Lexeo Therapeutics Corporate Overview

April 2024

Forward-Looking Statements

This Presentation contains forward-looking statements made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. These statements are based on the beliefs and assumptions and on information currently available to the management of Lexeo Therapeutics, Inc. (the "Company" or "Lexeo"). These statements may be identified by words such as “aims,” “anticipates,” “believes,” “could,” “estimates,” “expects,” “forecasts,” “goal,” “intends,” “may” “plans,” “possible,” “potential,” “seeks,” “will,” and variations of these words or similar expressions that are intended to identify forward-looking statements. All statements other than statements of historical fact contained in this presentation are forward-looking statements, including statements concerning: the timing, progress and results of preclinical and clinical trials of Lexeo’s gene therapy product candidates, expected cash runway, and the completion of the equity financing announced in March 2024. Forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause Lexeo’s actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. This information was factually accurate on the date it was published.

The safety and efficacy of Lexeo’s product candidates and/or uses under investigation have not been established. There is no guarantee that any of the Company’s product candidates will receive regulatory authority approval or become commercially available in any country for the uses being investigated or that any such product candidate will achieve a particular revenue level. In particular, Lexeo’s expectations could be affected by, among other things, uncertainties involved in the development of new therapeutic products; unexpected clinical trial results or unexpected new clinical data; unexpected regulatory actions or delays or government regulation generally; Lexeo’s ability to obtain or maintain patent or other proprietary intellectual property protection; competition in general; and government, industry, and general public pricing and other political pressures. The actual results may vary from the anticipated results and the variations may be material. Other factors that may cause the Company’s actual results to differ from current expectations are discussed in the Company’s filings with the SEC, including the sections titled “Risk Factors,” “Management’s Discussion and Analysis of Financial Condition and results of Operations” and “Special Note Regarding Forward-Looking Statements” in the Company’s Annual Report on Form 10-K for the annual period ended December 31, 2023, filed with the SEC on March 11, 2024, as well as other filings the Company may make with the SEC in the future. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this presentation is given. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Except as required by law, Lexeo undertakes no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

This Presentation does not constitute an offer to sell or a solicitation of an offer to buy securities, nor shall there be any sale of any securities in any state or jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction.

LEXEO therapeutics

Lexeo Therapeutics Team

Management Team & Key Advisors

R. Nolan Townsend
Chief Executive Officer

Pfizer · Lehman Brothers

Eric Adler, M.D.
Chief Medical Officer and
Head of Research

UC San Diego School of Medicine Division of Cardiology · Boston University

Sandi See Tai, M.D.
Chief Development Officer

Pfizer · Wyeth

Jenny R. Robertson
Chief Business and Legal Officer

Pfizer · Hogan Lovells

Jordan Shin, M.D., Ph.D.
SVP, Clinical Development and
Translational Science, Cardiology

Renovacor · Lung Biotechnology · MGH Massachusetts General Hospital

Rajiv Patni, M.D.
Senior Advisor to the CEO
and Board of Directors

REATA Pharmaceuticals · GBT · Portola

Chair and Scientific Founder

Steven Altschuler, M.D.
Chairman

The Children's Hospital of Philadelphia · Spark Therapeutics

Ronald Crystal, M.D.
Founder & Chief Scientific Adviser

Weill Cornell Medicine Genetic Medicine

Professor and Chairman, Weill Cornell Medicine
Director, Belfer Gene Therapy Core Facility

Former Chief,
Pulmonary Branch
NIH National Heart, Lung, and Blood Institute

Founder / Co-founder Adverum Biotechnologies · GenVec · XyloCor Therapeutics

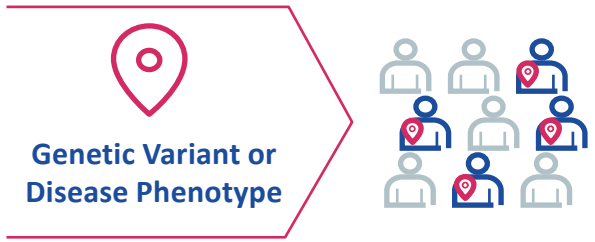
Management team with broad leadership experience in gene therapy and rare disease

LEXEO therapeutics

Revolutionizing Genetic Medicines for Cardiovascular Diseases and APOE4-Alzheimer's

Attractive Disease Area Strategy

- Genetically-defined cardiovascular and APOE4-associated Alzheimer's disease
- Well established biomarkers potentially allowing for early signs of clinical activity



Evolving Regulatory Environment	Shift towards surrogate endpoints could circumvent need for large cardiovascular outcome trials
Targeted Delivery Platform	Improvements in modern AAV delivery technology, including AAVrh10 allows for greater targeting of the heart
Increased Genetic Screening	Increased screening has potential to expand awareness and increase opportunity
Lower Efficacy for APOE4 Patients[1]	APOE4 homozygotes demonstrated lower efficacy results compared to heterozygotes and noncarriers
Higher Risk of ARIA-E[1]	APOE4 homozygotes were associated with a higher incidence of ARIA-E compared to heterozygotes and noncarriers

Focused on genetically-defined cardiovascular diseases
with data from Alzheimer's disease driving business development

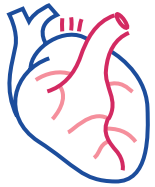
Note: ARIA-E = amyloid-related imaging abnormalities characterized by edema and effusion.

(1) Van Dyck, et al. New England Journal of Medicine, 2022. Sims, et al. JAMA, 2023.

LEXEO therapeutics

Lexeo Investment Highlights

Clinical-stage genetic medicine company addressing larger-rare and prevalent patient populations



Cardiac Portfolio

- Ongoing Phase 1/2 trial for **Friedreich's ataxia cardiomyopathy**, with early clinical data demonstrating potential clinical benefit
- Cleared IND for LX2020 for the treatment of **arrhythmogenic cardiomyopathy caused by mutations in the PKP2 gene (PKP2-ACM)**
- Potential to be one of the first genetic medicine companies **with data from two cardiac gene therapy programs in 2024**

APOE4 Alzheimer's Portfolio

- **Significant optionality on portfolio of approaches to treat the genetics underlying APOE4-associated Alzheimer's disease**; estimated 900,000 E4 homozygotes in the US
- **Observed a decline in CSF biomarkers** in initial clinical data from Cohort 1 of ongoing Phase 1/2 clinical study of LX1001
- Phase 1/2 data readout in 2H 2024 **potentially driving business development**

LEXEO therapeutics

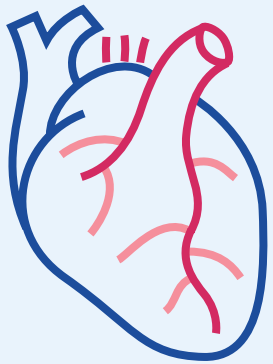
Advancing Genetic Medicines in Larger-Rare and Prevalent Patient Populations

	Programs:	Indication:	Gene of Interest:	Preclinical: Discovery	Preclinical: Preclinical	Clinical: Phase 1/2	Clinical: Phase 2/3	Upcoming Milestones:
Cardiovascular	LX2006	FA Cardiomyopathy	FXN	~5k US Pts				Mid-2024: Interim Data Readout
	LX2020	PKP2-ACM[1]	PKP2	~60k US Pts				1H 2024: First Patient Dosed 2H 2024: Interim Cohort 1 Data
	LX2021	DSP[2] Cardiomyopathy	CX43	~35k US Pts				2024: Initiate IND-Enabling Studies
	LX2022	Hypertrophic Cardiomyopathy	TNNI3	~25k US Pts				2024: Candidate Selection
APOE4-Associated Alzheimer's Disease	LX1001	Alzheimer's: APOE4 homozygotes	APOE2+	~900k US Pts				2H 2024: Phase 1/2 Data Readout
	LX1021	Alzheimer's: APOE4 homozygotes	Christchurch[3] APOE2+	~900k US Pts				2024: Pre-IND Meeting
	LX1020	Alzheimer's: APOE4 homozygotes	APOE2+ APOE4-	~900k US Pts				2024: Candidate Selection

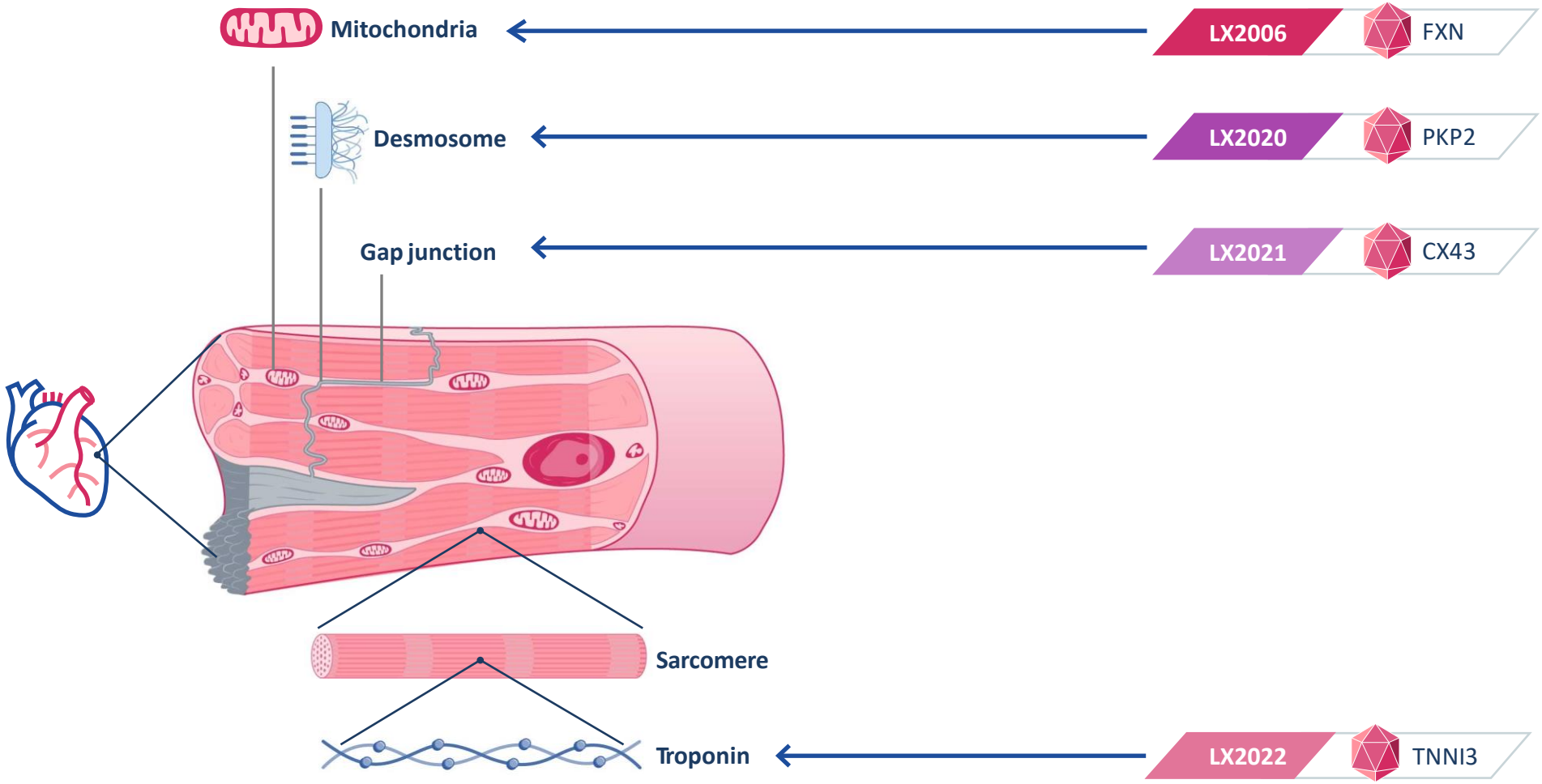
(1) Arrhythmogenic Cardiomyopathy
(2) Desmoplakin
(3) Christchurch Modified APOE2 gene

LEXEO therapeutics

Cardiovascular diseases



Several Targets in Cardiac Organelles that are Dysregulated in Cardiomyopathy; Potential Readthrough to Other Therapeutic Indications

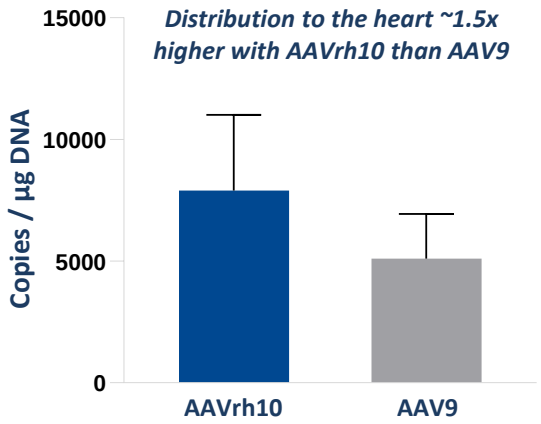


Utilizing AAVrh10 for Initial Genetic Cardiac Indications

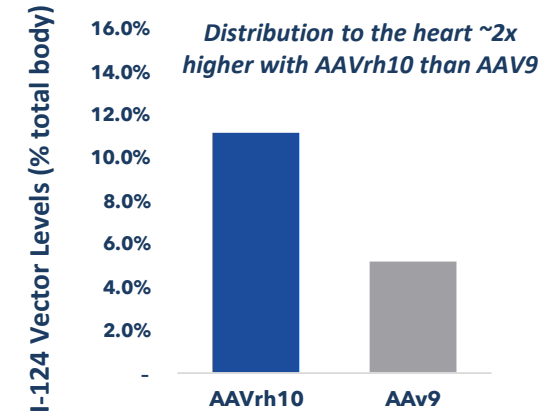
- ✓ Observed ~1.5x to 2.0x greater biodistribution in the heart compared to AAV9 in multiple large animal models
- ✓ Observed greater trends of functional improvements in PKP2-murine model compared to AAV9
- ✓ AAVrh10 cardiac tropism may allow for lower doses compared to other vector serotypes while achieving targeted transgene biodistribution

Compelling Cardiac Tropism

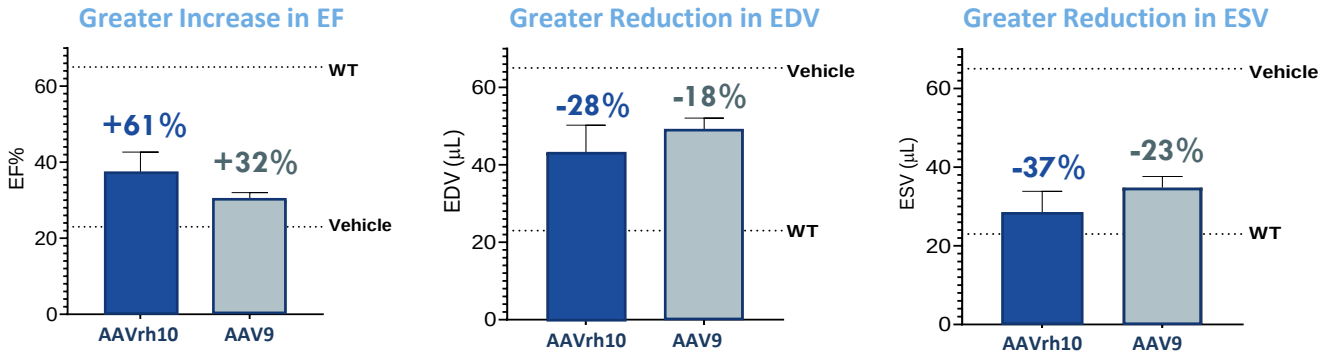
Yucatan Minipig Biodistribution(1)



NHP Biodistribution(2)



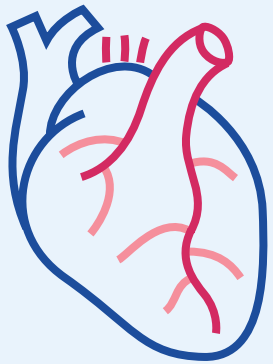
Greater Trends of Functional Improvement Versus AAV9 in PKP2-ACM Model(1)



Note: PKP2 homozygous mouse model administered with human PKP2 (N = 5 mice / group).

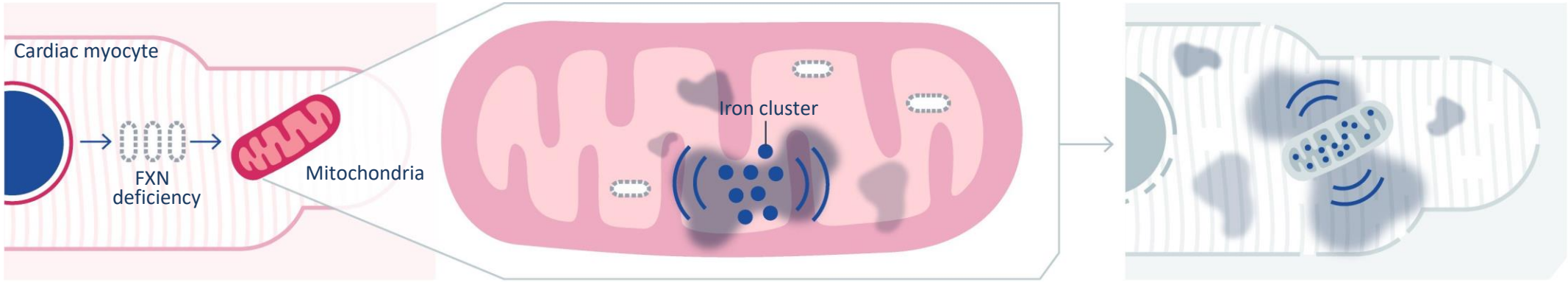
(1) Data presented at ASGCT 2023.
(2) Ballon DJ et al, Human Gene Therapy, 2020.

LX2006 (FA Cardiomyopathy)



Friedreich's Ataxia Cardiomyopathy and How LX2006 is Designed to Treat It

Disease mechanism

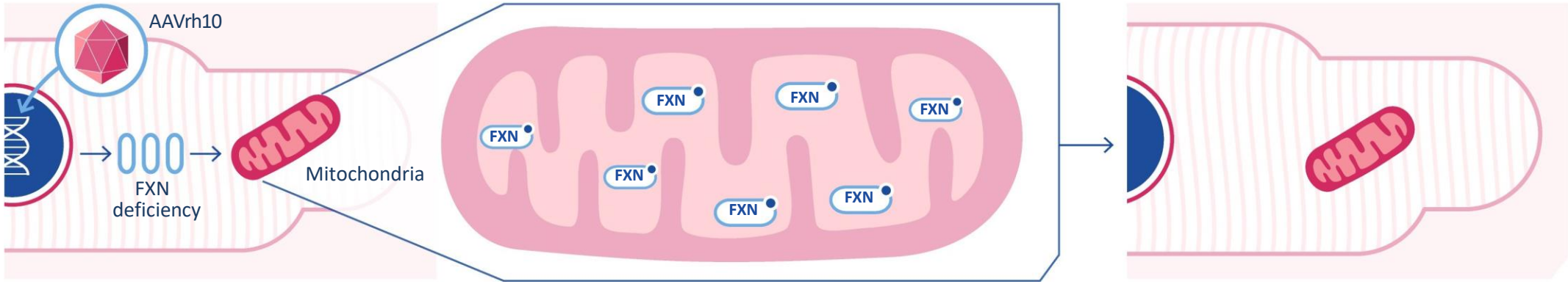


Frataxin deficiency in the mitochondria of myocardial cells

Disruption of normal iron-sulfur cluster biosynthesis

Results in mitochondrial dysfunction and deficient energy production in cardiac myocytes

LX2006 mechanism



AAV-mediated transfer of FXN gene to myocardial cells

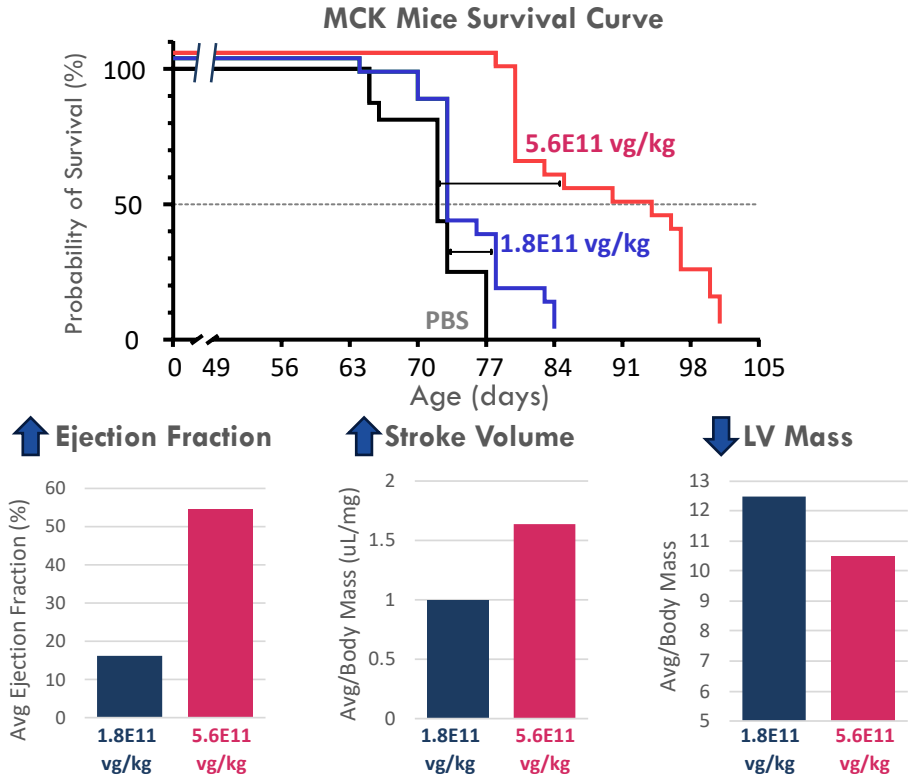
Intended to restore frataxin levels in mitochondria

Restored mitochondrial function and improved cardiac myocyte function

LEXEO therapeutics

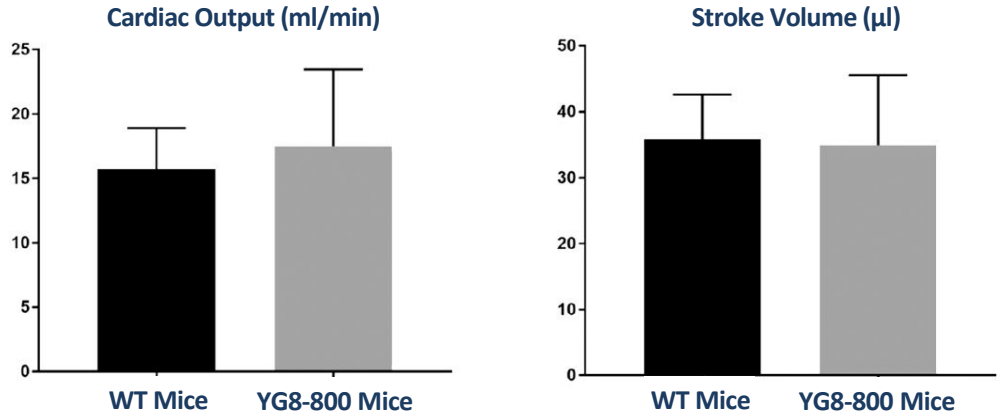
Preclinical Models Suggest Low Levels of FXN May be Sufficient for Physiological Improvement

1 LX2006 Dose-Escalation Resulted in Survival and Functional Improvement in MCK Preclinical Studies



Dose-escalation resulted in improvement in survival and cardiac function with no toxicity signals in preclinical MCK model

2 Recently Developed FA Murine Models Suggest Potential FXN Therapeutic Target Level



YG8-800 mouse, with approximately 800 GAA repeats, has ~5% of normal FXN in the heart and displays near normal cardiac output and stroke volume(1)

(1) A promising mouse model for Friedreich Ataxia progressing like human patients, Behavioral Brain Research, 2023.

LX2006 Friedreich's Ataxia Phase 1/2 (SUNRISE-FA) Overview

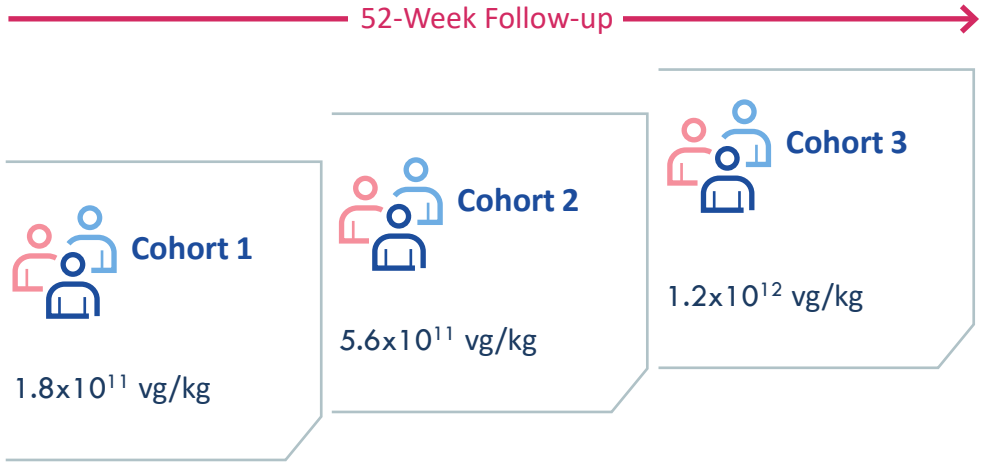
Key Features:

- 52-week, dose-ascending, open-label trial with long-term follow-up (5-years post dose)
- Vector: AAVrh10
- Route of Administration: systemic administration
- Immune Suppression: prednisone

Key Inclusion Criteria:

- Adults with FA identified based on genotyping
- Left ventricular hypertrophy / EKG changes
- Ejection fraction ≥40%

Trial Design



Endpoints:

Primary Endpoint: Safety

Additional Endpoints:

- **Cardiac biopsy: FXN expression**(1)
- **Cardiac imaging: Structure + function**
- **LV hypertrophy**
- Cardiac strain
- Ejection fraction
- Symptoms during CPET
- CPET Peak VO2
- Cardiac arrhythmias
- Cardiac serum biomarkers
- FA neurologic scales

Patients Treated with LX2006 Across Trials(2), as of April 22, 2024

Dose	Combined Enrollment Update and Months of Follow-up		
	>12 Months	6-12 Months	<6 Months
Cohort 1 $1.8x10^{11}$ vg/kg	3	3	-
Cohort 2 $5.6x10^{11}$ vg/kg	-	2	3

(1) Cardiac biopsies performed only in Lexeo sponsored SUNRISE-FA trial.
(2) Includes patients from Cornell sponsored IIT and Lexeo sponsored SUNRISE-FA trial. LX2006 mid-2024 data readout to include data set from both clinical trials.

LEXEO therapeutics

LX2006: We Believe the First and Only Clinical Stage Program to Demonstrate Increased FXN in Target Organ with Potential Early Clinical Benefit

	Cohort 1 ($1.8x10^{11}$ vg/kg)			Cohort 2 ($5.6x10^{11}$ vg/kg)		
	Hypertrophy	**Troponin**	**CPET**	**Hypertrophy**	**Troponin**	**CPET**
Cardiac Biomarkers[1]	Average LVMI improvement of 9g/m^2 or ~10% (n=2)	Average reduction of 41% (n=2)	Improvement in peak VO2 of 43% (n=1)[2]	Cohort 2 biomarker data expected in mid-2024 readout		
	LCMS	**IHC**		**LCMS**	**IHC**	
Cardiac Tissue Biopsy Analyses	Increase of 0.22 ng/mg (+29% from baseline) (n=1)	+65% increase in area stained from baseline (n=1)		Avg. increase of 1.10 ng/mg (+79% from baseline) (n=2)	IHC analysis expected in mid-2024 readout	

- ✓ We believe LX2006 is the first ever clinical stage program to show target organ increase in FXN protein
- ✓ Improvements in cardiac biomarkers observed in low dose Cohort 1 demonstrate potential of LX2006 to impact disease pathology
- ✓ Cardiac biopsy analysis demonstrated increase in FXN protein following treatment by two independent methods: LCMS (n=3) and IHC (n=1)
- ✓ To date, LX2006 has been well tolerated with no reported treatment-related serious adverse events

Note: LCMS = Liquid chromatography mass spectrometry, FXN = Frataxin, IHC = immunohistochemistry.

(1) Two subjects treated in Weill Cornell Medicine investigator initiated trial using product candidate referred to as LX2006 at Lexeo; results presented at Friedreich's Ataxia Research Alliance webinar hosted July 10, 2023.

(2) One subject was not evaluated for peak VO2 at the 6-month visit.

LEXEO therapeutics

Ongoing Study of AAVrh.10hFXN(1) at 1.8x10^{11} vg/kg Dose: Early Signs of Potential Clinical Benefit

	Left Ventricular Mass Index (g/m^2)				High Sensitivity Troponin I (ng/L)			
Subject	Baseline (g/m^2)	6 Month (g/m^2)	Nominal Change	Percent Change	Baseline (ng/L)	6 Month (ng/L)	Nominal Change	Percent Change
Subject 1 (Male)	109	98	**-11** ↓	**-10%** ↓	148	39	**-109** ↓	**-74%** ↓
Subject 2 (Female)	81	73	**-8** ↓	**-10%** ↓	224	203	**-21** ↓	**-9%** ↓

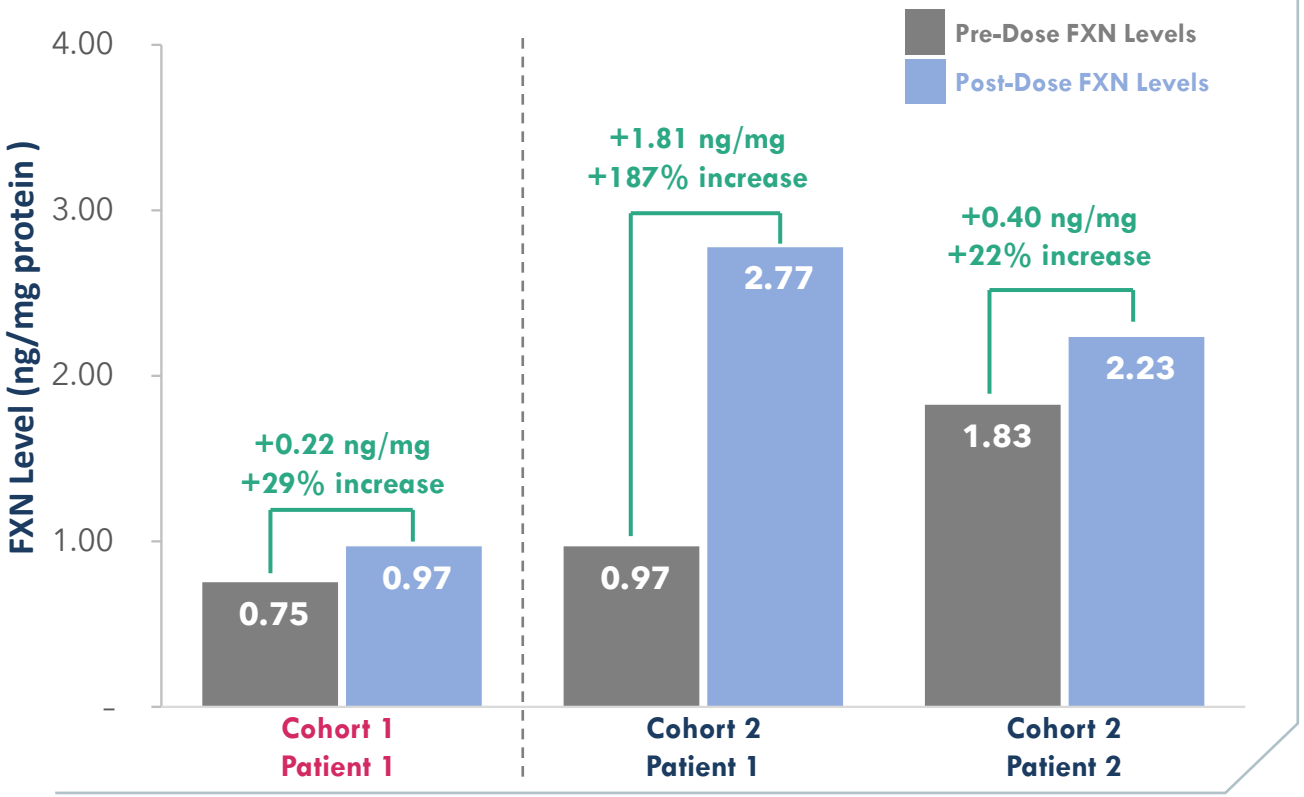
- Observed reduction in both LVMI and Troponin at interim 6-month visit from ongoing AAVrh10hFXN clinical trial (NCT05302271)
- (Male) Upper limit of Normal LVMI / Troponin: 93 g/m^2 / 58 ng/L
- (Female) Upper limit of Normal LVMI / Troponin: 77 g/m^2 / 40 ng/L
- Subject 2 demonstrated an approximately 43% increase in peak VO2 as measured by CPET at 6-month visit(2)

(1) Both subjects treated in Weill Cornell Medicine investigator initiated trial using product candidate referred to as LX2006 at Lexeo; results presented at Friedreich's Ataxia Research Alliance webinar hosted July 10, 2023.
(2) Subject 1 was not evaluated for peak VO2 at the 6-month visit.

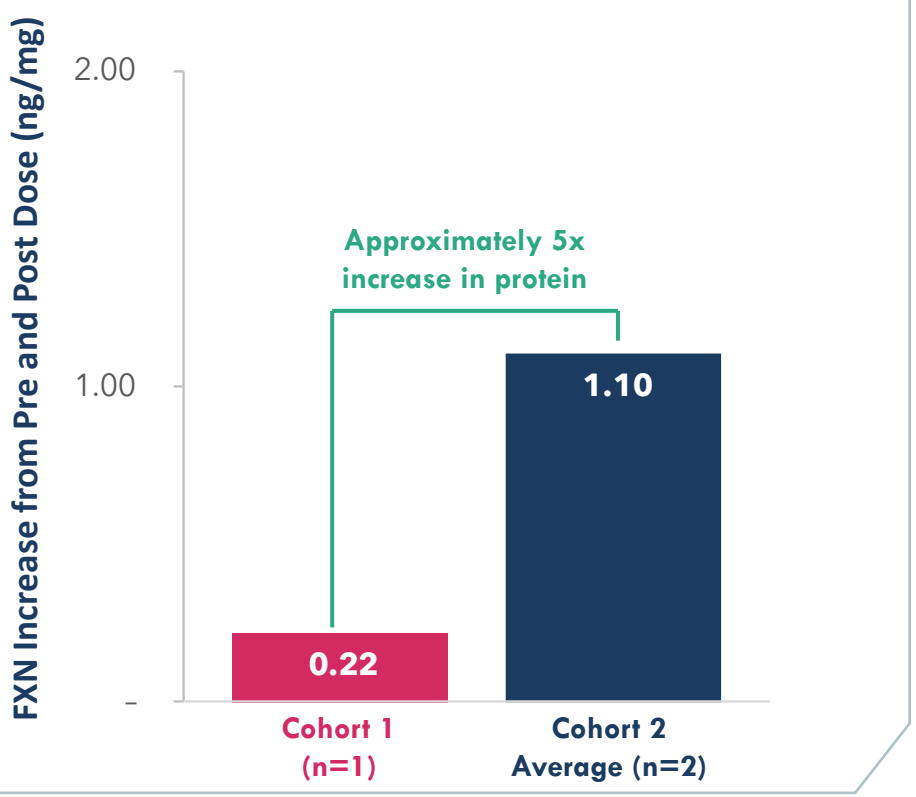
LEXEO therapeutics

Cohort 2 Cardiac Biopsies Have Demonstrated Increased Frataxin Expression in Target Organ and Dose-Response Between Cohorts

Post-Treatment FXN Levels Increased in All Patients



Observed Dose Response Between Cohorts



- Observed increase in FXN levels as measured by LCMS relative to pre-treatment baseline levels in all patients evaluated to date via cardiac biopsies (n=3)
- Observed an approximately 5x increase in protein on average in Cohort 2 relative to Cohort 1
- Pre-treatment baseline FXN levels of approximately 2% in the heart relative to healthy controls(1)

Note: Frataxin levels measured by ultrahigh performance liquid chromatography-multiple reaction monitoring/mass spectrometry (UHPLC-MRM/MS). LCMS = Liquid chromatography mass spectrometry, FXN = Frataxin.
Note: Cohort 1 dose of $1.8x10^{11}$ vg/kg and Cohort 2 dose of $5.6x10^{11}$ vg/kg.
(1) Lexeo data on file from healthy cadaver samples (n=29 tissue samples from 16 patients) with mean and median FXN levels of 56.97 ng/mg and 50.84 ng/mg, respectively.

LEXEO therapeutics

Regulatory Precedents for Select Functional Cardiac Measures and Protein Expression Observed to Date

Select Regulatory Framework Precedents		LX2006 Cohort 1 Observations
Left Ventricular Mass Index	• Various approved therapies for hypertension – 5g/m^2 has been noted as an important threshold[1]	**Improved (2 of 2)[4]** **Average improvement of 9g/m^2**
Troponin I	• RP-A501 (Danon disease)[2] – Decrease in Troponin I (secondary endpoint in registrational trial) • Mavacamten (obstructive HCM) – Decrease in Troponin I versus placebo (supportive data)[3]	**Improved (2 of 2)[4]** **Average Troponin I decline of 41%**
Peak VO2 (CPET)	• Mavacamten (obstructive HCM)[3] – Improvement in Peak VO2 versus placebo (part of composite endpoint)	**Improved (1 of 1) [4]** **Improvement of 43%**
Protein Expression	• SRP-9001 (Duchenne Muscular Dystrophy) – Protein expression utilized for accelerated approval pathway	**Protein Increase (3 of 3)[5]** **Increase observed in all patients**

Regulatory precedents suggest potential composite endpoint of cardiac functional measures and increased protein expression

Note: Prior regulatory frameworks and approved therapies may not be indicative of the regulatory process for our gene therapy candidates

(1) Lønnebakken et al, Left Ventricular Hypertrophy Regression During Antihypertensive Treatment in an Outpatient Clinic, JAHA, 2017.

(2) RP-A501 has not completed registrational studies.

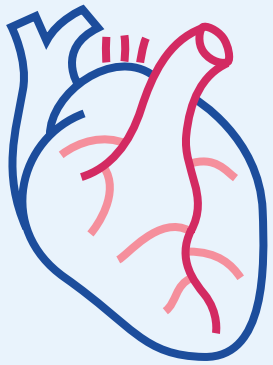
(3) Camzyos (mavacatmen) US Prescribing Information accessed August 2023.

(4) Both subjects treated in Weill Cornell Medicine Investigator Initiated Trial; results presented at Friedreich's Ataxia Research Alliance webinar hosted July 10, 2023.

(5) Includes Cohort 1 and Cohort 2.

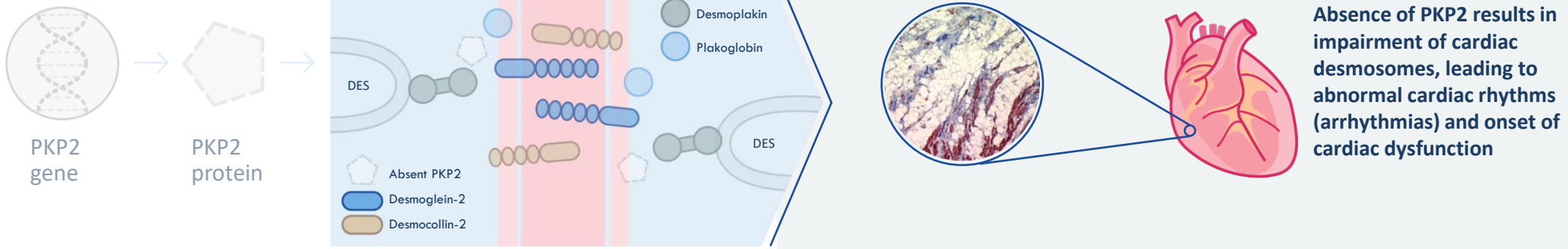
LEXEO therapeutics

LX2020 (PKP2-ACM)



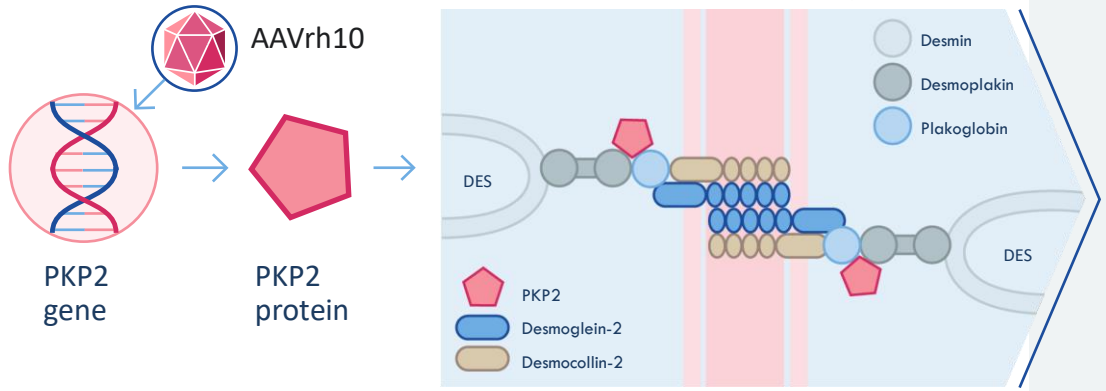
ACM Caused by Mutations in PKP2 and How LX2020 is Designed to Treat It

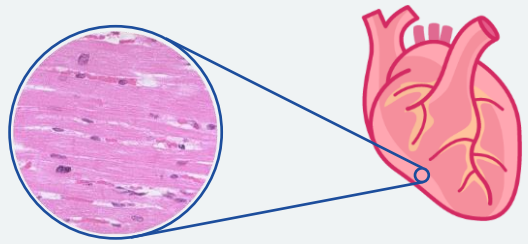
Disease mechanism



Absence of PKP2 results in impairment of cardiac desmosomes, leading to abnormal cardiac rhythms (arrhythmias) and onset of cardiac dysfunction

LX2020 mechanism





Delivering a functional PKP2 gene to cardiac muscle to increase desmosomal PKP2 protein levels, reassemble desmosomes and restore myocardial cell function

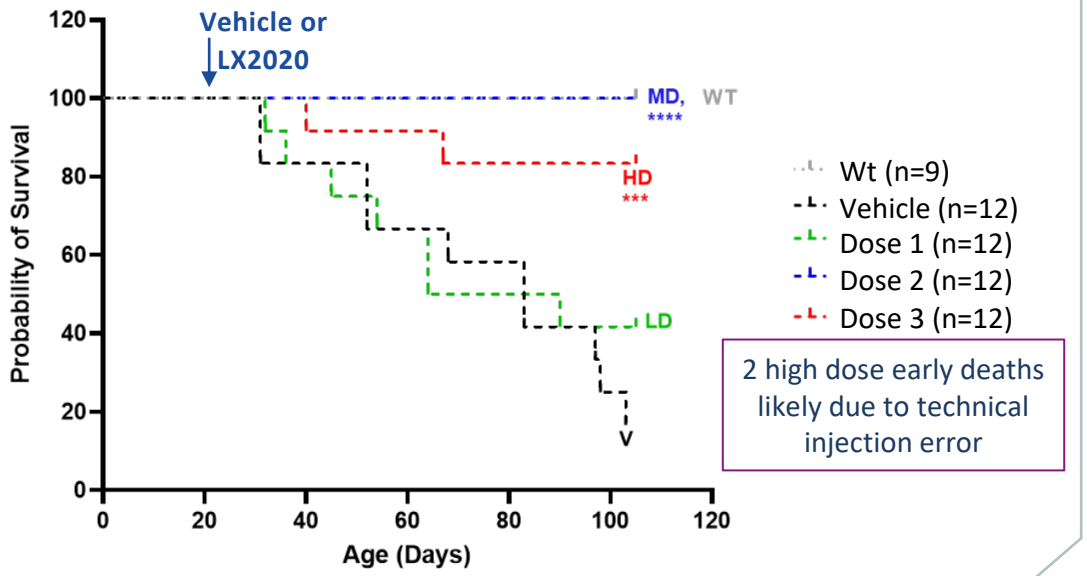
LEXEO therapeutics

IND Clearance Supported by Robust Preclinical Package

Robust Preclinical Package

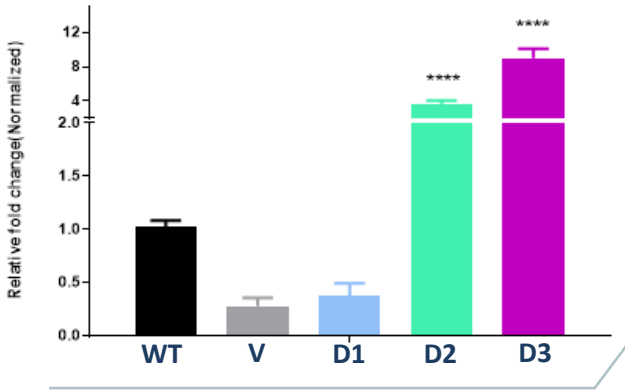
- Murine studies utilizing CRISPR-Cas 9 edited model recapitulating PKP2-ACM disease features
- NHP safety study showed no toxicity at highest evaluated dose levels (low x10^{14} vg/kg)

LX2020 Significantly Extended Survival in Severe Mouse Model

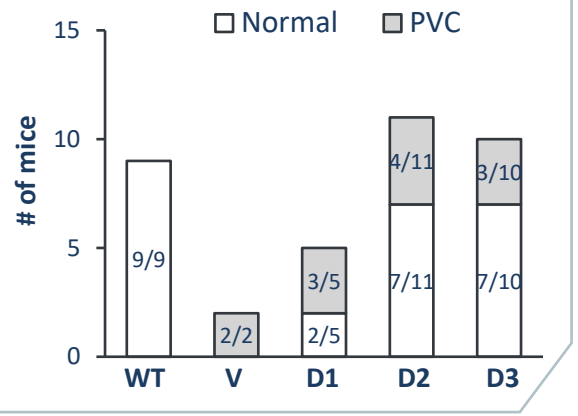


2 high dose early deaths likely due to technical injection error

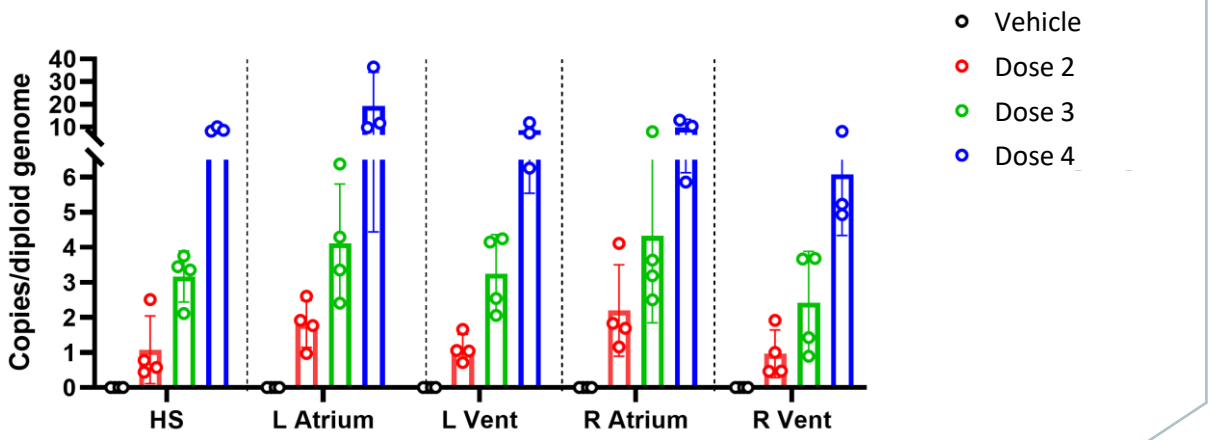
Quantification of PKP2 Expression in Severe Mouse Model



PVC Analysis in Severe Mouse Model



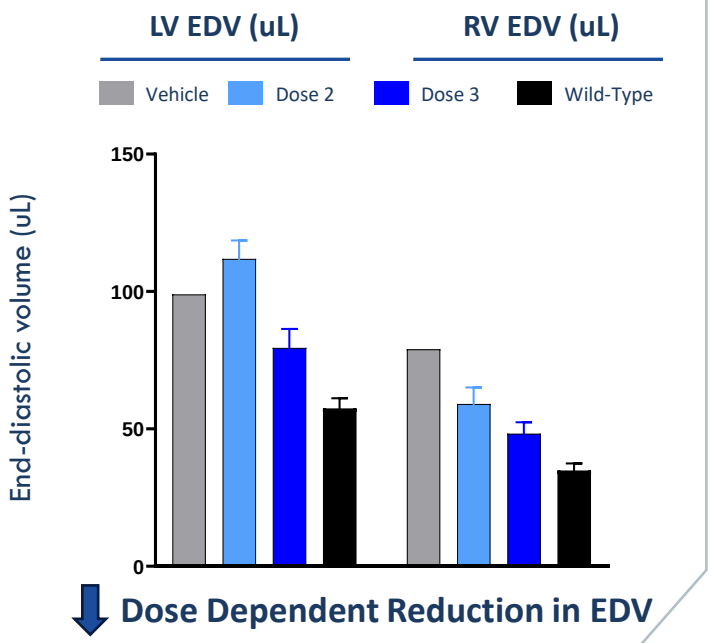
IND-Enabling NHP: VCN in Various Heart Regions



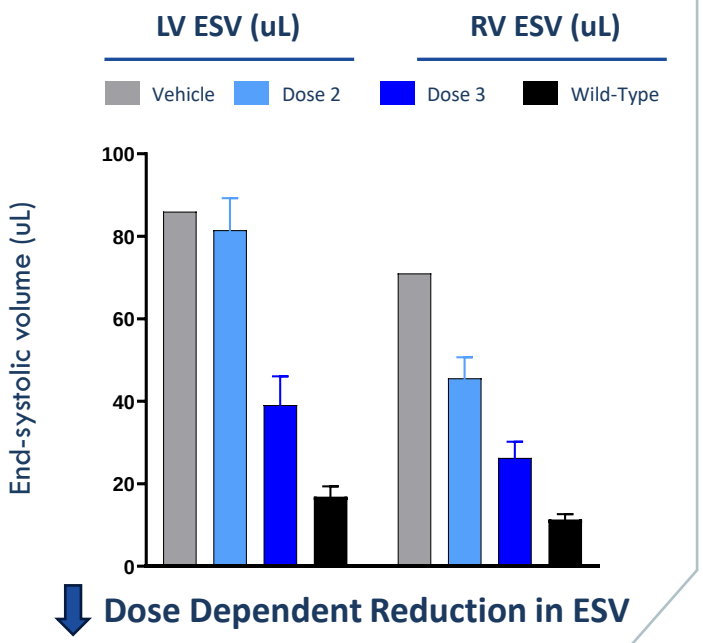
Note: PVC = premature ventricular contractions; VCN = vector copy number.

Quantitative MRI Analysis Showed Improvement in Cardiac Function in Homozygous Mouse Model

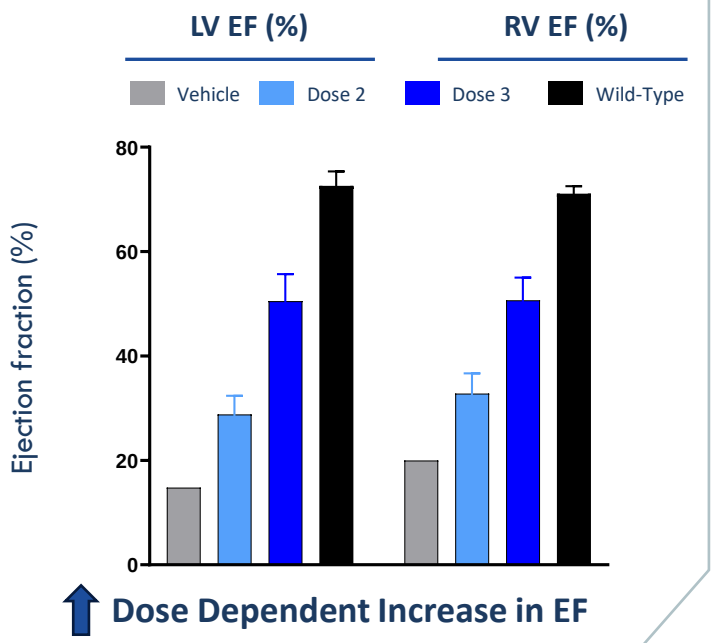
End-Diastolic Volume



End-Systolic Volume



Ejection Fraction



Quantitative MRI showed improved LV and RV ejection fraction and cardiac function in mid and high dose groups. RV improvements are most relevant as it is the primary ventricle impacted in PKP2-ACM

Note: LV = left ventricle; RV = right ventricle.

In Preclinical Studies LX2020 Successfully Impacted All Modifiable Elements of ACM Diagnosis and Risk Calculator

		LX2020 Preclinical Evidence
Arrhythmias	**Arrhythmia Burden** Daily Premature Ventricular Contraction (PVC) Count	↓ **Ectopic Beats** (7/10 without PVC)
	Life-threatening Arrhythmia Events SCD, ICD Shocks, VT/VF Events	↑ **Survival** (100%)
Repolarization & Depolarization	**Depolarization/Repolarization Abnormalities** T-wave Inversions/ QRS Complex	↓ **QRS Interval** (18% reduction)
Cardiac Structure & Function	**Cardiac Contractility** RV Dysfunction and Enlargement	↑ **Cardiac Fxn/EF** ↓ **Cardiac Dilation**
	Cardiac Structure/Function Myocardial Tissue Integrity (Fibrosis, Calcifications, Fragility)	↓ **Fibrosis, Calcifications, & Tissue Tearing**

LX2020 preclinical data demonstrated improvement across key areas for determining ACM diagnosis and risk profile

Note: PVC = premature ventricular contractions; VCN = vector copy number.

LX2020 PKP2-ACM Phase 1/2 (HEROIC-PKP2) Overview

Key Features:

- 52-week, dose-ascending, open-label trial with long-term follow-up (5-years post dose)
- Vector: AAVrh10
- Route of Administration: systemic administration
- Immune Suppression: prednisone + rapamycin

Key Inclusion Criteria:

- Male or female 18-65 years of age
- Confirmed diagnosis of ACM with either 2010 Task Force Criteria or 2020 International Criteria for ACM as affected
- Documented PKP2 mutation
- Existing implantable cardioverter defibrillator (ICD) that is MRI compatible
- Minimum threshold of PVCs/24-hr

Trial Design

52-Week

Endpoints

Primary Endpoint: Safety

Additional Endpoints:

- Change in ventricular arrhythmias and associated clinical events
- Change in 12-lead ECG
- Change in cardiac MRI and ECHO
- Change in cardiac biomarkers (including troponin and BNP)
- Change in Patient Symptoms (NYHA Functional Class and PROs)
- Change in PKP2 cardiac transduction & protein expression (cardiac biopsy)

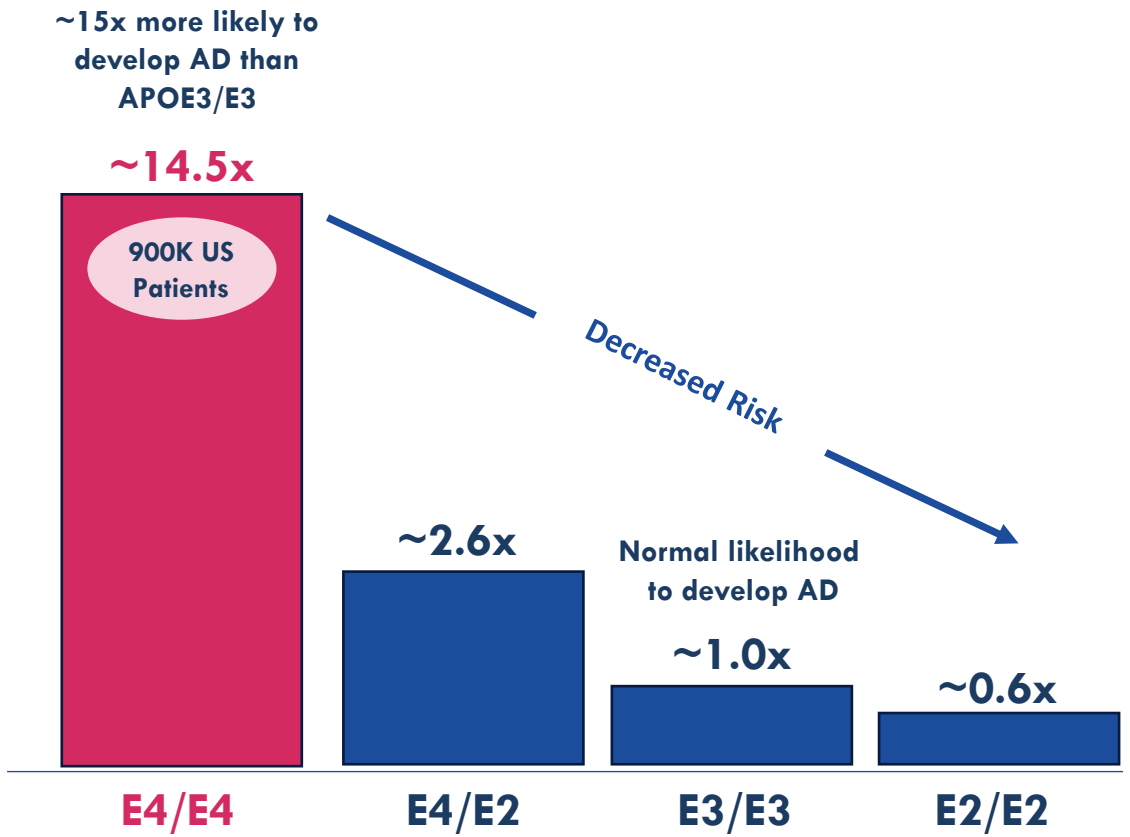
LEXEO therapeutics

LEXEO therapeutics

APOE4-Associated Alzheimer's Disease

APOE4 Homozygotes Represent a Patient Subgroup with Continued Unmet Need

Alzheimer's Disease Risk by APOE Genotype[1]



Unmet Need Persists Despite Recent Approvals

APOE4 homozygotes demonstrated lower efficacy results compared to heterozygotes and noncarriers and were associated with a higher incidence of ARIA-E

Reduced Efficacy[2]

CDR-SB[3]	APOE4 Noncarrier	APOE4 Heterozygote	APOE4 Homozygotes
Lecanemab	-0.75	-0.50	0.28
Donanemab	-0.76	-0.73	-0.41

Increased Incidence of Adverse Events[2]

ARIA-E	APOE4 Noncarrier	APOE4 Heterozygote	APOE4 Homozygotes
Lecanemab	5.4%	10.9%	32.6%
Donanemab	15.7%	22.8%	40.6%

Note: ARIA-E = amyloid-related imaging abnormalities characterized by edema and effusion.

(1) Yamazaki Y, et al. Nature Neurology Review, 2019.
(2) Van Dyck, et al. New England Journal of Medicine, 2022. Sims, et al. JAMA, 2023.
(3) Reduction in CDR-SB is improvement.

LEXEO therapeutics

LX1001 Phase 1/2 Trial in APOE4 Homozygotes

Key Features:

- 52-week, dose-ranging, open-label trial with long-term follow-up (5-years post dose)
- Vector: AAVrh10
- Route of Administration: C1-C2 (between cervical vertebrae 1 and 2) or intracisternal injection
- Immune Suppression: corticosteroids prior to treatment and tapering following dosing

Key Inclusion Criteria:

- ≥50 yr APOE4 homozygotes
- Mild cognitive impairment to moderate dementia with biomarkers consistent with Alzheimer's disease

Trial Design

52-Week Follow-up

Cohort 1	Cohort 2	Cohort 3	Cohort 4
n=5	n=4	n=3	n=3
$1.4x10^{10}$ vg/ml CSF	$4.4x10^{10}$ vg/ml CSF	$1.4x10^{11}$ vg/ml CSF	
$5.7x10^{12}$ Total VGs	$1.8x10^{13}$ Total VGs	$5.7x10^{13}$ Total VGs	$1.4x10^{14}$ Total VGs

Vector genomes measured using ddPCR.
Assumes average CSF in patient of 408.7 ml.

Endpoints

Primary Endpoint: Safety

Secondary Endpoint: Conversion of CSF APOE4 to APOE2/APOE4

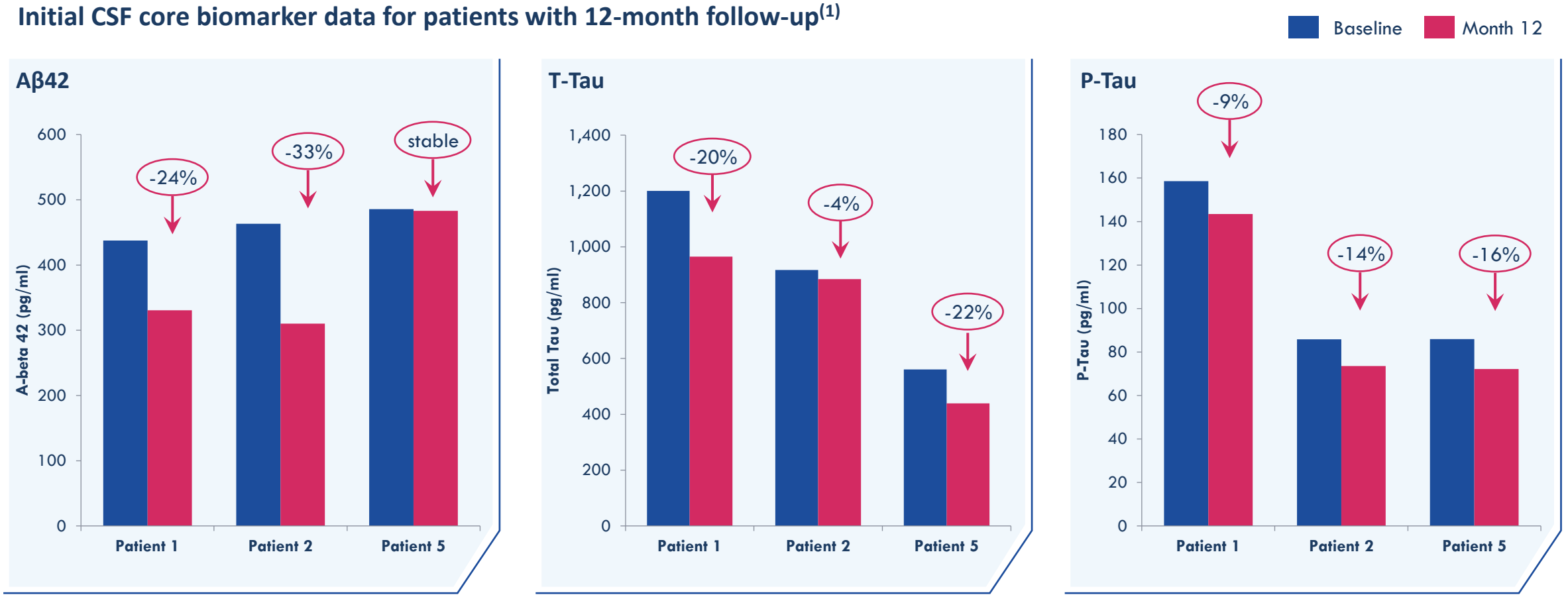
Other Secondary Endpoints:

- CSF biomarkers: Aß42, T Tau and P Tau
- Amyloid and tau PET scans
- Quantitative MRI
- Cognitive testing

LEXEO therapeutics

Initial LX1001 Low-Dose Cohort Data: CSF Core Biomarkers

Initial CSF core biomarker data for patients with 12-month follow-up(1)



Reduction in CSF total tau and phospho-tau were observed in all 3 patients, reduction in CSF Aβ42 was observed in 2 of the 3 patients

Note: Patient 3 transitioned to a long-term care-facility prior to Month 3 and unlikely to have follow-up efficacy data. Patient 4 was unable to travel to the study site for the month 6 and 12 visits.
(1) If a screening and baseline value was obtained the mean was used as the baseline.

Significant Catalysts Across Lead Programs in 2024 Supported by Strong Balance Sheet

Program	2024 Upcoming Milestone	US Prevalence
LX2006 FA Cardiomyopathy	• Mid 2024: Interim Data Readout	~5K
LX2020 PKP2-ACM	• 1H 2024: First Patient Dosed • 2H 2024: Interim Data Readout (Cohort 1)	~60K
LX1001 Alzheimer's: APOE4	• 2H 2024: Interim Phase 1/2 Data Readout (All Cohorts)	~900K
LX2021 DSP Cardiomyopathy	• 2024: Initiate IND-enabling Studies	~35K

Pro forma cash and marketable securities[1]

~$210M

Balance sheet as of December 31, 2023 pro forma for financing announced March 2024

Projected runway into

2027

More than 2 years of runway following key catalysts

Pro Forma shares of common stock[2]

32.9M

Expected pro forma shares outstanding

(1) Cash, cash equivalents and investments in marketable securities of $121.5 million as of December 31, 2023. Pro forma for expected $88.5 million net proceeds from equity financing announced in March 2024, unaudited.

(2) Shares outstanding as of March 7, 2024, pro forma for approximately 6.3M additional shares of common stock expected from equity financing announced in March 2024.

LEXEO therapeutics