Update from INGLAXA Phase 1/2 Clinical Trials & Development Plans for 4D-310 Genetic Medicine for Fabry Disease Cardiomyopathy

February 22, 2023

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This Presentation shall not constitute an offer to sell or the solicitation of an offer to buy securities.

We are Boldly Innovating to Unlock the Full Potential of Genetic Medicines for Millions of Patients

COMPANY	Nasdaq **FDMT**	Emeryville, CA	~140 Employees **GMP** Facilities **R&D** Headquarters
PLATFORM	**Directed Evolution**	**~1 BILLION** capsid sequences	Targeted & Evolved Vectors Payload Design GMP Manufacturing
PRODUCT ENGINE	Vector **Modularity**	**Clinical-Stage Vectors** in **3 Therapeutic Areas**	
CLINICAL DEVELOPMENT	**5** Clinical Candidates for **7** Patient Populations		
STRATEGY	**Fully Integrated Large Market** Genetic Medicines Company		

Pipeline: 5 Clinical-Stage Product Candidates incl. Large Markets

THREE THERAPEUTIC AREAS, RARE & LARGE SUSTAINABLE PATIENT POPULATIONS

VECTOR *Delivery*	PRODUCT CANDIDATE	INDICATION	RESEARCH CANDIDATE	IND-ENABLING	PHASE 1 / 2	PHASE 3	PRODUCT RIGHTS
R100 *Intravitreal* 	**OPHTHALMOLOGY**						
	4D-150	Wet AMD					4DMT
		Diabetic Macular Edema					4DMT
	4D-125	XLRP					4DMT
	4D-110	CHM					4DMT
	4D-175	Geographic Atrophy					4DMT
A101 *Aerosol* 	**PULMONOLOGY**						
	4D-710	CF modulator-ineligible					4DMT
		CF eligible for modulators					4DMT
	4D-725	A1AT Deficiency					4DMT
C102 *IV* 	**CARDIOLOGY**	**Today's Focus**					
	4D-310	Fabry Disease Cardiomyopathy					4DMT

Key Takeaways for Today: 4D-310

1. **Heart**: Clinical POC for single low dose IV delivery, transgene expression & efficacy
   - **3rd 4DMT Vector with Clinical POC**
2. **Safety**: Generally well-tolerated with no liver, heart or DRG toxicity observed
3. **Transient acute atypical hemolytic uremic syndrome ("aHUS")**:
   - **Understood & manageable**
4. **Approval pathway clear**

Fabry Disease Cardiomyopathy: Leading Cause of Death

HIGH UNMET MEDICAL NEED RESULTS IN 75% OF DEATHS

- **Fabry Disease (FD):** monogenic disease due to AGA (*GLA*) mutations
  - Prevalence: **>50,000** (U.S. & EU-5)
- **FD Cardiomyopathy:** leading cause of death (**~75%**)[1]
- **Current therapies (e.g., ERT):** do NOT adequately address FD cardiomyopathy[2–4]
- **Major unmet medical need**

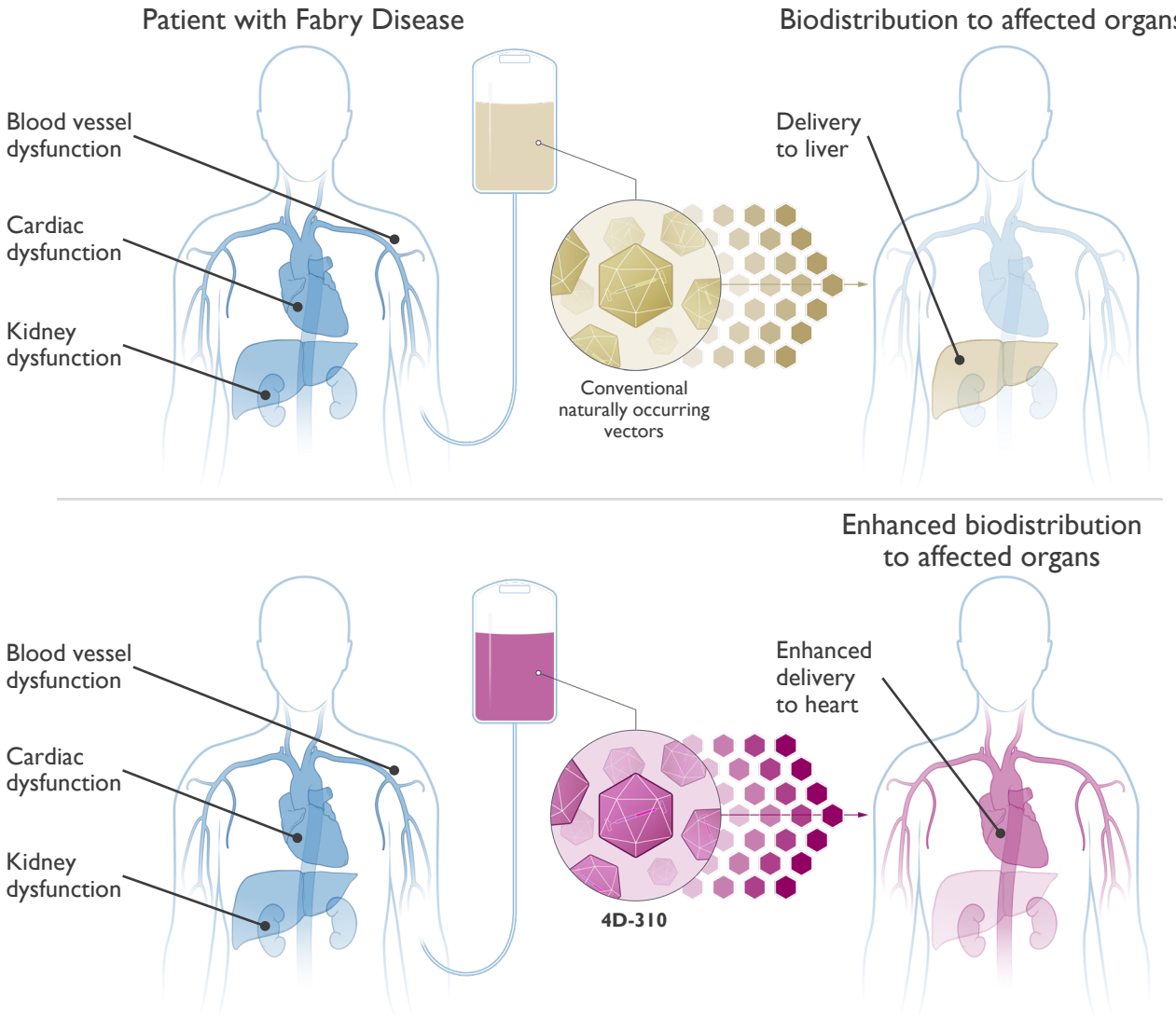
1. Baig S et al. *Europace* 2018;20:153–61. 2. Waldek S et al. *Genet Med* 2009;11:790–796. 3. Banikazemi M et al. *Ann Intern Med* 2007;14:77–86. 4. Tsukimura T et al. *Mol Genet Metab Rep* 2020;25:100650.

Challenges with IV AAV Gene Therapy for Cardiac, Skeletal Muscle & CNS

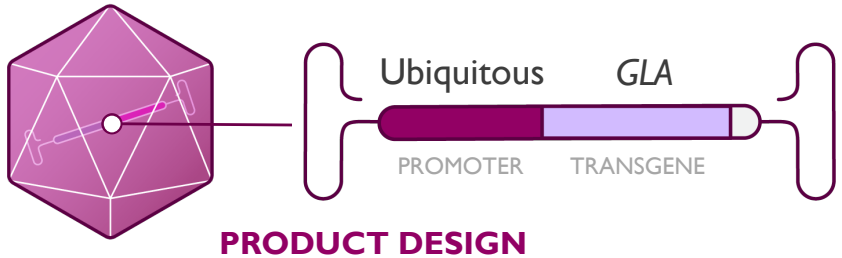
- Inefficient delivery
- Inefficient transduction
- High doses required (7E13–4E14 vg/kg)
- Organ toxicities: Liver, heart & dorsal root ganglion (DRG)
- Pre-existing anti-capsid antibodies (“NAbs”)

4D-310 Product Design & C102 Target Vector Profile for Heart

INVENTED FOR LOW DOSE IV DELIVERY TO THE HEART IN NHP & HUMANS



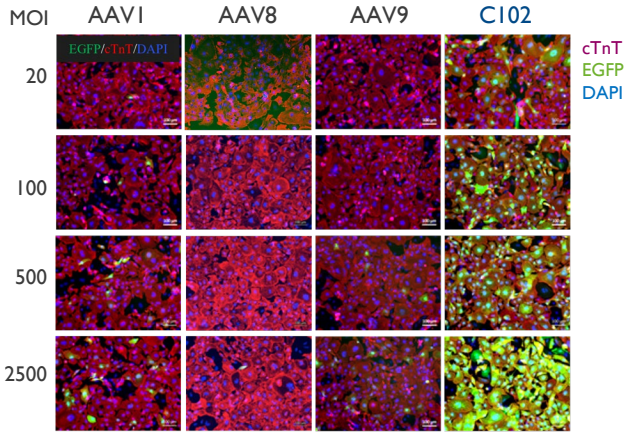
Our Product



PRODUCT DESIGN

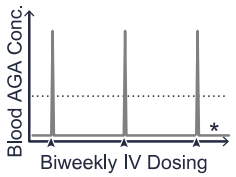
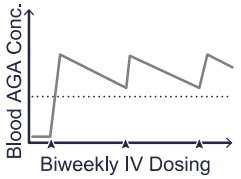
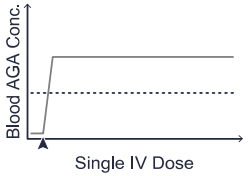
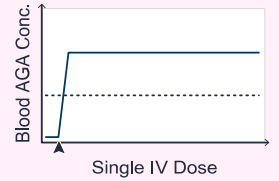
- **Vector:** C102
- **Transgene:** *GLA* (encodes AGA enzyme)
- **Promoter:** Ubiquitous

Improved Transduction: **Human** Cardiomyocytes



Human ventricular cardiomyocytes EGFP immunofluorescence *in vitro*

4D-310 for Fabry Disease Cardiomyopathy: Unique MOA Well-Differentiated Versus ERT & Genetic Medicines Competition

		ERT (Blood)		Genetic Medicine	
MOA	**Product Design**	AGA Enzyme Infusions	PEGylated AGA	AAV-mediated Liver-directed	**4D-310**
AGA Delivery Through the Bloodstream	Pharmacokinetics (··· Normal; ▲ Time of dose; * Lifelong)				
	Single dose administration	–	–	+	+
	Liver secretion of AGA	–	–	+	+
Cardiovascular Treatment & AGA Production in Target Cells	Heart (cardiomyocytes)	–	–	–	+
	Kidney (glomeruli, including podocytes)	–	–	–	+
	Blood vessels	–	–	–	+
Antibody Resistance	Intracellular production in target tissues (anti-AGA antibody avoidance)	–	–	–	+
	Capsid evolved for resistance to preexisting NAb	–	–	–	+

Abbreviations: Ab, antibodies; AGA, aspartylglucosaminidase; AAV, adeno-associated virus; ERT, enzyme replacement therapy; IV, intravenous.

Phase 1/2 Open Label Clinical Trials: 4D-310 for FD Cardiomyopathy

	INGLAXA-1	INGLAXA-2
Geography	U.S. multicenter	Taiwan & Australia multicenter
Patient Population	Male or female adults; classic or late onset Fabry disease; cardiac involvement* (on or off ERT)	
4D-310 Dose	**1E13 vg/kg**	
Immune Regimen	**Corticosteroid prophylactic immunosuppression**	
Primary Endpoint	Incidence and severity of adverse events	
Secondary Endpoints	**Cardiac imaging, function, QoL status**	
Cardiac Biopsy Endpoints	n.a.	**Transgene delivery, RNA expression & AGA protein expression**
C102 NAb Screening	Exclude pts with HIGH titer NAb to C102 (titer >1:1,000)	
AGA Ab Screening	Exclude pts with HIGH titer antibodies to AGA (titer ≥1:25,000)	

*Eligibility for INGLAXA-2 required evidence of left ventricular hypertrophy on ECHO or CMR within 12 months prior to screening. AGA, a-galactosidase A; ERT, enzyme replacement therapy; NAb, neutralizing antibody.

Baseline Characteristics: INGLAXA-1 & INGLAXA-2

COHORT 1 (1E13 vg/kg + CORTICOSTEROID IMMUNE REGIMEN; N=6)

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age, years	51	32	26	19	57	69
Race/ethnicity	Hispanic/Latino	White	White	NR	Asian	White
Disease classification	**Classic**	**Classic**	**Classic**	**Late onset**	**Late onset**	**Late onset**
Variant	c.1023A>C	c.708G>T	c.974G>A	c.671A>G	c.639+919G>A	c.644 A>G
Serum AGA activity, nmol/hr/mL*	0.42	0.00	0.30	0.06	1.62	0.18
Serum lyso-Gb3, ng/mL†	6.28	101.0	8.78	45.0	3.79	2.03
ERT experience	Yes	Yes	Yes	No	Yes	Yes
ERT status at enrollment	**On**	**Off**	**On**	**ERT naïve‡**	**On**	**Off‡**
Anti-AGA antibody titer	1:947 (l)	1:99,900 (h)	1:13,900 (m)	Negative	Negative	Negative
Peak VO_2 mL/kg/min % predicted	 *21.4 (wk 26)* *72.0 (wk 26)*	 14.0 33.0	 23.0 66.1	 19.1 30.3	 24.8 76.0	 28.2 120.2
Global longitudinal strain, %	–17.10	–22.17	–18.83	–23.27	–21.95	–20.63
eGFR, mL/min/1.73m^2 ¶	107	130	125	142	77	62

*Reference range, 4.44–27.42 nmol/hr/mL. †Reference range, ≤1.0 ng/mL. ‡On migalastat. ¶Reference range, >60 mL/min/1.73m^2. AGA, α-galactosidase A; eGFR, estimated glomerular filtration rate; ERT, enzyme replacement therapy; Gb3, globotriaosylceramide.

INGLAXA

Cardiac Outcomes

Study Participants with ≥12 Months of Follow-Up (Data cutoff of December 5, 2022) & Biopsy Data

Cardiac Assessments: Central Reading Center & Independent Histopathology

IMAGING, FUNCTION, QOL & BIOPSY; WORSENING DESPITE ERT IN NHS

		ERT Natural History (12 months)
Exercise capacity (peak VO_2) *FDA-recommended primary endpoint*	CPET*	**Worsened**[1]
Cardiac quality of life (physical limitations, symptoms) *FDA-recommended primary endpoint*	KCCQ	**n.a.**
Cardiac contractility (global longitudinal strain) *FDA-recommended supportive endpoint*	Echocardiogram*	**Worsened**[2]
Substrate accumulation (native T1 signal) *Exploratory endpoint*	Cardiac MRI*	**Worsened**[2]
Transgene delivery & expression *Exploratory endpoint* (INGLAXA-2)	Cardiac Biopsy *(INGLAXA-2 trial only)*	**Negative**[3]

*Assessed by independent central reading center. CPET, cardiopulmonary exercise test; KCCQ, Kansas City Cardiomyopathy Questionnaire; MRI, magnetic resonance imaging;
1. Lobo et al. *Intern Med J* 2008;38:407; 2. Nordin et al. *Circ Cardiovasc Imaging* 2019:e009430; 3. Thurberg et al. *Circulation* 2009;119:2561–7.

Cardiac Assessments: Improvement in All FIVE Cardiac Endpoints (4 Patients)

SIGNIFICANT IMPROVEMENTS IN ALL 5 CARDIAC ENDPOINTS

		ERT Natural History (12 months)	4D-310 1E13 vg/kg 12 months (n=3) Biopsy wk 6 (n=1)
Exercise capacity (peak VO_2) *FDA-recommended primary endpoint*	CPET*	**Worsened**[1]	**Improved** in 2 of 3
Cardiac quality of life (physical limitations, symptoms) *FDA-recommended primary endpoint*	KCCQ	**n.a.**	**Improved** in 2 of 2, stable in 3rd
Cardiac contractility (global longitudinal strain) *FDA-recommended supportive endpoint*	Echocardiogram*	**Worsened**[2]	**Improved** in 3 of 3
Substrate accumulation (native T1 signal) *Exploratory endpoint*	Cardiac MRI*	**Worsened**[2]	**Improved** in 2 of 3
Transgene delivery & expression *Exploratory endpoint* (INGLAXA-2)	Cardiac Biopsy *(INGLAXA-2 trial only)*	**Negative**[3]	**Widespread delivery & expression**

*Assessed by independent central reading center. CPET, cardiopulmonary exercise test; KCCQ, Kansas City Cardiomyopathy Questionnaire; MRI, magnetic resonance imaging;
1. Lobo et al. *Intern Med J* 2008;38:407. 2. Nordin et al. *Circ Cardiovasc Imaging* 2019:e009430. 3. Thurberg et al. *Circulation* 2009;119:2561–7.

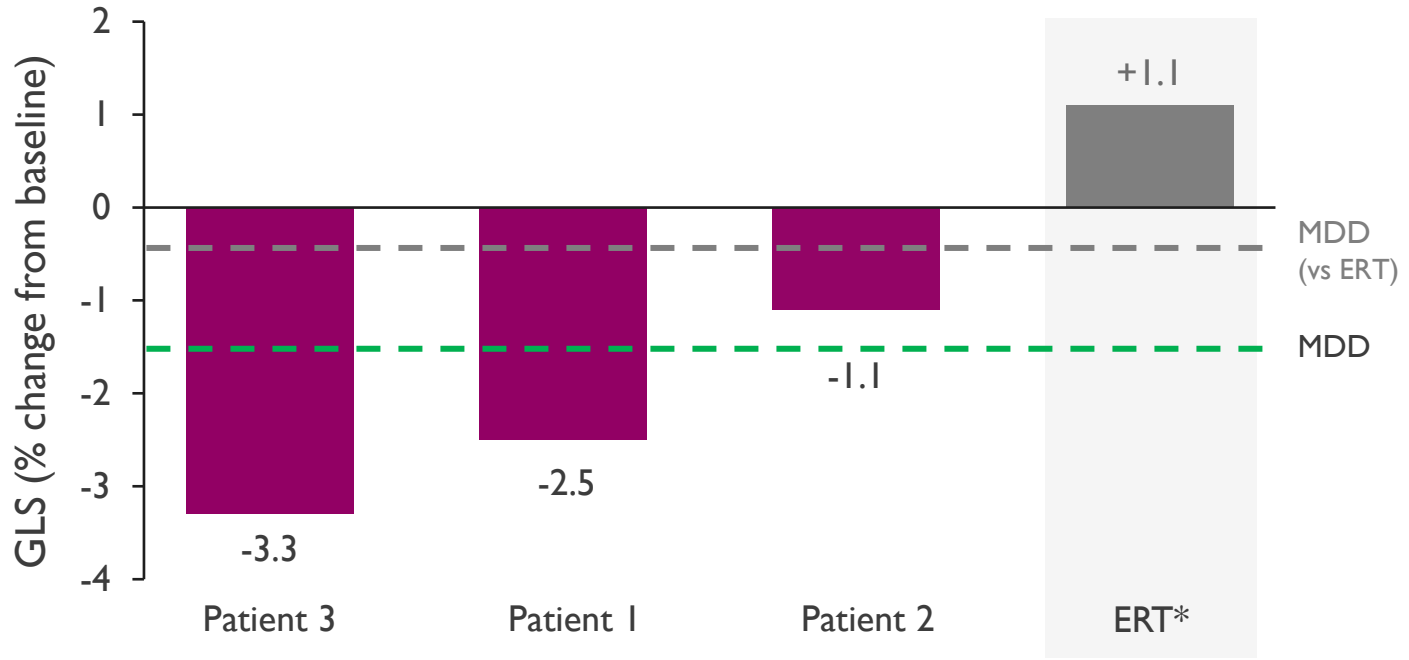
Global Longitudinal Strain (ECHO): 3 of 3 Responders

Global Longitudinal Strain (%)

Patient	Screening	Week 52	Change
1	−17.10 (Borderline)	−19.6	**−2.5**
2*	−22.17 (Normal)	−23.27	**−1.1**
3	−18.83 (Borderline)	−22.1	**−3.3**
ERT†	−13.2‡	−12.1‡	**+1.1‡**

GLS was measured in 3 apical views (4-, 3- and 2-chamber); the average value is shown.
GLS range (borderline), -16.0 to -18.0% [1]; (low), >-15.9%. Minimal detectable difference, 1.5% [2].
*High antibody titer, entered study off ERT.
†Historical control (N=18); median duration of ERT, 4.2 years (range, 1.4–12.2) [3].
‡Mean value.

Change from Baseline to Week 52 (Responders)



vs ERT	-4.4	-3.6	-2.2	n.a.

*Historical control (N=18); median duration of ERT, 4.2 years (range, 1.4–12.2) [3].

1. Yang H et al. JACC *Cardiovascular Imaging* 2018;11:1196–1201. 2. Lambert J et al. *Heart* 2020;106:817–23. 3. Nordin S et al. *Circ Cardiovasc Imaging* 2019:e009430. MDD, minimum detectable difference.

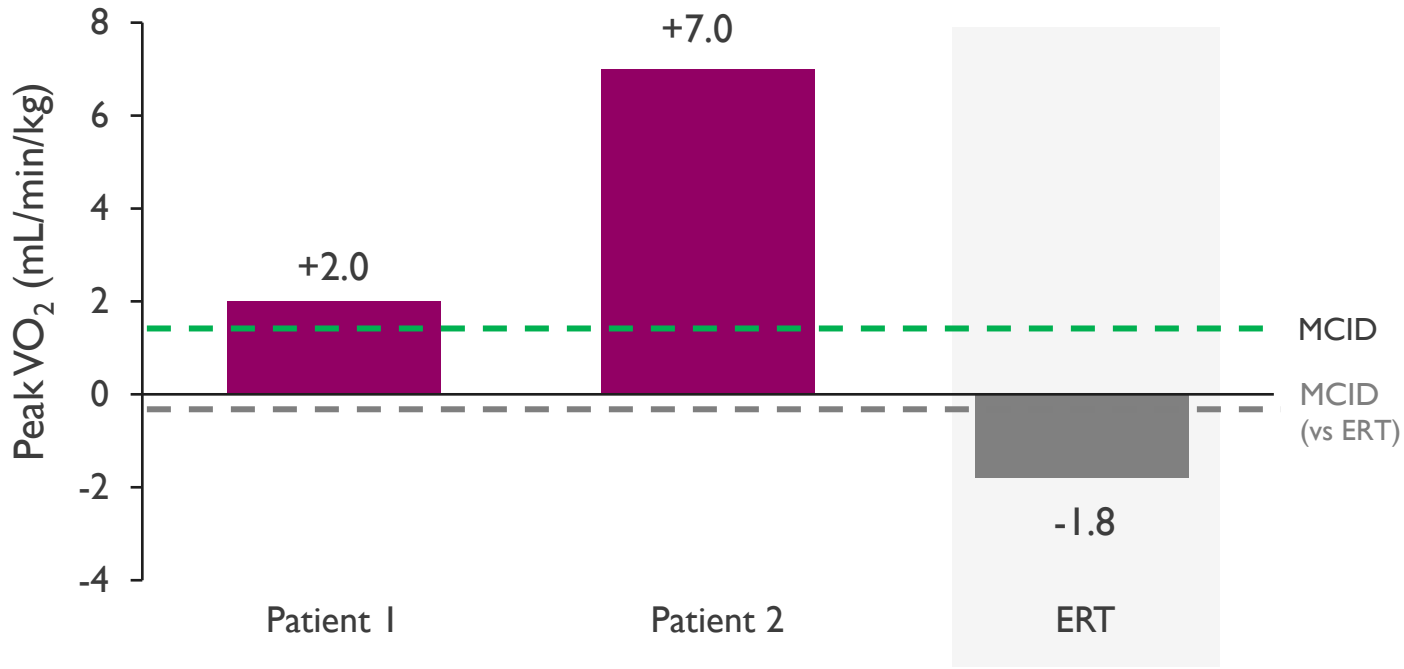
Peak VO_2 (Cardiopulmonary Exercise Testing): 2 of 3 Responders

Peak VO_2 Measurements

Pt	Peak VO_2	Screening	Week 26	Week 52	Change
1	mL/kg/min	na	21.4	23.4	+2.0*
	% predicted	na	72.0	78.3	+6.3*
2†	mL/kg/min	14.0	na	21.0	+7.0
	% predicted	33.0	na	50.0	+17.0
3	mL/kg/min	23.0	23.4	20.8	−2.2
	% predicted	66.1	65.8	58.3	−7.8
ERT‡	VO_2 max (mL/kg/min)	24.1⁋	NR	22.4⁋	−1.8⁋

Minimal clinically important difference, 1.5 mL/kg/min [1].
*Calculated as change from Week 26 to Week 52.
†High antibody titer, entered study off ERT.
‡Historical control (N=14); median duration of ERT, 48 months [2].
⁋Mean value.

Change from Baseline to Week 52 (Responders)



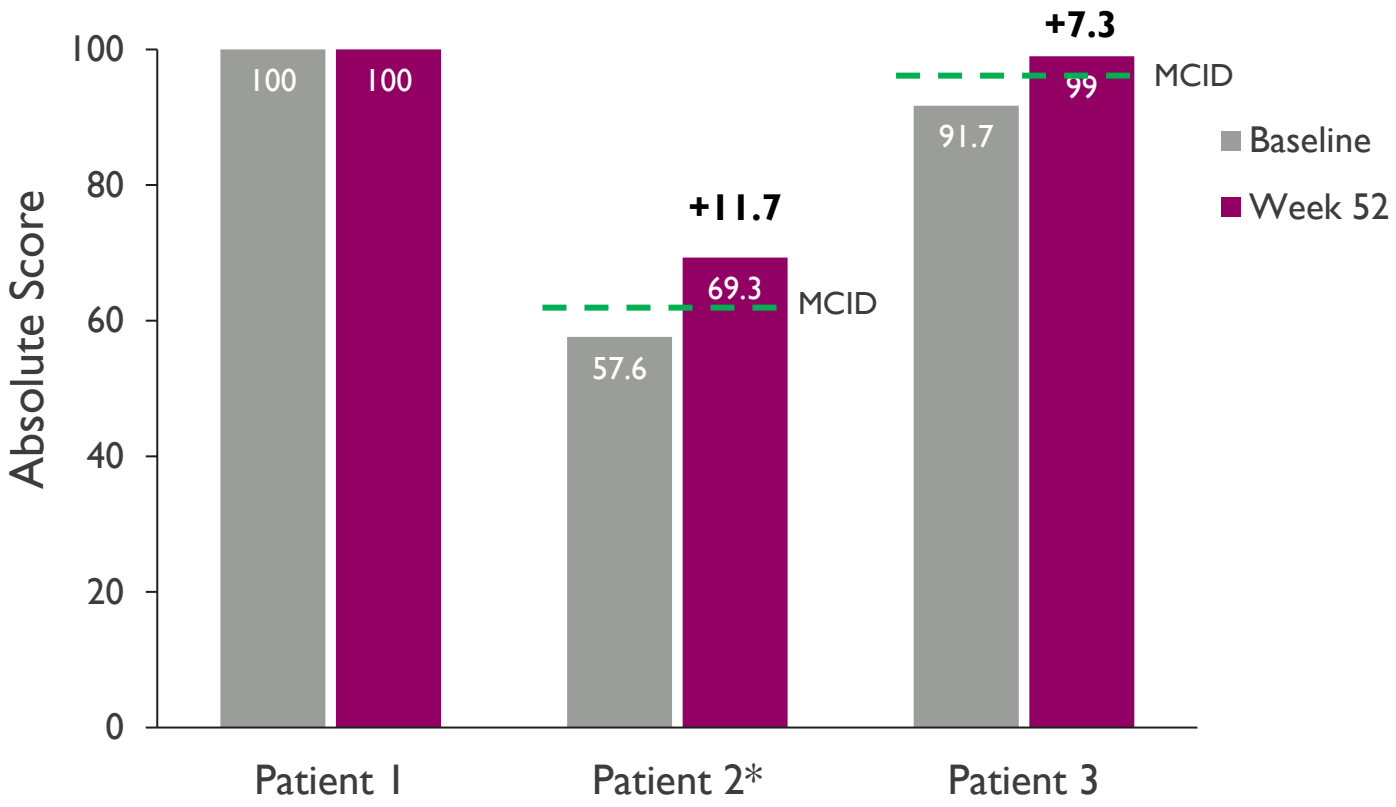
	Patient 1	Patient 2	ERT
vs ERT	+3.8	+8.8	n.a.

1. Wilkinson TJ et al. *Am J Phys Med Rehabil* 2019;98:431–7. 2. Lobo T et al. *Intern Med J* 2008;38:407–14.

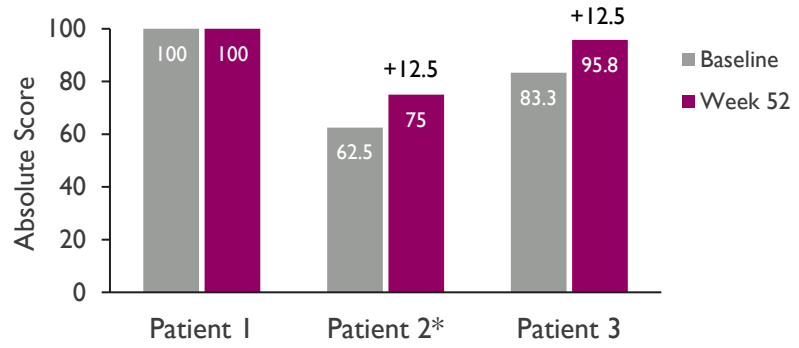
Cardiomyopathy QOL (KCCQ): 2 of 2 Responders; 1 Retained 100%

CHANGE CORRELATES WITH PEAK VO_2, 6-MINUTE WALK, HOSPITALIZATION & MORTALITY[1]

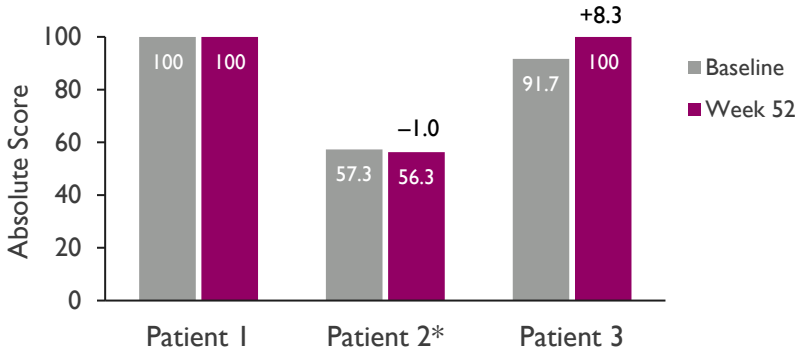
Overall Summary Score



Physical Limitation Score



Total Symptom Score



Scores range from 0 to 100 (higher scores = less severe); minimal clinically important difference (overall summary score), 5 points [1]. *High antibody titer; entered study off ERT. Abbreviations: ERT, enzyme replacement therapy.

1. Spertus JA et al. *JACC* 2020;76:2379–90.

4D-310 Cardiac Biopsy (Week 6): High-level Transgene Expression

FIRST PATIENT IN INGLAXA-2 CLINICAL TRIAL (1E13 VG/KG)*

Histology

- Healthy tissue, no inflammation
- 4 of 4 samples (+): cardiomyocytes only (ISH)
- **Est. 50% of cardiomyocytes (+) by ISH**
- 4 of 4 samples (+): AGA protein (IHC)

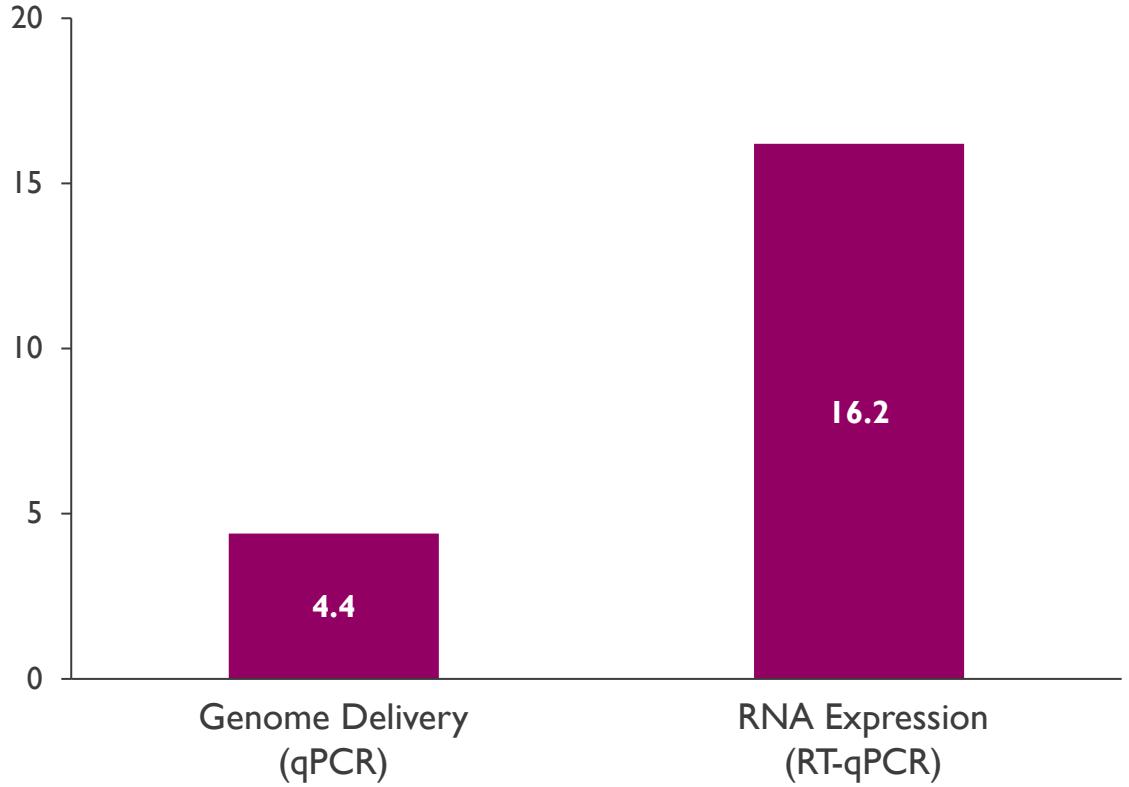
Genome Delivery (qPCR)

- 1.2 vector copies per cell
- **4.4 vector copies per cardiomyocyte**

RNA Expression (ISH & RT-qPCR):

- 4.3 transcript copies per cell
- **16.2 transcript copies per cardiomyocyte**

Cardiomyocyte Genome Delivery & RNA Expression**



*Male (57 y) with late onset Fabry disease (*GLA* variant: IVS4 + 919G>A); **As calculated based on cardiomyocyte to heart cell ratio (est. 30% of all cells); baseline anti-AGA antibody titer negative; baseline serum AGA activity, 1.62 nmol/hr/mL; baseline serum lyso-Gb3 concentration, 3.79 ng/mL; entered study on ERT. †Endogenous AGA protein observed in commercially acquired cardiac tissue samples. AGA, α-galactosidase A, ERT, enzyme replacement therapy; IHC, immunohistochemistry; ISH, *in situ* hybridization.

Safety, Tolerability & aHUS

Mechanism, Investigation & Mitigation Strategies

With Dr. PJ Utz, member of 4DMT Scientific Advisory Board

Interim Safety & Tolerability: aHUS & NO Liver, Cardiac, DRG Tox

COHORT 1, INGLAXA-1 & -2: 1E13 VG/KG & CORTICOSTEROID IMMUNE REGIMEN

- Total n=6 dosed
- Generally well-tolerated after aHUS "window"
- **NO liver, heart or DRG toxicity observed**
- **Transient acute aHUS** (n=3): admission & observation
  - Active aHUS process: ~Day 3-7 onset; resolution started within ~1-4 days
  - No intervention (n=1): discharge ~4 days
  - Eculizumab (n=1): discharge ~1 week
  - **Eculizumab & temporary dialysis (n=1): discharge ~1 week**
    - **DLT (Gr 4) - 4DMT hold on enrollment f/b FDA hold**
- 69 y.o. pt with DLT (Gr 4 aHUS) investigation

aHUS with Intravenous AAV Delivery: Mechanism & Risk Factors

- **Class effect:** Dose-related IV AAV for cardiac & muscle[1]
- **Timing:** Initiation ~3–7 days after administration
- **Mechanism:** Rapid IgM rise → capsid binding → complement activation
- **aHUS risk factors:**
  - **Known**: High AAV doses (~7E13 – 4E14 vg/kg)
  - **Investigation on:**
    - Patient 6 (DLT) vs other INGLAXA patients

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National Center for Research, Greece

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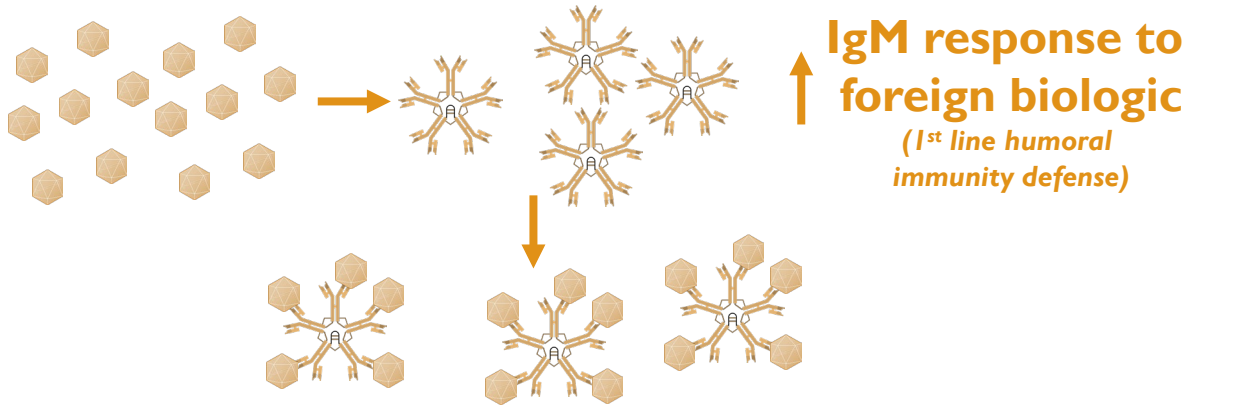
University of Florida

1. U.S. FDA Cellular, Tissue, and Gene Therapies Advisory Committee. September 2-3, 2021.

aHUS Following IV AAV: Mechanism of Complement Activation

RAPID IGM ANTIBODY INDUCTION WHILE CAPSID IN BLOOD LEADS TO CLASSICAL PATHWAY ACTIVATION



IgM response to foreign biologic

(1st line humoral immunity defense)

Activates Classical Pathway

Alternative Pathway

C3b

C3bBb

C3

C3a

C5

C5a

C5b-C9

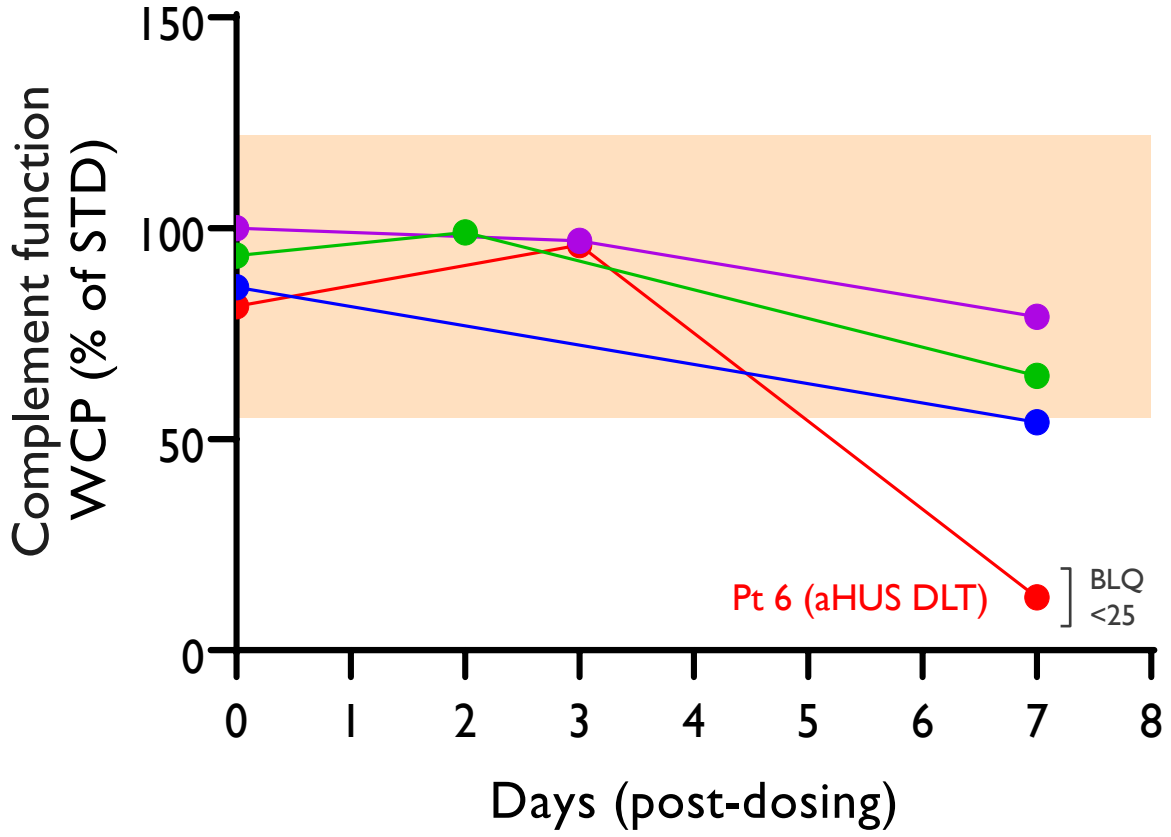
MAC

aHUS

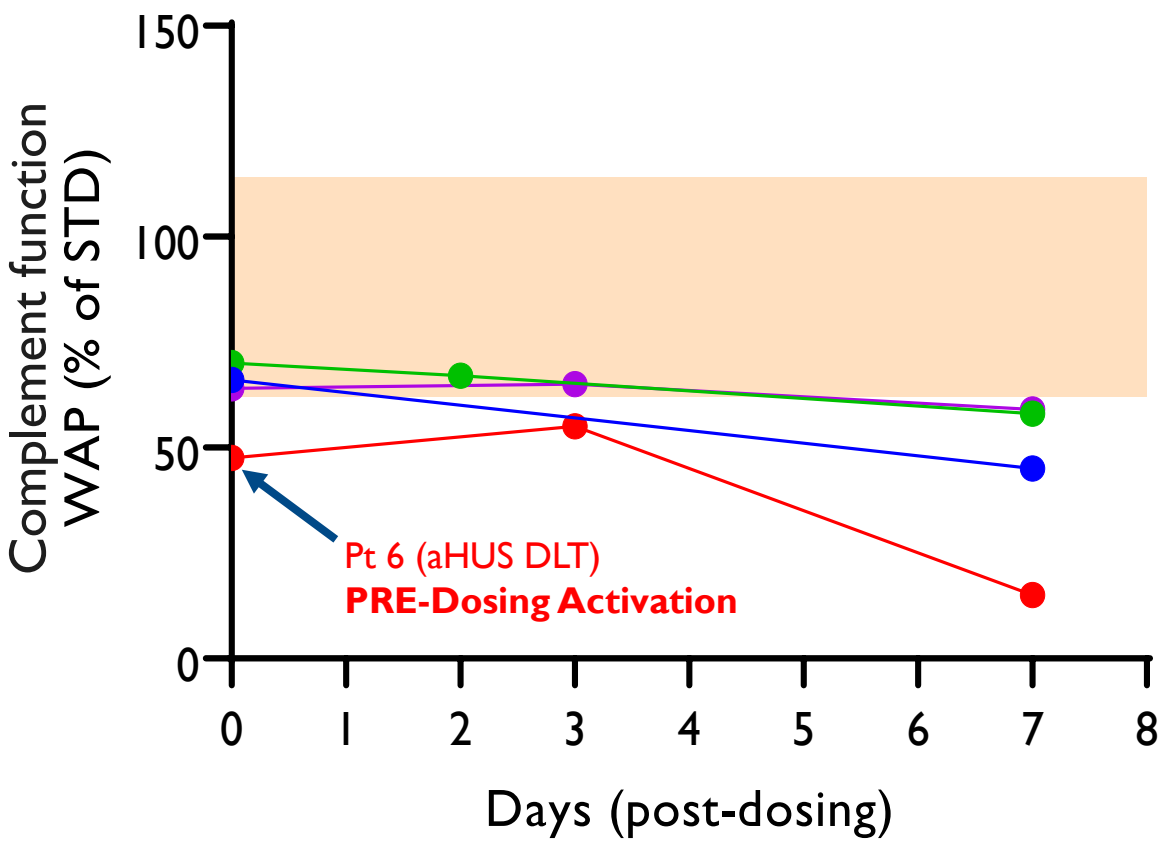
Patient 6 DLT Investigation: Functional Complement Assays

PRE-DOSING ACTIVATION IN PT 6 (DLT)

Classical
Pathway Activity



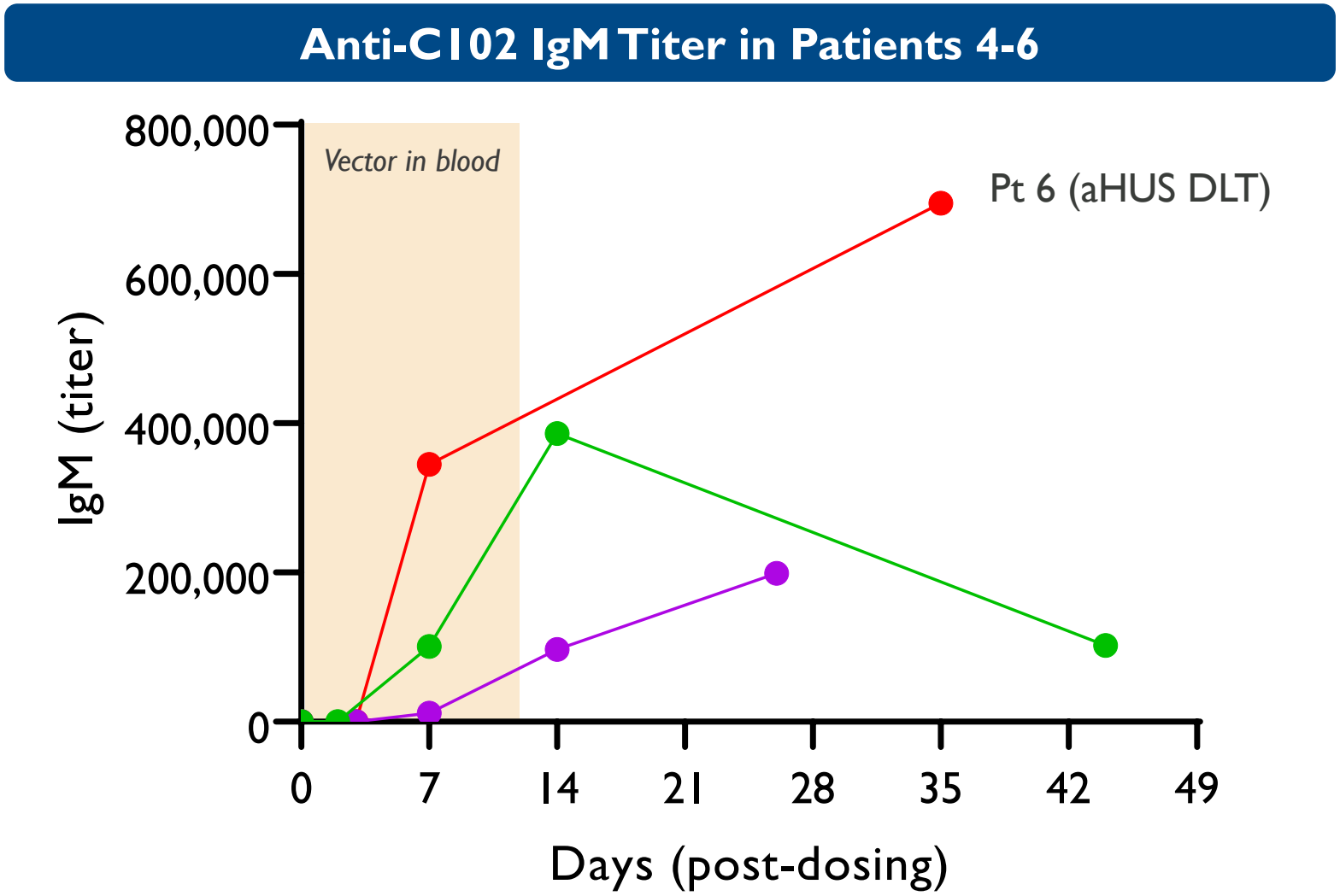
Alternative
Pathway Activity



aHUS, atypical hemolytic uremic syndrome; BLQ, below the level of quantitation. WCP (Wieslab classical pathway assay) / WAP (Wieslab alternative pathway assay) = measuring residual complement function.

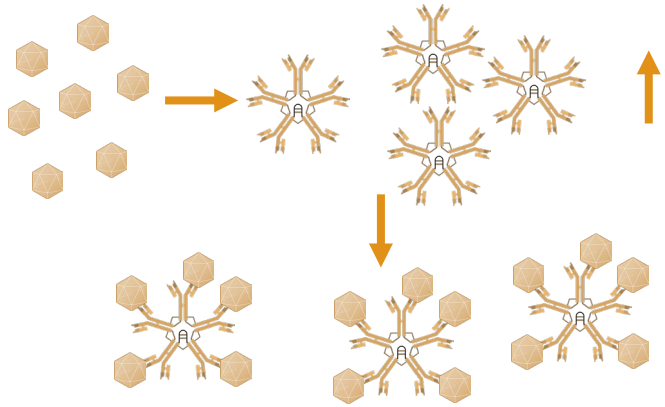
Patient 6 DLT Investigation: Post-Dosing IgM Antibody Titer Response

ELEVATED IN DLT PATIENT



Patient 6 DLT Investigation: Dual Risk Factors for Severe aHUS

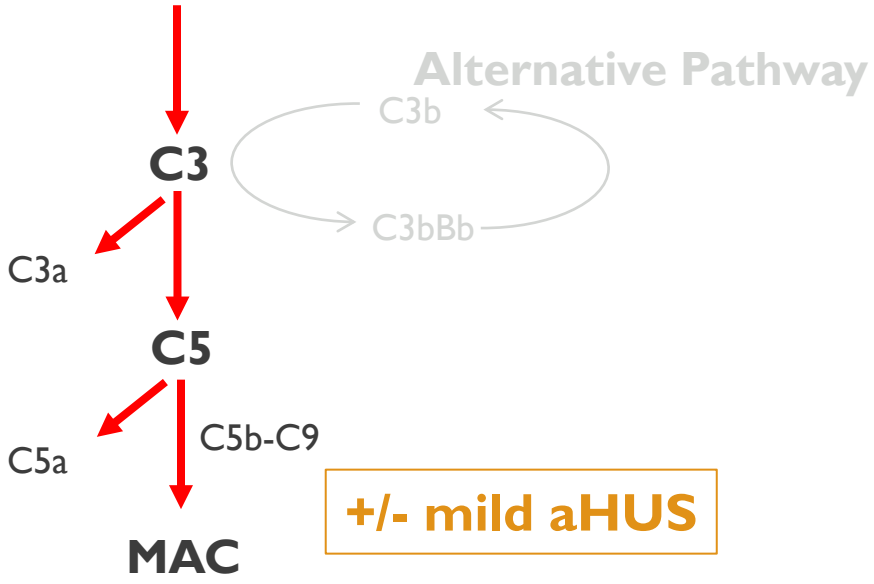
ALTERNATIVE PATHWAY ACTIVATION PRE-DOSING AND HYPER IGM RESPONSE POST-DOSING TRIGGER SEVERE AHUS RESPONSE



IgM response to foreign biologic

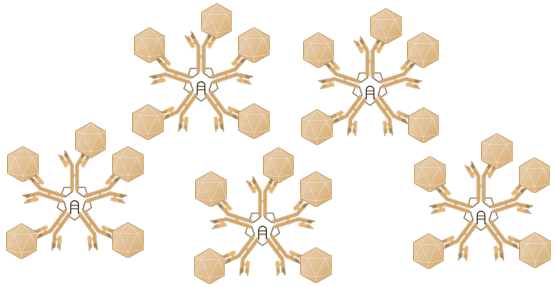
(1st line humoral immunity defense)

Activates Classical Pathway



+/- mild aHUS

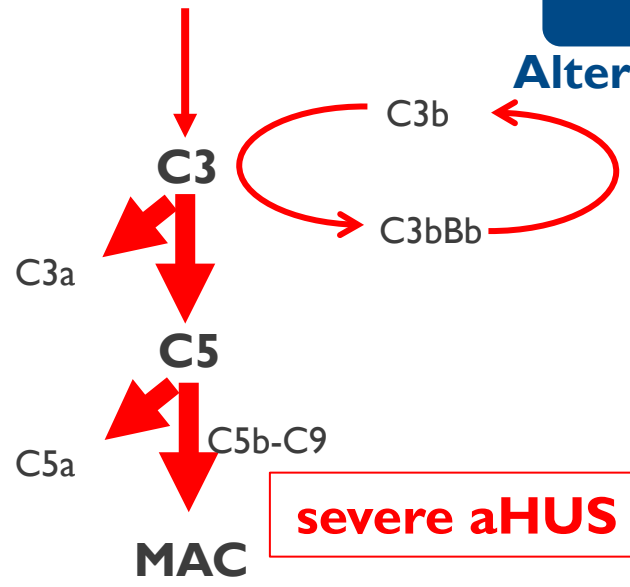
Hyper IgM Response Post-Dosing



Activates Classical Pathway

Complement Activation Pre-Dosing

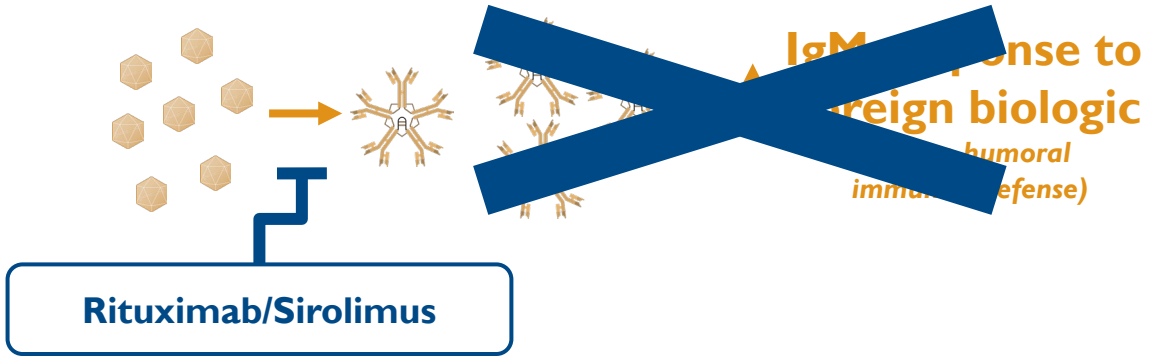
Alternative Pathway Activated



severe aHUS

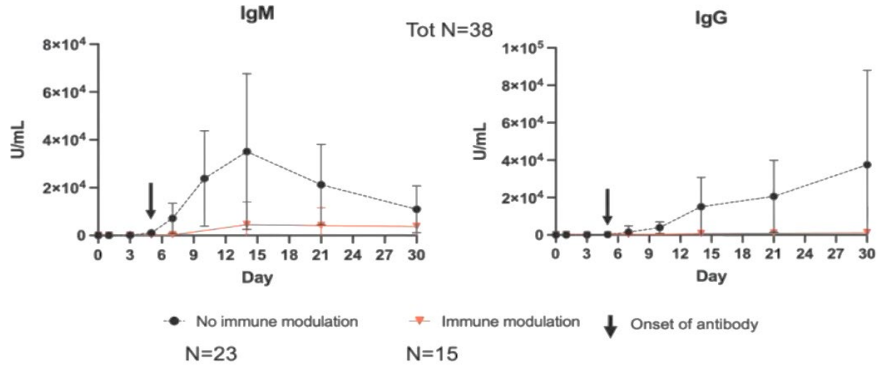
Prevention of AAV aHUS with Rituximab/Sirolimus: Strong Clinical Evidence

ASGCT-FDA JAN 2023; DRS. BYRNE & CORTI; N=38, 15 ON RITUXIMAB/SIROLIMUS (R/S)



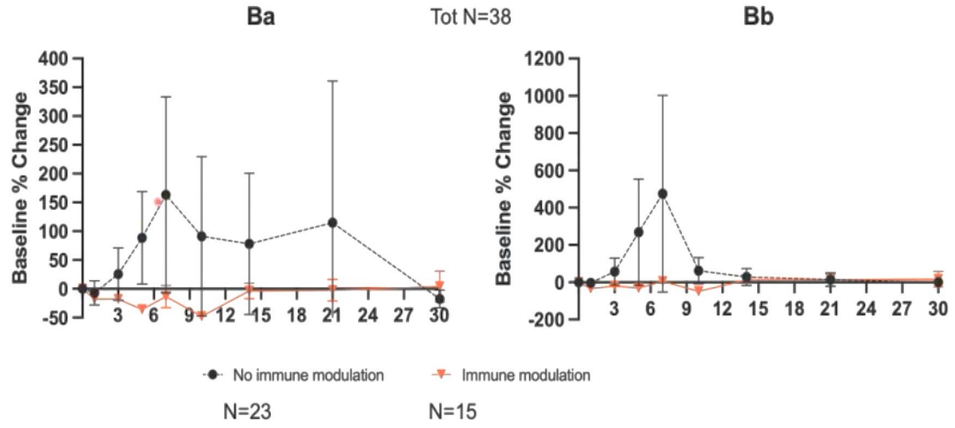
Classical Pathway NOT Activated

Anti-AAV9 IgM and IgG Following AAV9 Therapy



IgG, immunoglobulin G; IgM, immunoglobulin M; Immune modulation=Rituximab+Sirolimus

Ba and Bb Trend Following AAV9 Therapy



Prevention of AAV aHUS with Rituximab/Sirolimus: Strong Clinical Evidence

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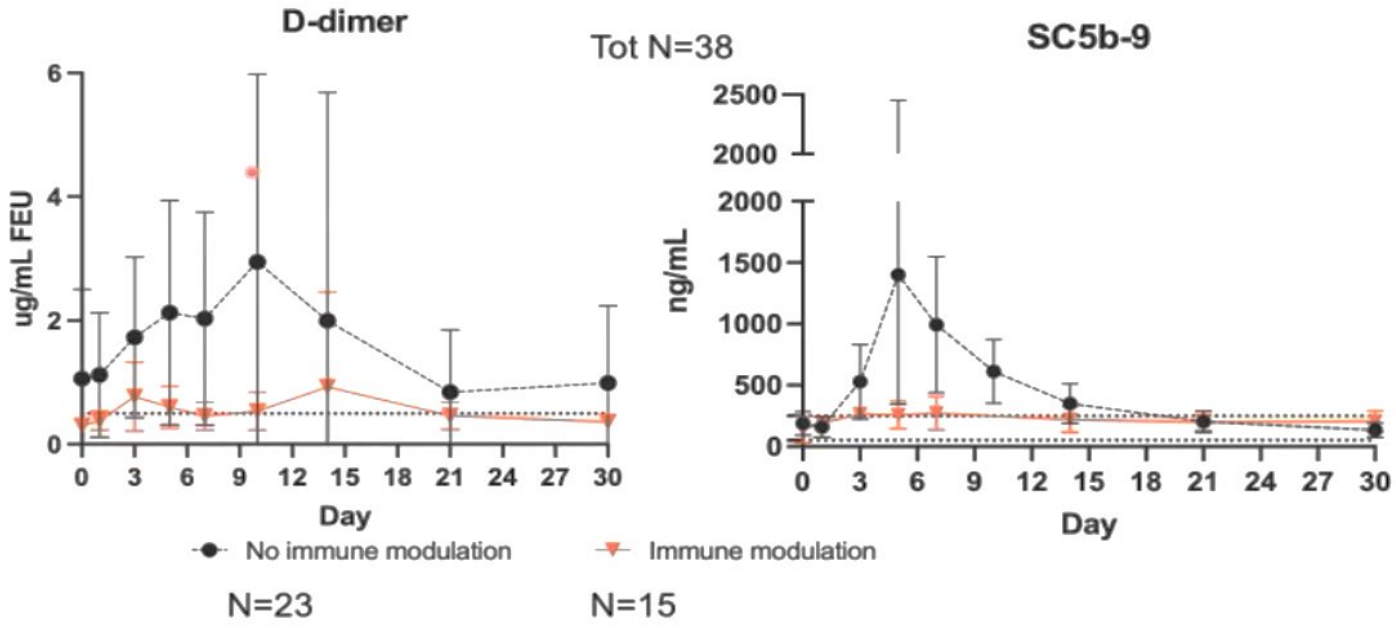
Pretreatment with R/S Prevents aHUS (TMA Process)



FEU, fibrinogen equivalent units; SC5b-9, serum complement protein complex C5b-9, also known as the membrane attack complex

aHUS Will Be Managed with:
1 Rituximab/Sirolimus and 2 Exclusion Criteria

IgM response to foreign biologic
(1st line humoral defense)

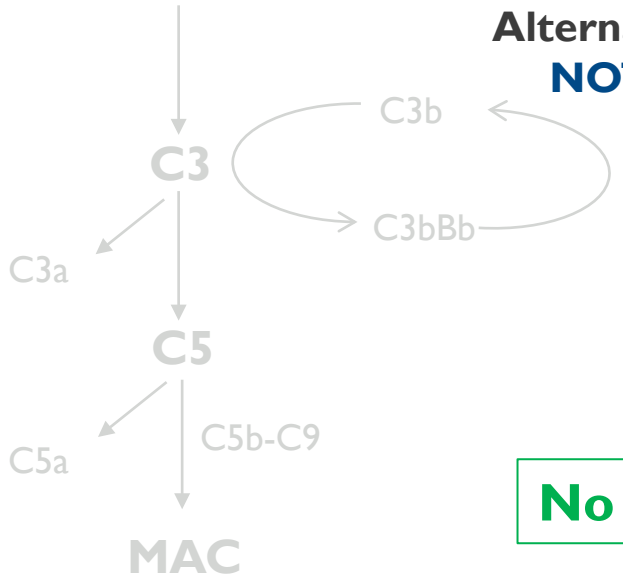
1 **Rituximab/Sirolimus**

2

Implement exclusion criteria:
- Prescreening for complement
- Exclusion of alternative pathway activation

Classical Pathway NOT Activated

Alternative Pathway NOT Activated



No aHUS

Summary: 4D-310 Phase 1/2 Clinical Trial Cardiac Proof-of-Concept

SIX PATIENTS (1E13 vg/kg) WITH CORTICOSTEROID PROPHYLAXIS

- **Cardiac activity demonstrated at 12 months:**
  - LV function (ECHO): 3 of 3
  - Exercise capacity (CPET): 2 of 3
  - Quality of life (KCCQ): 2 of 2 (1 SD 100%)
  - Activity despite Ab to AGA & C102 capsid
- **Positive cardiac biopsy:** Transgene delivery & expression in cardiomyocytes*
- **Generally well-tolerated:** No liver, heart or DRG toxicity observed
- **Transient acute aHUS on corticosteroids (n=3):**
  - Resolved in all 3
  - Investigation identified:
    - Pre-dosing: complement activation
    - Post-dosing: heightened production of IgM contributing to accelerated complement activation
    - C102 shown to not activate the complement pathways directly

*INGLAXA-2 clinical trial (n=1). aHUS, atypical hemolytic uremic syndrome; cMRI, cardiac magnetic resonance imaging; CPET, cardiopulmonary exercise test; KCCQ, Kansas City Cardiomyopathy Questionnaire; SAE, serious adverse event.

Next Steps for Development of 4D-310

- **Resume enrollment:**
  - Pre-screen for complement activation: exclude if (+)
  - Rituximab/sirolimus immunosuppressive regimen
  - Align with FDA to remove hold
- **Cardiac assessments continuing – endpoints supporting approval:**
  - Peak VO_2 (CPET)
  - Quality of life (KCCQ)
  - Left ventricular function (global longitudinal strain/echocardiography)
- **Phase 3 CMC & trial design aligned with FDA**

4D-310 for Fabry Disease Cardiomyopathy: Business Rationale

THIRD PROPRIETARY VECTOR VALIDATED IN CLINICAL TRIALS – PLATFORM VALIDATION & BD OPPORTUNITY

- **4D-310 for Fabry Disease Cardiomyopathy:**
  - No competition in heart disease
  - Significantly lower doses & COGs vs conventional AAV
- **C102 Vector Bus Dev Opportunities:**
  - Significant interest in cardiotropic vectors
  - Superior to conventional AAV including AAV8 & AAV9
- **Platform Validation:**
  - Three vectors/TAs: all validating in clinical trials
  - IV organ targeting with R/S immune regimen: safety readthrough all IV programs

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INGLAXA

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- Study staff
- Patients and their families

4DMT

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