FREELINE

Corporate Presentation

19 JANUARY 2022

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Examples include statements regarding upcoming milestones in its Phase 1/2 B-LIEVE dose-confirmation and potential pivotal Phase 3 clinical trials of FLT180a, Phase 1/2 MARVEL-1 clinical trial of FLT190 and Phase 1/2 dose-finding clinical trial of FLT201, including trial design and the timing of initiation, dosing of patients and data readouts; that its product candidates have the potential to be best-in-class, first-in-class and/or functional cures and to deliver transformative therapies; that a dose of 7.7e11 vg/kg with the Company’s immune management regimen will result in consistent Factor IX (“FIX”) levels in the normal range; regarding planned regulatory filings; or regarding the Company’s expectations regarding its use of cash and cash runway; as well as any other discussion of the Company’s strategies, financing plans, business plans and prospects, capital allocation objectives and manufacturing, research, pipeline and clinical trial plans, including anticipated development milestones for the Company’s product candidates. 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Freeline.

Transforming the lives of patients with systemic debilitating diseases using AAV-mediated gene therapies

Pioneering transformative gene therapies with best-in-class potential

High protein expression at low doses in humans enabled by proprietary capsid

Developing novel, high-quality gene therapy product candidates at commercial scale using proprietary protein engineering, analytics and CMC platform

Targeting diseases where high protein expression at low doses offers best-in-class potential and unlocks disease opportunities beyond the reach of first-generation AAV gene therapy

Three clinical-stage programs with best-in-class and/or first-in-class potential

FLT180a

For treatment of hemophilia B

Demonstrated Factor IX activity levels in normal range enabling potential for functional cure[1]

FLT190

For treatment of Fabry disease

Data from lowest dose cohort show promising and durable efficacy upon which to build

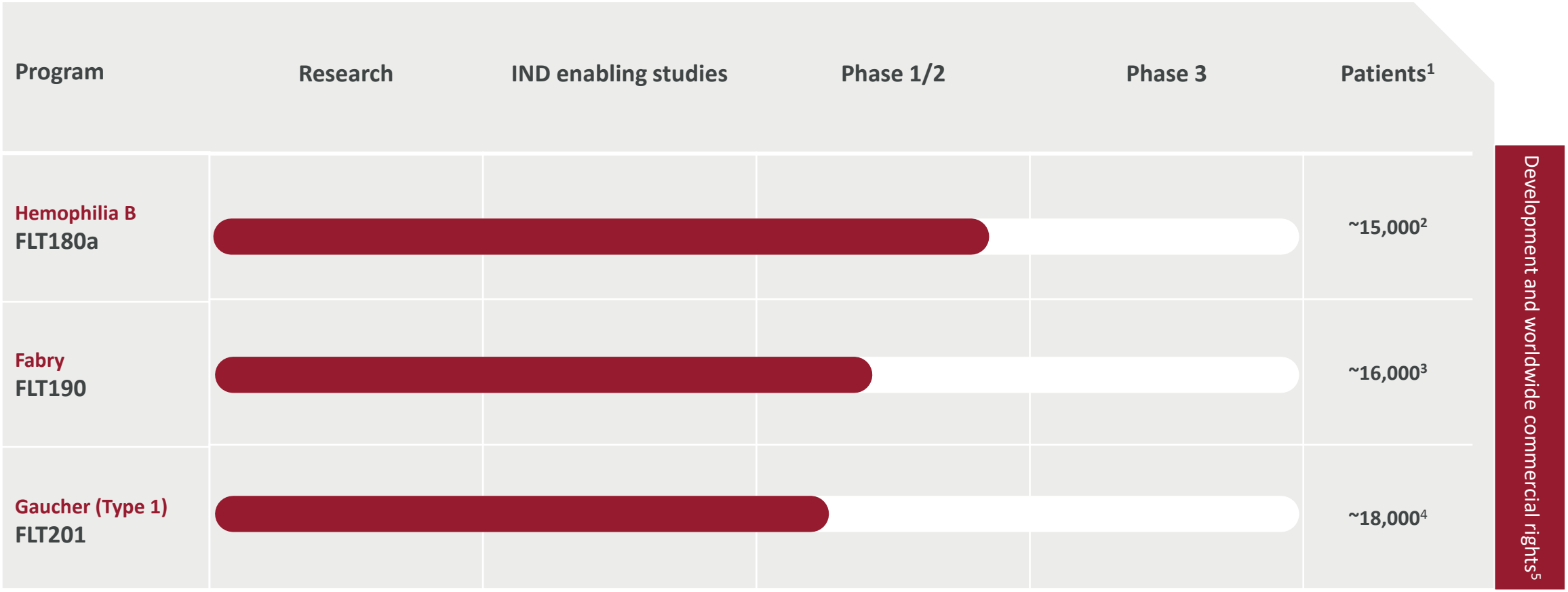
FLT201

For treatment of Gaucher disease[2]

Novel protein engineered treatment shows promising pre-clinical data; first AAV gene therapy program for Gaucher

[1] Certain adult hemophilia B patients
[2] Gaucher disease Type 1

Product Pipeline: Three clinical-stage programs with multiple 2022 milestones



[1] These figures represent the total approximate diagnosed population of Hemophilia B patients and the total theoretical genetic prevalence of the other indications. The seroprevalence of antibodies against the AAV capsid renders approximately 30-50% of patients currently not eligible for gene therapy.

[2] Hemophilia epidemiology: World Federation of Hemophilia 2020; Markets: EU4, UK, US, Japan, RoE.

[3] Ann Intern Med. 2003;138:338-346; Markets: EU4, UK, US, Japan.

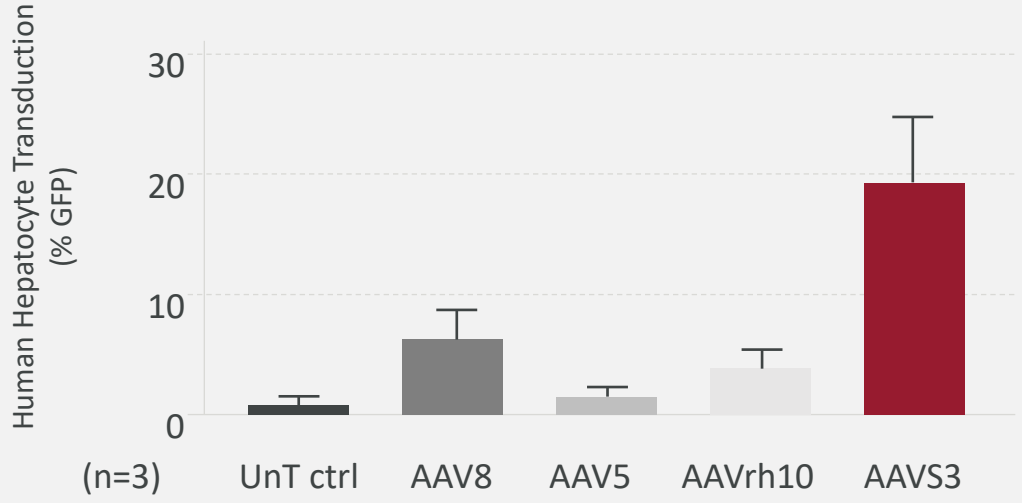
[4] Hematology. 2017 Mar;22(2):65-73. doi: 10.1080/10245332.2016.1240391; Markets: EU4, UK, US, Israel.

[5] Owned and in-licensed intellectual property rights.

Platform Technology: AAVS3 capsid engineered to more efficiently transduce human hepatocytes

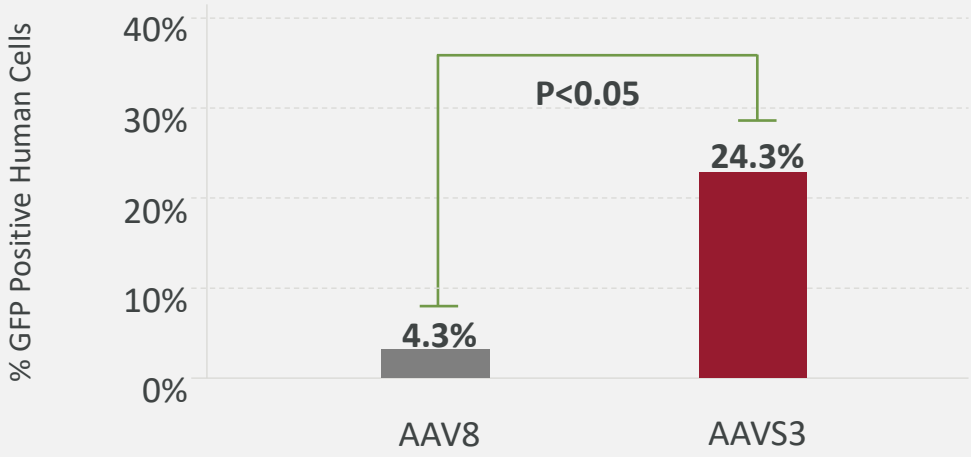
AAVS3 transduction significantly higher compared to other serotypes and untreated control

Transduction efficiency of AAVS3 in vitro compared with other vectors in primary human hepatocytes[1]



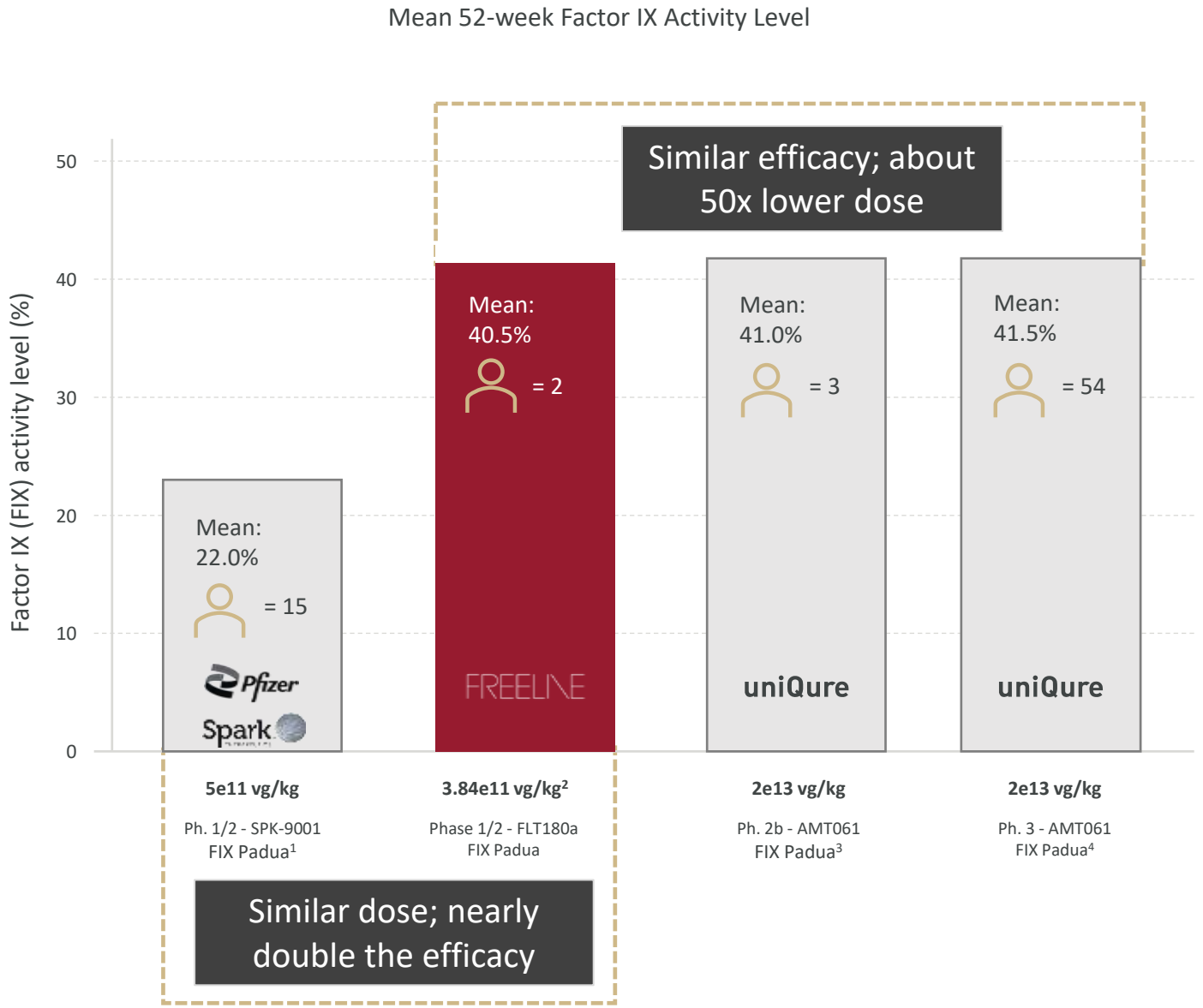
AAVS3 achieved nearly 6-fold higher transduction compared to AAV8

Transduction of human hepatocyte *in vivo* in AAV8 and AAVS3[2]



[1] Percentage of vectors containing GFP, green fluorescent protein, measured in primary human hepatocytes following transduction. AAVS3 pseudo typed vector used.
[2] Number of human hepatocytes expressing GFP following transduction. Measured in a human xenograft mouse model.

Clinical data demonstrates greater potency of AAVS3 relative to competitors



The FLT180a dose listed, 3.84e11 vg/kg, is equivalent to 4.5e11 vg/kg under the previous equivalent dosing nomenclature.

[1] Pfizer R&D Day Sep 2020 – up to four-year follow-up data in 15 patients from Phase 1/2 trial.

[2] As of the data cut-off date of September 20, 2021 measured using one-stage assay, central laboratory measurement. Two patients dosed at this dose level, with mean value calculated based on following Week 52 FIX activity levels: Patient 1, 45%, Patient 2, 36%.

[3] Miesback et al; Blood 2018 131:1022-1031.

[4] uniQure R&D day June 22, 2021 – 12-month follow-up data in 54 patients from the HOPE-B Phase 3 trial. Uncontaminated central laboratory data.

Company Strategy: Pioneering innovations that transform the lives of patients

1

Leverage highly potent capsid and platform capabilities to deliver best-in-class and first-in-class treatments

Clinical programs with potential to deliver transformative therapies.

2

Execute clinical programs to generate data demonstrating the value of Freeline's products and technology

Data anticipated across all clinical programs by mid-2022.

3

Invest in future innovations by leveraging Freeline's unique scientific capabilities

Next generation capsid design and development; novel protein variants engineered with enhanced therapeutic properties; CMC innovation.

Lean organization with core gene therapy discovery and development capabilities

Executive Team with deep experience in leading and running life science organizations.

Leading industry experts in clinical development.

Optimally-sized organization of ~190 FTEs.

Cash runway into Q2 2023.

Deep experience in gene therapy manufacturing.

Distinct in-house research capabilities and CMC technology.

Operational efficiency enhances financial flexibility for future growth and platform investments

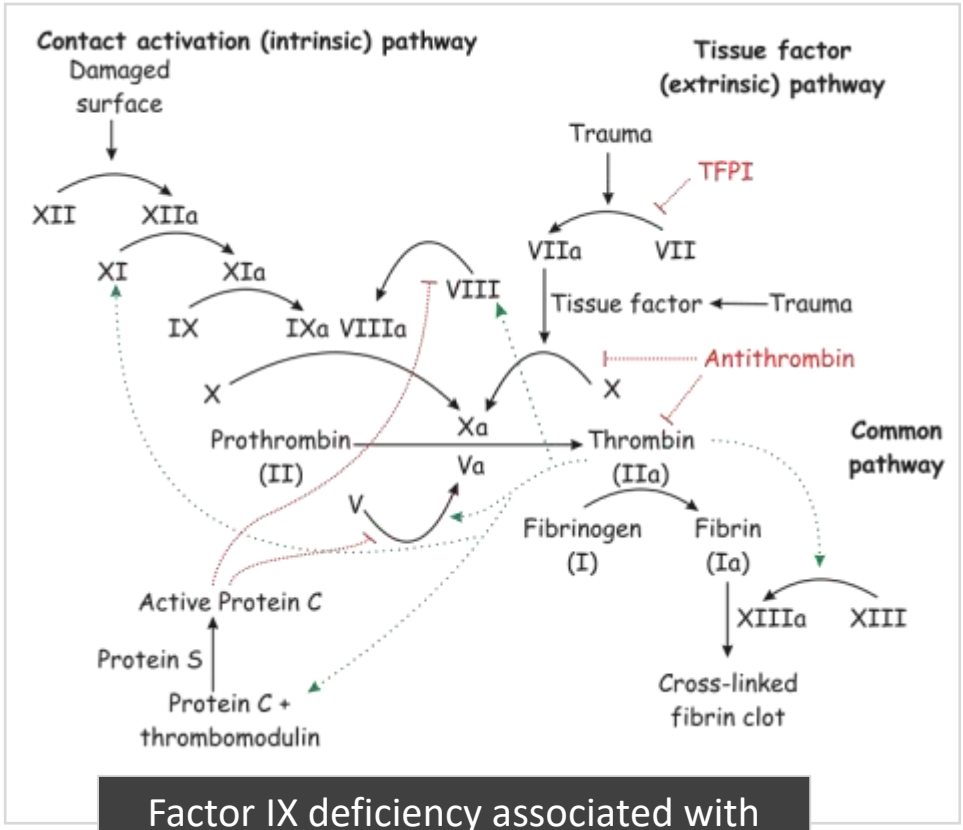
Hemophilia B

Hemophilia B Overview

Disease Characteristics:

- Rare, X-linked, congenital bleeding disorder caused by deficiency of coagulation Factor IX (FIX).
- Depending on the severity of the disease, symptoms can range from frequent, spontaneous bleeding into joints and muscles to abnormal bleeding from minor injuries.
- The majority of patients are male given that hemophilia is an X-linked disorder.

Blood Coagulation Cascade



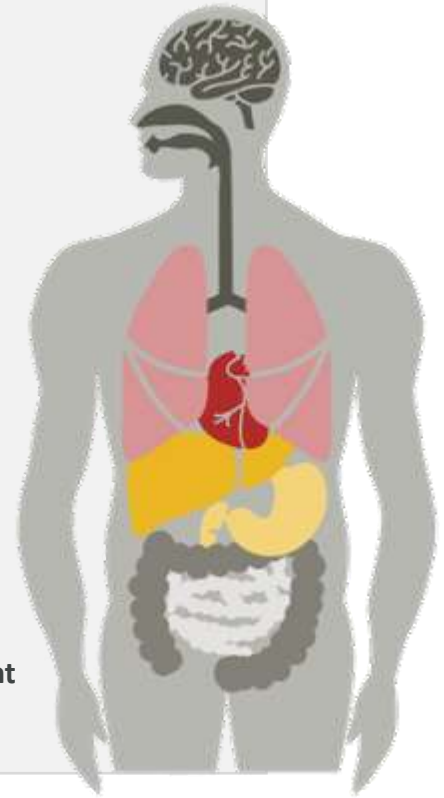
Factor IX deficiency associated with hemophilia B

Clinical Manifestations

Frequent, spontaneous bleeding

Abnormal bleeding from minor injuries

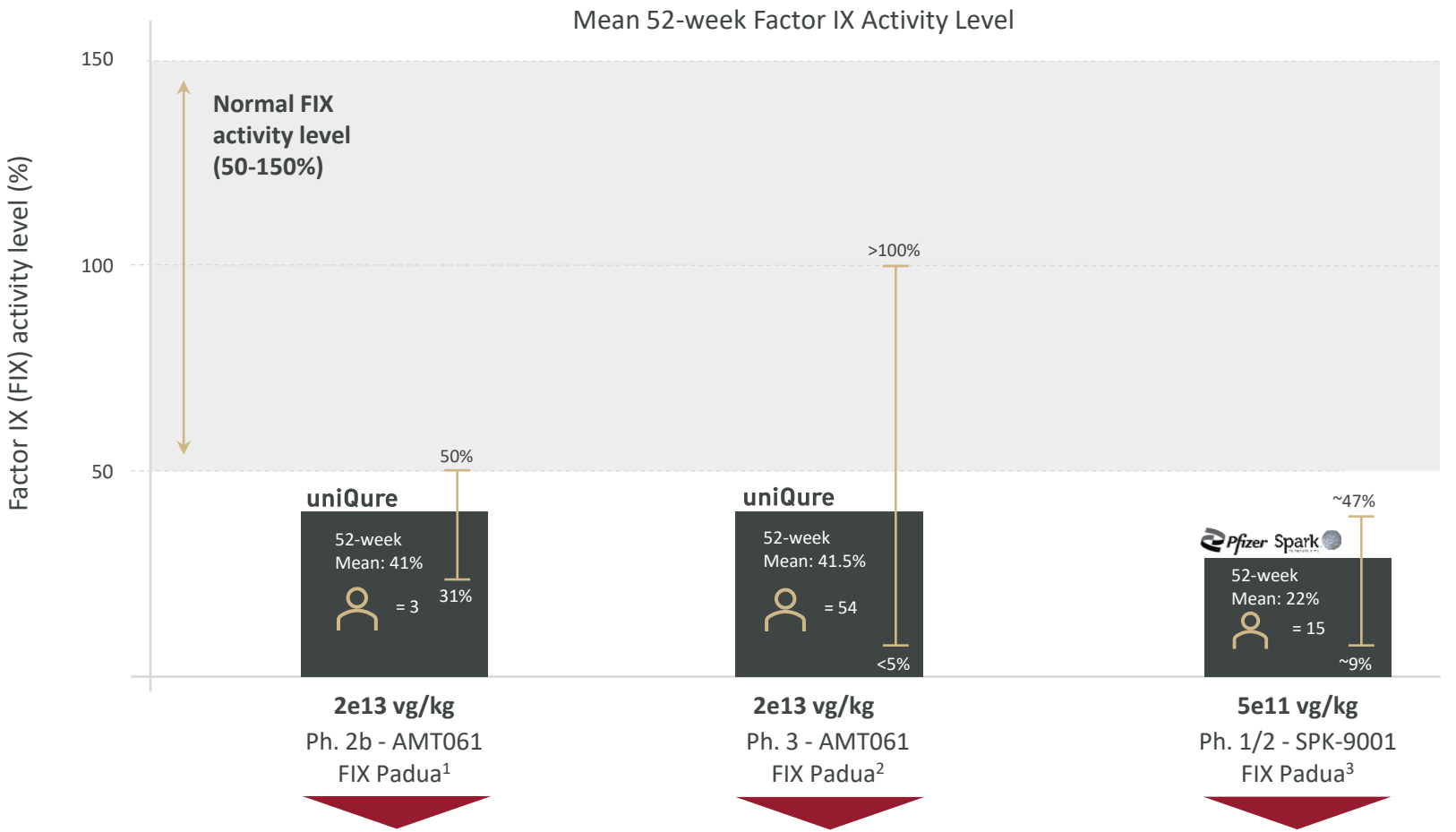
Risk of joint bleed which may result in permanent joint damage



Schulman (2012); World Federation of Haemophilia; Nilsson (1976); Thrombosis and Haemostasis; Orphanet.

Challenge: First generation capsids do not adequately protect patients from bleeds

FIX activity levels achieved using first generation capsids are in the mild range with large variability.



Treatment with FIX needed for high risk activities, surgery and trauma.

44% of patients experienced a bleed.

27.8% of patients had bleeds treated with FIX.

8 spontaneous bleeds required treatment with FIX.

20% of patients experienced a bleed.

[1] Miesback et al; Blood 2018 131:1022-1031.

[2] uniQure R&D day June 22, 2021 – 12 month follow-up data in 54 patients from the HOPE-B Phase 3 trial. Uncontaminated central laboratory data.

[3] Pfizer R&D Day Sep 2020 – up to four year follow-up data in 15 patients from Phase 1/2 trial.

Opportunity: Best-in-class therapy that achieves durable FIX activity levels in the normal range and protects patients from bleeds

Urgent need in the market[1]

Patients
Will wait for a gene therapy that delivers durable FIX activity levels in the normal range.

Physicians
Eliminating bleeds is an important consideration in selecting gene therapy.

Payers
Consider the curative and cost-saving potential of gene therapy attractive.

Hemophilia B target product profile

- Predictably provide FIX activity levels in the normal range (50-150%).
- Provide stable, durable FIX activity levels.
- Significantly reduce or eliminate the occurrence of spontaneous bleeds.

[1] Market research: Interviews, survey and analysis

B-AMAZE Phase 1/2 dose-finding trial design

Novel features of B-AMAZE

- Targeting normal range of FIX (50–150%)
- Adaptive dosing design to facilitate dose finding
- Prophylactic immune management plus reactive as needed for vector-related transaminitis

Duration

- 26 weeks for B-AMAZE
- 15 years for the LTFU study

Key inclusion

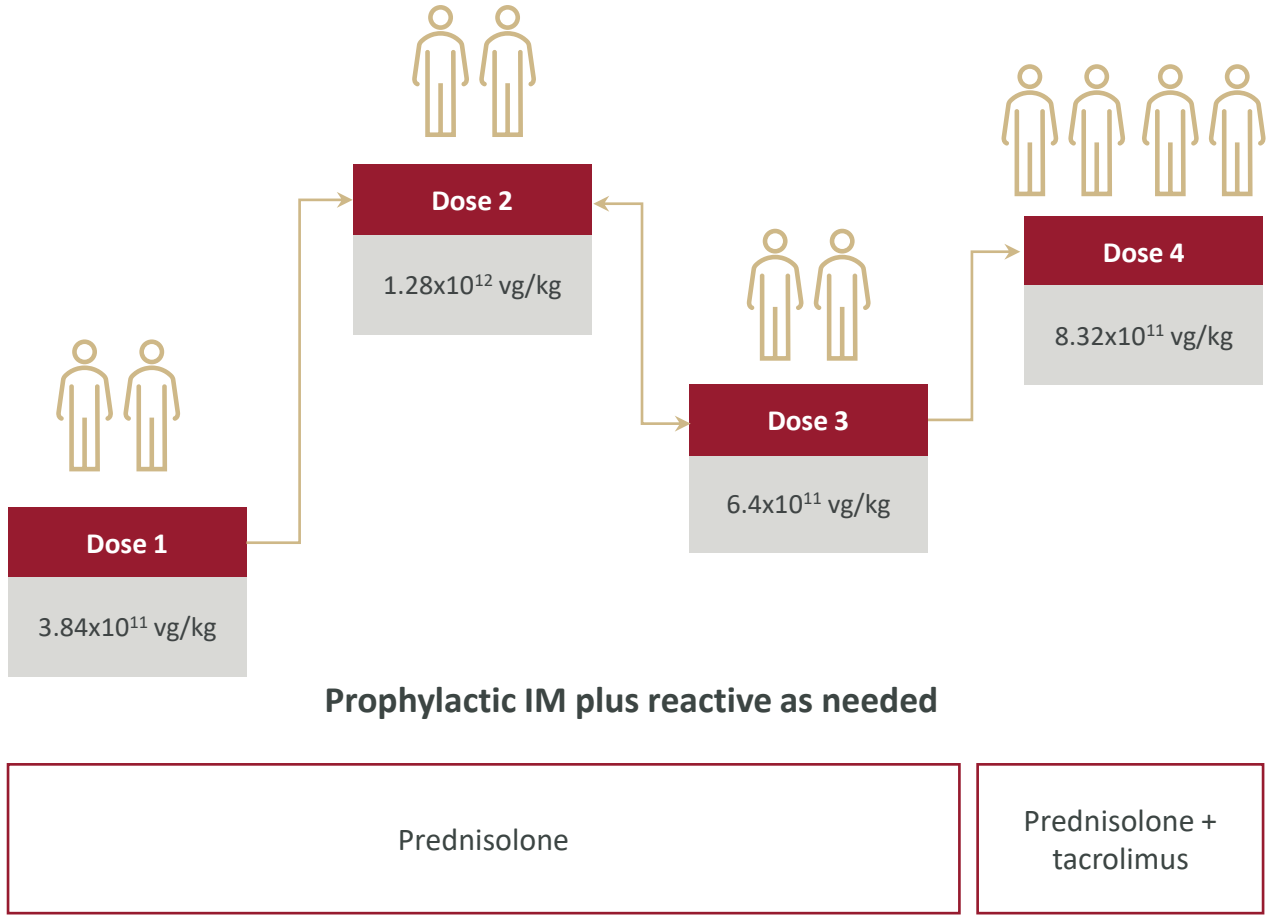
- Adults (aged ≥18 years)
- FIX levels ≤2%

Key exclusion

- Neutralizing antibodies to AAVS3
- Liver disease
- FIX inhibitors

B-AMAZE Week 26 endpoints

- Safety, as assessed by AEs
- FIX activity



FLT180a (verbrinacogene setparvovec) uses the potent, rationally designed AAVS3 capsid carrying a FIX-R338L 'Padua' variant transgene to enable high FIX expression with relatively low vector doses.

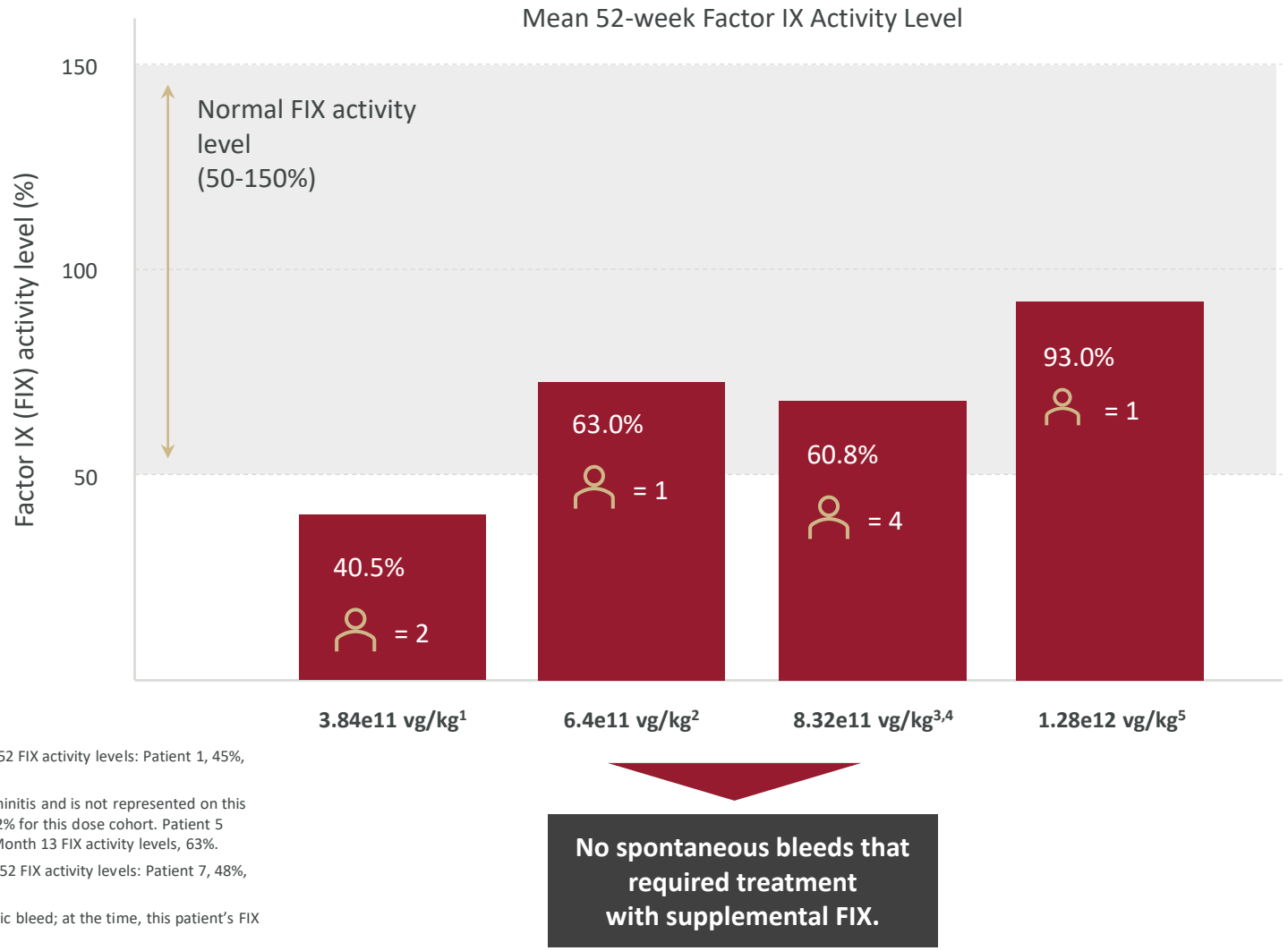
ClinicalTrials.gov identifiers: NCT03369444 for B-AMAZE and NCT03641703 for the LTFU study.

AAV, adeno-associated virus; AE, adverse event; FIX, Factor IX; IM, immune management; LTFU, long-term follow-up; vg, vector genomes.

FLT180a achieved FIX activity in the normal range and significantly reduced bleeds

Phase 1/2 B-AMAZE trial 52-week FIX activity level

As of September 20, 2021, measured using one-stage assay, central laboratory measurement.



[1] 3.84e11 vg/kg dose: Two patients dosed at this level, with mean value calculated based on following Week 52 FIX activity levels: Patient 1, 45%, Patient 2, 36%.

[2] 6.4e11 vg/kg dose: Two patients dosed at this level. Patient 4 experienced loss of expression due to transaminitis and is not represented on this chart. At Week 52, Patient 4 FIX activity level was 1.5%, which would result in a mean FIX activity level of 32.2% for this dose cohort. Patient 5 missing measurement Week 52 measurement. Was imputed with a later measurement made at Month 13. Month 13 FIX activity levels, 63%.

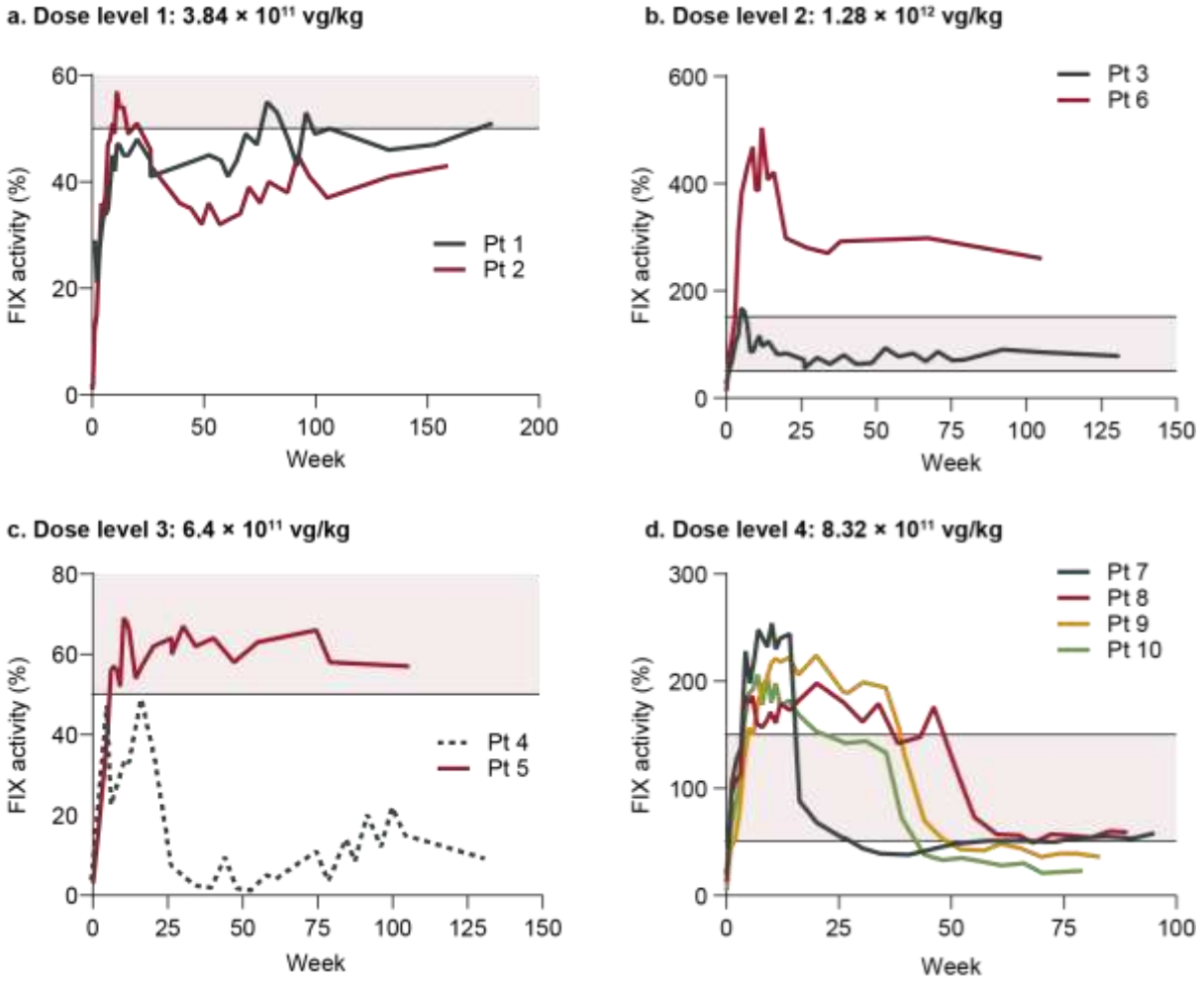
[3] 8.32e11 vg/kg dose: Four patients dosed at this level, with mean value calculated based on following Week 52 FIX activity levels: Patient 7, 48%, Patient 8, 117%, Patient 9, 43%, Patient 10, 35%.

[4] One patient in the 8.32e11 vg/kg dose cohort, Patient 8, received exogenous FIX for treatment of a traumatic bleed; at the time, this patient's FIX activity level was and remains in the low end of the normal range.

[5] Includes data from one patient, Patient 3. Data from patient with supranormal levels of FIX (Patient 6) not available for this timepoint due to pandemic-induced constraints, and was not imputed with a later measurement.

FLT180a has sustained expression up to 3.5 years after gene therapy

FIX expression has been sustained in 9 of 10 B-AMAZE patients.



Data as of September 20, 2021. FIX levels for each patient by dose are shown in panels a–d. The normal range of FIX (50–150%) is indicated in each panel in pink. Patient 4 resumed prophylaxis and is indicated with a dashed line. FIX was measured at a central laboratory with a one-stage assay.

FIX, Factor IX; Pt, Patient; vg, vector genomes.

9 of 10 patients did not receive FIX prophylaxis after FLT180a treatment:

- One patient lost FIX expression after transaminitis

Across 9 patients with sustained expression:

- One traumatic bleed was treated with FIX replacement
- No spontaneous bleeds required FIX replacement

Safety: FLT180a was generally well tolerated

- No infusion reactions or allergic reactions, and no evidence of FIX inhibitors
- Transient transaminitis with or without an associated decline in FIX was the most common FLT180a-related AE
- Late reductions in FIX were observed in patients in the 8.32×10^{11} vg/kg dosing group who received prolonged courses of prophylactic tacrolimus beyond the taper of corticosteroids
- One patient in the high-dose group with FIX expression above the normal range had an SAE of arteriovenous fistula thrombosis

Data as of September 20, 2021.

AE, adverse event; FIX, Factor IX; SAE, serious adverse event; vg, vector genomes.

FLT180a has potential to provide a functional cure by normalizing FIX activity levels

Key learnings from the Phase 1/2 B-AMAZE dose-finding clinical trial

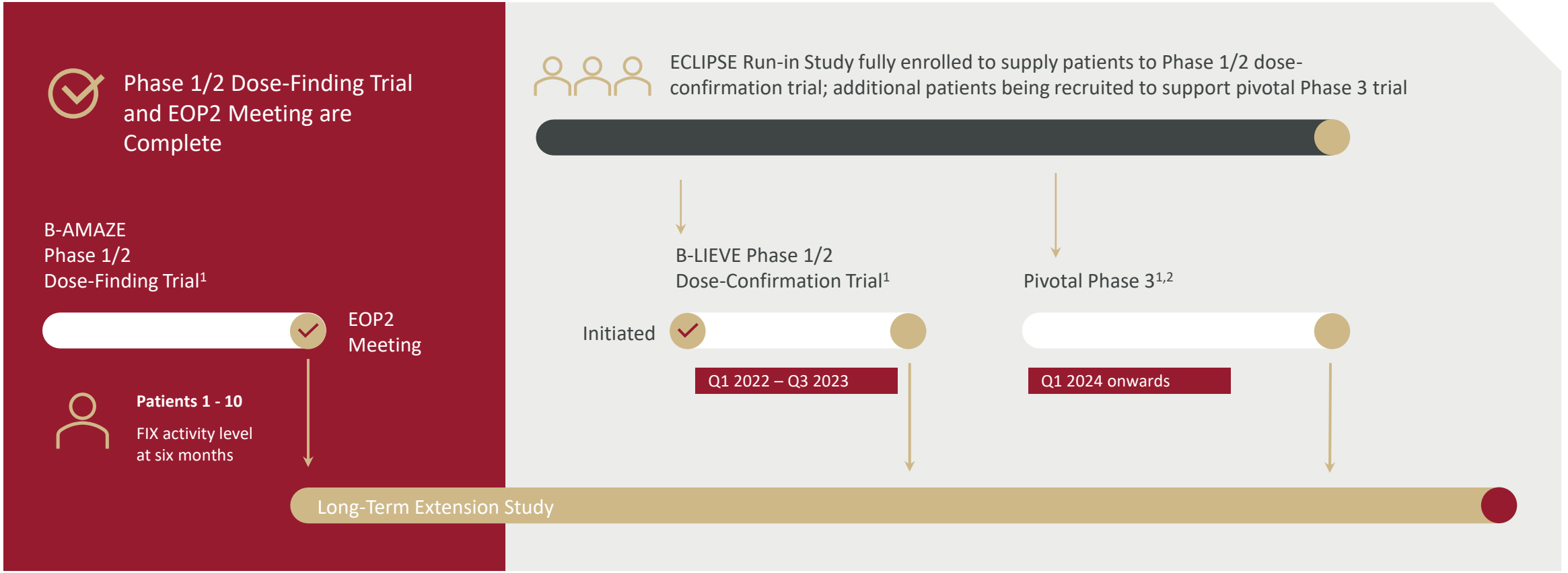
- Demonstrated efficacy with FIX activity in the normal range achieved with relatively low vector doses
- Stable and durable response up to 3.5 years post-treatment to date
- No spontaneous bleeds that required treatment with supplemental FIX[1]
- Favorable safety profile
- Proactive immune management approach expected to preserve FIX activity levels in the normal range
- Learnings from immune management regimen applied to other programs

A dose of 7.7e11 vg/kg with updated immune management regimen is expected to result in consistent FIX levels in the normal range and will be taken forward in the dose-confirmation study (B-LIEVE)

[1] One patient in the 6.4e11 vg/kg dose cohort (7.5e11 vg/kg under the previous equivalent dosing nomenclature) lost expression due to transaminitis and resumed FIX prophylaxis.

FLT180a clinical development plan for accelerated approval

- B-LIEVE: Phase 1/2 trial to confirm the dose and immune management regimen for use in a pivotal Phase 3 study
- Plan to file for accelerated approval using the surrogate endpoint of FIX activity levels combined with demonstration of a positive correlation between 26-week FIX activity levels and 52-week Annualized Bleeding Rate



[1] To measure safety, efficacy and durability.

[2] Pending discussion with regulators, the Company intends to file for accelerated approval based on six-month FIX activity level data in approximately 20 patients, along with annualized bleed rate data in a subset of these patients.

Anticipated FLT180a milestones

FLT180a – Hemophilia B

Phase 1/2 B-LIEVE dose-confirmation trial

H1 2022

- Complete dosing of first dose cohort.
- Report safety and biomarker data from first dose cohort.

H2 2022

- Complete dosing of second dose cohort or additional patients in the first dose cohort (if necessary) by end of Q3.
- Report updated safety and biomarker data.

H1 2023

- Report updated safety and biomarker data.
- Initiate Phase 3 pivotal trial start-up activities.

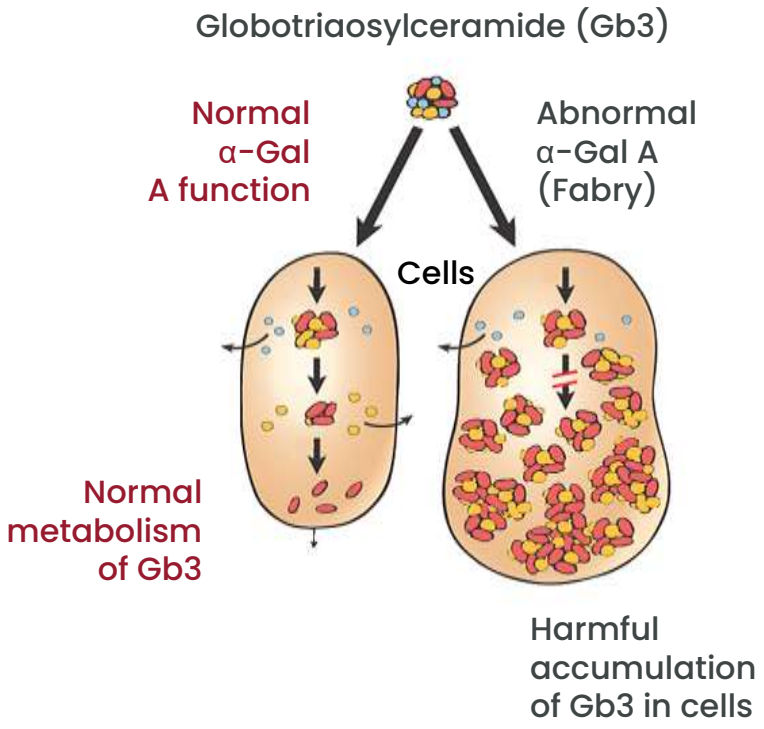
Fabry Disease

Fabry Disease Overview

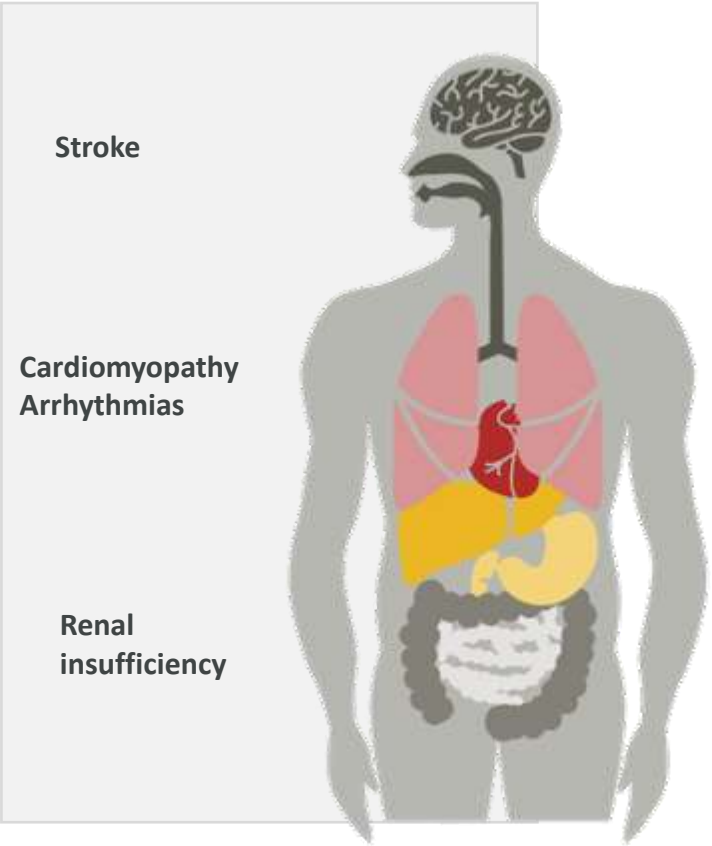
Disease Characteristics:

- Rare X-linked lysosomal storage disorder (LSD) resulting from deficient activity, or absence, of α Galactosidase A (α-Gal A).
- Deficiency of α-Gal A levels result in accumulation of substrates such as Gb3 and lyso-Gb3.
- Characterized by progressive multi-systemic damage including the kidney, heart and vasculature.

Fabry Disease Mechanism



Clinical Manifestations



NORD; National Fabry disease Foundation; Fabrydisease.org; Tsukimura et al 2018; GeneReviews; Waldek et al 2009; Mehta et al 2006.

Challenge: No curative therapy exists and high unmet medical need remains for Fabry disease patients receiving ERT

Fabry disease patient perspectives[1]

ERT treatment burden

"Travelling to and from the hospital, the time spent for infusions, and trying to keep my spirit up and adjusting to treatment is a significant challenge."

Need for greater efficacy

"My son who is 35 was having bi-weekly infusions with not much positive results."

Exhaustion

"The pain doesn't just affect me but so does the fatigue. Sometimes the pain literally wipes me out."

Limitations of ERT

- Lifelong, intravenous infusions every 2 weeks placing significant burden on patients and healthcare systems
- Despite treatment with ERT, patients continue to experience debilitating symptoms and disease progression resulting in a shortened lifespan

Average life expectancy[2]	General population	Fabry disease population
US Males	74.7 years	58.2 years

- ERT is seen as expensive and burdensome by physicians

ERT= Enzyme Replacement Therapy

[1] Market research - interviews, survey and analysis

[2] Waldek S, Patel MR, Banikazemi M, Lemay R, Lee P. Life expectancy and cause of death in males and females with Fabry disease: findings from the Fabry Registry. Genet Med. 2009 Nov;11(11):790-6.

Opportunity: Patients need a therapy that delivers sustained levels of α-Gal A eliminating the need for ERT

Competitor landscape

- Short half-life ERT therapies do not deliver sustained protein levels and patients continue to experience debilitating symptoms and disease progression.
- *Ex vivo* lentiviral gene therapy is more burdensome and invasive for the patient compared to AAV approaches. Manufacturing scale-up is also highly complex.

Urgent need in the market[1]

Patients
Attracted to freedom from bi-weekly infusions provided by gene therapy.

Physicians
Will prescribe gene therapy with long-lasting efficacy results that are comparable to or better than ERT.

Payers
Recognize potential for cost savings compared to chronic lifelong ERT.

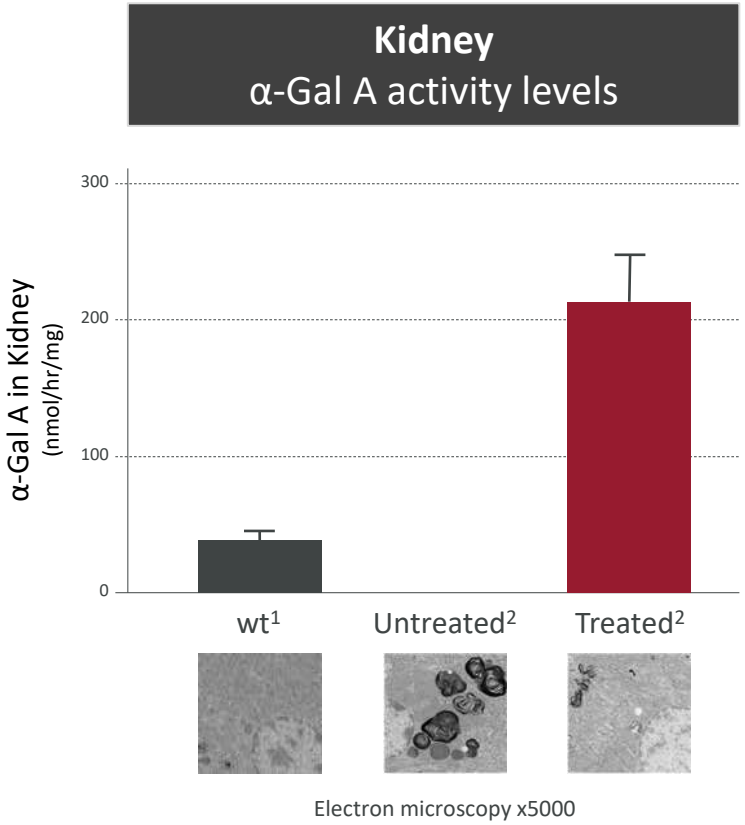
Fabry disease target product profile

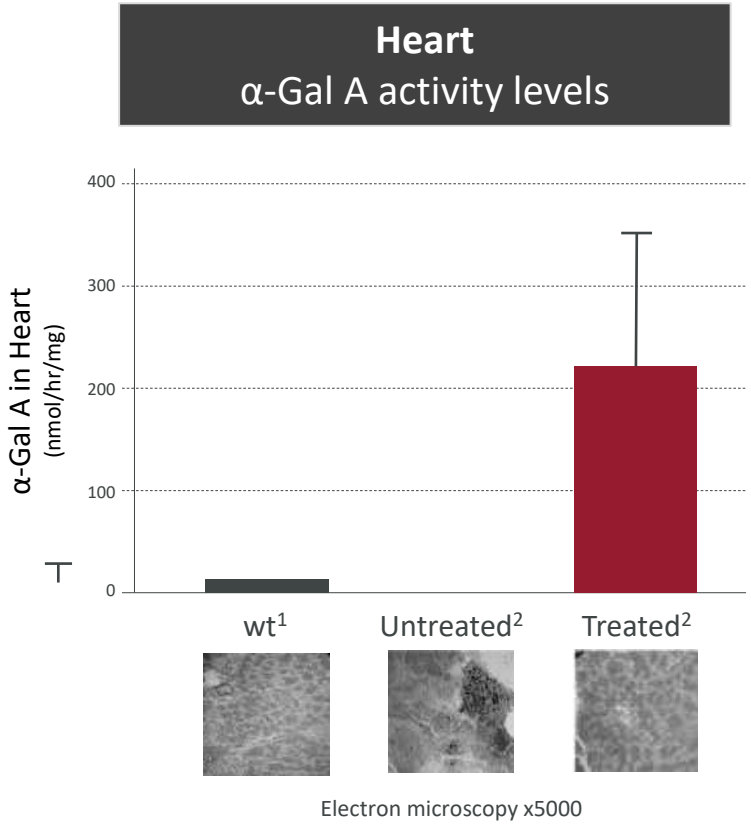
- Durable increased α-Gal A activity levels in or above the normal range.
- Sustained reduction in Gb3.
- Significantly reduce or eliminate the need for ERT.

[1] Market research - interviews, survey and analysis

FLT190 treatment for Fabry disease: Potential efficacy demonstrated in Fabry mouse model

Meaningful increase in α-Gal A expression leading to reduction in pathologic substrate in key tissues.





WORLD symposia 2019: Jey Jeyakumar et al. Liver-directed gene therapy corrects Fabry disease in mice.

FLT190 vector genome pseudo-typed with AAV8 in GLA knockout ("GLA KO") mice; Dose: 2e12 vg/kg. Error bars: mean ± SD.

Time point: 16-week disease development prior to treatment; analysis 14 weeks post-treatment. Gb3/Lyso-Gb3 data (n=4, 2 males and 2 females).

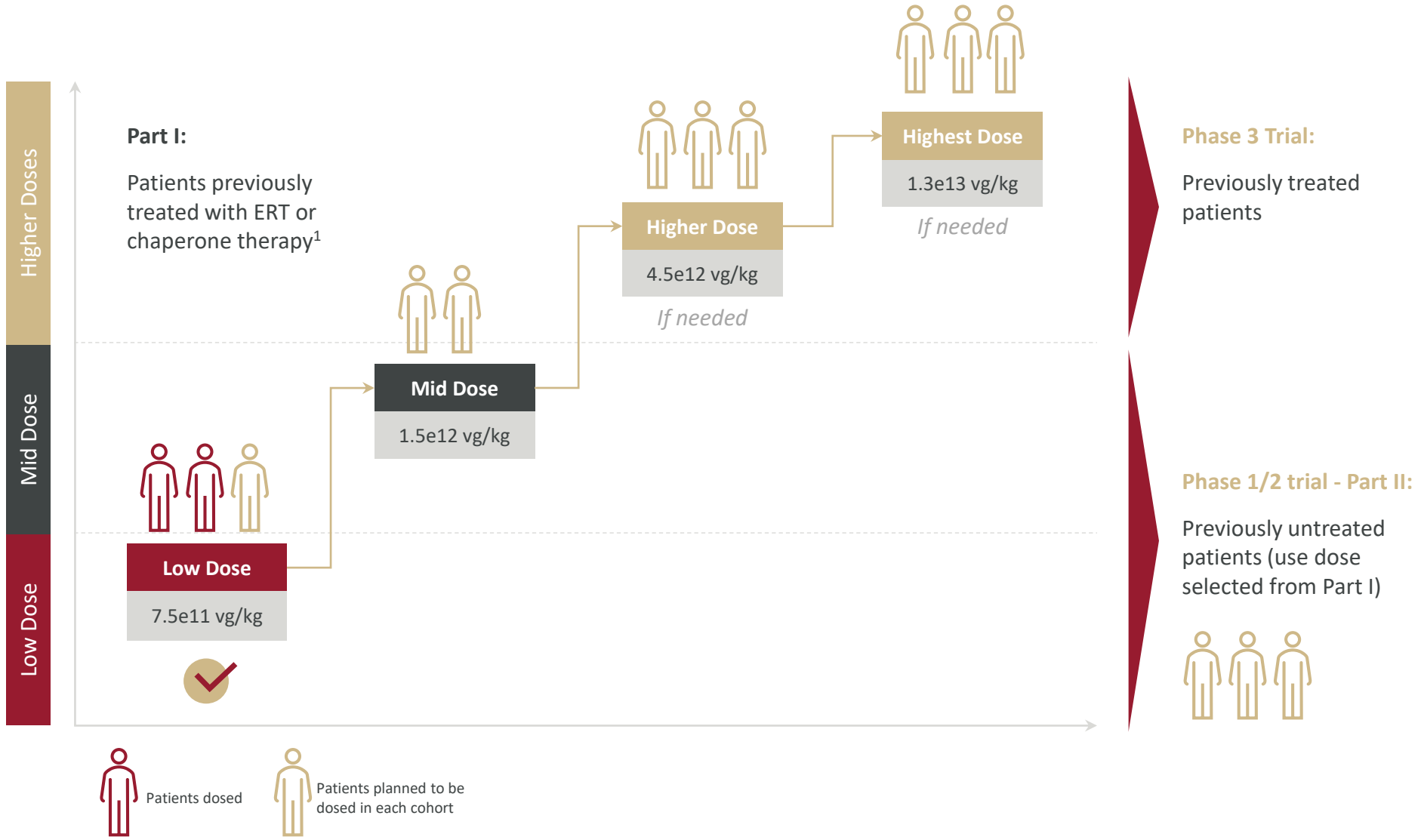
[1] Untreated wild-type (non-GLA KO) mice.

[2] GLA KO mice.

Fabry mouse model: Ohshima T, Murray GJ, Swaim WD, Longenecker G, Quirk JM, Cardarelli CO, Sugimoto Y, Pastan I, Gottesman MM, Brady RO, Kulkarni AB. (1997). alpha-Galactosidase A deficient mice: a model of Fabry disease. PNAS: 18;94(6):2540-4.

MARVEL-1 Phase 1/2 dose-finding trial design

Adaptive study:
To establish a dose of FLT190 that delivers sustained increased α-Gal A activity to levels that reduce substrate accumulation.



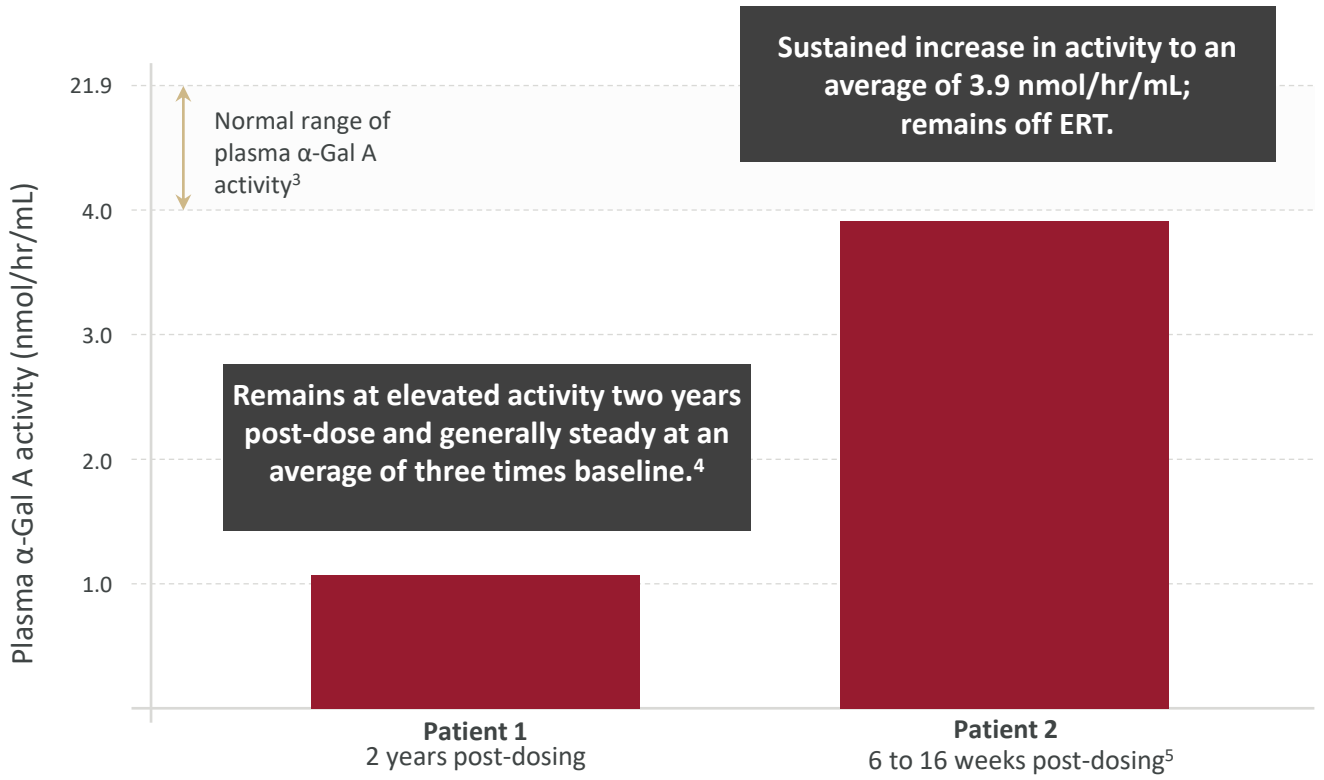
ERT= Enzyme Replacement Therapy.
✓ Marks cohort with patients dosed

Interim clinical data: Durable α-Gal A activity over 2 years in Patient 1 and near-normal levels in Patient 2

~40% increase in total dose with ~400% increase in activity[1]

[1] As of the data cut-off date of October 6, 2021.
[2] α-Gal A: Plasma α-galactosidase A, the missing enzyme in Fabry disease.
[3] Current assay normal range: 4.0-21.9 nmol/hr/mL.
[4] Patient 1 had a subtherapeutic response with plasma α-Gal A at 0.8-1.3 nmol/hr/mL.
[5] The total vector genome (vg) dose Patient 2 received was approximately 40% higher than Patient 1 due to differences in their weights

Average Plasma α-Gal A[2] Activity Following Treatment with 7.5e11 vg/kg of FLT190



Patient 2

- No rise in ALT/AST levels; received optimized immune management regimen.
- Treatment well-tolerated with no SAEs.
- Transient troponin-T elevation has returned to baseline consistent with mild myocarditis.

Patient 1

- Experienced no enduring clinical sequelae of the mild transient myocarditis episode previously reported in 2019.

Anticipated FLT190 milestones

FLT190 – Fabry disease

Phase 1/2 MARVEL-1 dose-finding trial

H1 2022

- Dose third patient in first dose cohort by end of Q1.
- Report safety and biomarker data from third patient dosed and updated safety and biomarker data from the first two patients dosed.

H2 2022

- Dose first patient in second dose cohort.
- Report updated safety and biomarker data.

H1 2023

- Dose additional patient in Phase 1/2 trial by end of Q1.
- Report updated safety and biomarker data.

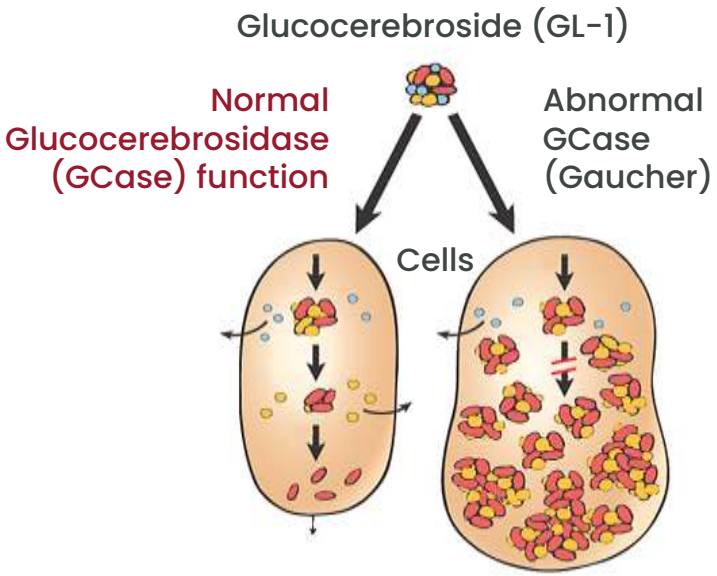
Gaucher Disease (Type 1)

Gaucher Disease (Type 1) Overview

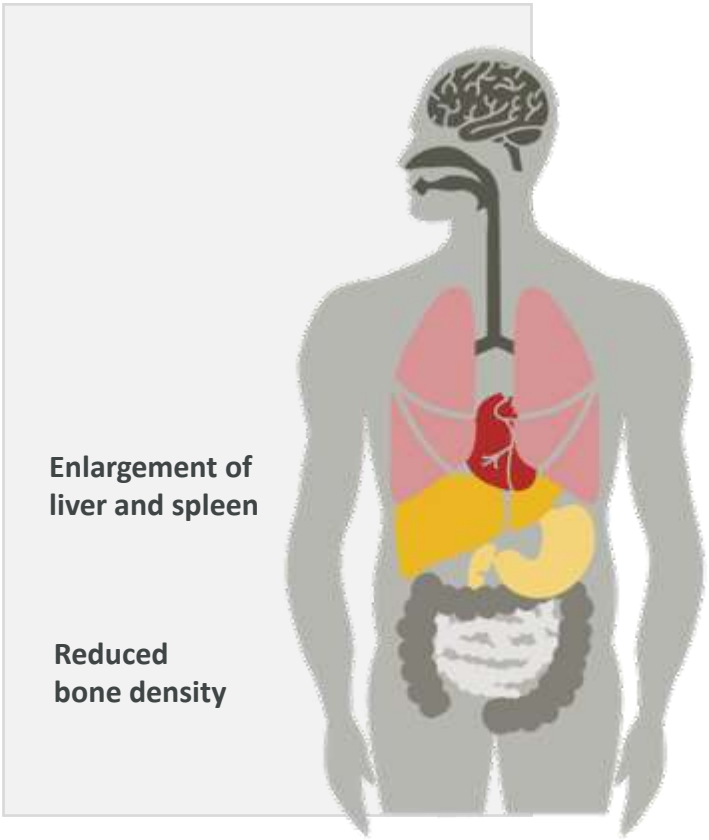
Disease Characteristics:

- Rare genetic LSD resulting from deficiency of glucocerebrosidase (GCase).
- Leads to accumulation of glucocerebroside (GL-1).
- Characterized by bone disease, hepatosplenomegaly, anaemia and thrombocytopenia with absence of primary CNS disease

Gaucher Disease Mechanism



Clinical Manifestations



National Gaucher Foundation; Orphanet, Medscape; GeneReviews; Weinreb et al 2008; Mignot et al 2003.

Challenge: No curative therapy exists for Gaucher disease (Type 1) patients receiving lifelong ERT or SRT

Gaucher disease patient perspectives[1]

ERT treatment burden

"… It would be a lot easier if I didn't have to do the infusions every two weeks…it takes two or three hours…"

"… The main reason for considering a gene therapy would be to be free of the infusions. It would be brilliant, absolutely brilliant…"

Limitations of ERT and SRT

- Lifelong intravenous infusions every two weeks placing significant burden on patients and healthcare systems
- Daily oral administration for SRT makes it difficult for patients to adhere to treatment
- SRT and ERT are viewed as expensive by physicians
- No curative therapy exists; patients continue to experience disease progression

ERT= Enzyme Replacement Therapy; SRT = Substrate Reduction Therapy

[1] Market research - interviews, survey and analysis

Opportunity: Freeline has most advanced AAV gene therapy program for Gaucher disease (Type 1)

Competitor landscape

- Limited competition in the Gaucher gene therapy market.
- Short-half life therapies do not deliver sustained protein levels. Gene therapy offers the potential of chronically sustained normal GCase levels.
- *Ex vivo* lentiviral gene therapy is more burdensome and invasive for the patient compared to AAV approaches. Manufacturing scale-up is also highly complex.

Urgent need in the market[1]

Patients
Attracted to freedom from bi-weekly infusions provided by gene therapy.

Physicians
Will prescribe durable gene therapy with efficacy comparable to or better than ERT.

Payers
Are attracted to the potential cost savings associated with the displacement of chronic ERT.

Gaucher disease target product profile

- Durable increased GCase activity levels.
- Sustained reduction in GL-1.
- Significantly reduce or eliminate the need for ERT / SRT.

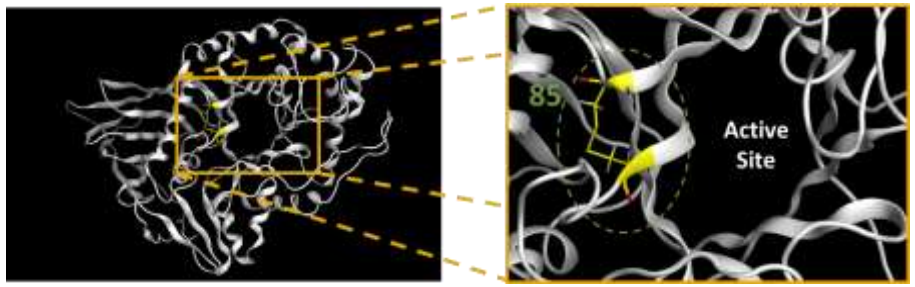
[1] Market research - interviews, survey and analysis

FLT201 produces a more stable GCase variant that achieves higher expression levels

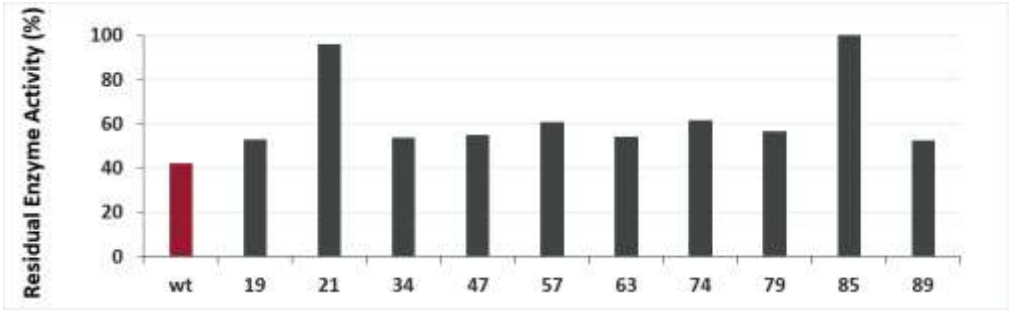
Key features of GCase variant

- > 20-fold increase in half-life in lysosomal pH compared with wild type (wt)
- Compared to wt, 6-10 fold increase in half-life in serum, resulting in a 20-fold increase in potency of the vector
- Unchanged kinetics or the anticipated antibody response.

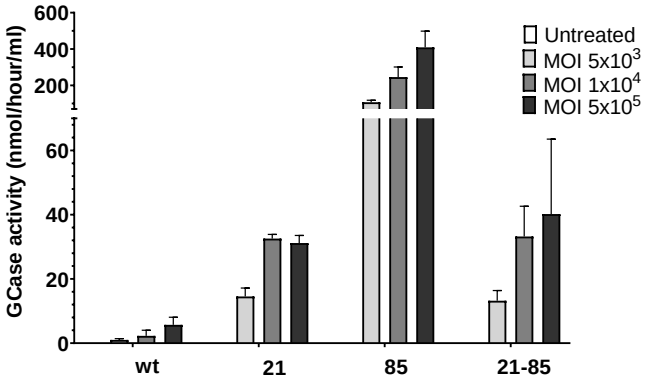
Structure of GCase variant: GCase variant 85



In vitro stability: 10 of 86 GCase protein variants showed improved stability compared with wt



Variant 85 showed the highest level of GCase activity when transduced using AAVS3 in Huh7 cells: >80-fold increase in activity vs. wt GCase

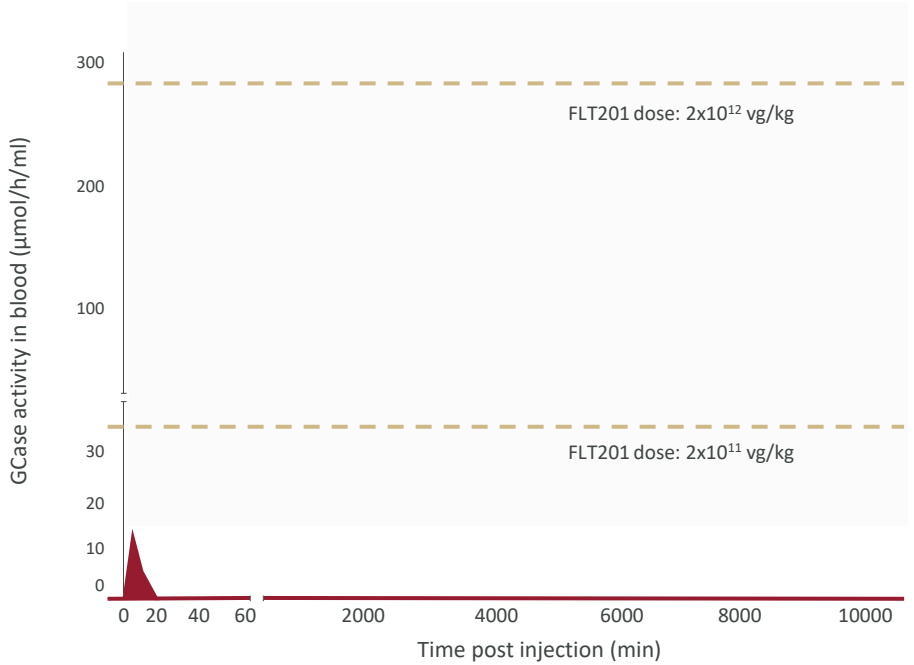


FLT201 produces more stable protein; higher uptake in Gaucher mice

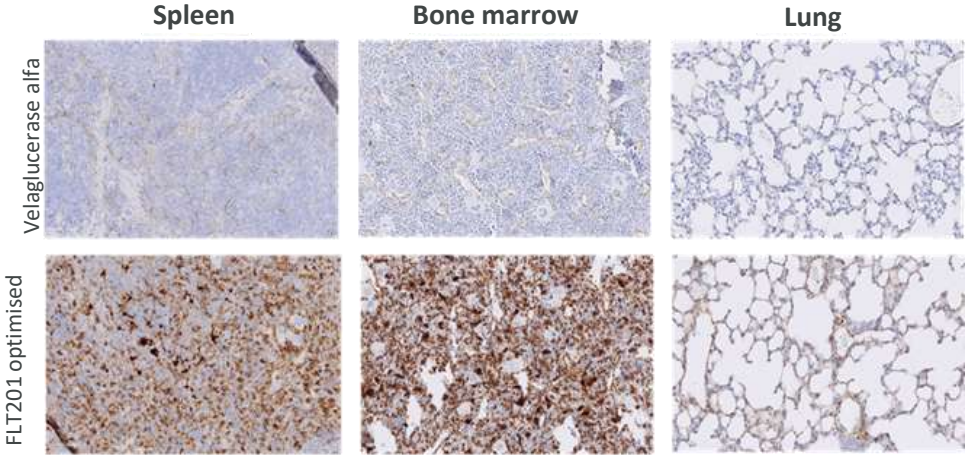
Higher GCase expression at low doses and increased uptake in key tissues

- Novel GCase variant produced by FLT201 is more stable in plasma than wild-type protein
- Greater than 20-fold increase in potency vs. vector expressing wild-type protein and better uptake in key tissues in Gaucher mice
- Enhanced potential for substrate clearance in key tissues in Gaucher mice

Increased protein stability sustains higher GCase levels compared with velaglucerase alpha[1]



Enhanced and sustained GCase uptake observed in key tissues as compared with velaglucerase alpha



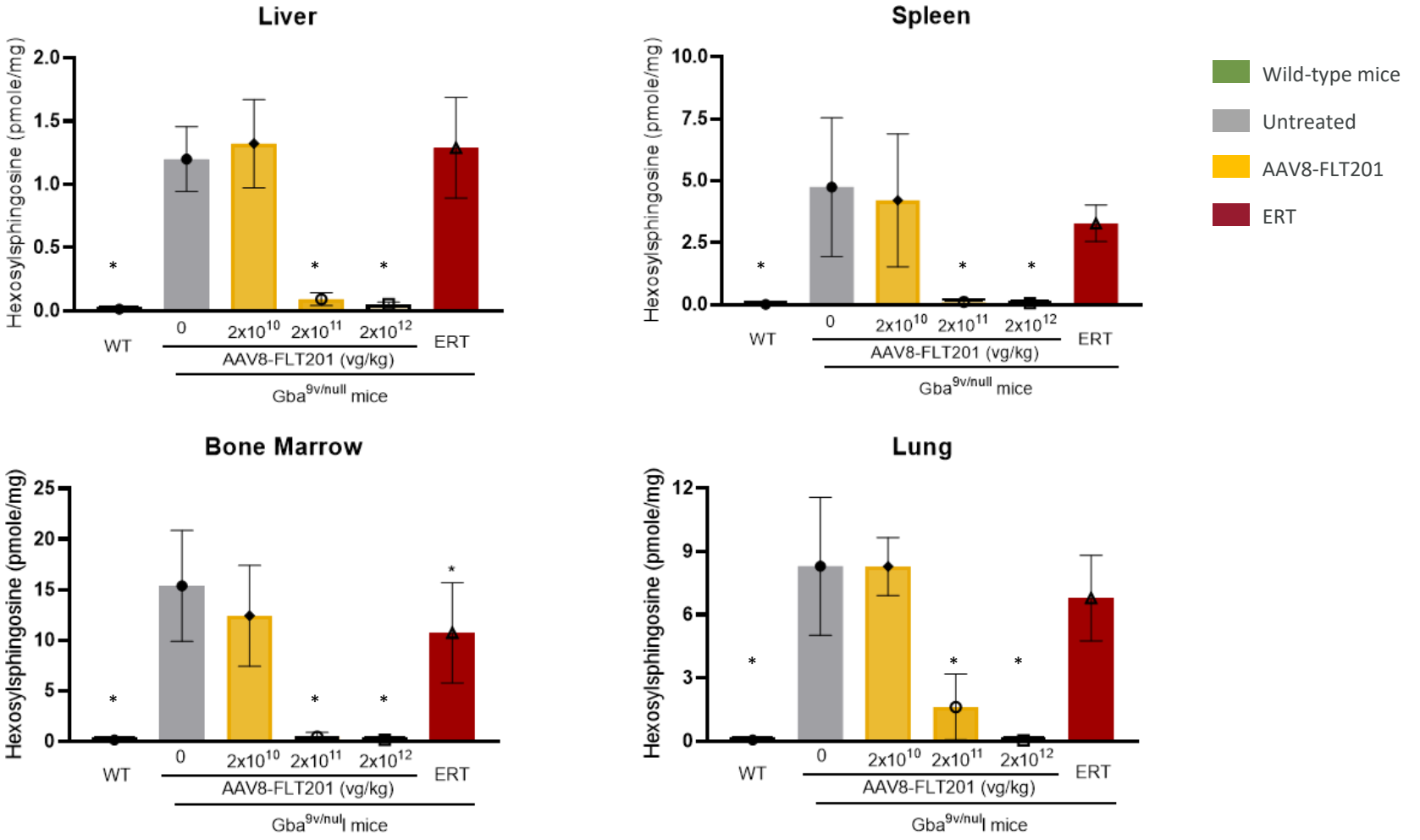
Anti-GCase – DAB, Haematoxylin counterstain, AAV8

[1] Velaglucerase alfa is an Enzyme Replacement Therapy (ERT) for Gaucher disease.
[2] FLT201 vector genome pseudo-typed with AAV8 in Gaucher mice

FLT201 clears substrate in key tissues in Gaucher mice

GCase tissue penetration and enzymatic activity leads to substrate level reduction across key tissues

- Dose-dependent reductions of lyso-GB1 observed in all tissues analyzed, including bone marrow and lung



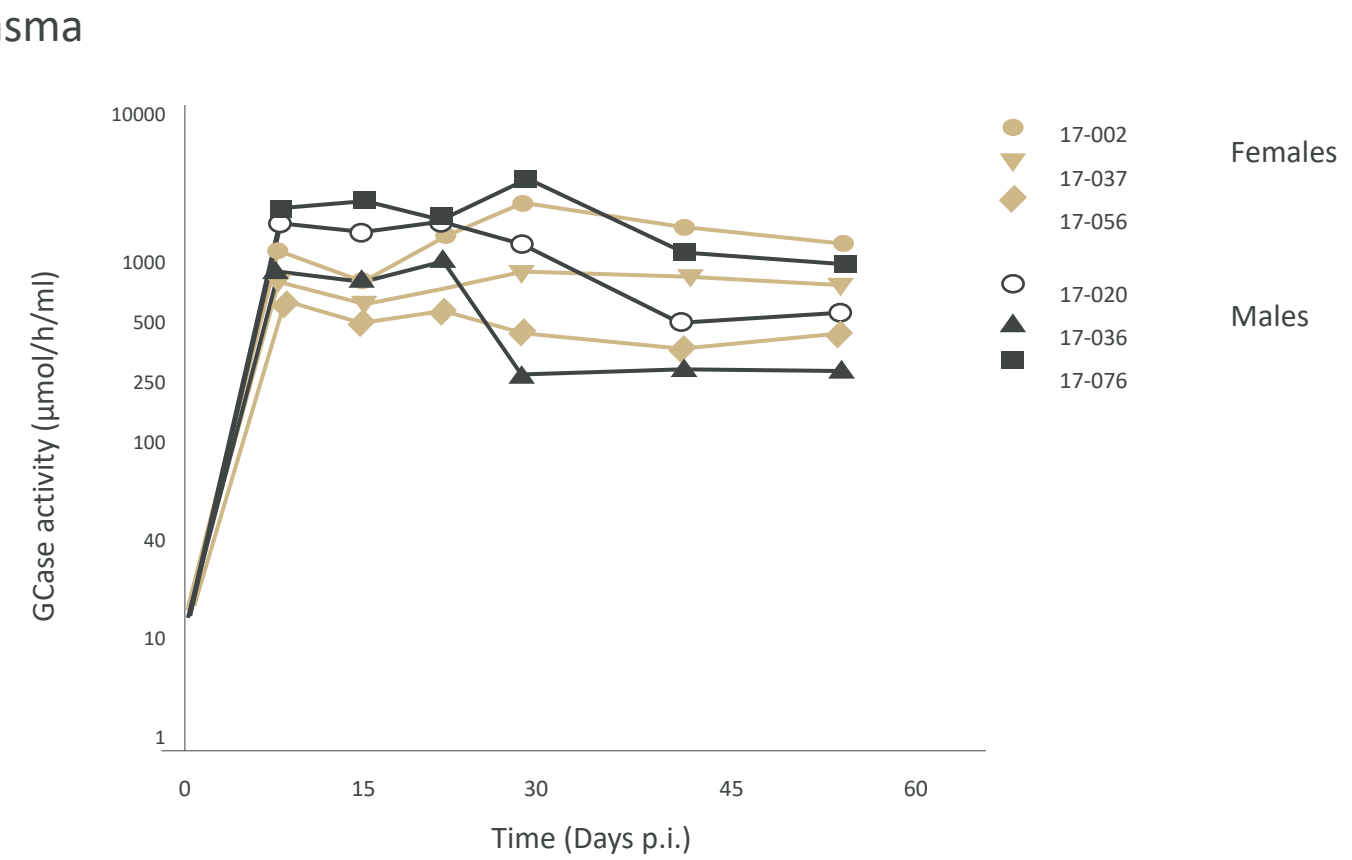
Data from study conducted in collaboration with Professor Ying Sun (Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, USA) and presented at the 17th Annual WORLDSymposium.
Velaglucerase alpha - an ERT used for Gaucher disease.
AAV8-FLT201 = AAV8 pseudo-typed FLT201 genome.
ERT = Velaglucerase alfa 60 U/kg biweekly (equivalent of the standard of care in humans).

WT = wild-type mice.
Evaluated 12 weeks post-injection.
[1] Lack of GCase enzyme in humans leads to the accumulation of lyso-GB1 and Gaucher disease.
*p ≤ 0.0001.

FLT201 demonstrates high levels of GCase expression in non-human primates

Achieves steady increases in GCase plasma levels.

- A single injection of FLT201 was well tolerated
- Resulted in a rapid increase of GCase in plasma that was sustained for at least two months (study ongoing)

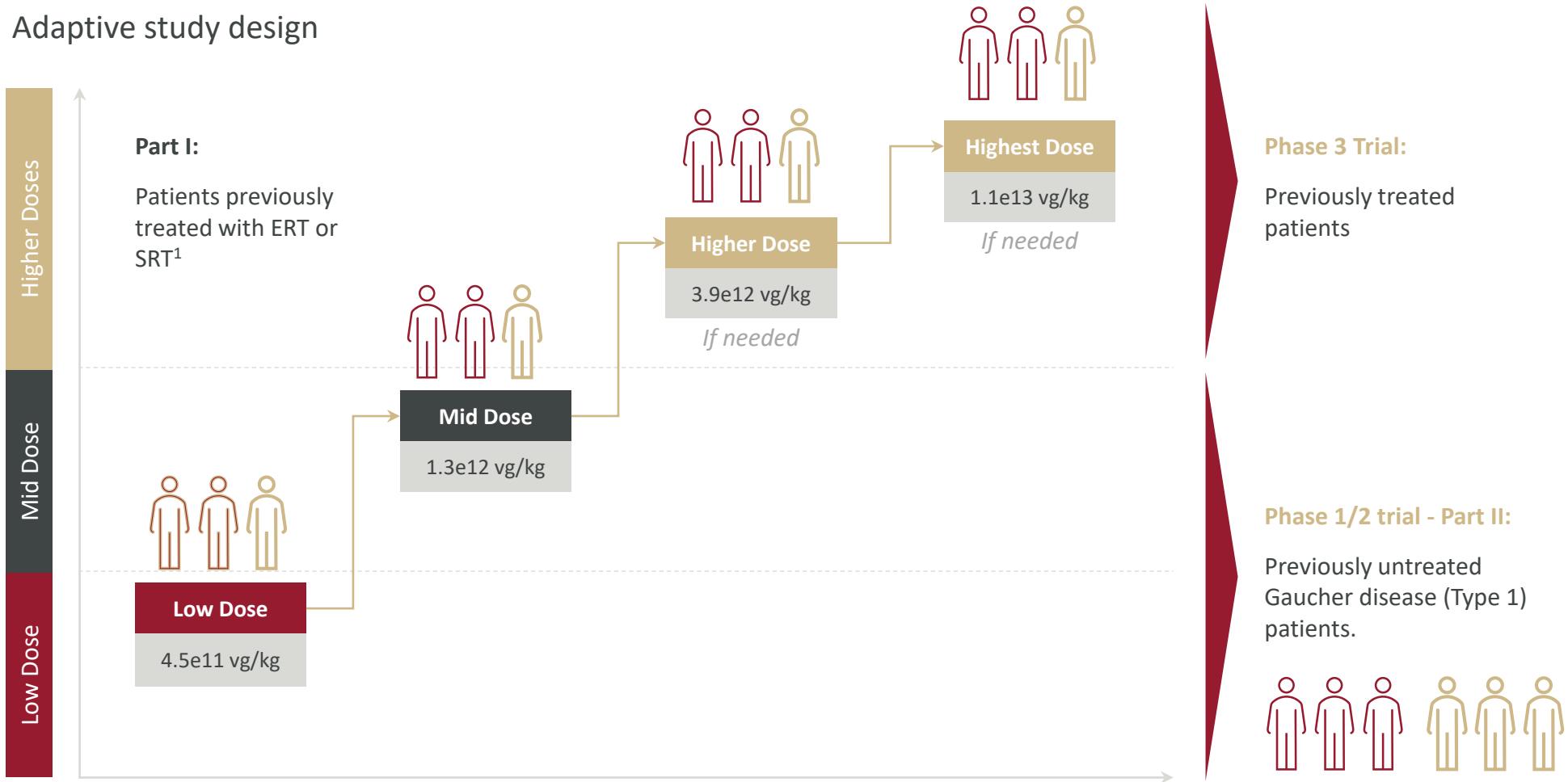


Dose: FLT201 $2x10^{12}$ vg/kg (long-term observational study)

Phase 1/2 dose-finding trial design

Study to evaluate safety and tolerability of FLT201 and establish a dose that delivers sustained increased GCase to levels that reduce substrate accumulation and improve clinical parameters

Adaptive study design



GCase = glucocerebrosidase

The study protocol allows for testing up to four doses in Part I of the clinical trial. However, depending on dose response in the initial cohorts, we may not ultimately need to escalate to the later doses.

The Data Monitoring Committee (DMC) may recommend the next dose level at the next planned dose level, at same, higher or lower dose level based on emerging safety/tolerability, PK, PD and efficacy data.

[1] ERT = Enzyme Replacement Therapy; SRT = Substrate Reduction Therapy

This symbol equates to one patient planned for dosing.

If appropriate, we may decide to expand the number patients dosed in a given cohort. This symbol represents an additional potential patient for dosing.

Anticipated FLT201 milestones

FLT201 – Gaucher disease (Type 1)

Phase 1/2 dose-finding trial

H1 2022

- Dose two patients at first dose cohort.

H2 2022

- Report safety and biomarker data from first dose cohort by end of Q3.
- Dose a further two patients by end of Q3.
- Report safety and biomarker data on all dosed patients by end of Q4.

H1 2023

- Dose a further two patients by end of Q1 (if needed).
- Report updated safety and biomarker data from first two dose cohorts.

Summary of anticipated upcoming milestones

	H1 2022	H2 2022	H1 2023
FLT180a – Hemophilia B Phase 1/2 B-LIEVE dose-confirmation trial	• Complete dosing of first dose cohort. • Report safety and biomarker data from first dose cohort.	• Complete dosing of second dose cohort or additional patients in the first dose cohort (if necessary) by end of Q3. • Report updated safety and biomarker data.	• Report updated safety and biomarker data. • Initiate Phase 3 pivotal trial start-up activities.
FLT190 – Fabry disease Phase 1/2 MARVEL-1 dose-finding trial	• Dose third patient at first dose cohort by end of Q1. • Report safety and biomarker data from third patient dosed and updated safety and biomarker data from the first two patients dosed.	• Dose first patient second dose cohort. • Report updated safety and biomarker data.	• Dose additional patient in Phase 1/2 trial by end of Q1. • Report updated safety and biomarker data.
FLT201 – Gaucher disease Phase 1/2 dose-finding trial	• Dose two patients at first dose cohort.	• Report safety and biomarker data from first dose cohort by end of Q3. • Dose a further two patients by end of Q3. • Report safety and biomarker data on all dosed patients by end of Q4.	• Dose a further two patients by end of Q1. • Report updated safety and biomarker data from first two dose cohorts.

FREELINE

Thank you

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