



# Targeting T1D

## Modifying Disease

## Moving Beyond Insulin

Corporate Presentation  
Q2 2026

# Forward-Looking Statements

The material in this presentation has been prepared by SAB Biotherapeutics, Inc., doing business as SAB BIO (“SAB”) and is general background information about SAB’s activities current as of the date of this presentation. This information is given in summary form and is not intended to be complete. Information in this presentation, including financial forecasts, should not be considered advice or a recommendation to investors or potential investors in relation to holding, purchasing, or selling securities or other financial products or instruments and does not take into account any particular investment objectives, financial situation or needs. This presentation is for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy any securities.

This presentation may contain forward-looking statements including statements regarding our intent, belief, or current expectations with respect to SAB’s businesses and operations, market conditions, the exercise of outstanding warrants for cash, results of operations and financial condition, capital adequacy, specific provisions, and risk management practices. Readers are cautioned not to place undue reliance on these forward-looking statements. SAB does not undertake any obligation to update any information herein for any reason or to publicly release the result of any revisions to these forward-looking statements to reflect events or circumstances after the date hereof to reflect the occurrence of unanticipated events unless required by law. While due care has been used in the preparation of forecast information, actual results may vary in a materially positive or negative manner and the presentation may contain errors or omissions. Forecasts and hypothetical examples are subject to uncertainty and contingencies outside SAB’s control. Past performance is not a reliable indication of future performance. The forward-looking statements contained or implied in this presentation are subject to other risks and uncertainties, including those discussed under the heading “Risk Factors” in SAB’s most recent Annual Report on Form 10-K with the Securities and Exchange Commission (the “SEC”) and in other filings and reports that SAB makes with the SEC.

Unless otherwise specified, information is current at the date hereof.

The SAB logo and other trademarks of SAB appearing in this presentation are the property of SAB. All other trademarks, services marks, and trade names in this presentation are the property of their respective owners.



# Transforming Treatment for People Living with Autoimmune Diseases through a Unique Disease-modifying Therapy

At SAB BIO, our mission is to dramatically redefine what it means to be diagnosed with Type 1 Diabetes by developing a medicine to change the course of disease, not just treat symptoms



# SAB BIO Investment Highlights

## Potential for Significant Value Creation and Patient Impact

### Leading Clinical-Stage Company Focused on Autoimmune Type 1 Diabetes



#### REDEFINING T1D TREATMENT LANDSCAPE

SAB-142, our lead product candidate, is a potentially best-in-class, disease-modifying therapy with a de-risked mechanism of action  
Currently conducting a registrational Phase 2b SAFEGUARD study for newly diagnosed Stage 3 autoimmune type 1 diabetes (T1D)



#### LARGE MARKET OPPORTUNITY WITH ESTABLISHED REGULATORY PATHWAY

T1D is a multi-billion market opportunity with a global prevalence of ~9.5M  
SAB-142 is initially focused on Stage 3 T1D (U.S. incidence of 64K) where the treatment landscape is expanding towards disease-modifying therapies along an informed regulatory pathway established by Tzield



#### UNIQUE MULTI-SPECIFIC ANTIBODY PLATFORM WITH HIGH BARRIERS TO ENTRY

Proprietary, wholly-owned, discovered in-house platform capable of generating a diverse repertoire of multi-specific, targeted, fully human immunoglobulins (hIgG)  
This unique platform leverages a multi-level IP strategy with no biosimilar pathway creating high barriers to entry



#### WELL CAPITALIZED WITH TOP-TIER INVESTORS

Backed by a syndicate of life science specialist investors  
Current cash position is expected to fully fund SAB-142's SAFEGUARD study with data expected 2H 2027



#### EXPERIENCED LEADERSHIP TEAM

Led by management team and board of directors with deep, proven biopharma experience spanning global clinical development, regulatory strategy, and commercialization

# SAB-142: Potential Disease-modifying Immunotherapy Being Developed to Delay the Onset and Progression of Type 1 Diabetes

Goal: develop a T1D therapy that immunomodulates T cells to preserve C-peptide while avoiding immunosuppression



SAB-142  
Anti-Thymocyte  
Globulin (Human)

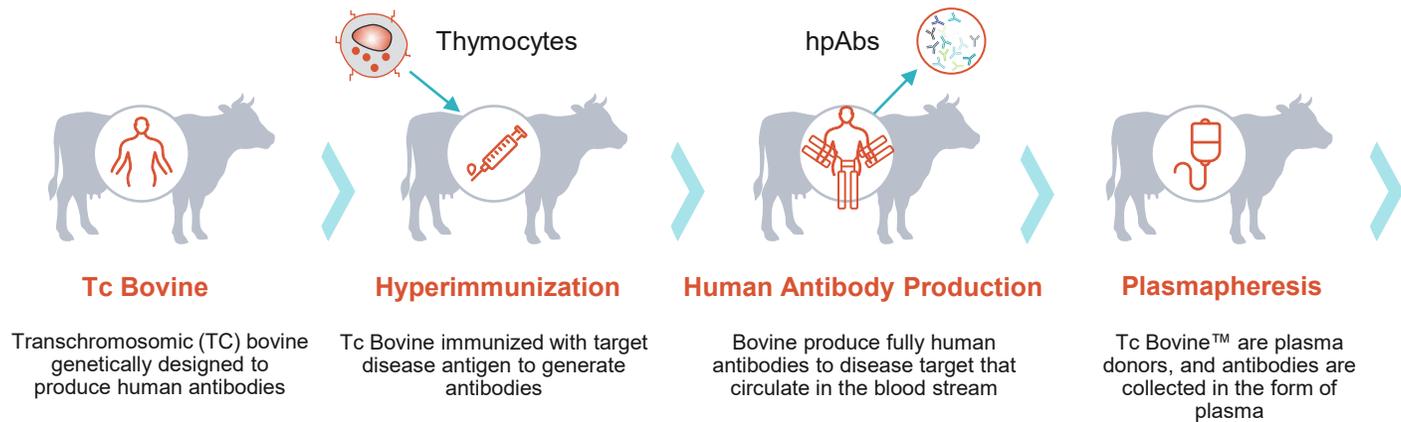
- › **SAB-142 is a multi-specific, fully human anti-thymocyte globulin (hATG)** disease-modifying immunotherapy to delay the onset and progression of T1D
- › SAB-142 works by directly **targeting multiple immune cells** involved in destroying pancreatic beta cells, including the modulation of “bad acting” T-lymphocytes
  - Mechanism of action, analogous to rabbit ATG (rATG), directly modulates multiple immune cells involved in destroying pancreatic beta cells
  - **SAB-142 provides a better safety profile** resulting in no serum sickness and low/no immunogenicity – this offers the potential for life-long disease modification through redosing safely, preserving C-peptide, and delaying the onset or progression of T1D
  - By stopping immune cells from attacking beta cells, this treatment has the **potential to preserve insulin-producing beta cells**
  - **C-peptide** is a stable marker of endogenous insulin production
  - Preserving beta cell function and thus insulin production as measured by C-peptide is **key to delaying progression of T1D**

Early intervention is essential and life-long disease modification is possible with effective, safe, and reliable redosing

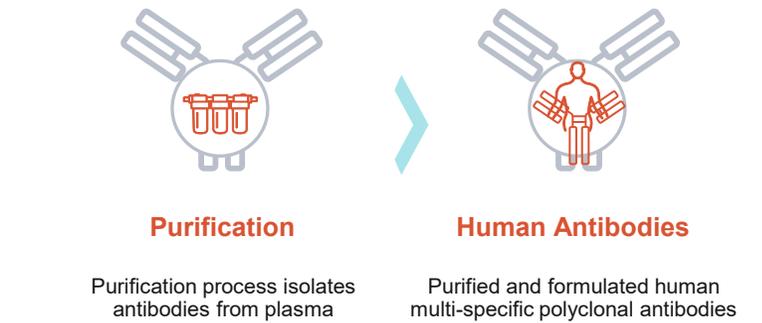
# SAB-142 is Manufactured Using our Fully Human Immunoglobulin G (hIgG) to Treat and Prevent Autoimmune Disorders

SAB has a proprietary platform that can produce targeted, fully human, multi-specific polyclonal antibodies without the need for human donors through its Tc Bovine platform

## Upstream at SAB's Biosecure Pharm Facility



## Downstream at SAB's cGMP Facility



# Significant Unmet Need in Type 1 Diabetes

An evolving treatment landscape with a regulatory pathway informed by Tzield

## Stage 2 (Delay Onset) Market



Approved in Stage 2 patients to delay the onset of T1D



\$2.9B Sanofi acquisition of Provention Bio (Tzield in Phase 3 for Stage 3 at time of acquisition)

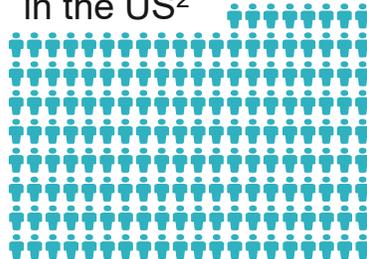
## Stage 3 (Newly Diagnosed) Market



64,000 U.S. Patients Diagnosed Annually<sup>1</sup>

2.0M

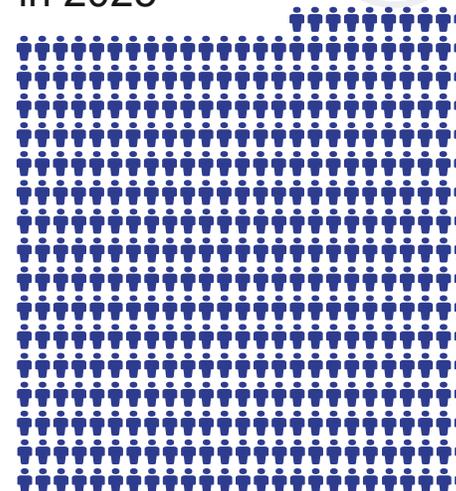
People with T1D cases in the US<sup>2</sup>



500,000 Patients WW Diagnosed Annually<sup>2</sup>

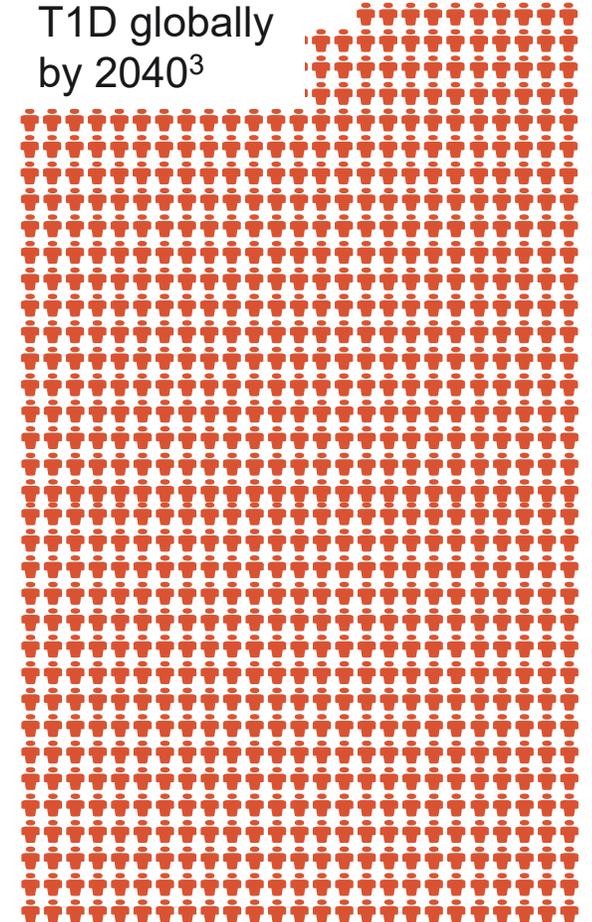
9.5M

People with T1D globally in 2025<sup>3</sup>



14.7M

People with T1D globally by 2040<sup>3</sup>



# SAB-142: A Clinically Validated De-risked Mechanism of Action

rATG has been tested across multiple studies in T1D patients

**2013** 

rATG Tested in Academic Setting (START Study)

Dosage	6.5mg/kg
Lymphodepletion	⚠️
C-peptide	✅
HbA1c	✅
Serum Sickness	⚠️
Ability to redose	🚫

*(Gitelman et al., 2013)*

**2018** 

rATG Tested in Academic Setting (TN-19 Study)

Dosage	2.5mg/kg
T-cell Exhaustion	✅
Lymphodepletion	⚠️
C-peptide	✅
HbA1c	✅
Serum Sickness	⚠️
Ability to redose	🚫

*(Haller et al., 2018)*

**3Q 2025** 

rATG Tested in Academic Setting (MELD-ATG Study)

Dosage	Multiple
C-peptide	✅
HbA1c	✅
Lymphodepletion not an efficacy driver	✅
Serum Sickness	⚠️
Ability to redose	🚫

*(Mathieu et al., Lancet, 2025)*

**SAB-142: hATG tested in healthy volunteers and T1D patients**

**4Q 2025** 

SAB-142 tested in Healthy Volunteers And T1D patients

Dosage	Multiple
T-cell Exhaustion	✅
C-peptide*	N/A
HbA1c*	N/A
Serum Sickness	🚫
Ability to redose	✅

*(January and December 2025)*



Leveraged clinical findings showing low-dose rATG (2.5 mg/kg) effectively mitigates disease

SAB applied the clinical findings that exhaustion correlated with efficacy rather than depletion

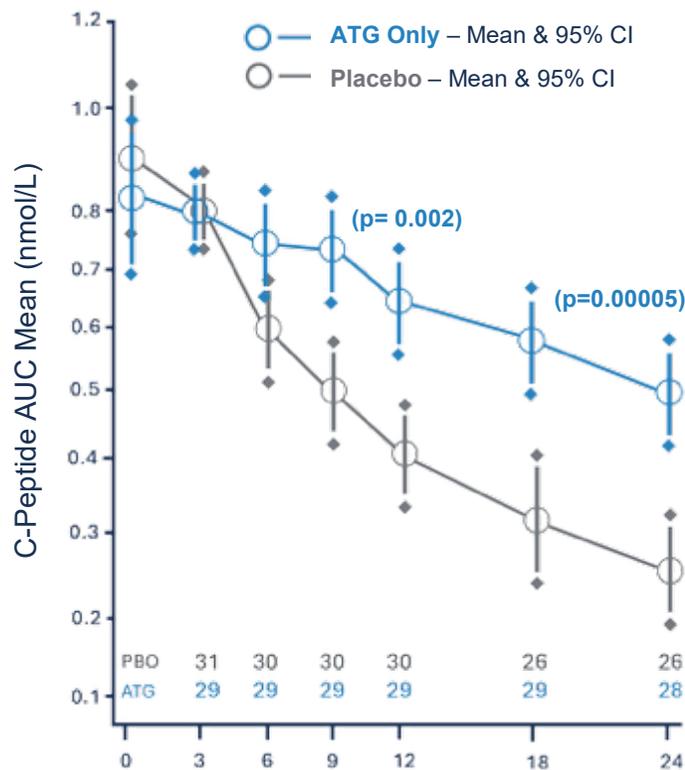
# Rabbit ATG: De-Risked Mechanism of Action



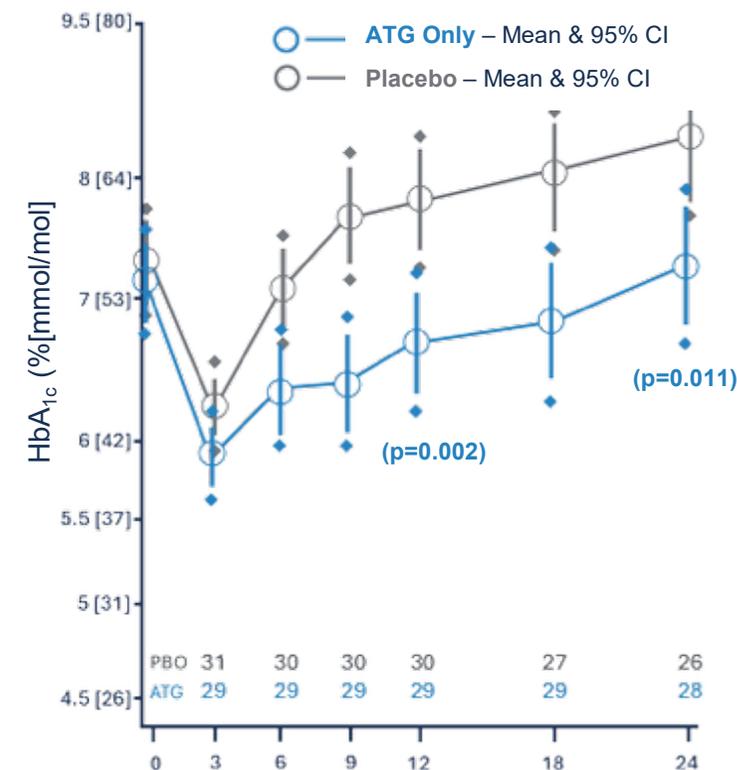
**TN-19: Low-Dose rATG\* Preserved C-peptide in New Onset T1D 1 and 2 Years Post Treatment**

C-peptide, the cleavage product of proinsulin, released in equal amounts with insulin, reflects a person's ability to produce endogenous insulin and is the standard biomarker of pancreatic  $\beta$ -cell function

**Decline in C-peptide AUC Mean Over Time by Treatment Group**



**HbA<sub>1c</sub> Over Time by Treatment Group**



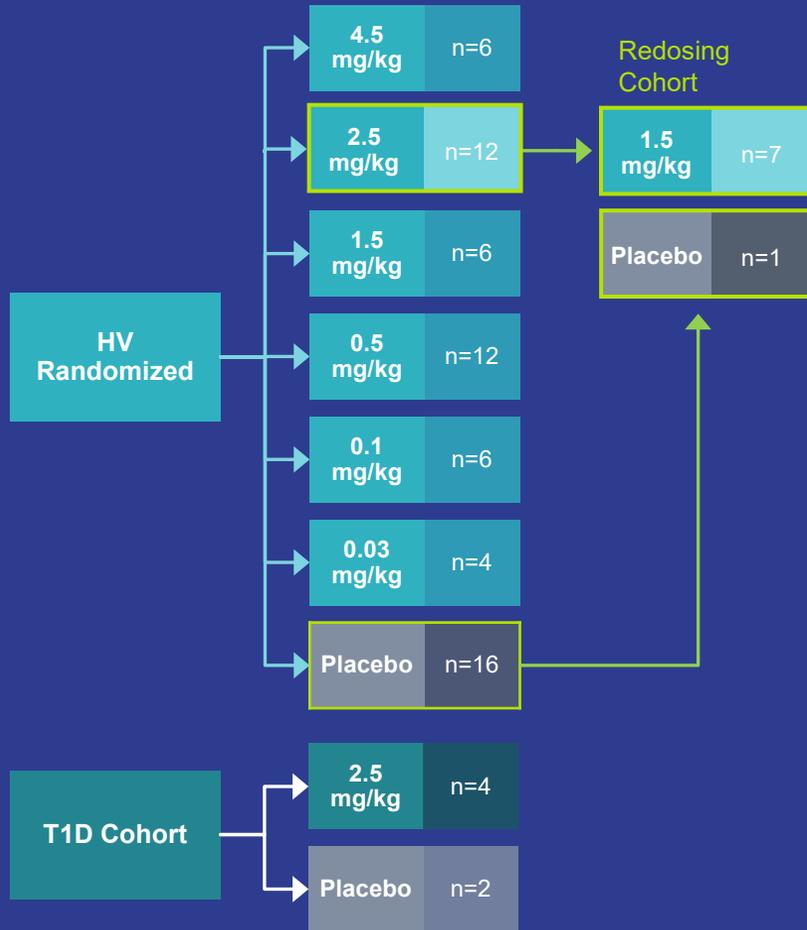
Time on Study (months)

Haller et al. Diabetes. 2019 Jun;68(6):1267-1276

\*Haller MJ, Long SA, Blanchfield JL, Schatz DA, Skyler JS, Krischer JP, Bundy BN, Geyer SM, Warnock MV, Miller JL, Atkinson MA, Becker DJ, Baidal DA, DiMeglio LA, Gitelman SE, Goland R, Gottlieb PA, Herold KC, Marks JB, Moran A, Rodriguez H, Russell WE, Wilson DM, Greenbaum CJ; Type 1 Diabetes TrialNet ATG-GCSF Study Group. Low-Dose Anti-Thymocyte Globulin Preserves C-Peptide, Reduces HbA<sub>1c</sub>, and Increases Regulatory to Conventional T-Cell Ratios in New-Onset Type 1 Diabetes: Two-Year Clinical Trial Data. Diabetes. 2019 Jun;68(6):1267-1276.

# Phase 1: HUMAN\* Trial Study Design

Randomized, double-blind, placebo-controlled, single- and multiple- ascending dose, adaptive design clinical study



# SAB-142's Phase 1 Data Demonstrated Encouraging Efficacy Signals with Clinically Validated, Multi-specific MOA with Sustained Immunomodulation



## Efficacy

Encouraging early signals of C-peptide preservation in established T1D patients

- ✓ Early C-peptide signal consistent with beta cell preservation with 3 out of 4 T1D participants with super responder status
- ✓ Glycemic control improvement not associated with increases in exogenous insulin use



## Immunogenicity

Confirm SAB-142 not immunogenic

- ✓ Not observed to cause anti-drug antibodies
- ✓ Enables safe and reliable redosing



## PK/PD

Demonstrate sustained "T-cell exhaustion" signature

- ✓ Clinically validated by rATG and other T1D T-cell targeting biologics
- ✓ Demonstrated correlation with C-peptide preservation based on precedent rATG studies and natural course of T1D



## Safety and Tolerability

Position SAB-142 for a convenient, potentially twice a year dosing regimen

- ✓ No sustained lymphodepletion leading to immuno-suppression; no neutropenia observed
- ✓ No serum sickness

# Complete Phase 1 Safety Data Support Outpatient Dosing and Chronic Treatment Potential

Phase 1 data confirmed advancement into a registrational Phase 2b study in newly diagnosed adult, adolescents, and pediatric T1D patients (age 5-40)

HV and T1D

## Key Safety Outcomes

- › No serum sickness (0%, N=0/68)
- › No ADA-related AEs at any dose or cohort (0%, N=0/68)
- › No drug-related SAEs

## Observed AEs

- › Headaches, a typical AE for T-cell modifying therapies
- › Mild CRS (Grade 1 only)
- › Transient infusion-site reactions (erythema, pruritus, tenderness, phlebitis)

## Transient On-Target PD Effects

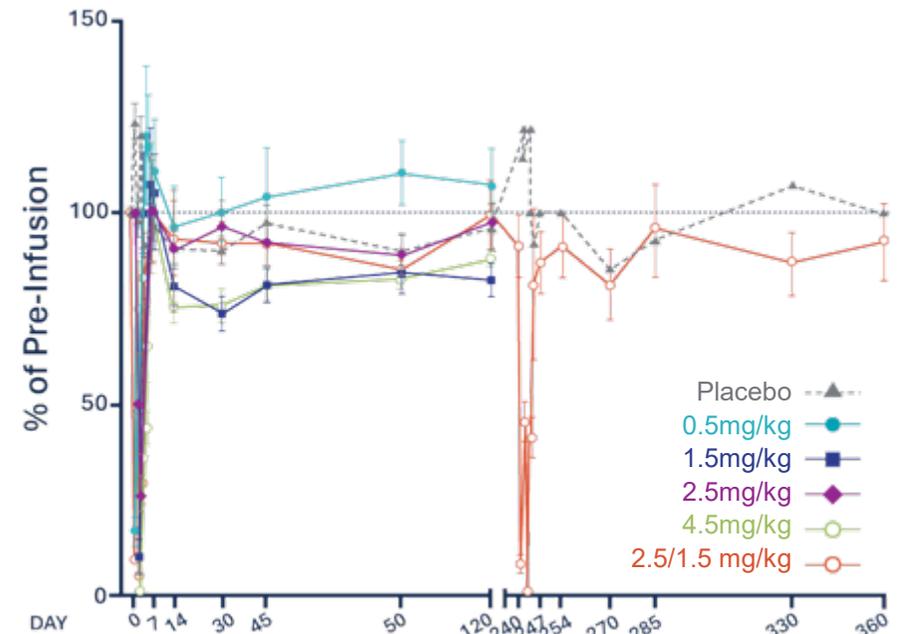
- › Transient lymphopenia indicative of target engagement, a desired PD effect
- › Lymphopenia shown to self-resolve in 1–3 days
- › No lymphocyte killing/sustained lymphodepletion, a key risk factor of immunosuppression – supports chronic redosing potential

## Hematologic Safety

- › No reductions in RBCs, neutrophils, lymphopenia, or thrombocytopenia from Day 7 onward



Mean Absolute Lymphocytes  $\pm$  SEM Normalized to Original Pre-SOI in HV



Lymphocytes were shown to fully recover to baseline across all doses including 4.5mg/kg and after repeat dosing

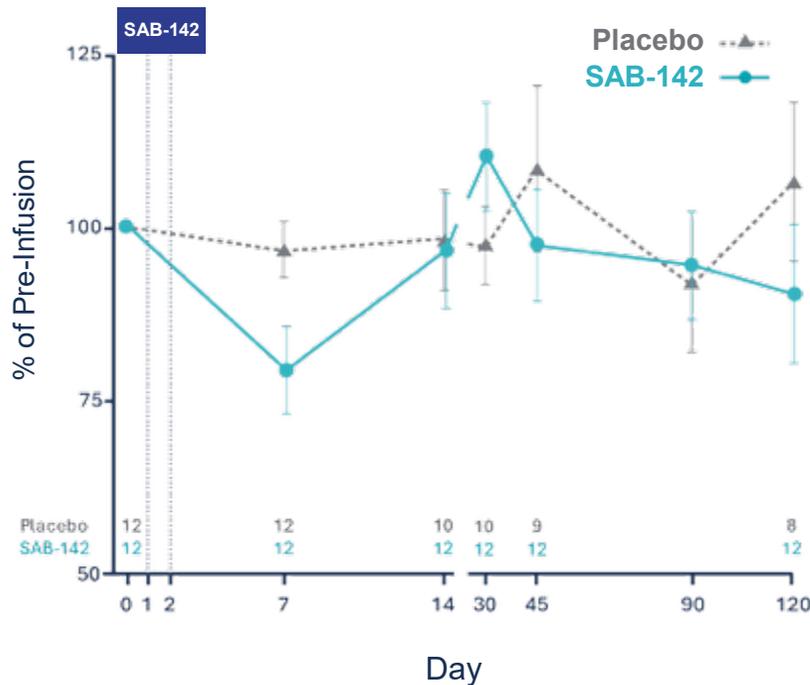
# SAB-142 Demonstrates a Comparable MOA to rATG, including Induction of Key T-cell Exhaustion Markers that have been Correlated with C-peptide Preservation in Prior rATG Studies



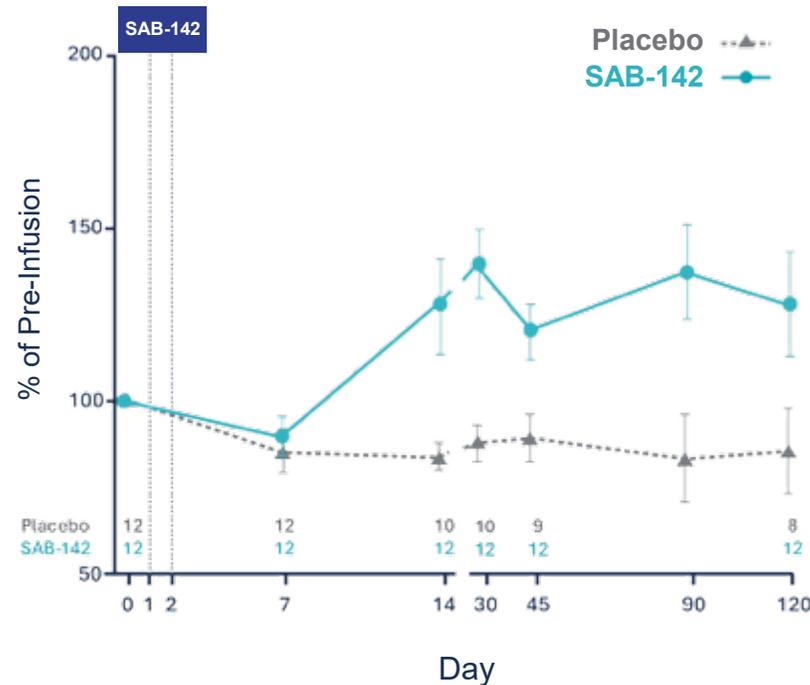
## 1 Treg preservation

## 2 Increase in CD4 exhaustion

Treg (CD3<sup>+</sup> CD4<sup>+</sup> CD127<sup>lo</sup> CD25<sup>+</sup> FoxP3<sup>+</sup>) ± SEM



Relative PD-1<sup>+</sup> Tconv Cells ± SEM



## rATG Published Data:

Responders to low-dose ATG induce CD4<sup>+</sup> T-cell exhaustion in type 1 diabetes - PubMed



### Responders to low-dose ATG induce CD4<sup>+</sup> T cell exhaustion in type 1 diabetes

Laura M. Jacobsen,<sup>1,2</sup> Kirsten Diggins,<sup>3</sup> Lori Blanchfield,<sup>3</sup> James McNichols,<sup>2</sup> Daniel J. Perry,<sup>2</sup> Jason Brant,<sup>2</sup> Xiaoru Dong,<sup>2,4</sup> Rhonda Bacher,<sup>4</sup> Vivian H. Gersuk,<sup>2</sup> Desmond A. Schatz,<sup>1</sup> Mark A. Atkinson,<sup>1,2</sup> Clayton E. Mathews,<sup>1,2</sup> Michael J. Haller,<sup>1</sup> S. Alice Long,<sup>3</sup> Peter S. Linsley,<sup>5</sup> and Todd M. Brusko<sup>1,2</sup>

<sup>1</sup>Department of Pediatrics, College of Medicine, University of Florida, Gainesville, Florida, USA. <sup>2</sup>Department of Pathology, Immunology, and Laboratory Medicine, University of Florida Diabetes Institute, Gainesville, Florida, USA. <sup>3</sup>Benaroya Research Institute at Virginia Mason, Seattle, Washington, USA. <sup>4</sup>Department of Biostatistics, University of Florida, Gainesville, Florida, USA.

**RESULTS.** Treatment with low-dose ATG preserved regulatory T cells (Tregs), as measured by stable methylation of FOXP3 Treg-specific demethylation region (TSDR) and increased proportions of CD4<sup>+</sup>FOXP3<sup>+</sup> Tregs (P < 0.001) identified by flow cytometry. While treatment effects were consistent across participants, not all maintained C-peptide. Responders exhibited a transient rise in IL-6, IP-10, and TNF-α (P < 0.05 for all) 2 weeks after treatment and a durable CD4<sup>+</sup> exhaustion phenotype (increased PD-1<sup>+</sup>KLRG1<sup>+</sup>CD57<sup>+</sup> on CD4<sup>+</sup> T cells [P = 0.011] and PD1<sup>+</sup>CD4<sup>+</sup> Temra MFI [P < 0.001] at 12 weeks, following ATG and ATG/G-CSF, respectively). ATG nonresponders displayed higher proportions of senescent T cells (at baseline and after treatment) and increased methylation of EOMES (i.e., less expression of this exhaustion marker).

**CONCLUSION.** Altogether in these exploratory analyses, Th1 inflammation-associated serum and CD4<sup>+</sup> exhaustion transcript and cellular phenotyping profiles may be useful for identifying signatures of clinical response to ATG in T1D.

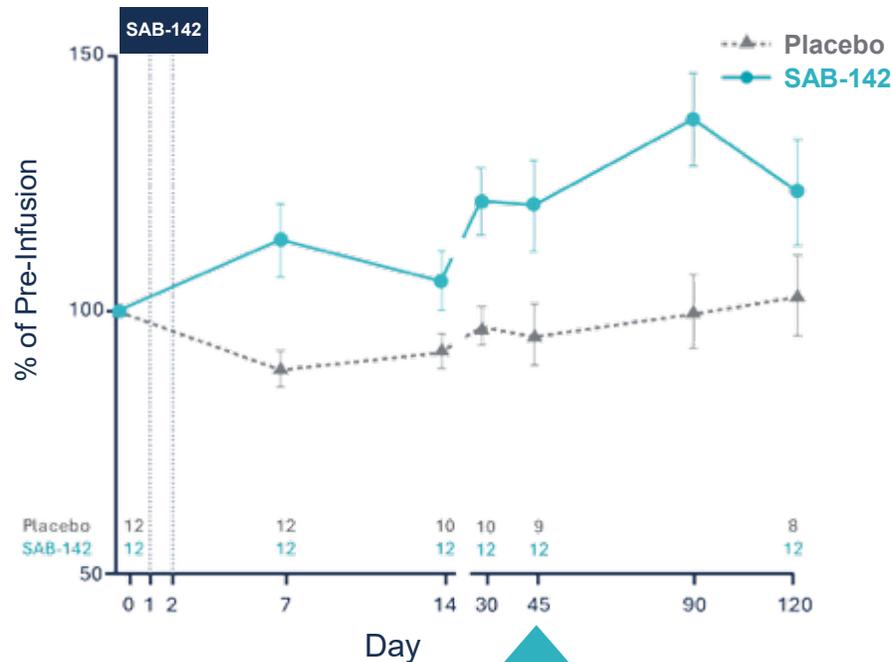
# SAB-142 Induces Operational Tolerance as Indicated by Single and Dual-Exhaustion Markers

HV

## SAB-142 Phase 1 Top-Line Data:

### 1 SAB-142 CD4+ T Conv Cell Single Exhaustion Markers

Relative TIGIT+ Tconv Cells  $\pm$  SEM



SAB-142 induced sustained expression of inhibitory receptors (TIGIT+) on CD4+ T conv cells indicative of an exhausted phenotype

### 2 SAB-142 CD4+ T conv Cell Dual Exhaustion Markers

Tconv Median % Change from Baseline



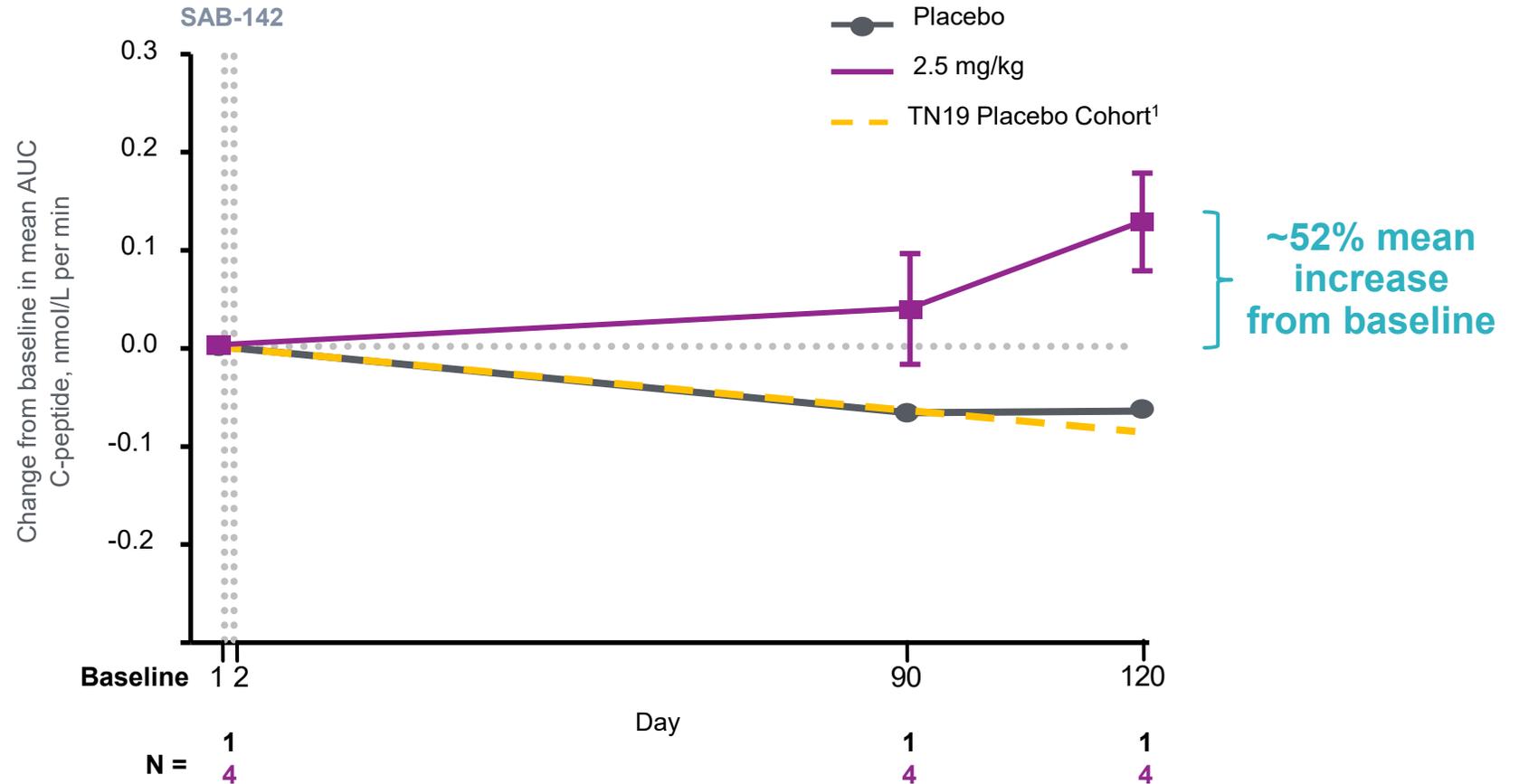
SAB-142 induced persistent expression of co-inhibitory receptors on CD4+ Tconv cells

# Adult Established T1D Cohort: C-Peptide Responses

Established T1D

- › C-peptide demonstrated preservation over time in the SAB-142 T1D cohort vs. study placebo and TN19 placebo cohort<sup>1</sup>
- › Supports a PD effect consistent with preservation of beta cell function in adults with established T1D

### MMTT C-Peptide Mean AUC ± SEM



<sup>1</sup>Predicted decline was estimated by first calculating the incremental AUC of TN19 placebo subject C-peptide MMTT data (n=26) for Weeks 48-96 with post-2hr values masked. The linear slope was used to calculate the predicted rate of decline in days:  $AUC_{BL} - (-0.6108 * (Study Day / 7))$ .

Source: Haller et al. Diabetes. 2019 Jun;68(6):1267-1276.

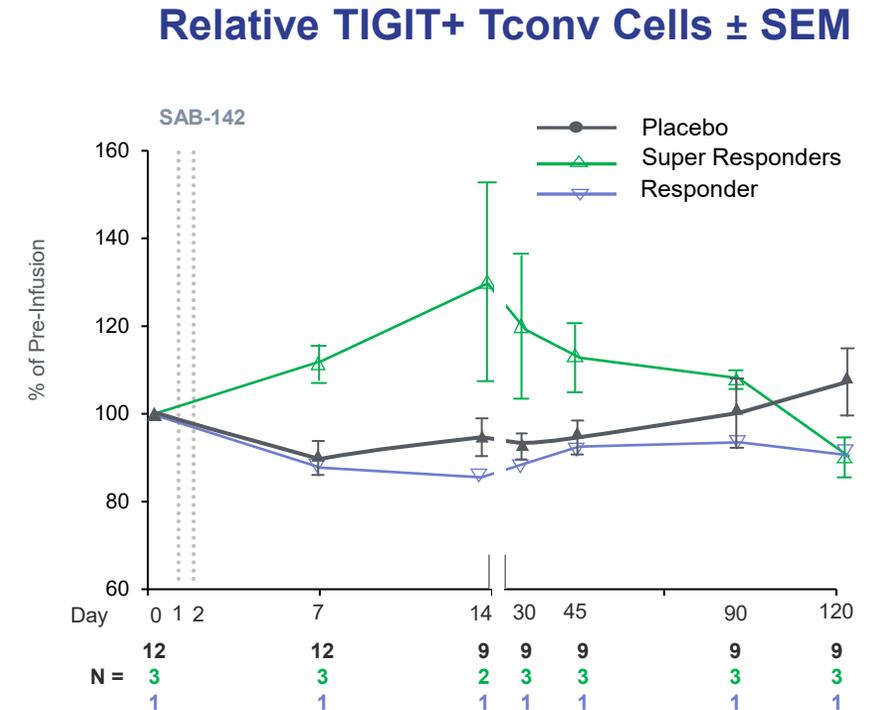
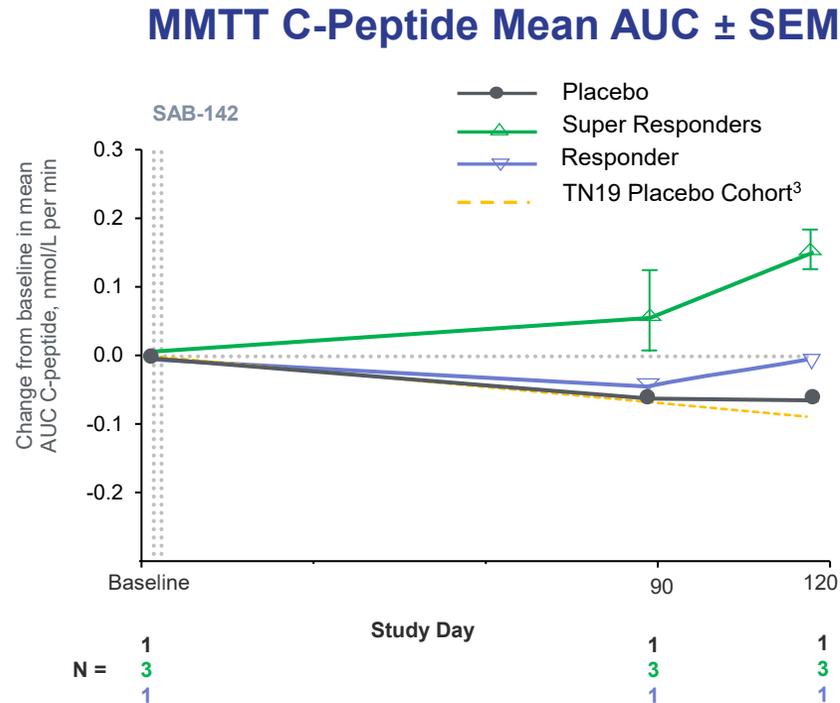
MMTT = Mixed Meal Tolerance Test; AUC = Area Under the Curve; SEM = Standard Error the Mean.

# C-Peptide Responses and T Cell Exhaustion

## Super Responders vs. Responder

Established T1D & HV

- > In SAB-142 T1D cohort, 3 of the 4 participants exhibited mean C-peptide values consistent with super responder status as previously categorized in the study of rATG<sup>1</sup>
- > SAB-142 induced CD4+ Tconv cell exhaustion contributing to C-peptide preservation, which is consistent with MOA observed with rATG in previous studies<sup>1,2</sup>



**Super responder C-peptide profile concomitant with T cell exhaustion**

<sup>1</sup>In that rATG study, super responders were defined as participants with C-peptide at or above the baseline at the end of the study (Month 24). Similarly, in the SAB-142 Phase 1 trial, participants with C-peptide response above the baseline at the end of the study (Day 120) were defined as super responders. Source: Haller et al. Diabetes. 2016 Dec;65(12):3765-3775.

<sup>2</sup>Source: Jacobsen et al. JCI Insight. 2023 Jul;8(16):e161812.

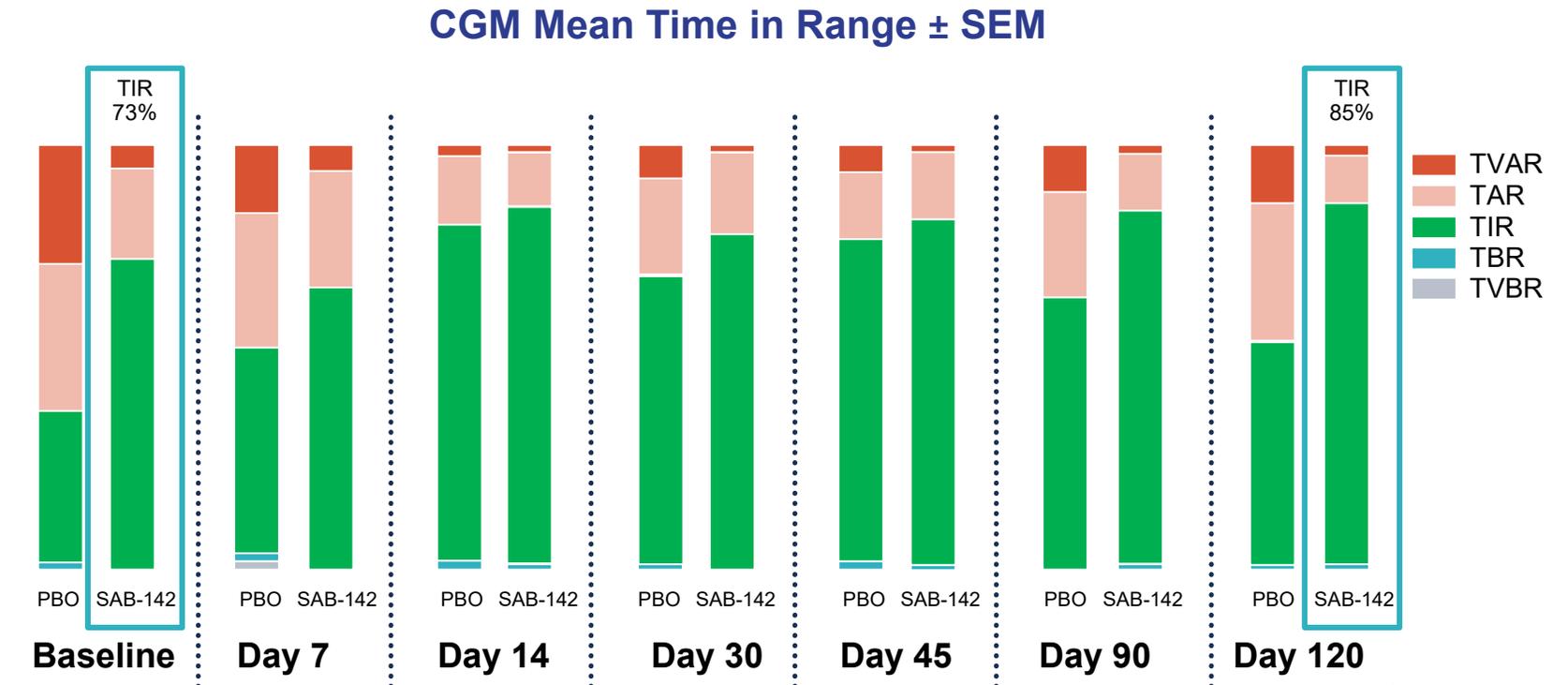
<sup>3</sup>Predicted decline was estimated by first calculating the incremental AUC of TN19 placebo subject C-peptide MMTT data (n=26) for Weeks 48-96 with post-2hr values masked. The linear slope was used to calculate the predicted rate of decline in days:  $AUC_{BL} - (-0.6108 * (Study Day / 7))$ . Source: Haller et al. Diabetes. 2019 Jun;68(6):1267-1276.

MMTT = Mixed Meal Tolerance Test; AUC = Area Under the Curve; SEM = Standard Error of the Mean.

# Adult Established T1D Cohort: Continuous Glucose Monitoring (CGM) Time in Range

Established T1D

- › Participants receiving SAB-142 (2.5 mg/kg) demonstrated an increase in mean TIR from 73% at baseline to 85% at Day 120
- › SAB-142 effects on glycemic control are aligned with the observed C-peptide response
- › Both effects, increase in C-peptide and improvement in TIR relative to baseline, correlate with immune PD activity



Improvement in glycemic control was not associated with increases in exogenous insulin use

# SAB-142: Potential for Broadest Use with Superior Safety, Efficacy, and Patient Convenience in a Broad Age Range of T1D Patients\*

	 <b>Tziel</b> *** (teplizumab-mzwv)	 <b>Thymoglobulin</b> Anti-thymocyte Globulin (Rabbit)	 <b>SAB BIO</b> SAB-142
CD4+ exhaustion signature			
Treg preservation			
No sustained lymphodepletion (Tconv and/or Tregs)			
No anti-drug antibodies (ADAs)			
No serum sickness			
C-peptide preservation			 SAFEGUARD**
HbA1C			 SAFEGUARD**
Dosing	<b>12 DAYS x 2</b> (Annual, not redosable)	<b>1-2 DAYS</b> (Not redosable)	<b>2 DAYS x 2</b> (Annual, redosable)
Infusing timing	<b>1 HOUR</b>	<b>4-12 HOURS</b>	<b>4-6 HOURS</b>
Redosable (with potential for chronic dosing without ADAs)			
Age range	<b>8-17</b>	<b>5-45</b>	<b>5-40*</b> 

**SAB-142 exhibits multi-specific T-cell exhaustion profile and Treg-sparing without sustained lymphodepletion**

mimicking immunologic cellular profiles that naturally occur during the initial spontaneous partial remission period (“honeymoon period”)

**T-cell exhaustion phenotype is universally linked to C-peptide preservation**

\* Not head-to-head studies.

\*\* Potential benefits on HbA1c and C-peptide are informed by prior rATG outcomes, though this translatability to SAB-142 will be confirmed with the Phase 2b SAFEGUARD study results. Registrational Phase 2b SAFEGUARD trial includes T1D patients ages 5-40.

\*\*\* Marketed by Sanofi through its \$2.9bn acquisition of Provention Bio

# SAFEGUARD: Multicenter, Global Phase 2b for SAB-142 in Stage 3 Type 1 Diabetes Patients



**SAF**ety and **E**fficacy of human anti-thymocyte immunoglobulin SAB-142 **AR**resting progression of Type 1 **D**iabetes

## Trial design:

- > 159 pediatric, adolescent, and adult patients (5-40 years)
  - **Part A:** 12 patients – dose-ranging study for 12 months
  - **Part B:** 147 patients – randomized, double-blind, placebo-controlled, dose-ranging study for 12 months
- > All patients, including placebo, eligible for 12-month long-term extension study (Part C) upon completion

## Inclusion criteria:

- > New onset Stage 3 T1D: within 100 days of diagnosis
- > Baseline C-peptide:  $\geq 0.2$  nmol/L

## Dosing regimen:

- > Intravenous (IV)
- > 0.5 mg/kg on Day 1 and remainder of dose Day 2
- > 1<sup>st</sup> dose at study start and 2<sup>nd</sup> dose at month 6
- > Induction dose levels: 1.5 and 2.5mg/kg; Maintenance dose: 1.5mg/kg

Global study initiated with **topline results expected 2H 2027**



**United States**  
(FDA) NCT07187531



**Europe**  
(EMA)



**United Kingdom**  
(MHRA)

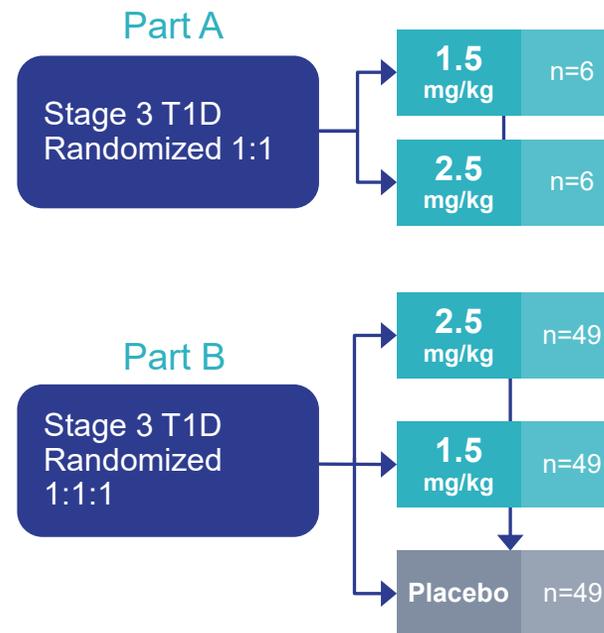


**Australia**  
(TGA)



**New Zealand**  
(MEDSAFE)

## Phase 2b Study Design



## Primary Endpoint:



### Stimulated C-peptide

following 2-hr MMTT at 12 months  
(detect at least 40% difference with 80% power)

## Secondary Endpoint:



### Leading Clinical Endpoint: HbA1C

## Other secondary Endpoints:

- > Time in Tight Range, Time in Range, Time Above and Below Range
- > Hypoglycemic episodes
- > Safety
- > Insulin use

# Strong Balance Sheet with Committed Strategic Partners

## Recent Financings Fully Fund Phase 2b SAFEGUARD Study and Pre-Commercial Activities

Potential for an additional \$284 million if milestone-based warrants from July 2025 financing are exercised in full

## T1D Clinical Development Partner:

SAB-142 clinical development plan designed in partnership with Breakthrough T1D (formerly JDRF)

### Financial Snapshot:



### Key Clinical and Strategic Partners:

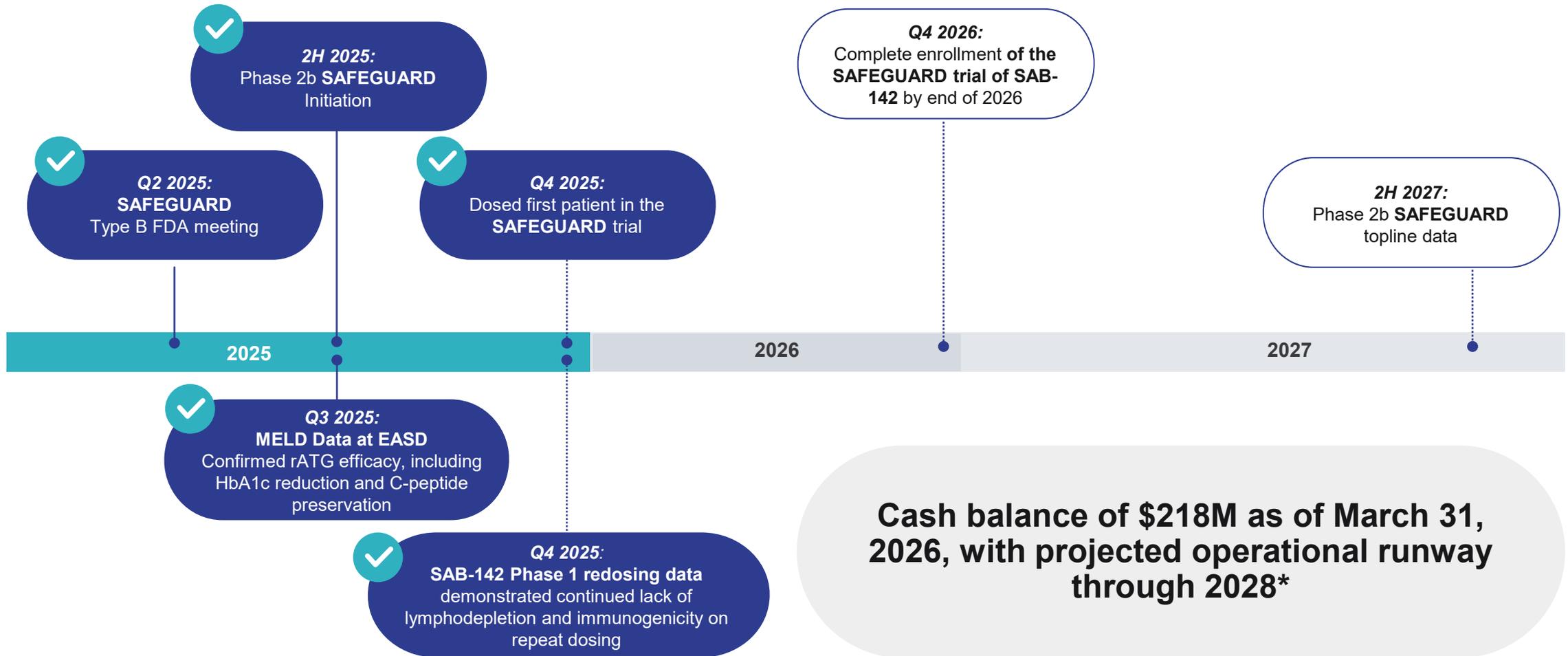


# Focused on T1D with Potential to Advance in Other Autoimmune Diseases

	Preclinical	Phase 1	Phase 2	Phase 3	Milestones:
<b>Type 1 Diabetes</b>					Q4'25: SAFEGUARD initiated Q1'26: Part A enrollment completed; Part B initiated
Delaying progression of T1D in new onset T1D patients (Stage 3)	Registrational Phase 2b				
Maintenance of Stage 3 T1D (LTE Part C SAFEGUARD)					
Delaying onset of Stage 3 T1D (Stage 2)					
<b>Transplantation</b>					In vivo and in vitro pre-clinical and Phase 1 SAB-142 data <b>support direct progression into Phase 2 in other autoimmune indications</b>
Transplant Maintenance in Islet Cell Transplantation					
<b>Autoimmunity</b>					
Celiac Disease					
SLE, Scleroderma, Polymyositis, Dermatomyositis					

-  Current Studies
-  Potential future studies SAB is not currently funding

# Strong 2025 Execution with Significant Catalysts Expected in 2026-2027



\*Note: Cash, cash equivalents, and available for sale securities as of March 31, 2026.

# SAB BIO Investment Highlights

## Potential for Significant Value Creation and Patient Impact

### Leading Clinical-Stage Company Focused on Autoimmune Type 1 Diabetes



#### REDEFINING T1D TREATMENT LANDSCAPE

SAB-142, our lead product candidate, is a potentially best-in-class, disease-modifying therapy with a de-risked mechanism of action  
Currently conducting a registrational Phase 2b SAFEGUARD study for newly diagnosed Stage 3 autoimmune type 1 diabetes (T1D)



#### LARGE MARKET OPPORTUNITY WITH ESTABLISHED REGULATORY PATHWAY

T1D is a multi-billion market opportunity with a global prevalence of ~9.5M  
SAB-142 is initially focused on Stage 3 T1D (U.S. incidence of 64K) where the treatment landscape is expanding towards disease-modifying therapies along an informed regulatory pathway established by Tzield



#### UNIQUE MULTI-SPECIFIC ANTIBODY PLATFORM WITH HIGH BARRIERS TO ENTRY

First-ever, wholly-owned, discovered in-house platform capable of generating a diverse repertoire of multi-specific, targeted, fully human immunoglobulins (hIgG)  
This unique platform leverages a multi-level IP strategy with no biosimilar pathway creating high barriers to entry



#### WELL CAPITALIZED WITH TOP-TIER INVESTORS

Backed by a syndicate of life science specialist investors  
Current cash position is expected to fully fund SAB-142's SAFEGUARD study with data expected 2H 2027



#### EXPERIENCED LEADERSHIP TEAM

Led by management team and board of directors with deep, proven biopharma experience spanning global clinical development, regulatory strategy, and commercialization

# THANK YOU

Targeting T1D

Modifying Disease

Moving Beyond Insulin



# Appendix

# T1D Occurs in 3 Progressive Stages Identified by the Presence of Multiple Autoantibodies and Increasing Glycemic Levels

T1D is associated with serious long-term complications, including diabetic nephropathy, neuropathy, and retinopathy, as well as increased risk of cardiovascular disease, peripheral arterial disease, cerebrovascular events, and diabetic foot complications.

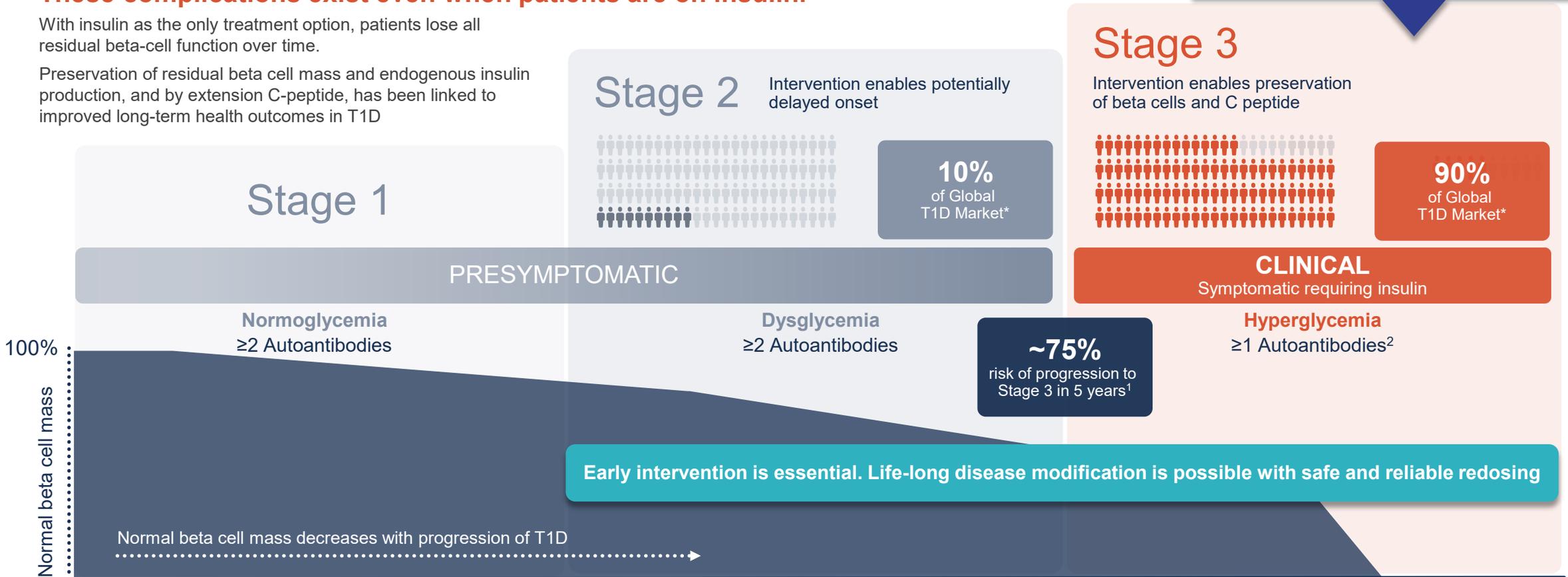
**These complications exist even when patients are on insulin.**

With insulin as the only treatment option, patients lose all residual beta-cell function over time.

Preservation of residual beta cell mass and endogenous insulin production, and by extension C-peptide, has been linked to improved long-term health outcomes in T1D



SAB-142, currently in Phase 2b, has a de-risked MOA to delay the progression of T1D



\*Based on Company estimates.

Source:

1. <https://pmc.ncbi.nlm.nih.gov/articles/PMC5321245/>

2. American Diabetes Association Professional Practice Committee. Diagnosis and classification of diabetes: standards of care in diabetes—2024. Diabetes Care. 2024;47(Suppl 1):S20—S42

# Rabbit ATG: De-Risked Mechanism of Action



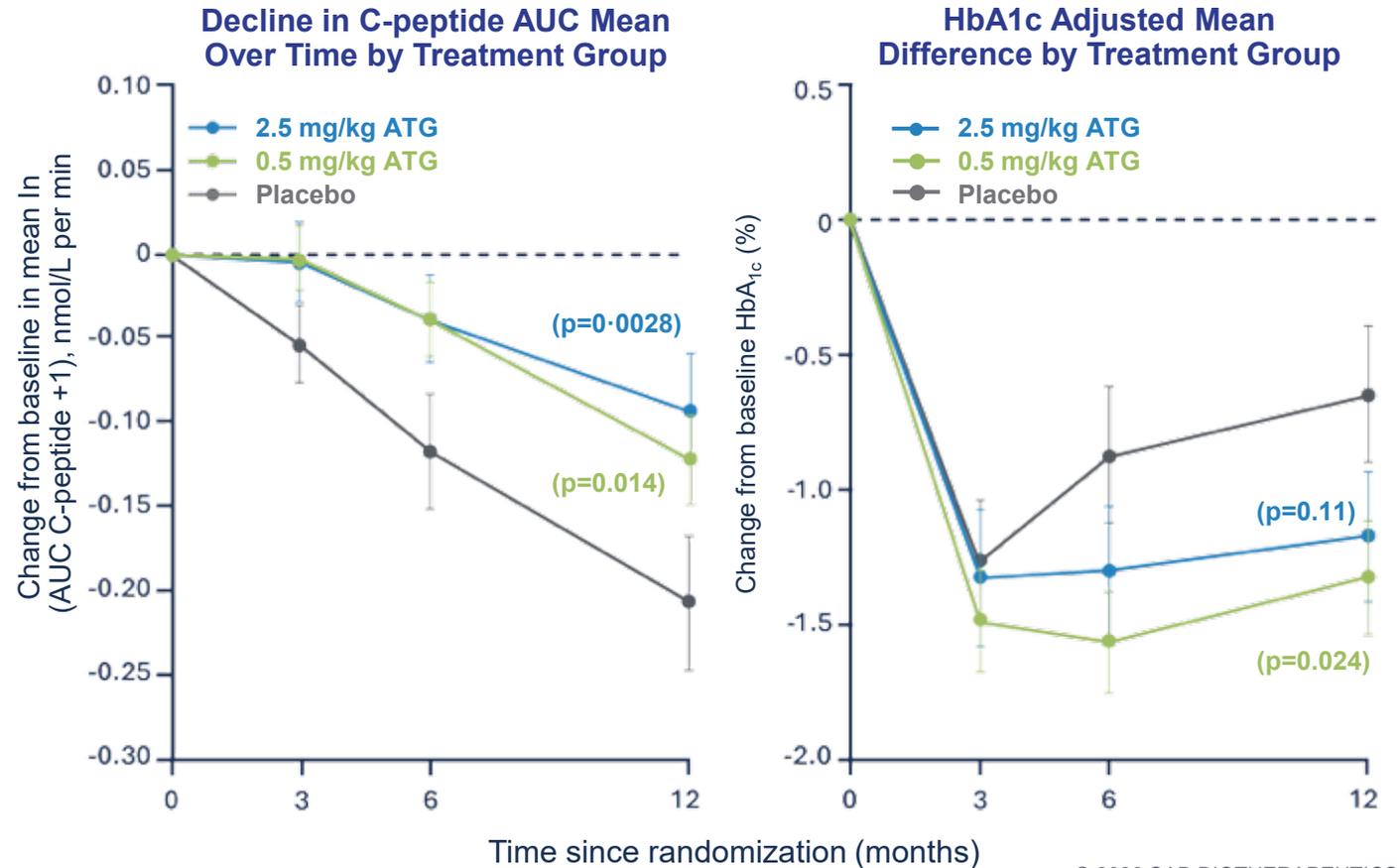
**MELD-ATG:** Minimal Effective Low Dose of rATG\* Preserved C-peptide in New Onset T1D 1 Year Post Treatment

MELD-ATG replicated results from Haller’s TN19 study with  $\leq 2.5$  mg/kg with statistically significant C-peptide preservation and glycemic control

\*Mathieu C, Wych J, Hendriks AEJ, Van Ryckeghem L, Tree T, Chmura P, Möller C, Casteels K, Danne T, Reschke F, Šmigoc Schweiger D, Battelino T, Johannesen J, Rami-Merhar B, Pieber T, De Block C, Evans M, Hilbrands R, Bosi E, Willemsen RH, Basu S, Pulkkinen MA, Knip M, Cnop M, Nitsche A, Schulte AM, Niemöller E, Peakman M, Wilhelm-Benartzi C, Gillespie D, Overbergh L, Mander AP, Marcovecchio ML; INNODIA. Minimum effective low dose of antithymocyte globulin in people aged 5-25 years with recent-onset stage 3 type 1 diabetes (MELD-ATG): a phase 2, multicentre, double-blind, randomised, placebo-controlled, adaptive dose-ranging trial. Lancet. 2025 Sep 18:S0140-6736(25)01674-5. doi: 10.1016/S0140-6736(25)01674-5. Epub ahead of print. PMID: 40976248.

**ATG is the only mechanism of action that has consistently reproduced clinical data demonstrating preservation of C-peptide and improvements in glycemic control**

(Mathieu et al., Lancet. 2025 Sep 18:S0140-6736(25)01674-5)



# Phase 1 Adult Established T1D Cohort: Baseline Characteristics

All study participants met SAFEGUARD inclusion criteria related to residual beta cell function and at least one T1D autoantibody at baseline

**Established T1D**

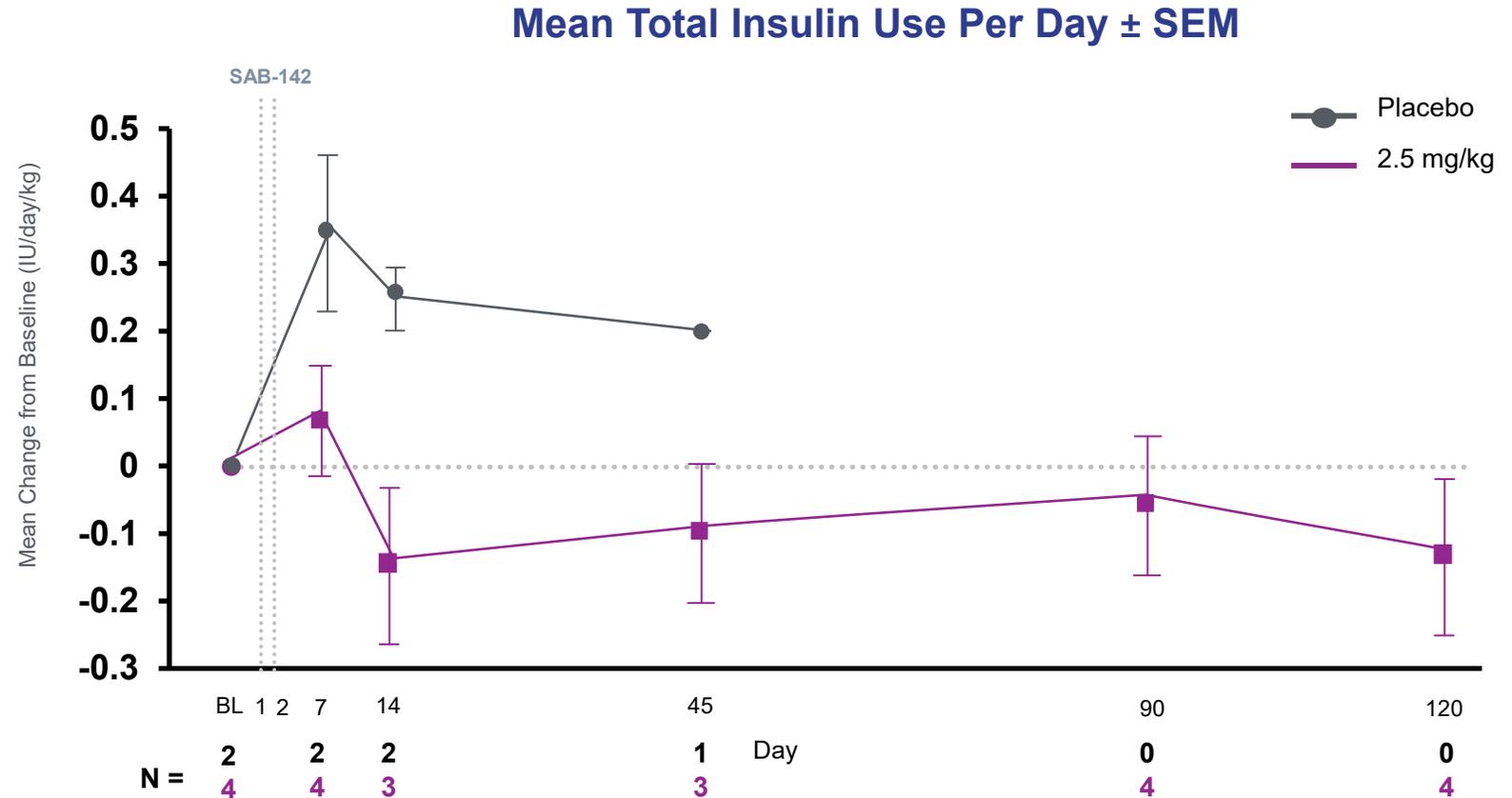
	SAB-142 2.5 mg/kg T1D (n=4)	Placebo T1D (n=2)*
<b>Age range (mean)</b>	19-40 (28.75)	19-34 (26.5)
<b>Sex</b>	2 Female & 2 Male	2 Female
<b>C-peptide AUC for 2-hr MMTT (nmol/L) / min</b>		
n	4	1*
Mean (SD)	0.302 (0.032)	0.432
<b>Fasting Glucose (mmol/L)</b>		
n	4	2
Mean (SD)	6.53 (1.773)	5.95 (1.344)
<b>Time in months from T1D diagnosis to randomization</b>		
n	4	2
Mean (SD)	40.2 (11.39)	28 (9.9)
<b>Average Total Insulin per day by Weight (IU/day/kg)</b>		
n	4	2
Mean (SD)	0.406 (0.287)	0.323 (0.225)
<b>GAD Autoantibodies Positive N (%)</b>	4 (100%)	0 (0%)
<b>IA-2 Autoantibodies Positive N (%)</b>	2 (50%)	2 (100%)
<b>ZNT8 Autoantibodies Positive N (%)</b>	3 (75%)	0 (0%)

\*One placebo subject discontinued after Day 45  
Note: SD = Standard deviation

# Adult Established T1D Cohort: Total Exogenous Insulin Use

Established T1D

- › Improvement in glycemic control was not associated with increases in exogenous insulin use
- › Phase 1 study participants are beyond their T1D “honeymoon” period



# SAB-142 Offers Distinct Advantages Over rATG

## SAB-142 / hATG

### Mechanism of Action:

- › **SAB-142 is a human alternative** to rATG (Thymoglobulin)
- › SAB-142's mechanism of action is comparable to the PD profile of rATG shown to correlate with C-peptide preservation
- › SAB-142, like rATG, modulates immune function resulting in sustained exhaustion in T cells likely involved in destroying pancreatic beta cells



SAFETY



EFFICACY



LYMPHO-DEPLETION

### Thymoglobulin<sup>®</sup> DISADVANTAGES



Majority of patients develop **grade 3 serum sickness**



**Inability to safely and reliably redose** due to serum sickness and antibodies



**Leads to lymphodepletion up to 2 years** which may increase risk in immunosuppression



Our Solution:



**No risk of serum sickness** due to fully human product. **Enables opportunity to safely redose**



Low/no immunogenicity due to fully human nature. **Enables opportunity to safely and reliably redose**



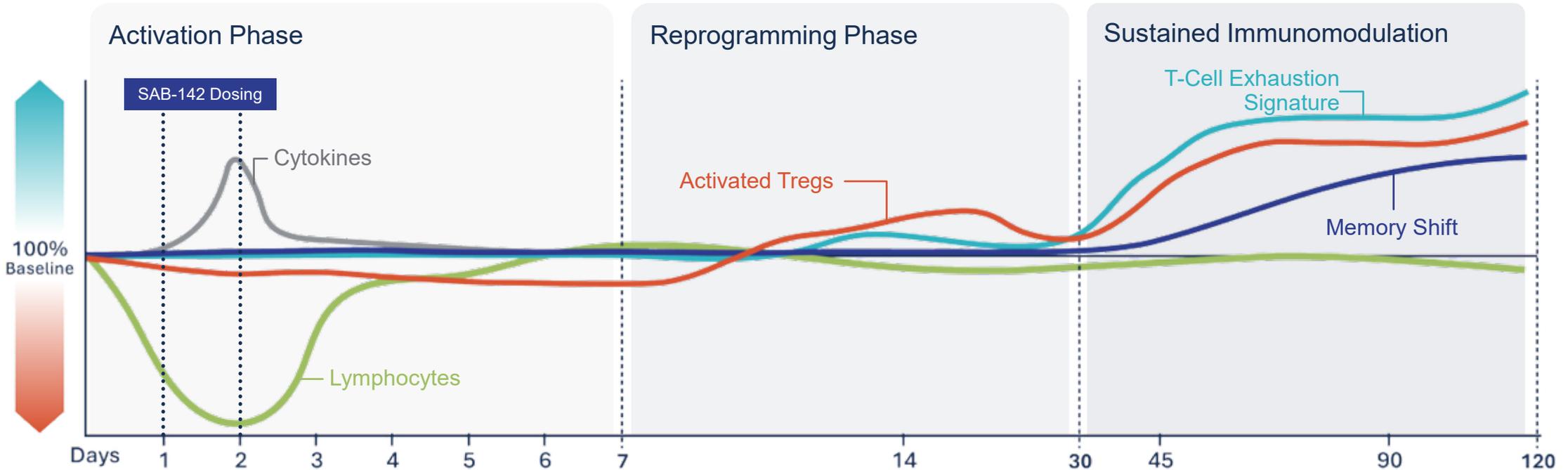
**Does not lead to lymphodepletion/ immunosuppression** based on Phase 1 data

# SAB-142: The Next Generation of Beta Cell Guardians

*Sustained Immunomodulation without Lymphodepletion*



## Pharmacodynamic Profile of SAB-142:



## Mechanism of Action of SAB-142:

- Transient Cytokine Increase
- Treg preservation & activation
- Sustained T-cell exhaustion signature
- Transient Lymphocyte Margination
- Initiation of memory phenotype shift
- Supporting restoration of immune tolerance

# Thymoglobulin's Therapeutic and Dosing Profile is Superior to Tzielid in Stage 3 T1D Patients\*

	 PHASE 3 (PROTECT) DATA	  PHASE 2 TN-19 and MELD DATA	 ADVANTAGES
C-peptide	Primary end point of C-peptide levels <b>met at Month 18</b>	Primary end point of C-peptide AUC <b>met at Month 12</b>	 <b>Statistically significant on C-peptide like Tzielid</b>
HbA1c	<b>Missed statistical significance</b>	<b>Statistically significant</b> at Month 12	 <b>Statistically significant on HbA1C where Tzielid missed</b>
Dosing	<b>Two courses of IV therapy:</b> Each course is 12 days of consecutive IV therapy administered at Randomization and at Month 6	<b>A single dose of IV</b> administered over 2 days	 <b>More Convenient Dosing</b>
Patient Population	Children and adolescents <b>8-17 years</b>	Adolescents and adults <b>12-45 years (TN-19)</b> <b>5-25 years (MELD-ATG)</b>	 <b>Shown to work in Broader range of patients</b>
Study Period	<b>18 months</b>	<b>12 months</b>	 <b>Required one course and less time to primary endpoint</b>
Sample Size n =	200 on Tzielid vs. 100 on placebo	29-34 on Thymoglobulin (per dose level) vs. 29-31 on placebo	 <b>Superior statistically significant efficacy with smaller sample size</b>