Selecta Biosciences

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Selecta Biosciences Corporate Presentation

March 2023

Safe harbor / disclaimer

Any statements in this presentation about the future expectations, plans and prospects of Selecta Biosciences, Inc. (the "Company"), including without limitation, statements regarding the Company's cash runway, the unique proprietary technology platform of the Company, and the unique proprietary platform of its partners, the potential of ImmTOR to enable re-dosing of AAV gene therapy and to mitigate immunogenicity, the potential of ImmTOR and the Company's product pipeline to treat chronic refractory gout, MMA, IgAN, other autoimmune diseases, lysosomal storage disorders, or any other disease, the anticipated timing or the outcome of ongoing and planned clinical trials, studies and data readouts, the anticipated timing or the outcome of the FDA's review of the Company's regulatory filings, the Company's and its partners' ability to conduct its and their clinical trials and preclinical studies, the timing or making of any regulatory filings, the potential treatment applications of product candidates utilizing the ImmTOR platform in areas such as gene therapy, gout and autoimmune disease, the ability of the Company and its partners where applicable to develop gene therapy products using ImmTOR, the novelty of treatment paradigms that the Company is able to develop, whether the observations made in non-human study subjects will translate to studies performed with human beings, the potential of any therapies developed by the Company to fulfill unmet medical needs, the Company's plan to apply its ImmTOR technology platform to a range of biologics for rare and orphan genetic diseases, the potential of the Company's technology to enable repeat administration in gene therapy product candidates and products, the ability to re-dose patients and the potential of ImmTOR to allow for re-dosing, the potential to safely re-dose AAV, the ability to restore transgene expression, the potential of the ImmTOR technology platform generally and the Company's ability to grow its strategic partnerships, and other statements containing the words "anticipate," "believe," "continue," "could," "estimate," "expect," "hypothesize," "intend," "may," "plan," "potential," "predict," "project," "should," "target," "would," and similar expressions, constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including, but not limited to, the following: the uncertainties inherent in the initiation, completion and cost of clinical trials including proof of concept trials, including the uncertain outcomes, the availability and timing of data from ongoing and future clinical trials and the results of such trials, whether preliminary results from a particular clinical trial will be predictive of the final results of that trial or whether results of early clinical trials will be indicative of the results of later clinical trials, the ability to predict results of studies performed on human beings based on results of studies performed on non-human subjects, the unproven approach of the Company's ImmTOR technology, potential delays in enrollment of patients, undesirable side effects of the Company's product candidates, its reliance on third parties to manufacture its product candidates and to conduct its clinical trials, the Company's inability to maintain its existing or future collaborations, licenses or contractual relationships, its inability to protect its proprietary technology and intellectual property, potential delays in regulatory approvals, the availability of funding sufficient for its foreseeable and unforeseeable operating expenses and capital expenditure requirements, the Company's recurring losses from operations and negative cash flows, substantial fluctuation in the price of its common stock, risks related to geopolitical conflicts and pandemics and other important factors discussed in the "Risk Factors" section of the Company's most recent Annual Report on Form 10-K, and in other filings that the Company makes with the Securities and Exchange Commission. In addition, any forward-looking statements included in this presentation represent the Company's views only as of the date of its publication and should not be relied upon as representing its views as of any subsequent date. The Company specifically disclaims any intention to update any forward-looking statements included in this presentation.

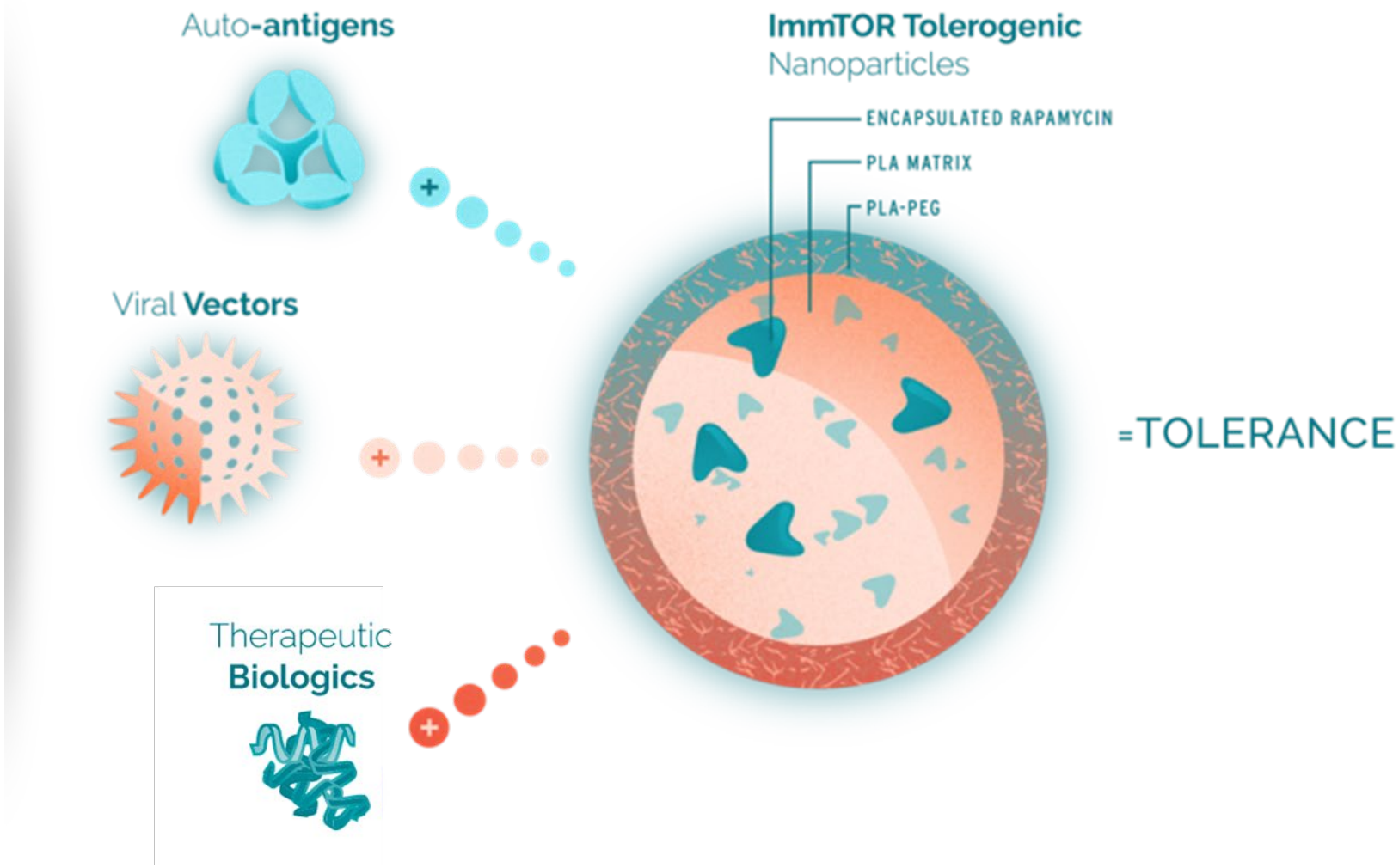
Pioneering Precision Immune Tolerance

1. **ImmTOR® and ImmTOR-IL™ immune tolerance platforms** have potentially broad applicability to address the challenges of **autoimmunity and immunogenicity**
2. **Diversified pipeline** of novel therapeutic candidates; proof of concept in biologics and gene therapy, **expanding into autoimmune disease**
3. **Topline Phase 3 data for SEL-212** in chronic refractory gout expected in **Q1 2023**, with additional value-generating milestones anticipated in 2023
4. **Multiple strategic partnerships designed to** validate platform and maximize its potential
5. Strong balance sheet, positioned to reach multiple value inflection points with expected **runway into mid-2024**

Proprietary precision immune tolerance platform with potentially broad applicability

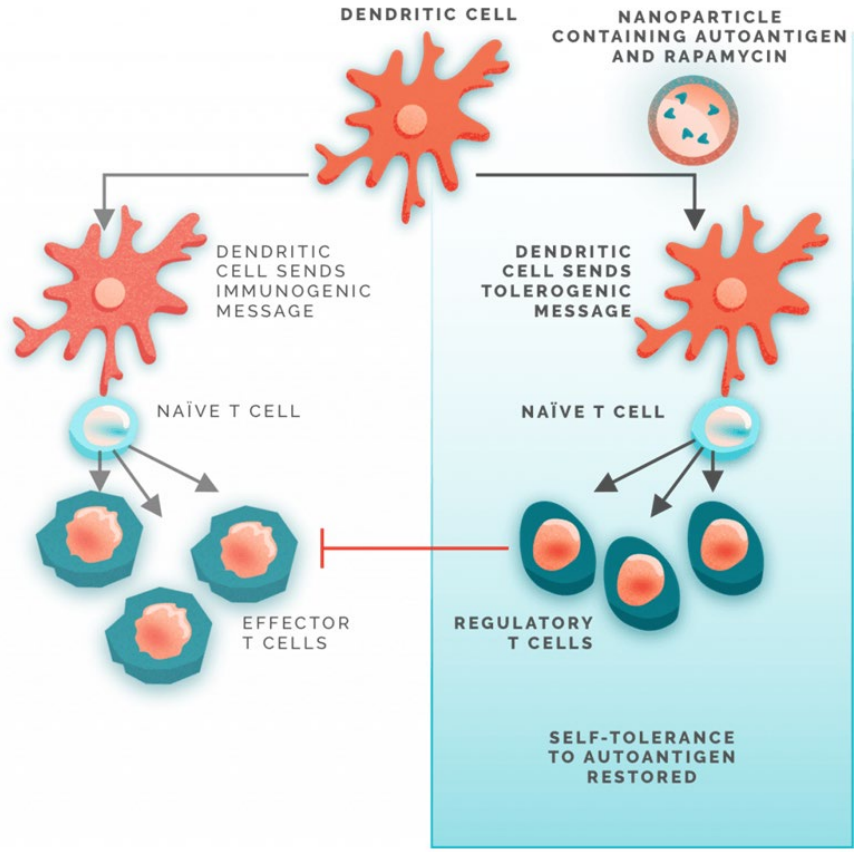
ImmTOR combines nanoparticle technology with an FDA approved anti-inflammatory and immunomodulatory drug

Designed to generate **antigen-specific immune tolerance** when combined with an antigen of interest

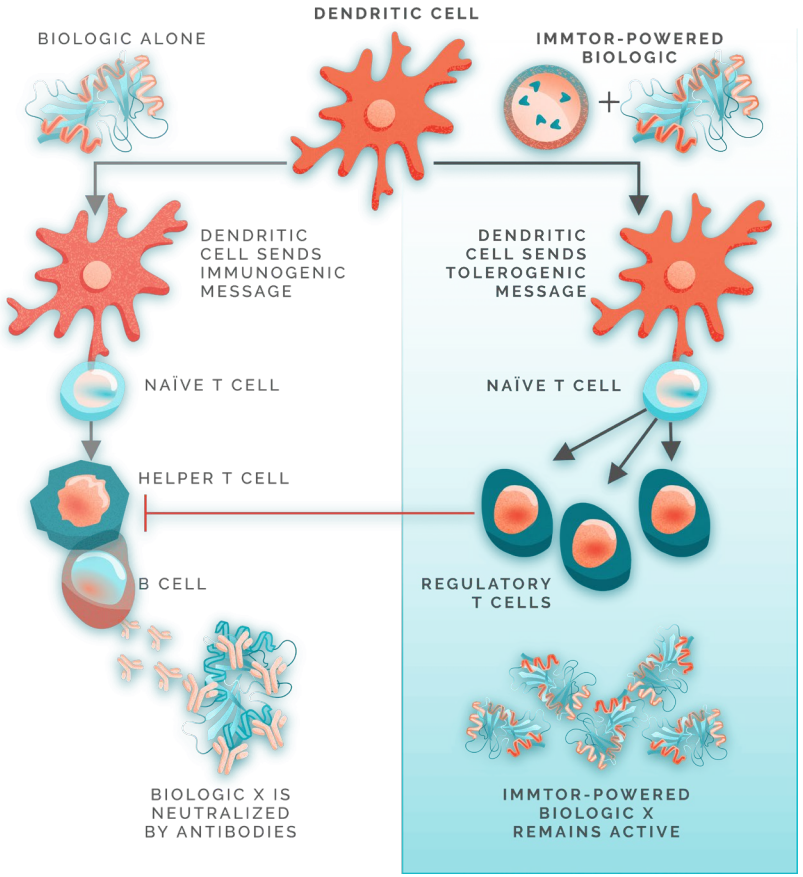


ImmTOR aims to restore self tolerance in autoimmune disease and overcome immunogenicity of gene therapies and biologics

Autoimmune Disease



Gene Therapies/Biologics



A diversified and growing wholly-owned pipeline

Indication	Antigen	Preclinical	Phase 1	Phase 2	Phase 3	Recent and Expected Upcoming Milestones
TOLEROGENIC THERAPIES						
Autoimmune indications	PDC-E2					
Undisclosed						
GENE THERAPIES						
Methylmalonic acidemia (MMA)	AAV (serotype undisclosed)	SEL-302				Phase 1 start Q4 2022
BIOLOGIC THERAPIES						
IgA nephropathy (IgAN)	IgA protease*					

*Option & License Agreement with IGAN Biosciences

Unlocking the potential of our platform through collaborations

Indication	Antigen	Preclinical	Phase 1	Phase 2	Phase 3	Upcoming Milestones	Commercial Rights
BIOLOGIC THERAPIES							
Chronic Refractory Gout	Pegadricase			SEL-212		DISSOLVE I & II Topline Data Q1 2023	sobi rare strength
GENE THERAPIES							
Pompe disease	IgG protease (Xork)			SEL-018			astellas
Duchenne muscular dystrophy (DMD)	Undisclosed						SAREPTA THERAPEUTICS
Limb-girdle muscular dystrophy (LGMD)	Undisclosed						SAREPTA THERAPEUTICS
Two indications for lysosomal storage disorders	Undisclosed						Takeda

Restoring Self-Tolerance in Autoimmune Disease

Striving to restore self-tolerance in autoimmune diseases

ImmTOR + IL-2 has the potential to be a best-in-class approach

THE **Challenges**

- Roughly **80 autoimmune conditions** affect as much as **4.5% of the world's population*; 24M+ individuals in the US** alone are affected by autoimmune diseases**
- The current standard of care is broad immunosuppression, which is associated with side effects and leaves patients vulnerable to serious infection and malignancies
- There is a significant need for **antigen-specific** therapies that can induce immune tolerance to pathogenic autoantigens without the need for chronic and systemic immune suppression

THE **Solution**

- Approach: restore **natural self-tolerance by administering ImmTOR with nanoparticle-encapsulated self-antigens** and avoid the need for chronic and systemic immune suppression
- **Aiming to expand antigen-specific Tregs and enhance durability of tolerance by developing a proprietary Treg-selective IL-2 to combine with ImmTOR and autoantigens**

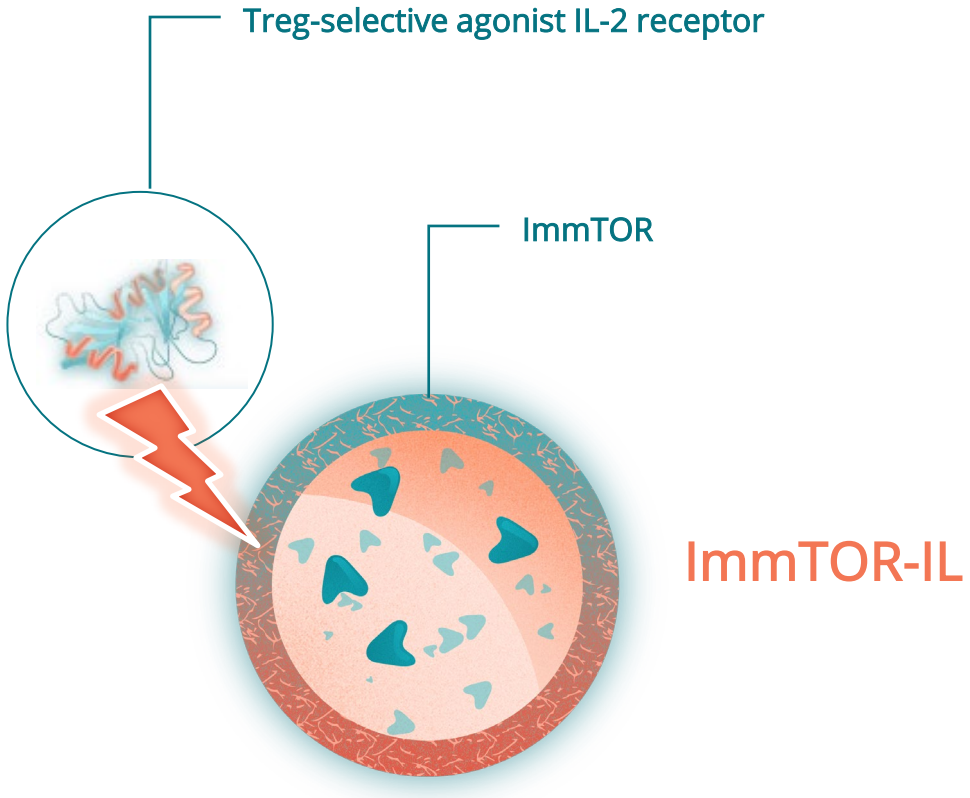
*Autoimmune Disease, by the Numbers" in Scientific American 325, 3, 31-33 (September 2021), doi:10.1038/scientificamerican0921-31
**https://www.niehs.nih.gov/health/topics/conditions/autoimmune/index.cfm

ImmTOR-IL : ImmTOR plus IL-2 receptor agonist

Evolution of the ImmTOR Platform

- Synergistic mechanism of ImmTOR and a Treg-selective IL-2
- Identified an **interleukin-2 (IL-2) cytokine with plans to advance it through the next stage of development**
- Observed to greatly increase the magnitude and durability of **antigen-specific Treg** expansion when compared to either ImmTOR or IL-2 alone
- Proof of concept human data in which we observed ImmTOR alone and IL-2 alone lowers the translational risk and provides further confidence in the clinical utility of this potentially synergistic approach
- Potential to enable **lower and fewer doses of ImmTOR**

	IL-2 mutein	ImmTOR	ImmTOR-IL
Induce Treg	✗	✓	✓
Expand existing Tregs	✓	✗	✓
Antigen-specific	✗	✓	✓
	Expansion of all pre-existing Tregs	Induction of target antigen-specific Tregs	Induction and expansion of antigen-specific Tregs

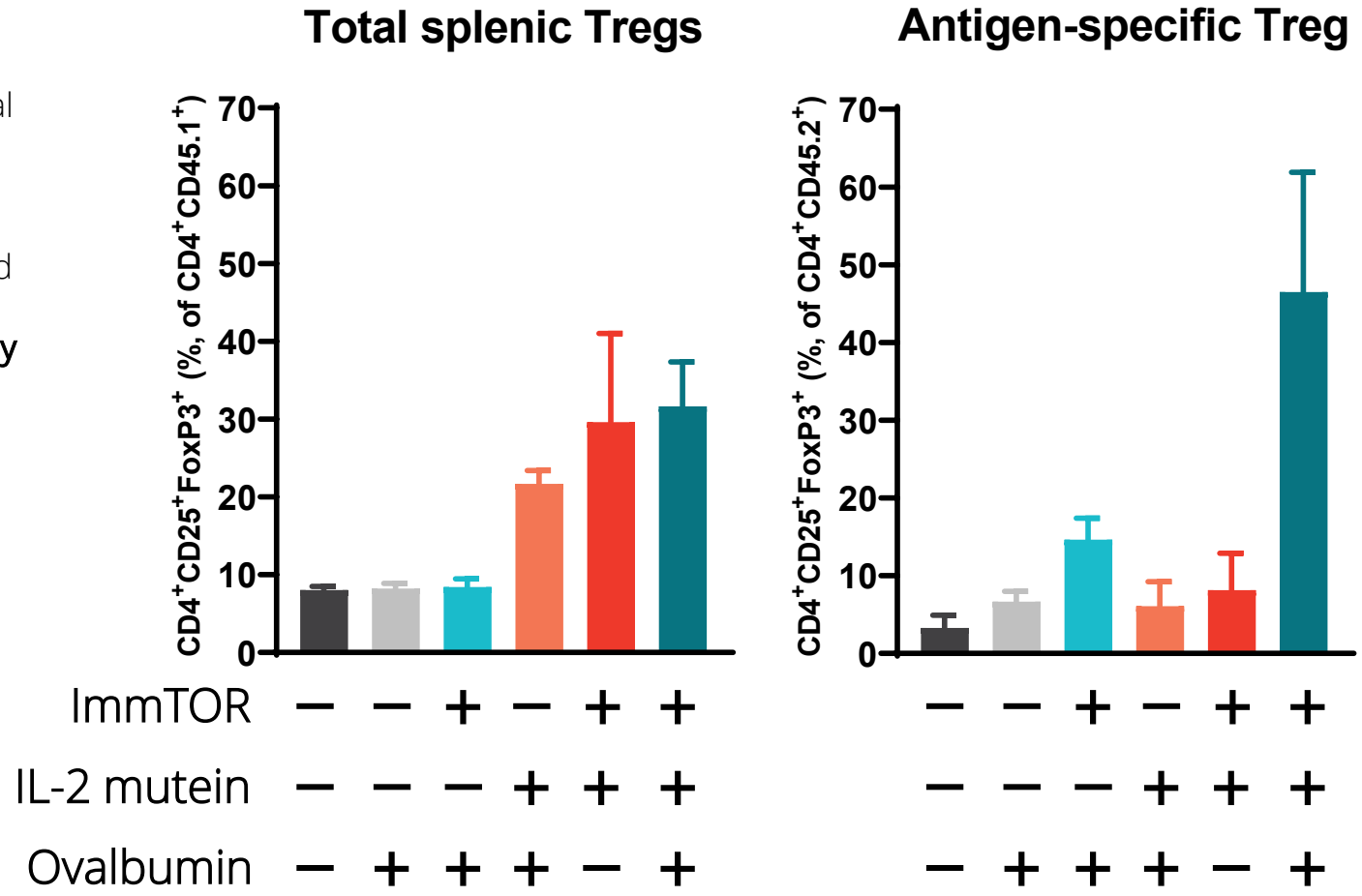


Induction and expansion of antigen-specific Treg

Observed a significant expansion of antigen-specific Treg* with a single dose of ImmTOR in combination with an IL-2 mutein + antigen

With superior expansion and durability of total Tregs, Selecta potentially **has a best-in-class IL-2 therapy**

Data believed to show an approximately 3-fold increase in antigen-specific Tregs, and an opportunity to enable a **"first in class" therapy for autoimmune disorders**



*study conducted in wildtype mice after adoptive transfer of ovalbumin specific transgenic T-cells

ImmTOR-IL mitigates immunogenicity of high vector dose AAV gene therapy

ImmTOR + 4 monthly doses of IL-2 mutein inhibits anti-AAV antibodies at 5E13 vg/kg dose

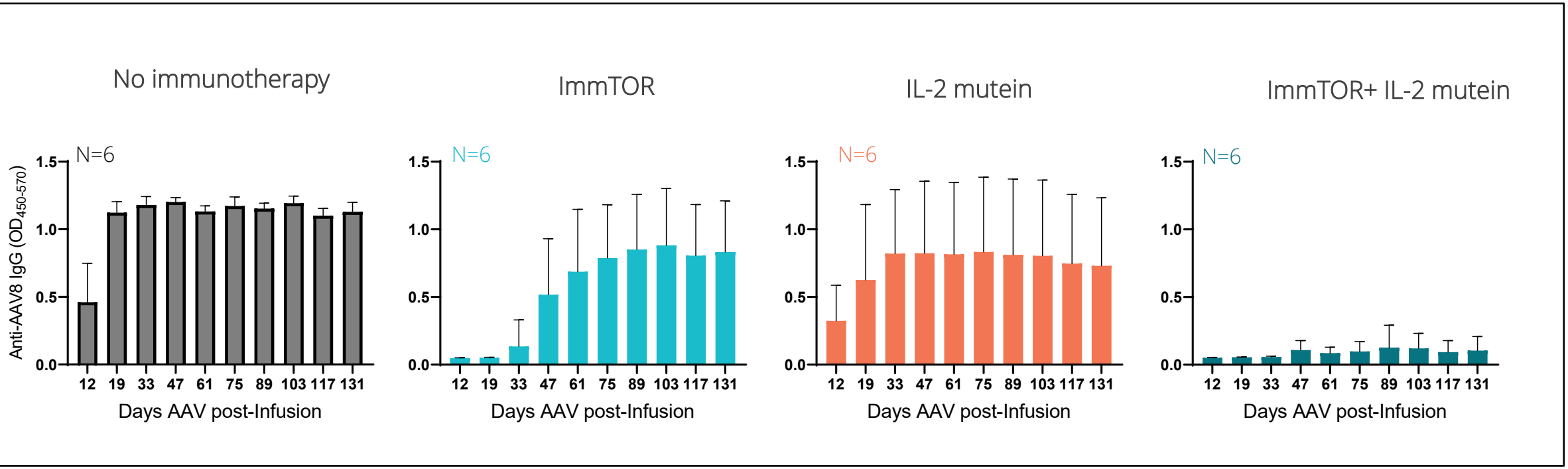
200 μg ImmTOR
9 μg IL-2 mutein

AAV8-SEAP
+/-ImmTOR
+/-IL-2 mutein — Day 0

+/-IL-2 mutein — Day 28

+/-IL-2 mutein — Day 56

+/-IL-2 mutein — Day 84

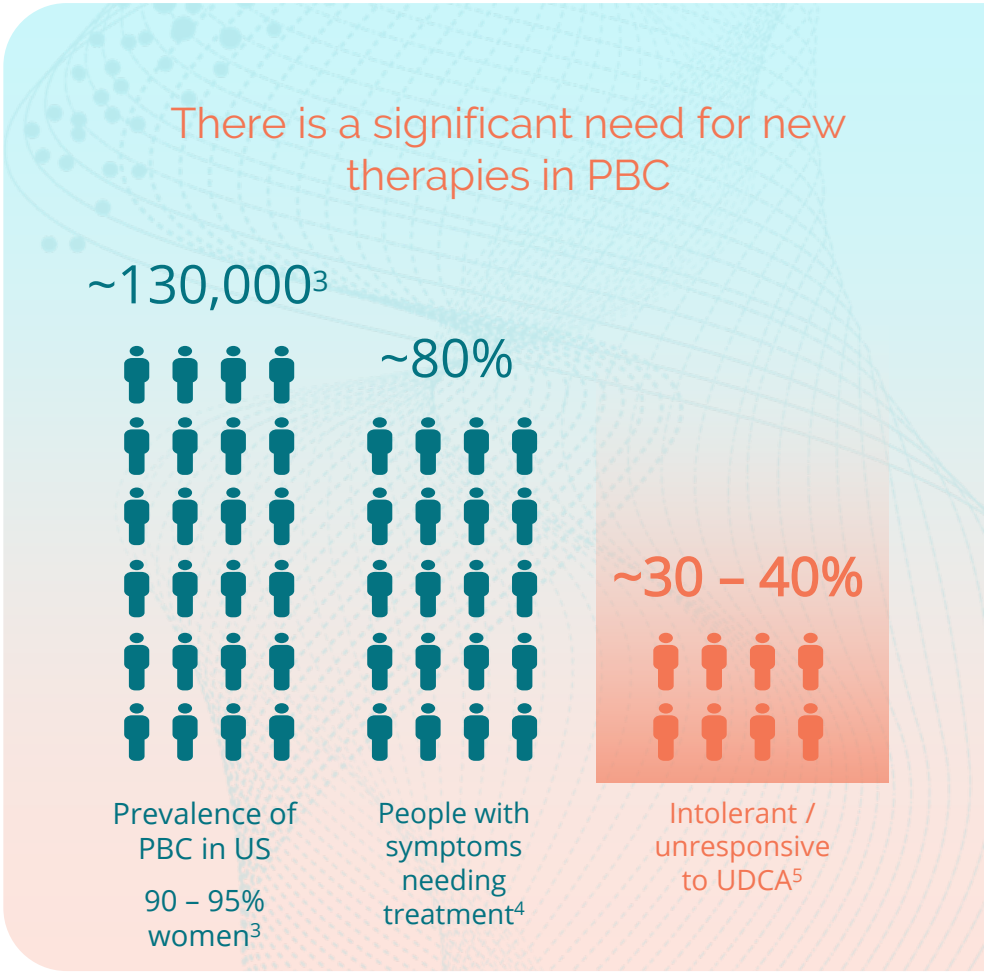


Preclinical data presented at ASGCT 2022, Poster title: Combination of ImmTOR Tolerogenic Nanoparticles and IL-2 Mutein Synergistically Inhibits the Formation of Anti-AAV Antibodies

Initial autoimmune disease focus: Primary Biliary Cholangitis (PBC)

ImmTOR + PDC-E2 antigen has the potential to address underlying autoimmune disease

- PBC is a rare T-cell mediated autoimmune liver disease
  - It is driven by a well-defined pathogenic antigen: **PDC-E2**
  - Leads to bile duct damage, progressive inflammation, scarring (cirrhosis) and eventually, liver failure
- Current therapies do not address underlying disease or key symptoms
  - **30 - 40%** of patients are intolerant / unresponsive to current SoC (UDCA[1]), and OCA[2] is marred by high AE rates and black box warnings
- Our approach has the potential to directly address underlying disease
  - In preclinical studies ImmTOR induced a **strong tolerogenic environment and showed hepatoprotective properties in liver injury models**
  - Co-administration of ImmTOR with PDC-E2 has the potential to **restore immune tolerance in the liver**



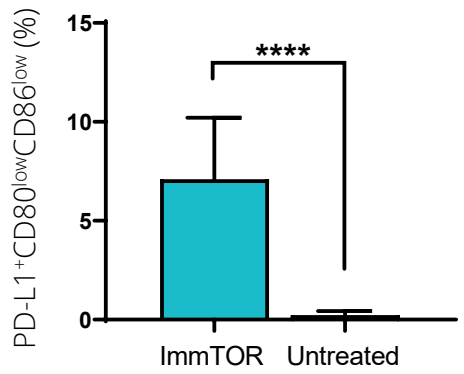
1. Ursodeoxycholic acid, 2. Obeticholic acid
3. Purohit & Cappell (2015) 4. Lu et al (2018); Lammers et al (2014); Marzioni et al (2019); 5. Floreani & Mangini (2018)

An ImmTOR-based approach to treating primary biliary cholangitis (PBC)

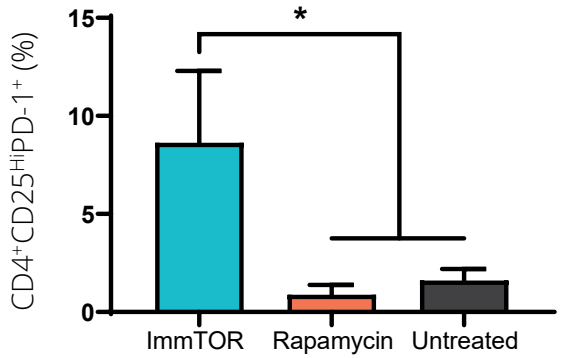
Selecta intends to co-administer ImmTOR with PDC-E2, the autoantigen implicated in PBC

We believe ImmTOR is ideally suited to address the root cause of PBC

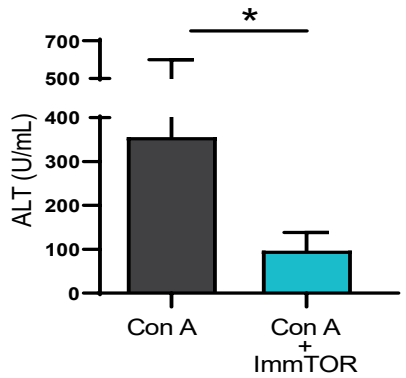
Tolerogenic liver sinusoidal endothelial cells



Hepatic regulatory T cells



Hepatoprotective in Con A liver inflammation model



* P=0.05, ****P=0.0001

PBC is a T-cell mediated disease driven by a well-defined antigen, ImmTOR designed to biodistribute to the liver and induce a tolerogenic environment, ImmTOR observed to exhibit hepatoprotective properties in liver injury models[1]

1. https://rarediseases.org/rare-diseases/primary-biliary-cholangitis/

Gene Therapy

AAV gene therapies are coming of age but still have challenges

Selecta has platform technologies to potentially address many key challenges facing the modality

THE
Challenges

- While most gene therapy trials use AAV vectors, the formation of neutralizing antibodies (NAbs) after AAV vector administration prevents redosing
  - Adverse patient events related to high vector doses is inextricably linked to immunogenicity*
  - **Pre-existing immunity to AAV vectors excludes significant numbers of patients**

THE
Solution

- **ImmTOR and Xork offer independent value creation opportunities with existing and new partners**
  - **ImmTOR** – Human proof of concept shows the possibility for ImmTOR to inhibit the formation of NAbs to AAV vectors. Extensive preclinical work shows the potential for **improved and more durable transgene expression upon the first dose** and potential hepatoprotective benefits
  - **Xork** – Cleaves human IgG specifically, efficiently and shows low cross reactivity to human sera potentially **opening a potential treatment window for those with pre-existing immunity to AAV vectors**

*Flotte TR. 2020. Hum Gene Ther 31:398-399.

Aiming to have the leading toolkit to power AAV gene therapies

"Gene therapy is a one time only treatment"

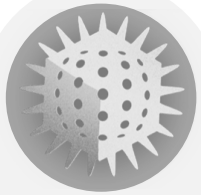
The ImmTOR platform has shown the ability to **mitigate the formation neutralizing antibodies (NAbs)** to empty capsids in humans

Preventing the formation of NAbs could enable redosing of gene therapies

"Patient eligibility is limited"

Xork can cleave IgG potentially opening a therapeutic window for gene therapy treatment and **enable redosing**

Increasing patients eligible for gene therapies can bring hope to those without treatment alternatives and make programs more commercially viable



"High doses are needed to ensure therapeutic benefit"

Low transduction efficiency and lack of organ specificity requires higher doses to ensure therapeutic benefit

Selecta has partnered with a leading synthetic biology company to engineer next generation capsids with improved transduction and organ specificity

ImmTOR could enable safer, more efficacious gene therapy treatments

ImmTOR is designed to be dose sparing – a key safety consideration and manufacturing benefit

Day 0

5e11 vg/kg AAV-SEAP

5e11 vg/kg AAV-SEAP + ImmTOR

25e11 vg/kg AAV-SEAP

Day 70

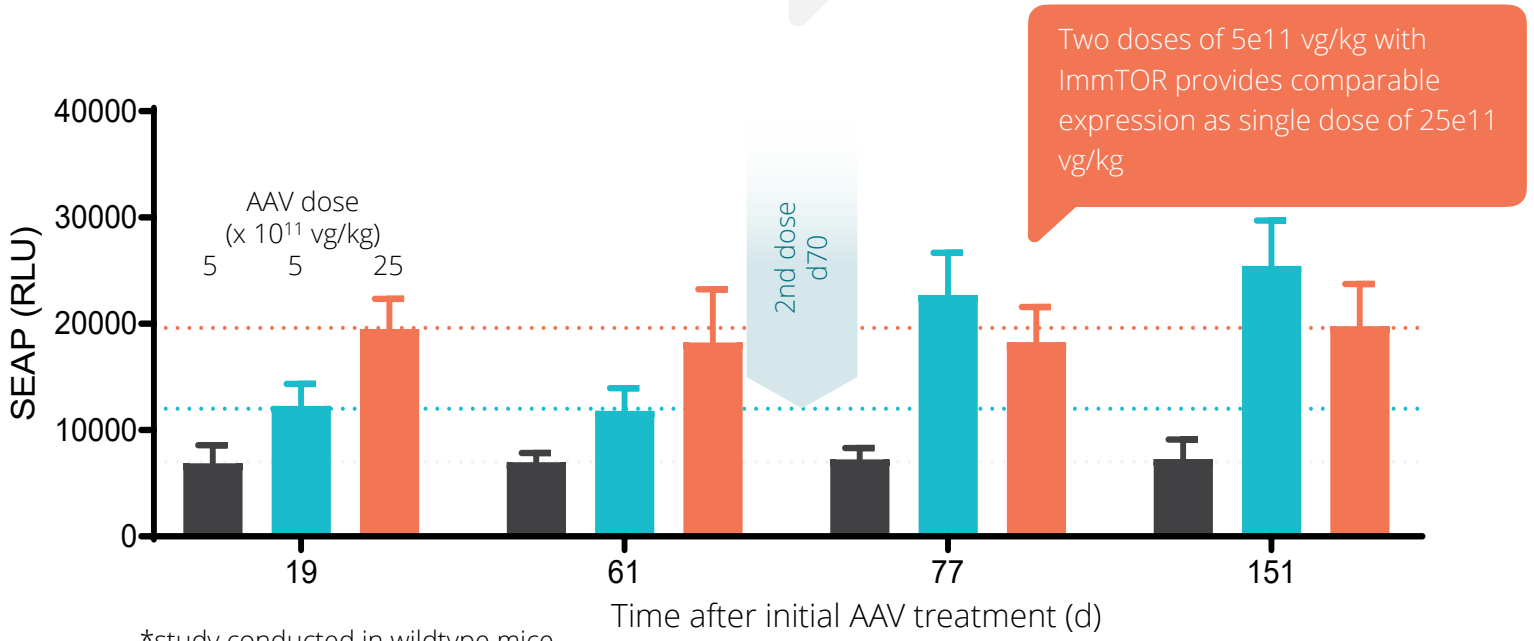
5e11 vg/kg AAV-SEAP

5e11 vg/kg AAAV-SEAP + ImmTOR

25e11 vg/kg AAV-SEAP

ImmTOR has been observed to **enhance transgene expression** after first and second doses of AAV

Repeat dosing enabled by ImmTOR is dose sparing



*study conducted in wildtype mice

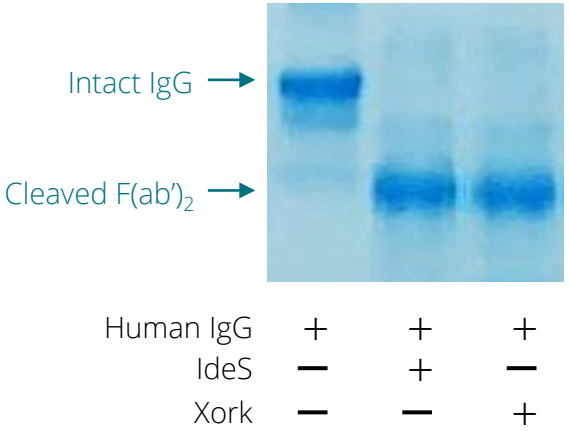
Ilyinskii et al., Science Advances, 2021

Aiming to simultaneously address two key challenges in AAV gene therapy

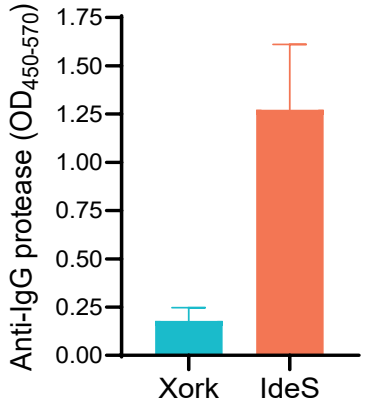
The combination of ImmTOR and Xork could make gene therapy both accessible and re-dosable

Xork is an IgG protease derived from a non-human pathogen

Cleavage of human IgG

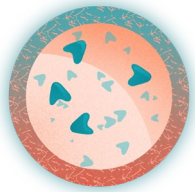


Pre-existing anti-IgG protease Antibodies in human serum



*IdeS is an IgG protease derived from the common human pathogen Streptococcus pyogenes

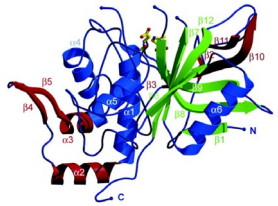
ImmTOR



- Mitigate NAb formation
- Address re-dosing issues
- Enhance expression

+

Xork

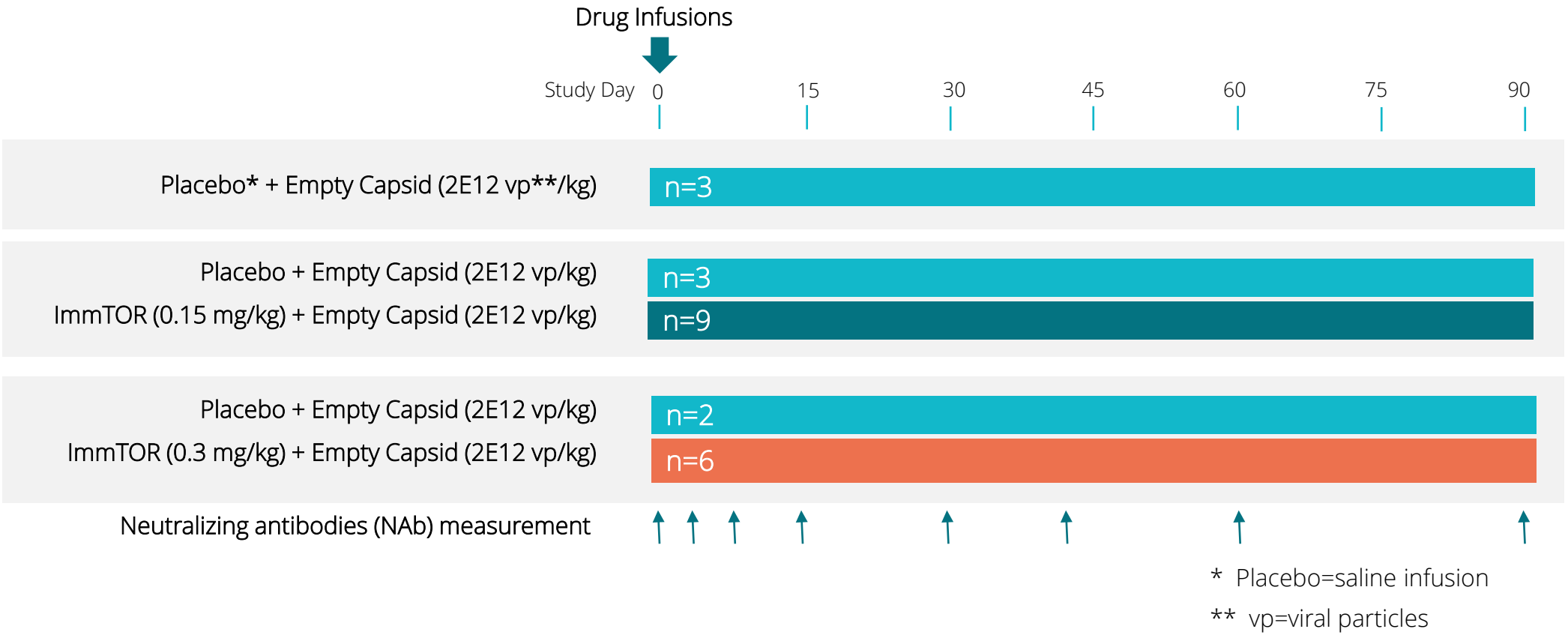


- Cleave pre-existing IgG

Potential to make gene therapy both accessible & re-dosable

SEL-399 (empty-AAV8 capsid) Phase 1 dose-escalation study

Total healthy volunteers enrolled: 23 (14 males and 9 females)
All subjects with anti-AAV8 NAb titers <1:5 at baseline
Randomized, placebo controlled and double-blind study

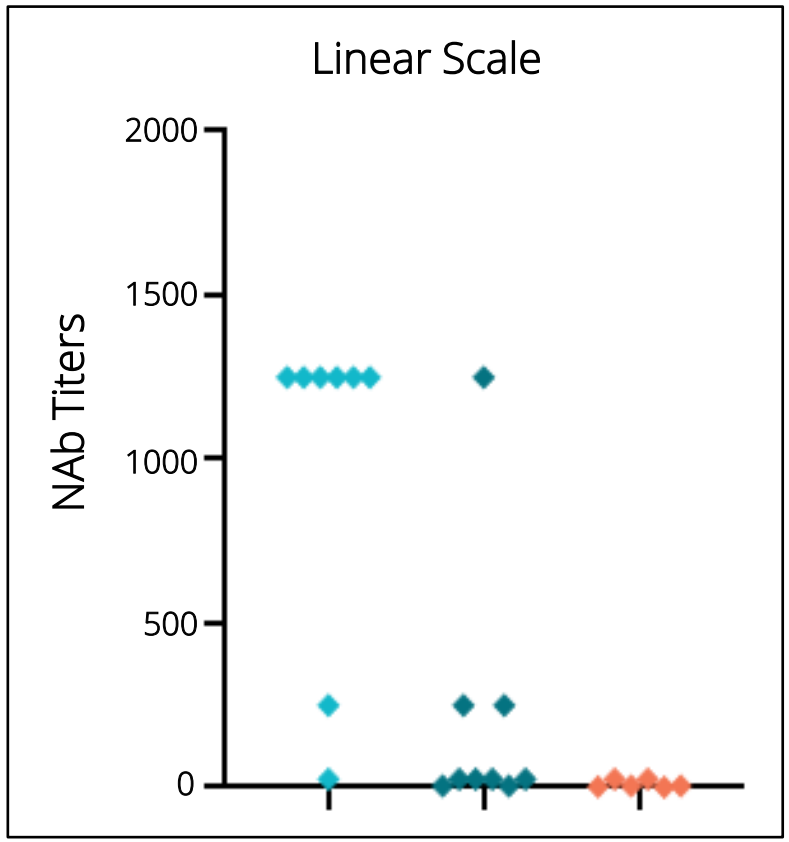


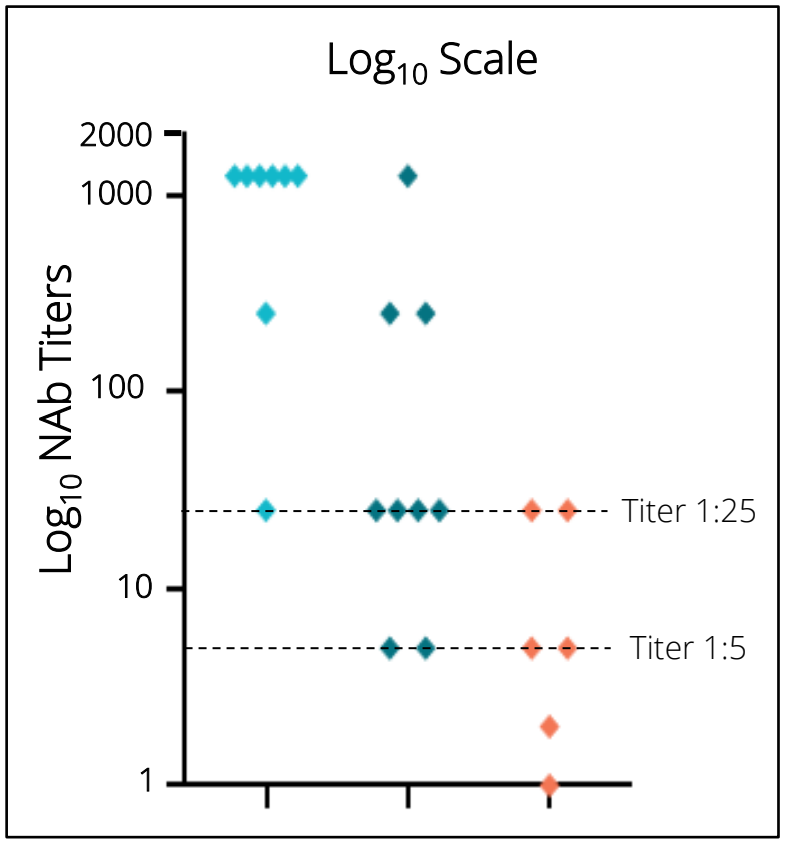
* Placebo=saline infusion

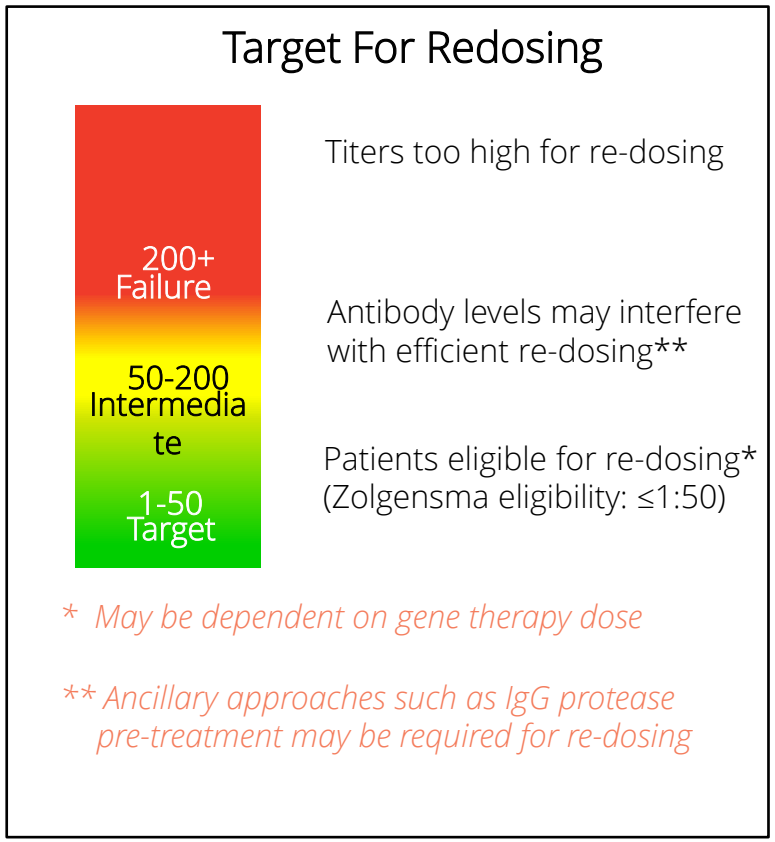
** vp=viral particles

Single dose ImmTOR observed to inhibit anti-AAV8 NAb formation at day 30

100% of subjects dosed with 0.3 mg/kg ImmTOR had NAb titers ≤1:25 at Day 30
67% of subjects dosed with 0.3 mg/kg ImmTOR had NAb titers ≤1:5 at Day 30







- Empty Capsid (n=8)
- Empty Capsid + 0.15 mg/kg ImmTOR (n=9)
- Empty Capsid + 0.3 mg/kg ImmTOR (n=6)

Summary and conclusions

- We observed AAV8 empty capsids **eliciting a strong immune response** with peak median anti-AAV8 NAb titers of 1:6875
- We observed **ImmTOR inhibiting the formation of anti-AAV8 NAb in a dose-dependent manner** at Day 30

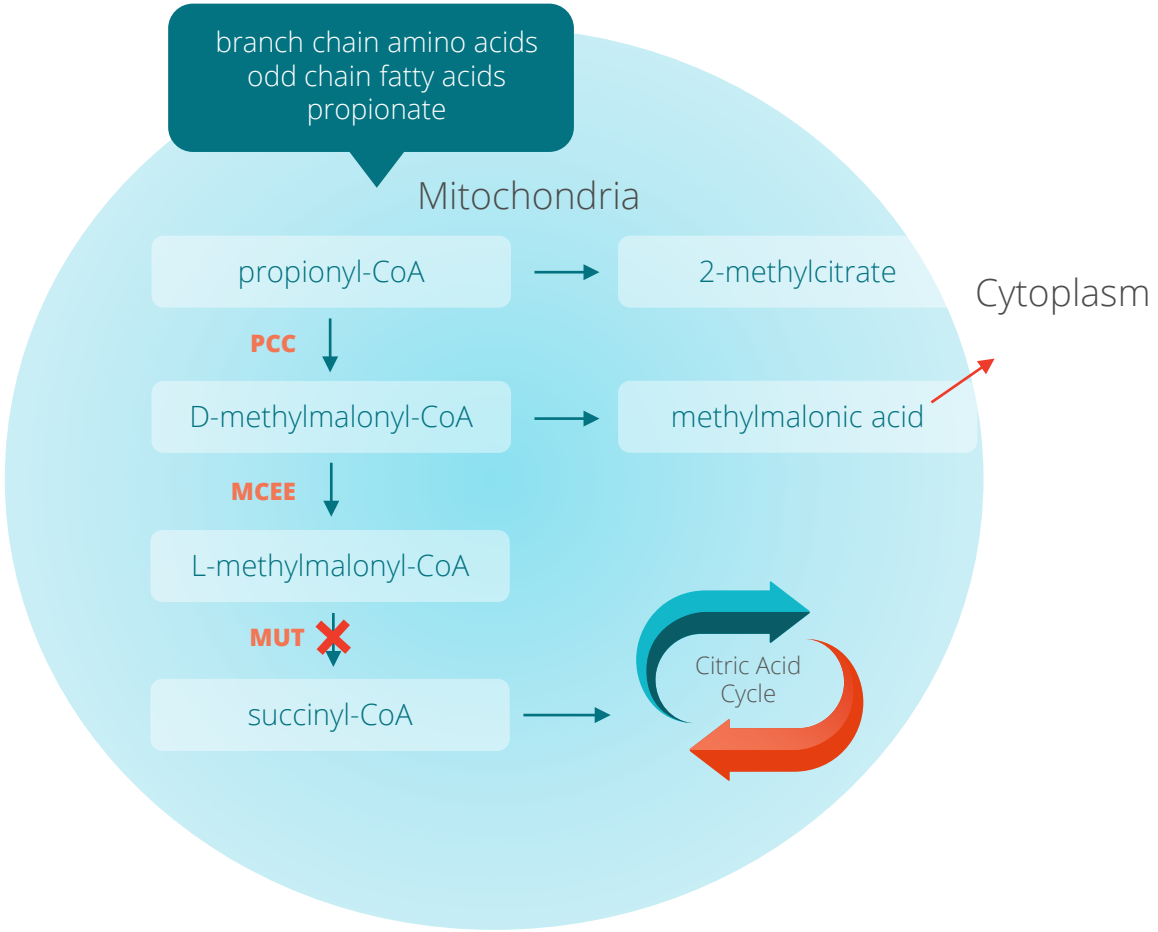
ImmTOR Dose	Subjects ≤ 1:5 NAb titer	Subjects ≤ 1:25 NAb titer	Median titers	Fold difference from control
0.15 mg/kg	22%	67%	1:25	50
0.30 mg/kg	**67%**	**100%**	**1:5**	**250**

- After Day 30, 2 of 6 subjects treated with 0.3 mg/kg ImmTOR maintained NAb titers ≤25, while remaining ImmTOR-treated subjects showed delayed formation of NAb reaching control levels by Day 90
- Animal studies suggest that if NAb are inhibited at Day 30, administration of two additional monthly doses of ImmTOR may maintain control of NAb beyond 90 days
- **Safety findings included AEs previously observed with ImmTOR** (Stomatitis & Rash). Asymptomatic and transient laboratory changes in subjects receiving ImmTOR were seen in 2 subjects with mild to moderate thrombocytopenia and 1 subject with grade 3 hypertriglyceridemia
- This promising study in healthy volunteers **provides support for the potential use of ImmTOR for the inhibition of NAbs to AAV8 in gene therapy clinical trials**

SEL-302 - Gene therapy program for the treatment of MMA

Phase 1 start expected in Q4 2022

- Methylmalonic acidemia (MMA) is a rare monogenic metabolic disease with a potential live birth incidence of between 1:25,000 and 1:48,000[1]
- Majority of patients have mutations in the mitochondrial methylmalonyl-CoA mutase (MUT) gene
- Metabolic instability, particularly in the liver, can cause hyperammonemia and production of other toxic metabolites
- Metabolic crisis can cause irreversible neurocognitive damage, stunted growth, chronic kidney disease and premature death
- Only effective treatment is liver transplantation at an early age
- **Selecta is developing an AAV gene therapy combined with ImmTOR for the treatment of MMA (SEL-302)**



1. https://www.genome.gov/Genetic-Disorders/MMA-Study-General-Information

MMA Clinical Trial Design: Schedule of Events for Individual Subjects

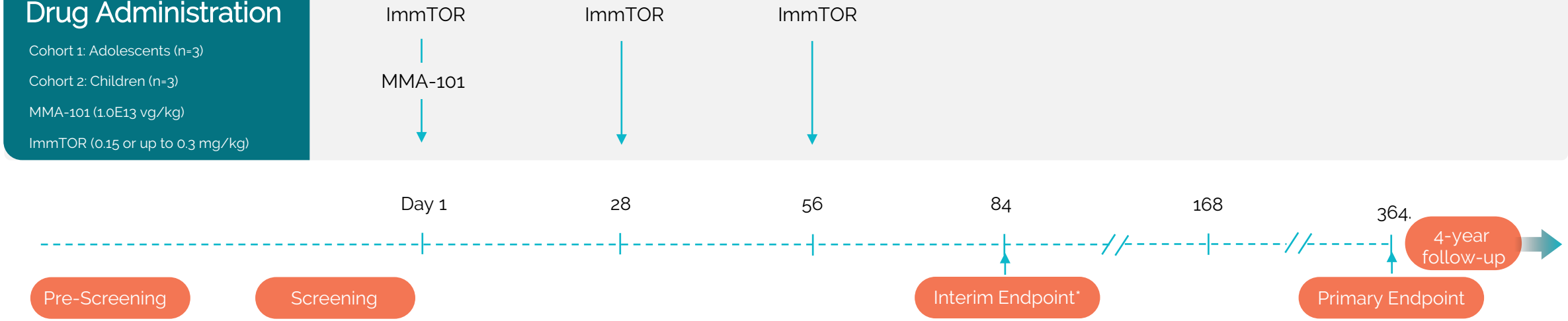
Drug Administration

Cohort 1: Adolescents (n=3)

Cohort 2: Children (n=3)

MMA-101 (1.0E13 vg/kg)

ImmTOR (0.15 or up to 0.3 mg/kg)



Key Biomarker Evaluations

POBT	Pre-dose, Day, 21, 42, 70, and 84
sMMA	Pre-dose, Day 7, 21, 42, 63, 70, 77, and 84
NAb	Pre-dose, Day 14, 28, 42, 56, 70, and 84

Clinical endpoints monitored throughout trial (hospitalizations, metabolic crises, growth/diet, patient- & caregiver-reported outcomes)

POBT=1-^{13}C -sodium propionate oxidative capacity using breath tesevaluationt; sMMA= serum methylmalonic acid levels; NAb=neutralizing anti-AAV8 antibodies
*Interim Endpoint= Data cutoff for Data Safety Monitoring Board

Biologics

ImmTOR designed to enhance biologic therapies

Unlocking their full potential by potentially ameliorating unwanted immune responses

THE **Challenges**

- Many **biologics can be highly immunogenic resulting in suboptimal responses to the standard of care** due to the development of anti-drug antibodies (ADAs) after multiple treatments
- Patients that develop an immune response to the current standard of care may be forced to discontinue treatment or experience adverse reactions

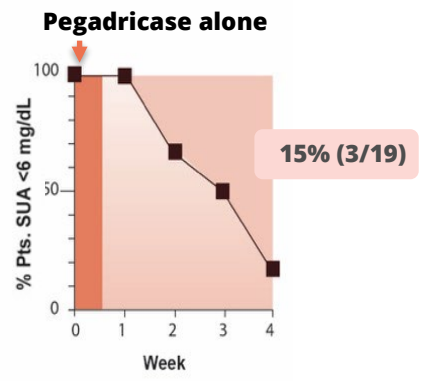
THE **Solution**

- ImmTOR, co-administered with immunogenic therapeutic enzymes, has the potential to ameliorate an immune response to the biologic treatment **allowing patients to stay on therapy longer**
- Extensive human data and significant safety database across multiple biologics demonstrates broad potential applicability and a promising approach **to minimize the healthcare and economic burden of ADAs**

Sands, E., Kivitz, A., DeHaan, W. et al. Tolerogenic nanoparticles mitigate the formation of anti-drug antibodies against pegylated uricase in patients with hyperuricemia. Nat Commun 13, 272 (2022). https://doi.org/10.1038/s41467-021-27945-7

SEL-212 is a late-stage enzyme therapy program in chronic refractory gout

ImmTOR markedly improved patient response to the enzyme pegadricase in a Phase 2 trial

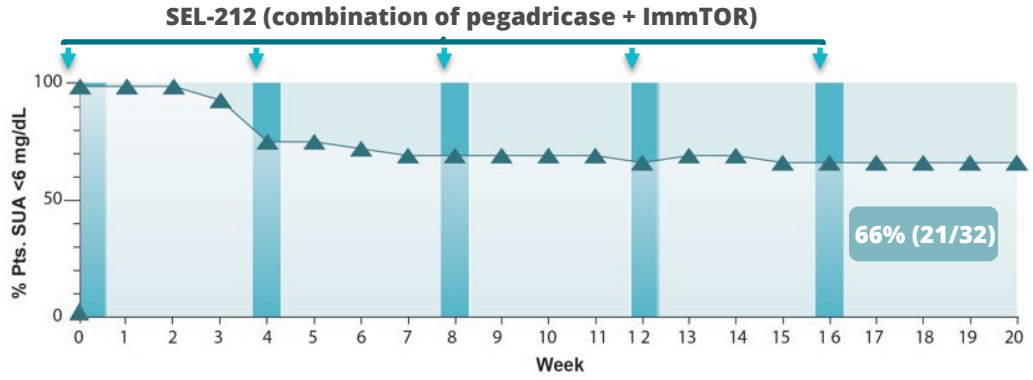


Pegadricase alone**

Pegadricase 0.2 or 0.4 mg/kg

Only 15% of patients treated with pegadricase alone maintain control of serum uric acid (SUA) after four weeks of therapy

Pegadricase is a highly immunogenic enzyme with most patients treated with pegadricase alone developing anti-drug antibodies within 2 weeks after a single treatment



5 monthly doses SEL-212*

Pegadricase 0.2 mg/kg

ImmTOR 0.1 or 0.15 mg/kg

ImmTOR was observed to ameliorate the immune response to pegadricase and was generally well-tolerated resulting in sustained control of serum uric acid (SUA)

* Data from 5 monthly dosing cohorts of the SEL-212/201 trial

**Data from pegadricase alone cohorts from the SEL-037/101, SEL-212/101, and SEL-212/201 trials

Patients most in need reaped greater benefits with SEL-212

Observed a delta of 19% points for SEL-212 versus pegloticase for patients with visible tophi at baseline

Patients with tophi at baseline:

- Represent the most severely affected population of gout patients
- Are less likely to achieve target SUA levels on conventional oral lowering therapies and have increased gout-related emergency room visits, hospitalizations, gout-related surgeries, and co-morbidities
- Have increased prevalence of swollen and tender joints and chronic kidney disease
- Have increased risk of mortality

Evaluation Period (Month)	Data Set	SEL-212		pegloticase		Treatment Difference[2]
		n[1]	Responder Percent	n[1]	Responder Percent	Percentage pts
Month 3 and 6 combined	PP	26	58%	26	39%	19
	ITT	35	57%	34	42%	16

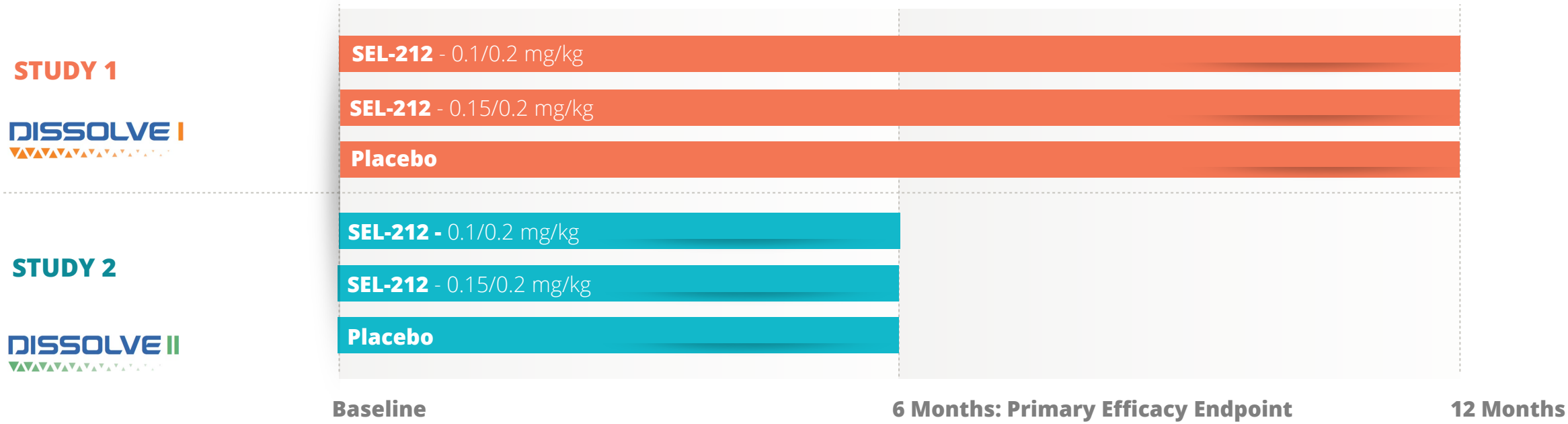
1. Number of patients with tophi with Responder Assessment
2. Treatment difference = SEL-212 percent responder - pegloticase percent responder. Rounded to nearest integer

Sources: Khanna et al Arthr Rheumatol 2016, 68, suppl 10; Edwards et al, Rheumatol 2019; Vincent et al J Rheumatol 2017; Perez-Ruiz, Ann Rheum Dis 2014

SEL-212 phase 3 DISSOLVE program design

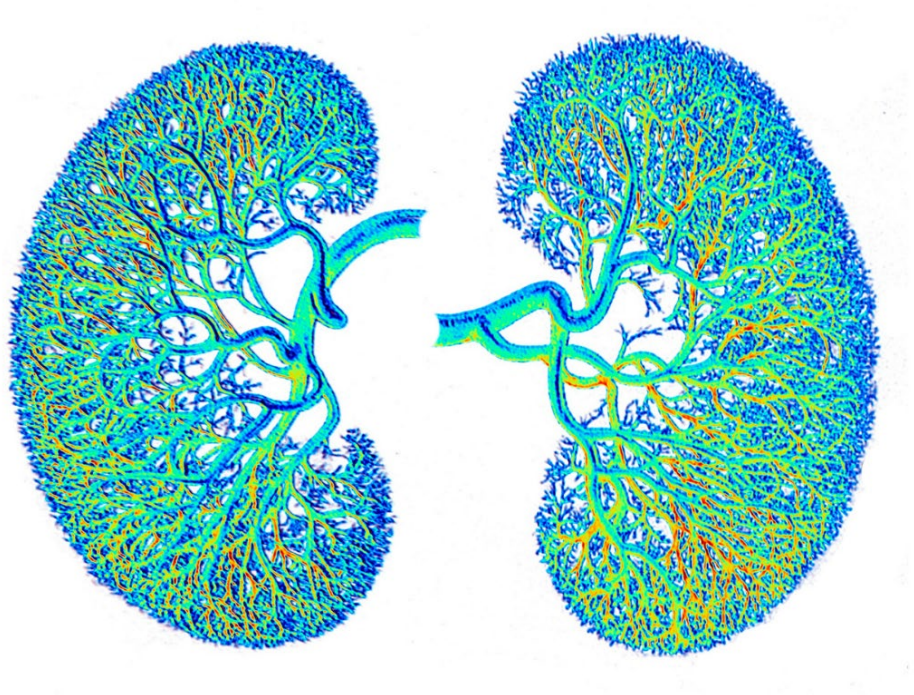
Evaluating SEL-212 in a pivotal phase 3 program vs. placebo, joint topline data expected in Q1 2023

- 2 double blinded placebo-controlled trials of SEL-212 (0.1 mg/kg & 0.15 mg/kg ImmTOR)
  - Both studies have a 6-month primary endpoint of serum uric acid (SUA) < 6 mg/dL at month 6, and DISSOLVE I has a 6-month safety extension; secondary endpoints include tender and swollen joint counts, tophus burden, patient reported outcomes of activity limitation and quality of life and gout flare incidence
- Randomized 1:1:1 against placebo with 265 treated subjects across both studies



Opportunity to address unmet medical needs for the treatment of IgAN

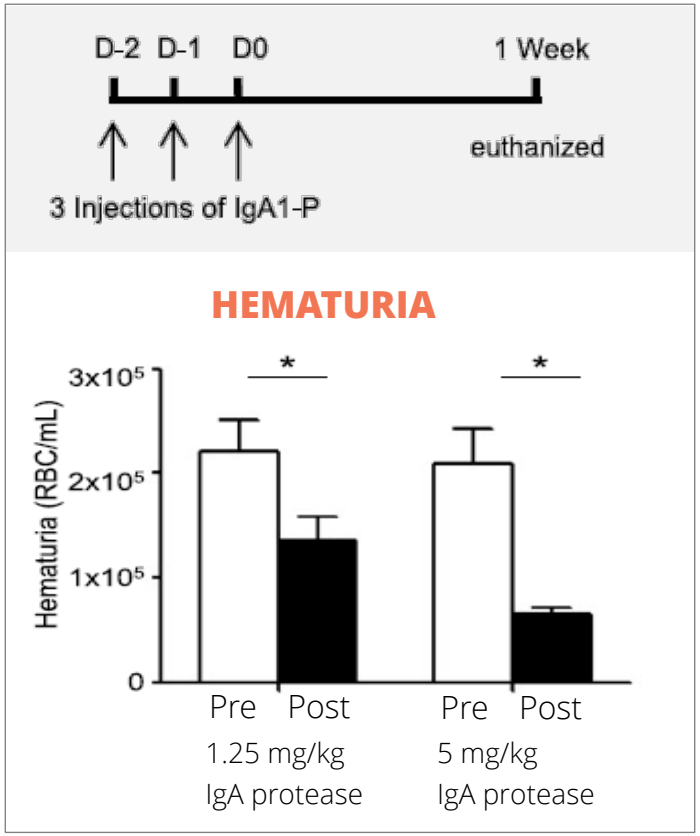
- Immunoglobulin A nephropathy (IgAN) is a leading cause of chronic kidney disease (CKD) and renal failure with 30-40% of patients reaching end-stage renal disease; approximately **100,000 patients in the U.S. and only one approved therapy**
- Caused by **deposits of the protein immunoglobulin A (IgA) inside the filters (glomeruli) in the kidney**
- **Current treatments fail to address the root cause of the disease** and are focused on protecting the kidney from further damage
- Selecta is **developing an entirely new class of IgA protease** from a commensal bacteria in collaboration with IGAN Biosciences for the treatment of IgAN
- Combining **ImmTOR with an IgA protease** has the potential to mitigate the formation of new ADAs and address the underlying pathophysiology of the disease



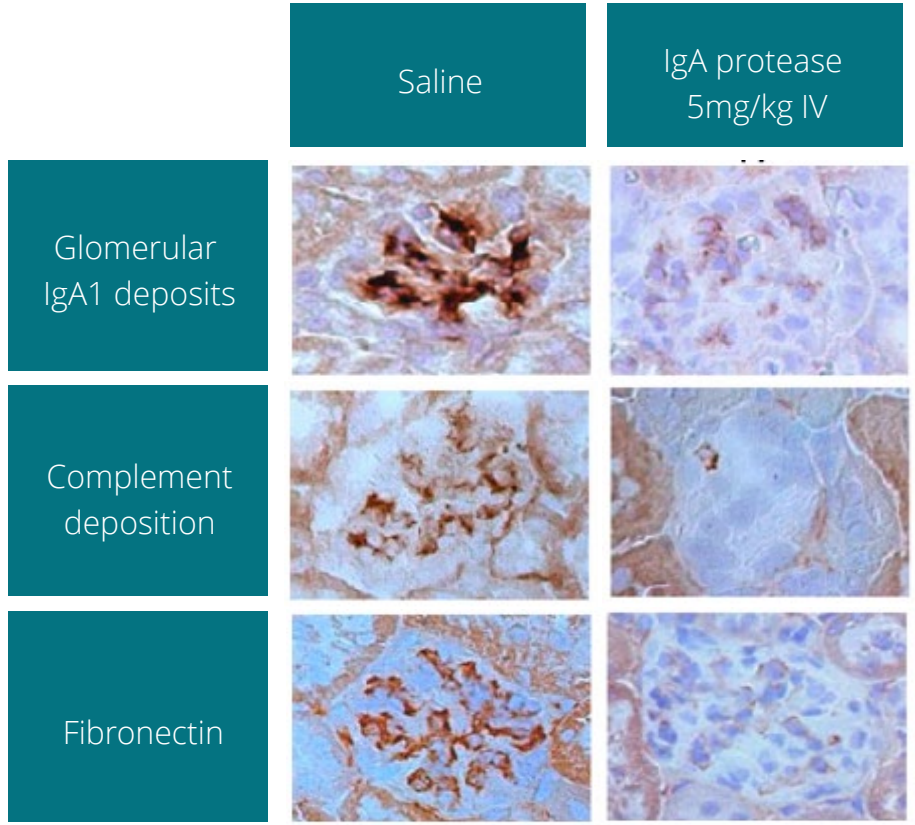
Combining ImmTOR with IgA protease for the treatment of IgAN

Building on the clinical data from the SEL-212 program and strong preclinical data in IgA

- **IND enabling studies underway for next generation Immunoglobulin A (IgA)** protease to address **IgA nephropathy**
- Mice expressing human IgA1 and sCD89 develop spontaneous IgA nephropathy
- Treatment with IgA protease **clears glomerular IgA1 deposits and associated inflammation and hematuria**



Adapted from Lechner et al., ***J Am Soc Nephrol***, 2016.



Corporate

Experienced management team positions Selecta for success

Carsten Brunn, Ph.D.
President and CEO

Blaine Davis
Chief Financial Officer

Lloyd Johnston, Ph.D.
Chief Operations Officer

Kei Kishimoto, Ph.D.
Chief Scientific Officer

Peter G. Traber, M.D.
Chief Medical Officer

Kristen Baldwin
Chief People Officer

Matthew Bartholomae
General Counsel

BAYER

PROTARA THERAPEUTICS

Alkermes

Momenta

Galectin Therapeutics

LIVEKINDLY

MINERVA NEUROSCIENCES

NOVARTIS

endo

Massachusetts Institute of Technology

MILLENNIUM

gsk

BAYER

senet

Lilly

Bristol Myers Squibb

Boehringer Ingelheim

Penn Medicine

Otsuka

Positioned for growth in 2023

~$136.2 MILLION

Cash on hand as of December 31, 2022[1]

Current funding expected to extend into mid 2024 and is expected to support anticipated development across pipeline programs including:

Key 2023 milestones:

- ✓ Licensed next-generation IgG protease candidate, Xork, for development with AT845, Astellas Gene Therapies' investigational product for the treatment of Pompe Disease
- ❑ Report top-line data from Phase 3 DISSOLVE I & II programs of SEL-212 in chronic refractory gout in Q1 2023
- ❑ Preliminary Phase 1 SEL-302 data in gene therapy for MMA
- ❑ Initiate IND enabling studies with the selected IL-2 candidate to further advance and expand the immune tolerance platform in autoimmune disease
- ❑ Initiate IND enabling studies with the selected IgA protease candidate from IGAN Biosciences

1. Includes cash, cash equivalents, marketable securities and restricted cash.

Pioneering Precision Immune Tolerance

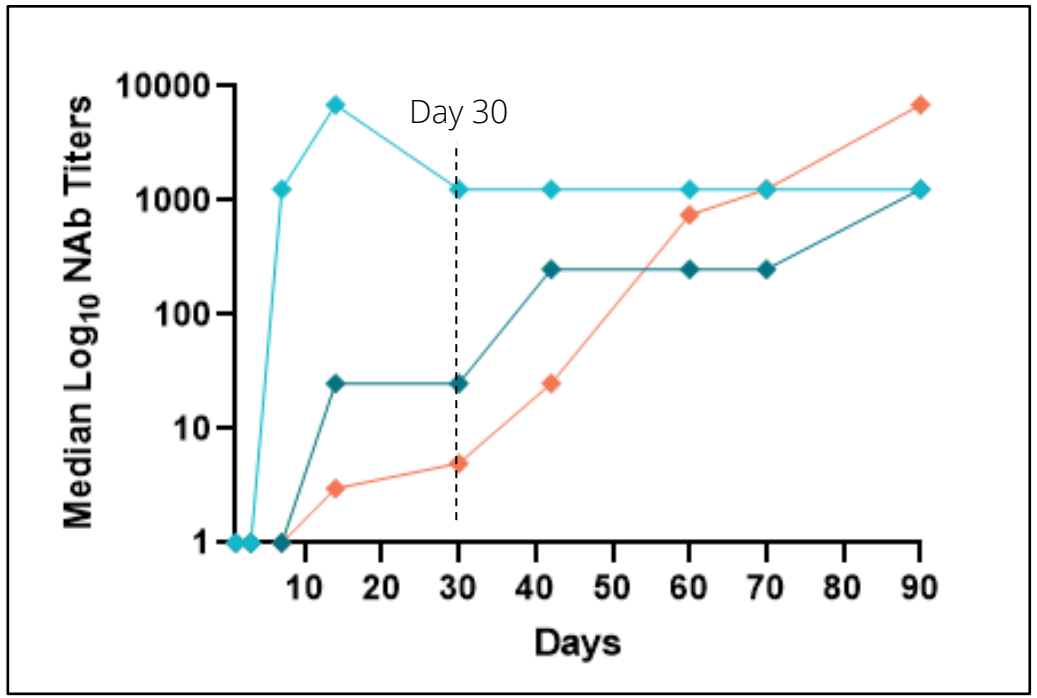
1. **ImmTOR® and ImmTOR-IL™ immune tolerance platforms** have potentially broad applicability to address the challenges of **autoimmunity and immunogenicity**
2. **Diversified pipeline** of novel therapeutic candidates; proof of concept in biologics and gene therapy, **expanding into autoimmune disease**
3. **Topline Phase 3 data for SEL-212** in chronic refractory gout expected in **Q1 2023**, with additional value-generating milestones anticipated in 2023
4. **Multiple strategic partnerships designed to** validate platform and maximize its potential
5. Strong balance sheet, positioned to reach multiple value inflection points with expected **runway into mid-2024**

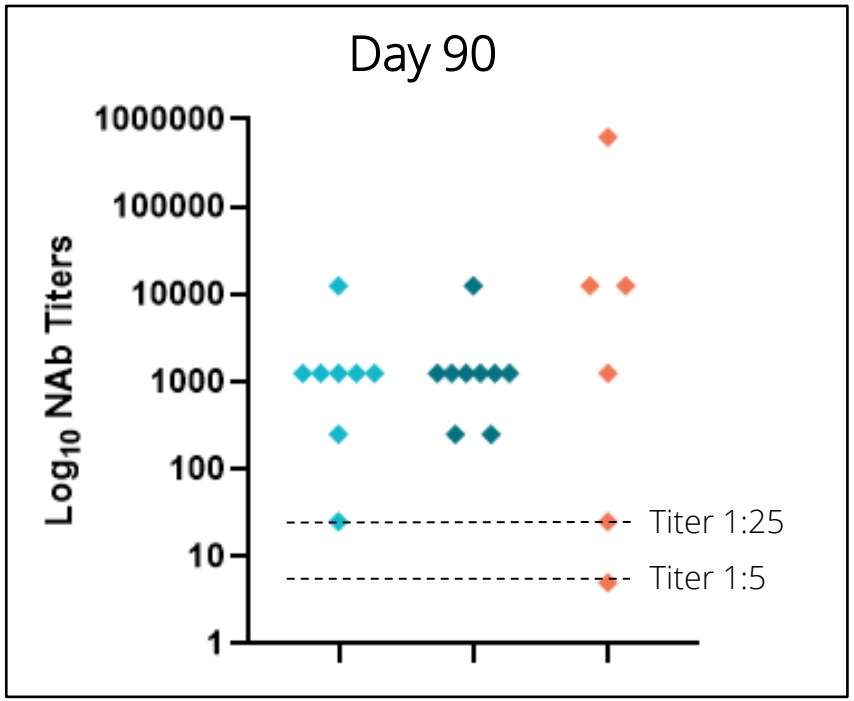
Appendix

Subjects treated with a single dose of ImmTOR developed delayed NAb formation by day 90

Additional doses of ImmTOR may be required to maintain control beyond Day 30

2 of 6 subjects dosed with 0.3 mg/kg ImmTOR had NAb titers ≤1:25 at Day 90
1 of 6 subjects dosed with 0.3 mg/kg ImmTOR had NAb titers ≤1:5 at Day 90



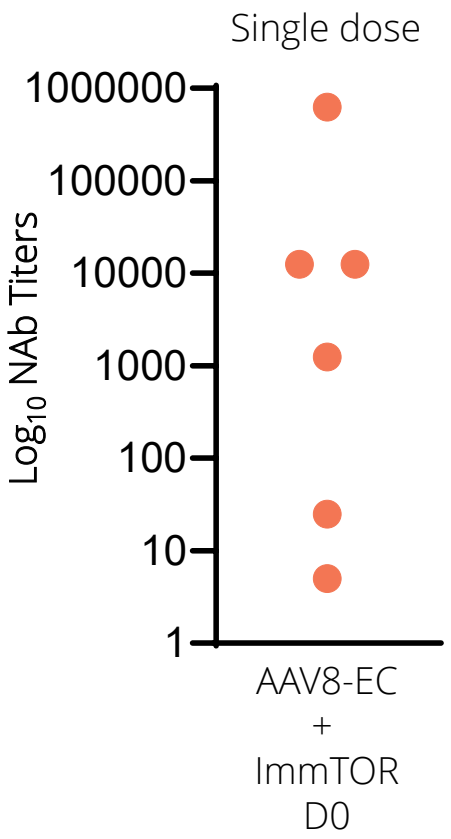


- Empty Capsid (n=8)
- Empty Capsid + 0.15 mg/kg ImmTOR (n=9)
- Empty Capsid + 0.3 mg/kg ImmTOR (n=6)

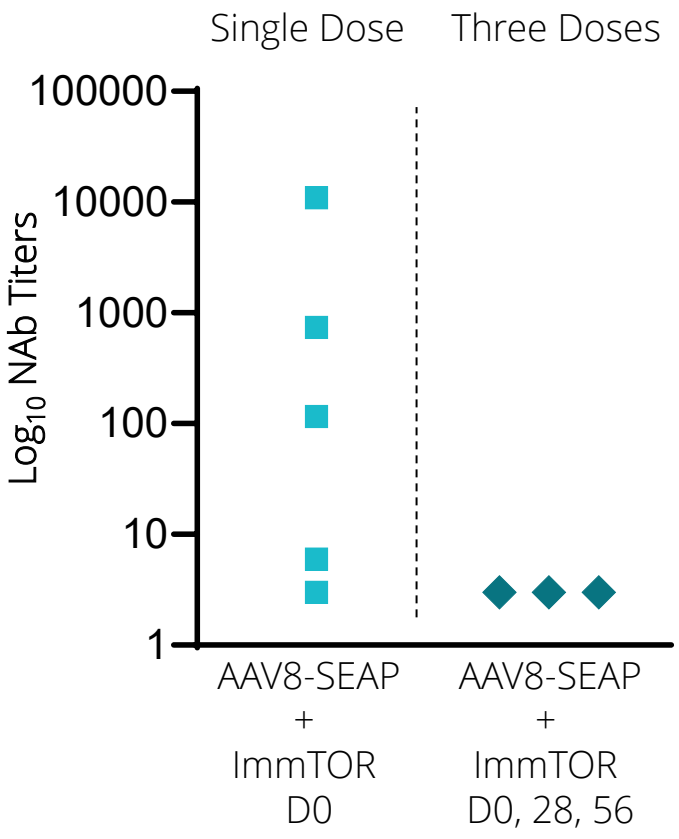
Empty capsid data in-line with single dose ImmTOR NHP data at day 90

Three monthly doses of ImmTOR provide inhibition of NAbs in NHP

Human



Nonhuman Primate[1]



Target For Redosing

Titers too high for re-dosing

Antibody levels may interfere with efficient re-dosing**

Patients eligible for re-dosing*
(Zolgensma eligibility: ≤1:50)

** May be dependent on gene therapy dose*

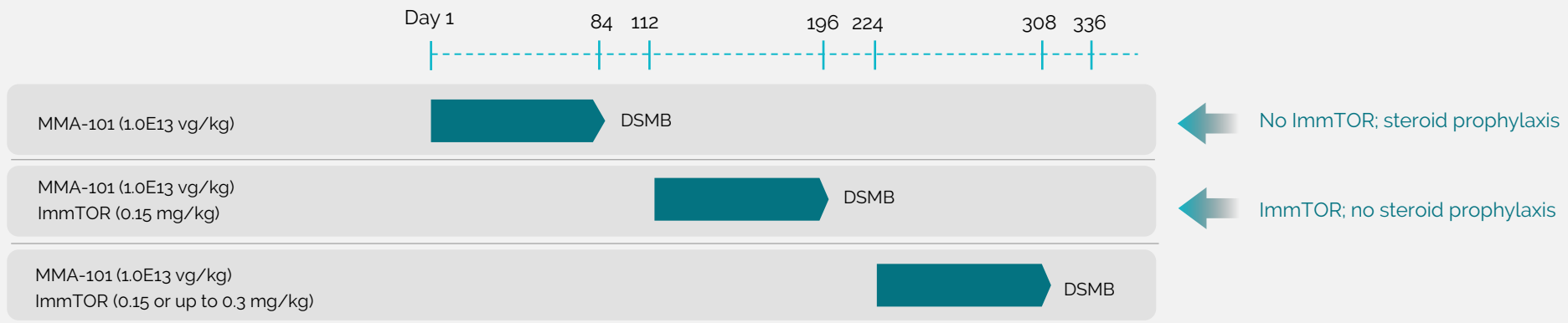
*** Ancillary approaches such as IgG protease pre-treatment may be required for re-dosing*

1. ESGCT 2021 Poster 003

MMA Clinical Trial Design: Schedule of Events

Cohort 1

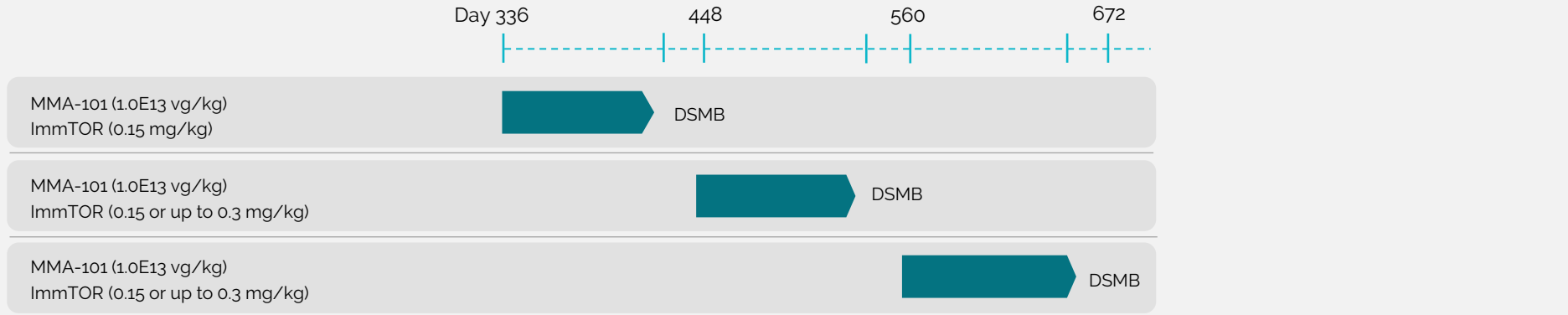
Cohort 1:
Adolescents (n=3)



Cohort 2

Children (n=3)

(ages ≥ 2 and <12)



Assumes 1 month (28 days) between Day 84 cutoff and subsequent participant enrollment to allow for DSMB report generation and review.