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 anticipated timing or outcome of selection of development candidates, 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, the anticipated timing for receipt of payments owed to the Company, and the Company's ability to grow its strategic partnerships, enrollment in the Company's clinical trials and the Company's plans with respect to areas affected by geopolitical conflict 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
Company Highlights

**ImmTOR™ platform has potentially broad applicability**

- Precision immune tolerance platform has potential to restore self tolerance in autoimmune disease and overcome immunogenicity of gene therapies and biologics
- Preclinical data indicates potentially profound synergy of ImmTOR and engineered Treg-selective IL-2 to expand antigen-specific Tregs and improve durability of immune tolerance (ImmTOR-IL)

**Proof of concept in biologics and gene therapy**

- SEL-212 in chronic refractory gout potentially serves as proof of concept for the ImmTOR platform in biologics with over 400 patients dosed – Phase 3 DISSOLVE I & II topline read out expected in Q1 2023
- Empty AAV capsid study data in healthy volunteers showed the potential ability of ImmTOR to inhibit the formation of neutralizing antibodies to AAV capsids

**Diversified pipeline expanding to autoimmune disease**

- SEL-302: Gene therapy program in methylmalonic acidemia (MMA), anticipated Phase 1 trial start in Q4 2022
- SEL-018: Plans to advance Xork IgG Protease to mitigate pre-existing anti-AAV antibodies through IND-enabling studies
- IgA nephropathy: clinical candidate selection & IND enabling studies in process
- Plans to advance the ImmTOR platform with IL-2 (ImmTOR-IL) for use in autoimmune disease
- Expected financial runway into mid 2024

**Targeted partnerships to maximize platform potential**

- Selecta Biosciences Investor Presentation – November 2022
- Sobi (Tec strengthens)
- GENOVIS
- AskBio
- Takeda
- SAREPTA (THERAPEUTICS)
- GINKGO BIOWORKS
- CYRUS BIOTECHNOLOGY
ImmTOR Platform

Precision Immune Tolerance
A precision immune tolerance platform with potentially broad applicability

ImmTOR combines nanoparticle technology with an FDA approved anti-inflammatory and immunomodulatory drug, and is designed to generate antigen-specific immune tolerance when combined with an antigen of interest.
ImmTOR could potentially be applied to restore self tolerance in autoimmune disease and overcome immunogenicity of gene therapies and biologics.
ImmTOR-IL: ImmTOR plus IL-2 receptor agonist

Evolution of the ImmTOR Platform

Synergistic mechanism of ImmTOR and a Treg-selective IL-2:

- 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, with applications across biologic therapies and autoimmune disease indications
Aiming to restore self tolerance to auto antigens and power biologics

**Tolerogenic Therapies**
ImmTOR could provide targeted immune tolerance to auto antigens

**Gene Therapies**
ImmTOR potentially enables redosing of transformative gene therapies

**Biologic Therapies**
ImmTOR is designed to address the immunogenicity of biologics

Autoimmune disease affects more than 24M people in the US alone⁶

80% of rare disease has a known monogenic cause⁵ and most gene therapy trials use AAV vectors

Over 160,000 patients between IgAN and chronic refractory gout in the US alone¹,²,³,⁴

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2. Arthritis & Rheumatology Vol. 71, No. 6, June 2019 pp 991-999
# A diversified and growing wholly-owned pipeline

<table>
<thead>
<tr>
<th>Indication</th>
<th>Antigen</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Recent and Expected Upcoming Milestones</th>
<th>Commercial Rights</th>
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<td>SEL-018</td>
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<td>IgA nephropathy (IGAN)</td>
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<td>Candidate Selection 2022</td>
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*Licensed Agreement with Ginkgo Bioworks & Option & License Agreement with IGAN Biosciences
Unlocking the potential of our platform through collaborations
Selecta has entered strategic transactions to further optimize the potential of the ImmTOR platform

<table>
<thead>
<tr>
<th>Collaboration</th>
<th>Year</th>
<th>ImmTOR Approach</th>
<th>Agreement</th>
<th>Indications</th>
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<td>License Agreement (Global)</td>
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<td>Lysosomal storage disorders</td>
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<th>Indication</th>
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<td>Chronic Refractory Gout</td>
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</table>
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 current standard of care for autoimmune diseases 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.

Our approach to autoimmune disease is designed to restore natural self-tolerance by administering ImmTOR with nanoparticle-encapsulated self-antigens thus avoiding the need for chronic and systemic immune suppression.

By developing a proprietary Treg-selective IL-2 to combine with ImmTOR and autoantigens we are advancing our precision immune tolerance platform with the aim of expanding antigen-specific Tregs and enhancing durability of tolerance.

There are roughly 80 autoimmune conditions that affect as much as 4.5% of the world's population*. 24M+ individuals in the US alone are affected by autoimmune diseases**

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*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
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 observed, Selecta potentially has a best-in-class IL-2 therapy.

Additionally, with an approximately 3-fold increase in antigen-specific Tregs, Selecta believes this data shows the 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
Superior anti-AAV antibody inhibition observed when IL-2 is combined with ImmTOR

Clear dose sparing effect seen when IL-2 mutein is combined with ImmTOR*

*study conducted in wildtype mice
Immunogenicity of high vector dose AAV gene therapy mitigated by ImmTOR-IL

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

200 µg ImmTOR
9 µg IL-2 mutein

AAV8-SEAP +/-ImmTOR
+/-IL-2 mutein

Day 0
Day 28
Day 56
Day 84

5E13 vg/kg AAV8-SEAP
5E13 vg/kg AAV8-SEAP + ImmTOR, d0
5E13 vg/kg AAV8-SEAP + IL-2 mutein, d0, 28, 56, 84
5E13 vg/kg AAV8-SEAP + ImmTOR, d0 + IL-2 mutein, d0, 28, 56, 84

N=6
N=6
N=6
N=6

Anti-AAV8 IgG (OD450-570)

Days AAV post-Infusion

Days AAV post-Infusion

Days AAV post-Infusion

Days AAV post-Infusion

200 µg ImmTOR
9 µg IL-2 mutein

Day 0
Day 28
Day 56
Day 84

5E13 vg/kg AAV8-SEAP
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N=6
N=6
N=6
N=6

Anti-AAV8 IgG (OD450-570)

Days AAV post-Infusion

Days AAV post-Infusion

Days AAV post-Infusion

Days AAV post-Infusion
Initial autoimmune disease focus: Primary Biliary Cholangitis (PBC)

We believe ImmTOR-IL + PDC-E2 antigen has the potential to restore immune tolerance in the liver

- PBC is a rare T-cell mediated autoimmune liver disease
  - Leads to bile duct damage, progressive inflammation, scarring (cirrhosis) and eventually, liver failure

- It is driven by a well-defined pathogenic antigen: PDC-E2
  - 95% of patients with PBC have auto-antibodies against PDC-E2, the E2 subunit of mitochondrial pyruvate dehydrogenase complex

- Current therapies do not address underlying disease or key symptoms
  - 30 - 40% of patients are intolerant / unresponsive to current SoC (UDCA), and OCA is marred by high AE rates and black box warnings

- Our approach has the potential to directly address the underlying disease
  - In preclinical studies ImmTOR induced a strong tolerogenic environment and showed hepatoprotective properties in liver injury models
  - Co-administration of ImmTOR-IL with PDC-E2 has the potential to restore immune tolerance in the liver

There is a significant need for new therapies in PBC

- ~130,000
- ~80%
- ~30 – 40%

Prevalence of PBC in US 90 – 95% women

Receive pharmacological treatment

Intolerant / unresponsive to UDCA

An ImmTOR-based approach to treating primary biliary cholangitis (PBC)
Selecta intends to co-administer ImmTOR-IL with PDC-E2, the autoantigen implicated in PBC

- Patients with PBC need a highly-targeted, liver-directed approach to treating the root cause of the disorder
- ImmTOR biodistributes to the liver and is designed to induce a tolerogenic environment and shows hepatoprotective properties in liver injury models

We believe ImmTOR is ideally suited to address PBC

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**

The formation of neutralizing antibodies (NAbs) after AAV vector administration prevents redosing due to the potentially dangerous immune response that would follow a second or third gene therapy administration. Adverse patient events related to high vector doses is inextricably linked to immunogenicity.*

Pre-existing immunity to AAV vectors excludes significant numbers of patients who would potentially benefit from treatment by AAV gene therapies.

**THE Solution**

**ImmTOR** – Human proof of concept shows the possibility for ImmTOR to inhibit the formation of neutralizing antibodies to AAV vectors. Extensive preclinical work shows the potential for improved and more durable transgene expression upon the first dose and potential hepatoprotective benefits of ImmTOR.

**Xork** – Cleaves human IgG specifically, efficiently and shows low cross reactivity to human sera potentially opening a treatment window for those with pre-existing immunity to AAV vectors.

**THE Opportunity**

ImmTOR, by inhibiting the formation of neutralizing antibodies, could make redosing of gene therapies possible. Functional benefit could be maintained or restored with additional doses. Safer and more efficacious dosing regimens could be implemented.

Xork could potentially make patients with pre-existing immunity to AAV vectors eligible for treatment.

Selecta has partnered its technologies with leading gene therapy companies.

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 of Nabs to empty capsids in humans

ImmTOR

"Patient eligibility is limited"

Xork can cleave IgG potentially opening a therapeutic window for gene therapy treatment

Xork

"High doses are needed to ensure therapeutic benefit"

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

Next Gen Capsids

Preventing the formation of neutralizing antibodies could enable redosing of gene therapies

Preventing the formation of neutralizing antibodies could enable redosing of gene therapies

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

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

Preventing the formation of neutralizing antibodies could enable redosing of gene therapies
Potential for ImmTOR to enhance AAV gene therapies
Safer, more durable AAV gene therapy treatments are within reach

- Prevention of AAV antibody production
- Potential to re-dose
- Inhibition of liver inflammation
- More durable transgene expression
- Enhanced AAV transduction
- Stabilization of hepatic stress
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

- **ImmTOR**

- **Xork**

- Potential to increase the number of patients eligible for gene therapy by mitigating pre-existing anti-AAV antibodies

- Potential to enable re-dosing by mitigating the de novo formation of anti-AAV antibodies

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

- Xork cleaves human IgG specifically and efficiently, but shows low cross reactivity to human sera compared to IdeS

*IdeS is an IgG protease derived from the common human pathogen Streptococcus pyogenes*
ImmTorch could enable safer, more efficacious gene therapy treatments

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

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

Repeat dosing enabled by ImmTorch is dose sparing

**Day 0**
- 5e11 vg/kg AAV-SEAP
- 5e11 vg/kg AAV-SEAP + ImmTorch
- 25e11 vg/kg AAV-SEAP

**Day 70**
- 5e11 vg/kg AAV-SEAP
- 5e11 vg/kg AAV-SEAP + ImmTorch
- 25e11 vg/kg AAV-SEAP

Two doses of 5e11 vg/kg with ImmTorch provides comparable expression as single dose of 25e11 vg/kg

*study conducted in wildtype mice

Ilyinskii et al., Science Advances, 2021
**SEL-399 Phase 1 dose-escalation study: subjects and design**

- 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

### Drug Infusions

<table>
<thead>
<tr>
<th>Study Day</th>
<th>Placebo* + Empty Capsid (2E12 vp**/kg)</th>
<th>Placebo + Empty Capsid (2E12 vp/kg)</th>
<th>ImmTOR (0.15 mg/kg) + Empty Capsid (2E12 vp/kg)</th>
<th>Placebo + Empty Capsid (2E12 vp/kg)</th>
<th>ImmTOR (0.3 mg/kg) + Empty Capsid (2E12 vp/kg)</th>
<th>Neutralizing antibodies (NAb) measurement</th>
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<tr>
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<td>n=3</td>
<td>n=3</td>
<td>n=9</td>
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* 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)
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

![Graphs showing NAb titers over time for different treatment groups]

- 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

AAV8-EC + ImmTOR D0

Human

AAV8-SEAP

+ ImmTOR D0

Target For Redosing

AAV8-SEAP

+ ImmTOR D0, 28, 56

Single dose

Single Dose

Three Doses

Log_{10} NAb Titers

Log_{10} NAb Titers

1000000

100000

10000

1000

100

10

1

AAV8-EC + ImmTOR D0

AAV8-SEAP + ImmTOR D0

AAV8-SEAP + ImmTOR D0, 28, 56

Titers too high for re-dosing

200+ Failure

50-200 Intermediate

1-50 Target

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
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

<table>
<thead>
<tr>
<th>ImmTOR Dose</th>
<th>Subjects ≤ 1:5 NAb titer</th>
<th>Subjects ≤ 1:25 NAb titer</th>
<th>Median titers</th>
<th>Fold difference from control</th>
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<tbody>
<tr>
<td>0.15 mg/kg</td>
<td>22%</td>
<td>67%</td>
<td>1:25</td>
<td>50</td>
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<tr>
<td>0.30 mg/kg</td>
<td>67%</td>
<td>100%</td>
<td>1:5</td>
<td>250</td>
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- 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 neutralizing antibodies 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.

• 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
### Key Biomarker Evaluations

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>POBT</td>
<td>Pre-dose, Day 21, 42, 70, and 84</td>
</tr>
<tr>
<td>sMMA</td>
<td>Pre-dose, Day 7, 21, 42, 63, 70, 77, and 84</td>
</tr>
<tr>
<td>NAb</td>
<td>Pre-dose, Day 14, 28, 42, 56, 70, and 84</td>
</tr>
</tbody>
</table>

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

POBT=1-13C sodium propionate oxidative capacity using breath test; sMMA= serum methylmalonic acid levels; NAb=neutralizing anti-AAV8 antibodies

*Interim Endpoint* = Data cutoff for Data Safety Monitoring Board evaluation

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**MMA Clinical Trial Design: Schedule of Events for Individual Subjects**

**Drug Administration**

- **MMA-101**
  - ImmTOR
  - Day 1, 28, 56, 84, 168, 364

**MMA Clinical Trial Design: Schedule of Events for Individual Subjects**

- **Pre-Screening**
- **Screening**
- **Interim Endpoint**
- **Primary Endpoint**

**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=1-13C sodium propionate oxidative capacity using breath test; sMMA= serum methylmalonic acid levels; NAb=neutralizing anti-AAV8 antibodies

*Interim Endpoint* = Data cutoff for Data Safety Monitoring Board evaluation
MMA Clinical Trial Design: Schedule of Events

### Cohort 1
Adolescents (n=3)
(ages ≥ 12 and < 18)

- **Day 1**: MMA-101 (1.0E13 vg/kg)
- **Day 84**: MMA-101 (1.0E13 vg/kg)
  - No ImmTOR; steroid prophylaxis
- **Day 196**: ImmTOR (0.15 mg/kg)
  - DSMB
- **Day 308**: MMA-101 (1.0E13 vg/kg)
  - ImmTOR (0.15 or up to 0.3 mg/kg)
  - DSMB

### Cohort 2
Children (n=3)
(ages ≥ 2 and < 12)

- **Day 336**: MMA-101 (1.0E13 vg/kg)
- **Day 448**: MMA-101 (1.0E13 vg/kg)
  - ImmTOR (0.15 mg/kg)
  - DSMB
- **Day 560**: MMA-101 (1.0E13 vg/kg)
  - ImmTOR (0.15 or up to 0.3 mg/kg)
  - DSMB
- **Day 672**: MMA-101 (1.0E13 vg/kg)
  - ImmTOR (0.15 or up to 0.3 mg/kg)
  - DSMB

Assumes 1 month (28 days) between Day 84 cutoff and subsequent participant enrollment to allow for DSMB report generation and review.
Biologics
Biologic therapies potentially enhanced by ImmTOR
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.

Human data in both immunogenic enzymes and gene therapy AAV empty capsids shows the promise of ImmTOR in enhancing biologics.

**THE Opportunity**

The use of ImmTOR as an adjunct to biologic therapies offers a promising approach to minimize the healthcare and economic burden of ADAs.

Extensive human data and significant safety data base across multiple biologics demonstrates the broad potential applicability of the technology in immunogenic biologics.

Pegadricase is highly immunogenic when given alone

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

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

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.

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

5 monthly doses SEL-212*

Pegadricase 0.2 mg/kg
ImmTOR 0.1 or 0.15 mg/kg

* 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 from our therapy
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

<table>
<thead>
<tr>
<th>Evaluation Period (Month)</th>
<th>Data Set</th>
<th>SEL-212</th>
<th>pegloticase</th>
<th>Treatment Difference$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n$^1$</td>
<td>Responder Percent</td>
<td>n$^1$</td>
</tr>
<tr>
<td>Month 3 and 6 combined</td>
<td>PP</td>
<td>26</td>
<td>58%</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>ITT</td>
<td>35</td>
<td>57%</td>
<td>34</td>
</tr>
</tbody>
</table>

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

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
- DISSOLVE I fully enrolled as of Q4 2021. Study completion anticipated Q4 2022
- DISSOLVE II fully enrolled as of Q2 2022. Study completion anticipated Q4 2022
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** which may lead to presence of blood (hematuria) and protein (proteinuria) in urine and progressive renal insufficiency/failure

- **Current treatments fail to address the root cause of the disease** and are focused on protecting the kidney from further damage by reducing IgA1 production, controlling blood pressure, cholesterol, and inflammation

- Selecta is developing a candidate for the treatment of IgAN combining **ImmTOR with an IgA protease** to remove injurious IgA from kidneys and improve markers of renal dysfunction
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

- Selecta intends to co-administer ImmTOR with its proprietary 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

- IgA Protease candidate selection and initiation of IND enabling studies in 2022

Experienced management team positions Selecta for success

Carsten Brunn, Ph.D.  
President and CEO

Kevin Tan  
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
Financial information at-a-glance

Expected financial runway into mid 2024

~$148.0 MILLION\(^{(1)}\)

Cash on hand as of September 30, 2022\(^{(2)}\)

- Top-line data from Phase 3 DISSOLVE I & II programs of SEL-212 in chronic refractory gout
- Phase 1 clinical trial initiation and preliminary SEL-302 data in gene therapy for MMA
- Enzyme candidate selection and IND enabling studies in IgA Nephropathy
- Advance proprietary IgG protease (Xork)
- Develop a proprietary IL-2 mutein to combine with ImmTOR. Advance and expand our immune tolerance platform into autoimmune disease
- Advance autoimmune disease program in PBC

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1. Unaudited
2. Includes cash, cash equivalents, marketable securities and restricted cash.
Company Highlights

ImmTOR™ platform has potentially broad applicability

- Precision immune tolerance platform has potential to restore self tolerance in autoimmune disease and overcome immunogenicity of gene therapies and biologics
- Preclinical data indicates potentially profound synergy of ImmTOR and engineered Treg-selective IL-2 to expand antigen-specific Tregs and improve durability of immune tolerance (ImmTOR-IL)

Proof of concept in biologics and gene therapy

- SEL-212 in chronic refractory gout potentially serves as proof of concept for the ImmTOR platform in biologics with over 400 patients dosed – Phase 3 DISSOLVE I & II topline read out expected in Q1 2023
- Empty AAV capsid study data in healthy volunteers showed the potential ability of ImmTOR to inhibit the formation of neutralizing antibodies to AAV capsids

Diversified pipeline expanding to autoimmune disease

- SEL-302: Gene therapy program in methylmalonic acidemia (MMA), anticipated Phase 1 trial start in Q4 2022
- SEL-018: Plans to advance Xork IgG Protease to mitigate pre-existing anti-AAV antibodies through IND-enabling studies
- IgA nephropathy: clinical candidate selection & IND enabling studies in process
- Plans to advance the ImmTOR platform with IL-2 (ImmTOR-IL) for use in autoimmune disease
- Expected financial runway into mid 2024

Targeted partnerships to maximize platform potential

- sobi
- GENOVIS
- AskBio
- Takeda
- SAREPTA THERAPEUTICS
- GINKGO BIOWORKS
- CYRUS BIOTECHNOLOGY
Contact:

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Watertown, MA 02472

617-923-1400
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