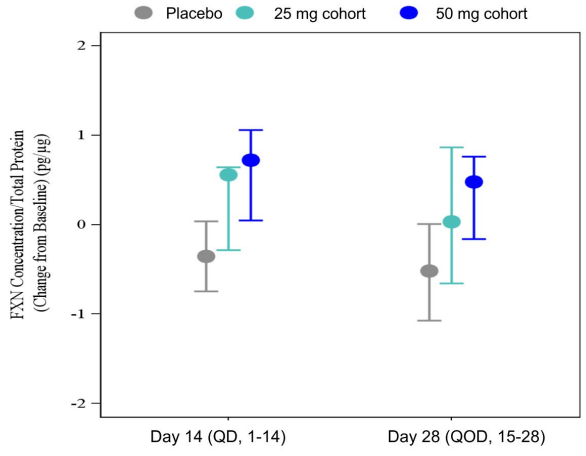
Larimar Therapeutics

*FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations are normalized to total cellular protein content in each sample. Data represent median and 25th and 75th percentiles. Only participants with quantifiable levels at both baseline and Day 14 are included in the figures.
**Median baseline FXN levels in patients were 3.5 pg/µg for the placebo, 3.7 pg/µg for the 25 mg cohort and 2.1 pg/µg for the 50 mg cohort.

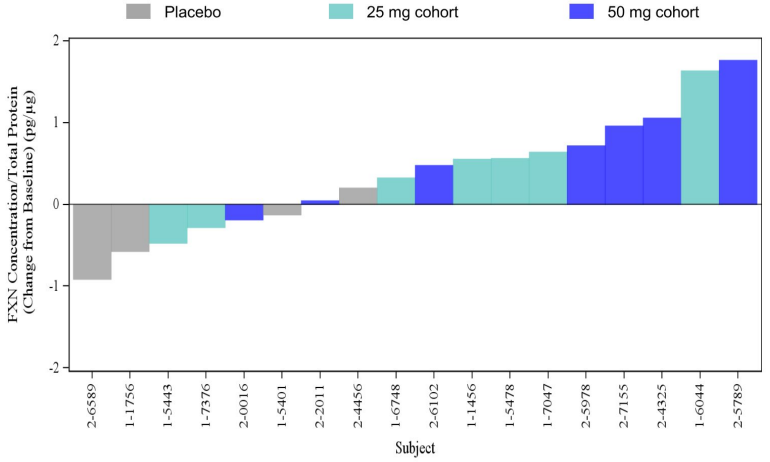
Dose-Dependent Increase in FXN Levels in Buccal Cells

Participants dosed daily for 14 days, then every other day until day 28

Buccal Cells
FXN Levels* Change from Baseline**



FXN Levels* in Buccal Cells
Change from Baseline at Day 14



*FXN levels measured via detection of peptide derived from mature FXN; FXN concentrations are normalized to total cellular protein content in each sample. Data represent median and 25th and 75th percentiles. Only participants with quantifiable levels at both baseline and Day 14 are included in the figures.
**Median baseline FXN level in patients were 2.1 pg/µg for the placebo, 1.8 pg/µg for the 25 mg cohort and 1.6 pg/µg for the 50 mg cohort.

Absolute Increases in Skin FXN Levels

Dose response in tissue FXN concentrations and increases from baseline after dosing

Day 14 Skin FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	3.70	3.38
	Day 14	5.53	6.40
	Change from Baseline	**2.81**	**3.02**
50 mg	Baseline	2.12	2.08
	Day 14	7.40	7.32
	Change from Baseline	**5.57**	**5.24**

Day 28 Skin FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	3.70	3.38
	Day 28	4.39	4.80
	Change from Baseline	**2.28**	**1.41**
50 mg	Baseline	2.12	2.08
	Day 28	5.23	5.24
	Change from Baseline	**3.14**	**3.17**

Larimar Therapeutics

Only participants with quantifiable levels at baseline and day 14 and day 28 are included in the tables.

Absolute Increases in Buccal FXN Levels

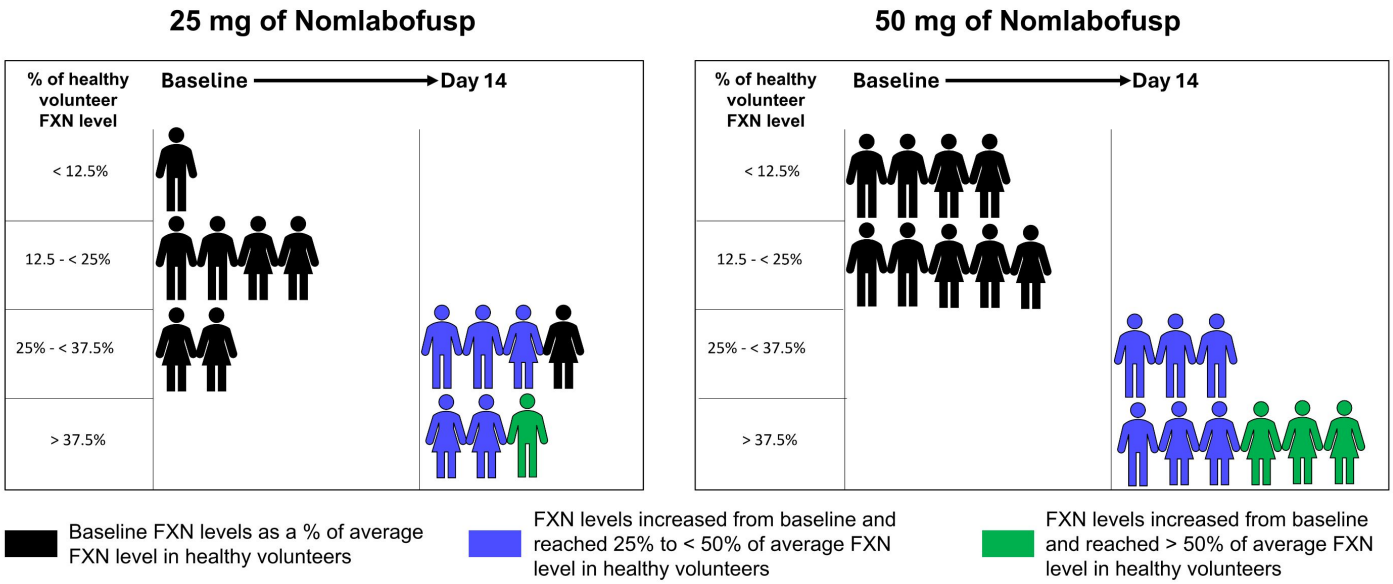
Dose response in tissue FXN concentrations and increases from baseline after dosing

Day 14 Buccal FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	1.78	1.80
	Day 14	2.24	2.22
	Change from Baseline	**0.56**	**0.42**
50 mg	Baseline	1.61	1.69
	Day 14	2.44	2.38
	Change from Baseline	**0.72**	**0.69**

Day 28 Buccal FXN Levels			
Dose	Visit	Absolute Values (pg/µg)	
		Median	Mean
25 mg	Baseline	1.70	1.65
	Day 28	1.73	1.76
	Change from Baseline	**0.03**	**0.11**
50 mg	Baseline	1.76	1.77
	Day 28	2.15	2.15
	Change from Baseline	**0.48**	**0.38**

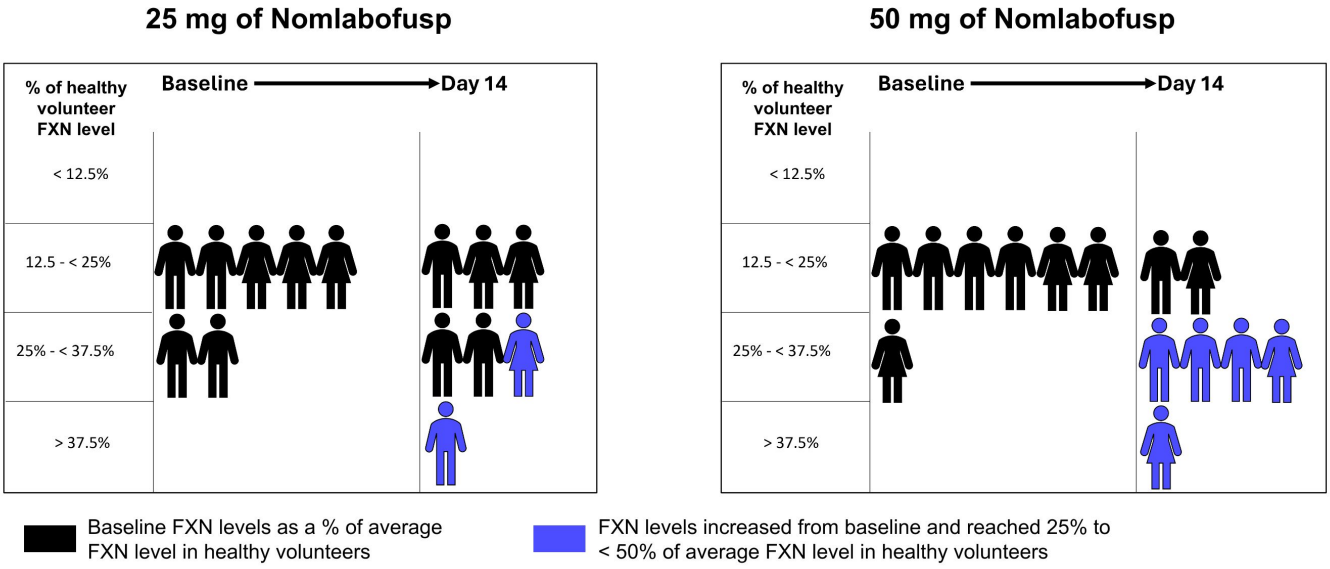
Only participants with quantifiable levels at baseline and day 14 and day 28 are included in the tables.

Skin Cell FXN Levels Achieve Higher % of Healthy Volunteers* Following 14 days of Daily Nomlabofusp



Only participants with quantifiable levels at baseline and day 14 are included in the figures.
*% of healthy volunteer FXN level is calculated by dividing each participant's FXN level by the average FXN level (16.34 pg/µg) from the noninterventional healthy volunteer study (N=60).

Buccal Cell FXN Levels Achieve Higher % of Healthy Volunteers* Following 14 days of Daily Nomlabofusp



Only participants with quantifiable levels at baseline and day 14 are included in the figures.
*% of healthy volunteer FXN level is calculated by dividing each participant's FXN level by the average FXN level (8.24 pg/µg) from Larimar's noninterventional healthy volunteer study (N=60).

Increasing FXN Levels May Slow Disease Progression

Disease Characteristics* Based on Literature**	Nomlabofusp Administration in Phase 2 Study
Patients with FXN levels 11% of average healthy volunteers • Median age of onset at 7 years • Deteriorate by 2.9 points/year as measured by FARS • Lose ambulation at a median of 11.5 years	**25 mg daily for 14 days shifted FXN levels in** • All but one patient to > 25% of average healthy volunteers in skin cells with a median value of 33.9%
Patients with FXN levels > 30% of average healthy volunteers • Median age of onset at 16 years • Deteriorate by 2.0 points/year as measured by FARS • Lose ambulation at a median of 18.3 years	**50 mg daily for 14 days shifted FXN levels in** • All patients from < 25% of average healthy volunteers to 33% to 59% (3 patients > 50%) in skin cells with a median value of 45%

H.L.Plasterer et al. PLoS ONE 2013 8(5):e63958; C. Rummey et al. EClinicalMedicine. 2020 18:100213

Larimar Therapeutics

*FARS: Friedreich's ataxia rating scale, measures disease progression with a higher score indicating a greater level of disability.
**FXN levels measured in peripheral blood mononuclear cells (PBMCs). FXN levels as measured by % of normal demonstrated to be equivalent in PBMCs, buccal cells, and whole blood.

Encouraging Therapeutic Potential for Nomlabofusp

Frataxin deficiency is the root cause of the disease

Lower levels of frataxin correlate with disease burden

Animal models show that increasing frataxin mitigates clinical outcomes

Dose-dependent increases in frataxin levels with nomlabofusp in several studies

Continue nomlabofusp clinical development

Nomlabofusp: Predictable Pharmacokinetics

1. Quick absorption after subcutaneous administration
2. Dose-proportional increases in exposure observed
3. Pharmacokinetic profile consistent with Phase 1 studies

Larimar Therapeutics

Ph1 & Ph2 Data: Nomlabofusp is Generally Well Tolerated

61 patients have participated in our Phase 1 and Phase 2 studies with no serious adverse events in any nomlabofusp clinical study. One severe adverse event (allergic reaction that resolved with standard treatment referenced below).

44 of 46 clinical trial participants dosed with nomlabofusp completed their respective study
One Phase 2 participant in the 25 mg cohort withdrew due to allergic reaction that resolved with standard treatment
One Phase 1 participant in the 50 mg cohort withdrew due to mild-to-moderate nausea and vomiting

Most common adverse events (AEs) were mild and moderate injection site reactions (ISRs)
No study discontinuations due to ISRs and all resolved

Open-label Extension Study: Initiated Q1 2024

Preliminary interim data expected in Q4 2024

Key Eligibility Criteria

Previous participation in Phase 1 or Phase 2 trials

Daily subcutaneous injection of 25 mg nomlabofusp;
self-administered or by a caregiver

1 site initiated and screening has begun

Key Study Objectives

- Safety and tolerability
- Long-term PK
- Tissue FXN concentrations and potential use as surrogate endpoint to support accelerated approval
- Clinical efficacy measures compared to the matched set of untreated patients from FACOMS* database

Screening Period ≤ 42 days** → Treatment Period Planned for ≥ 1 year → Potential extensions

Larimar Therapeutics

*FACOMS: Friedreich's Ataxia Clinical Outcome Measures Study.
**Estimated screening period may be extended for those study participants who have not been on a stable regimen of omaveloxolone for at least six months.

Nomlabofusp Clinical Development Plan

Intend to pursue accelerated approval pathway with potential BLA submission targeted for 2H 2025

Ongoing open-label extension study with 25 mg daily dosing for eligible patients who participated in SAD, MAD, and/or four-week dose exploration studies

Initial data expected Q4 2024

Planned pediatric MAD trial in patients 2 to 17 years of age*

Participants eligible to screen for OLE trial

Planned global double-blind placebo-controlled registration/confirmatory study**

BLA submission targeted for 2H 2025

*Company is discussing with FDA how to best include patients 2 to 17 years of age in clinical development.
**Company initiated discussions with FDA on the role of FXN levels to support accelerated approval. Also, the Company is planning discussions with regulators and investigators outside the U.S. to expand clinical program to international geographies.
Initiation of additional U.S. clinical trials is contingent on FDA review of clinical data due to partial clinical hold.

Positive Topline 50 mg & 25 mg Ph 2 Data and OLE Initiated in Q1 2024

Consistent Ph 1 and Ph 2 Findings	Nomlabofusp is generally well tolerated at doses tested up to 4 weeks Dose-dependent increases in FXN levels from baseline in evaluated tissues (skin and buccal cells) Baseline FXN levels in skin cells in the 50 mg cohort were < 17% of the average of healthy volunteers. After daily dosing for 14 days, FXN levels increased to 33% to 59%
Regulatory Updates	Initiated discussions with FDA regarding use of FXN as a surrogate endpoint to support accelerated approval Intend to pursue accelerated approval with potential BLA submission for 2H 2025 Beginning preparations to expand nomlabofusp clinical program to ex-U.S. geographies
Expected Milestones	**Q1 2024:** Dosing of first patient in OLE study **Q4 2024:** Initial data from OLE study; initiated in Q1 2024 **2H 2024:** Final Phase 2 data planned to be presented at a conference **2H 2025:** BLA submission

Larimar Therapeutics