Targeting T1D

Modifying Disease

Moving Beyond Insulin

Corporate Presentation
Q1 2026

Forward-Looking Statements

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

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)

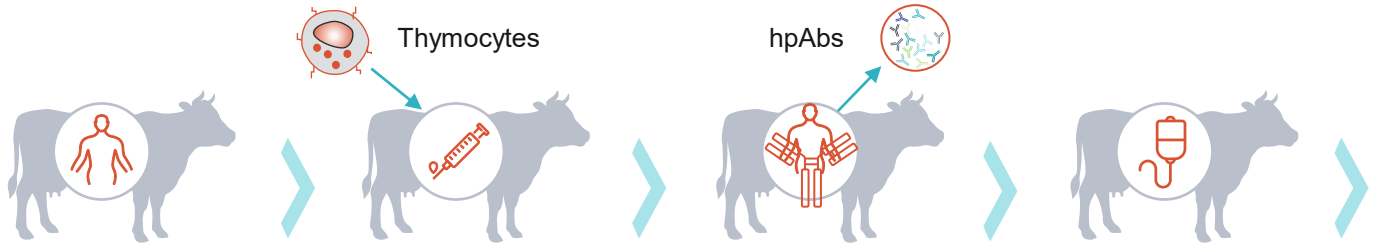
- **SAB-142 is a multi-specific, fully human anti-thymocyte globulin** (hATG) disease-modifying immunotherapy to delay the onset and progression of T1D
- SAB-142 works by directly **targeting multiple immune cells** involved in destroying pancreatic beta cells, including the modulation of “bad acting” 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



Tc Bovine

Transchromosomic (TC) bovine genetically designed to produce human antibodies

Hyperimmunization

Tc Bovine immunized with target disease antigen to generate antibodies

Human Antibody Production

Bovine produce fully human antibodies to disease target that circulate in the blood stream

Plasmapheresis

Tc Bovine™ are plasma donors, and antibodies are collected in the form of plasma

Downstream at SAB's cGMP Facility

Purification

Purification process isolates antibodies from plasma

Human Antibodies

Purified and formulated human multi-specific polyclonal antibodies

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

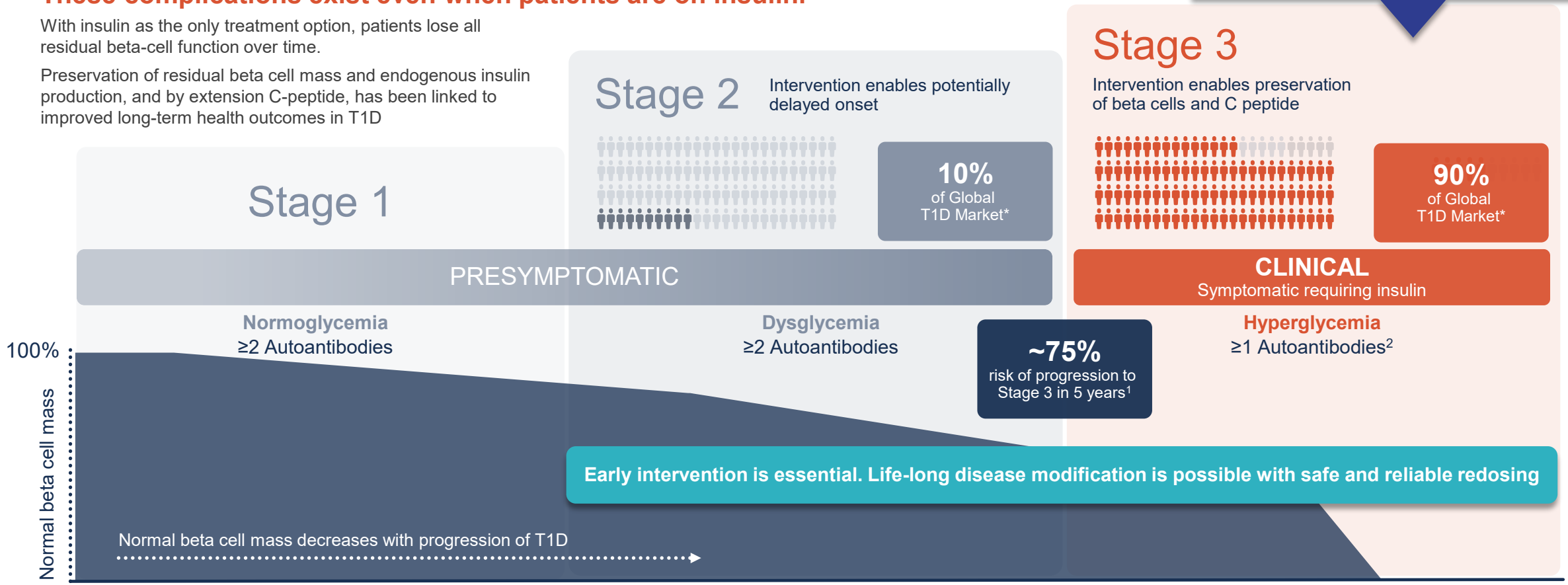
T1D is associated with serious long-term complications, including diabetic nephropathy, neuropathy, and retinopathy, as well as increased risk of cardiovascular disease, peripheral arterial disease, cerebrovascular events, and diabetic foot complications.

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

Significant Unmet Need in Type 1 Diabetes

An evolving treatment landscape with a regulatory pathway informed by Tzield

Stage 2 (Delay Onset) Market

Tzield™ (teplizumab-mzwv)

Approved in Stage 2 patients to delay the onset of T1D

sanofi

$2.9B Sanofi acquisition of Provention Bio

(Tzield in Phase 3 for Stage 3 at time of acquisition)

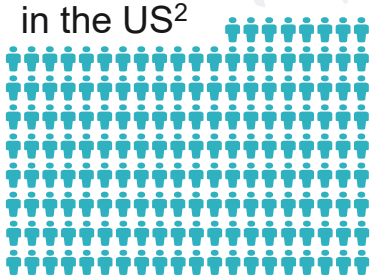
Stage 3 (Newly Diagnosed) Market

Tzield sBLA accepted with CNPV supported by PROTECT data

64,000 U.S. Patients Diagnosed Annually[1]

2.0M

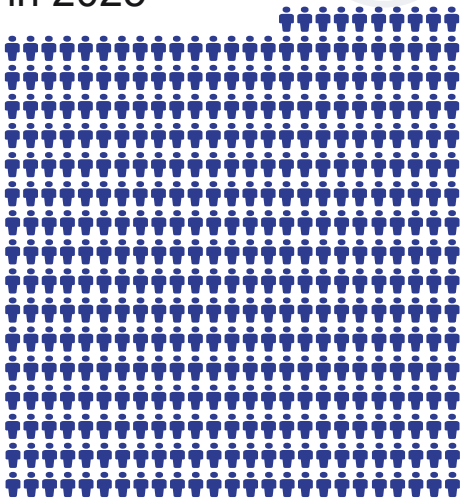
People with T1D cases in the US[2]



500,000 Patients WW Diagnosed Annually[2]

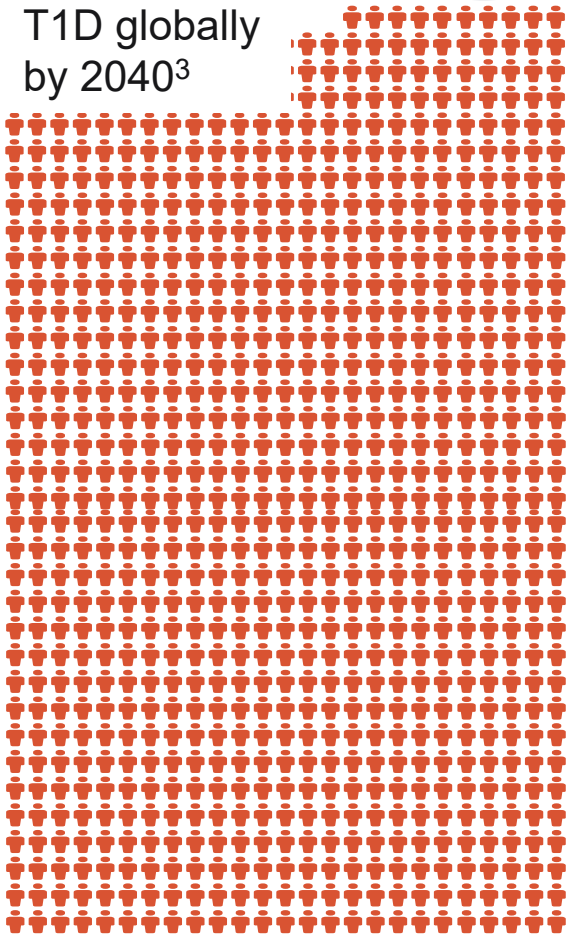
9.5M

People with T1D globally in 2025[3]



14.7M

People with T1D globally by 2040[3]



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

rATG has been tested across multiple studies in T1D patients

2013 — Thymoglobulin

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 — Thymoglobulin

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 — Thymoglobulin

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)

2013 — 2018 — 2025

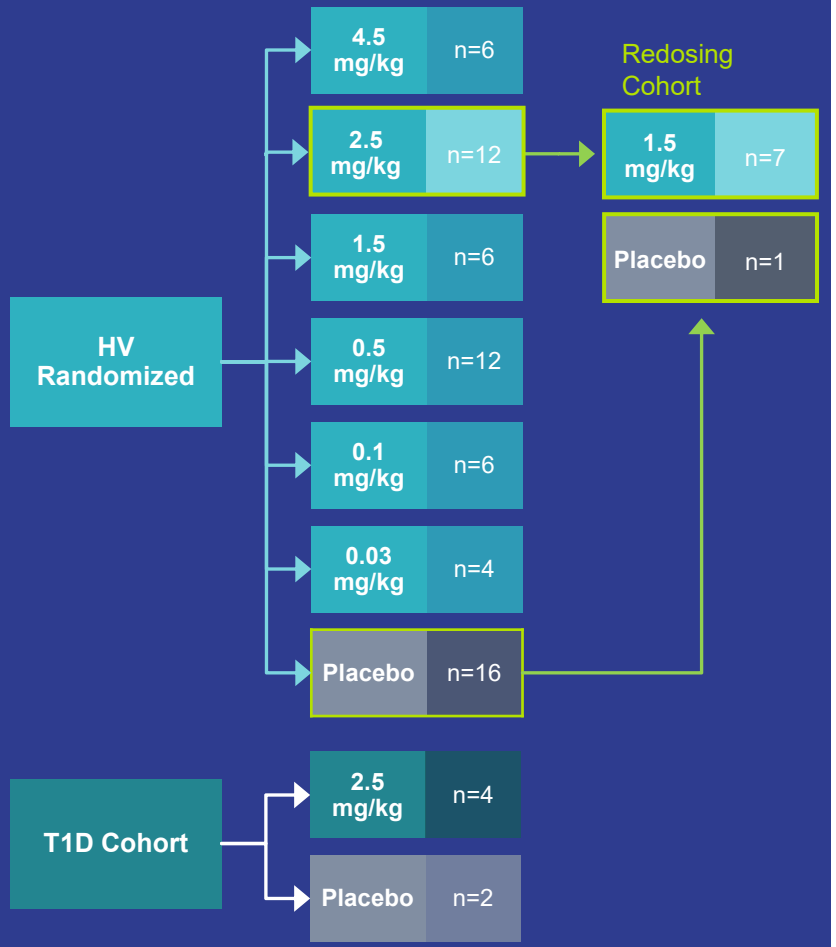
Leveraged clinical findings showing low-dose rATG (2.5 mg/kg) effectively mitigates disease

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

*Not measurable in healthy volunteers.

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

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

*HUman anti-thymocyte biologic in first-in-MAN clinical study

Adult Established T1D Cohort: Baseline Characteristics

Established T1D

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

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

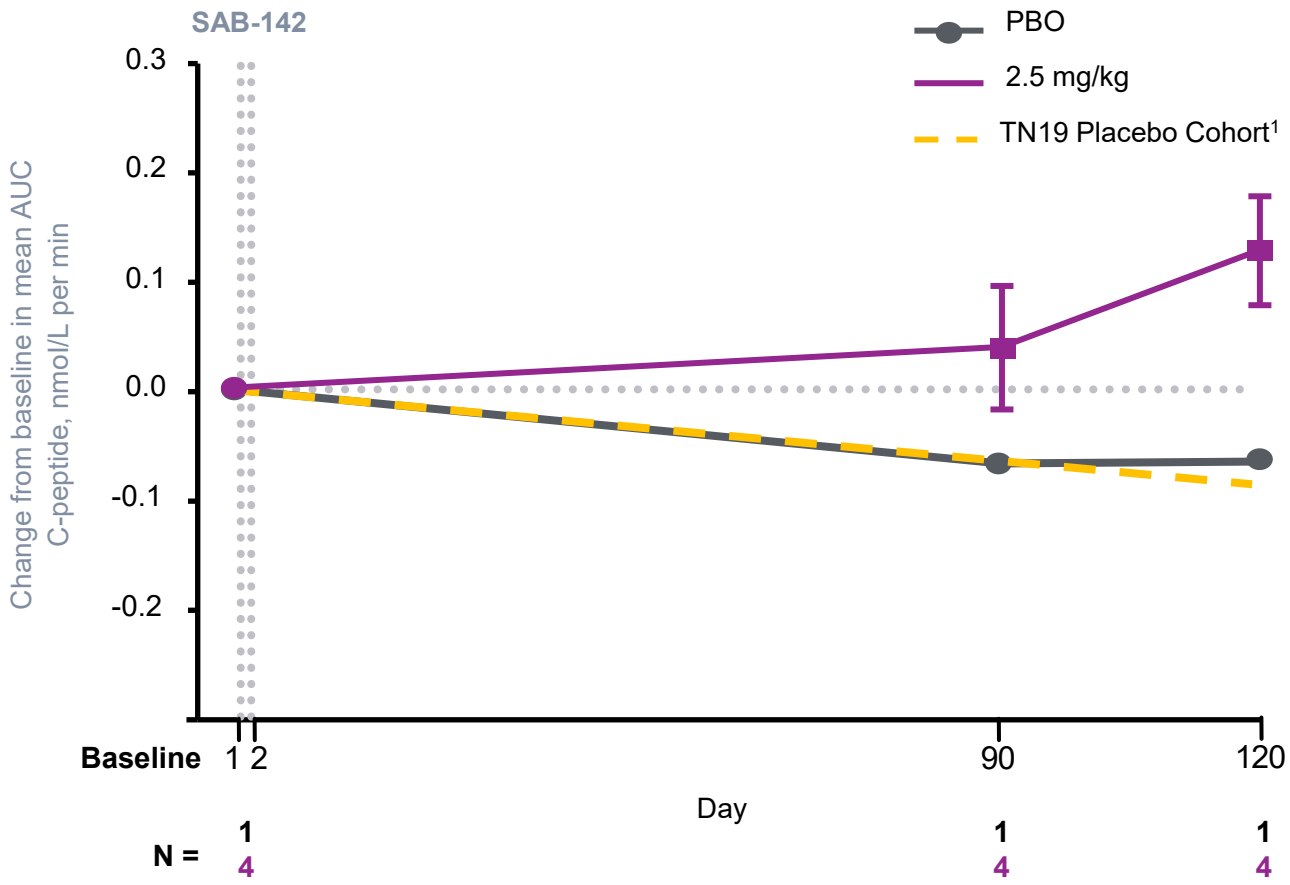
*One placebo subject discontinued after Day 45
Note: SD = Standard deviation

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[1]
- Supports a PD effect consistent with preservation of beta cell function in adults with established T1D

MMTT C-Peptide Mean AUC (nmol/L) / Min ± SEM



Note: [1] Predicted decline was estimated using placebo MMTT C-peptide AUC data from TN19 (n=31 at screening). Linear modeling of decline was based on Weeks 48, 72, and 96 (n=30, 26, and 26, respectively; post–2-hour values masked). A linear slope (–0.6108) was applied to estimate the rate of decline: 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.

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)

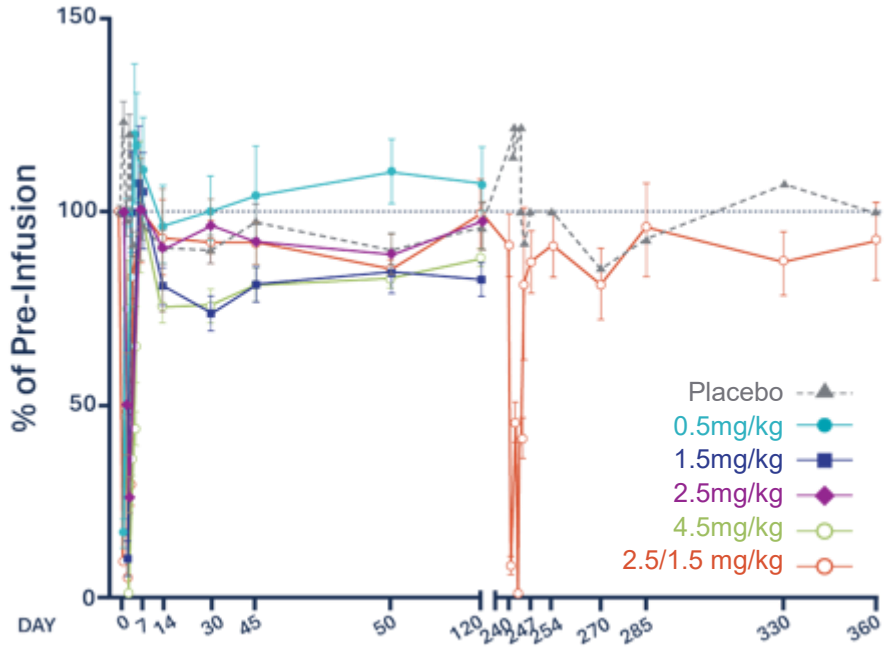
Transient On-Target PD Effects

- 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 ± 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

Note: AE = Adverse Events; SAE = Serious Adverse Events; CRS = Cytokine Release Syndrome; PD = Pharmacodynamics; RBCs = Red Blood Cells SEM = Standard Error of the Mean; SOI = Start of Infusion

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

	Tzield*** (teplizumab-mzwv)	Thymoglobulin Anti-thymocyte Globulin (Rabbit)	SAB BIO 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	**12 DAYS x 2 (Annual, not redosable)**	**1-2 DAYS (Not redosable)**	**2 DAYS x 2 (Annual, redosable)**
Infusing timing	**1 HOUR**	**4-12 HOURS**	**4-6 HOURS**
Redosable (with potential for chronic dosing without ADAs)	✗	✗	✓
Age range	**8-17**	**5-45**	**5-40***

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

SAFEGUARD Phase 2b Study

SAFety and Efficacy of human anti-thymocyte immunoGlobUlin SAB-142 ARresting progression of Type 1 Diabetes

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: ≥ 0.2 nmol/L

Dosing regimen:

- Intravenous (IV)
- 0.5 mg/kg on Day 1 and remainder of dose Day 2
- 1st dose at study start and 2nd 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

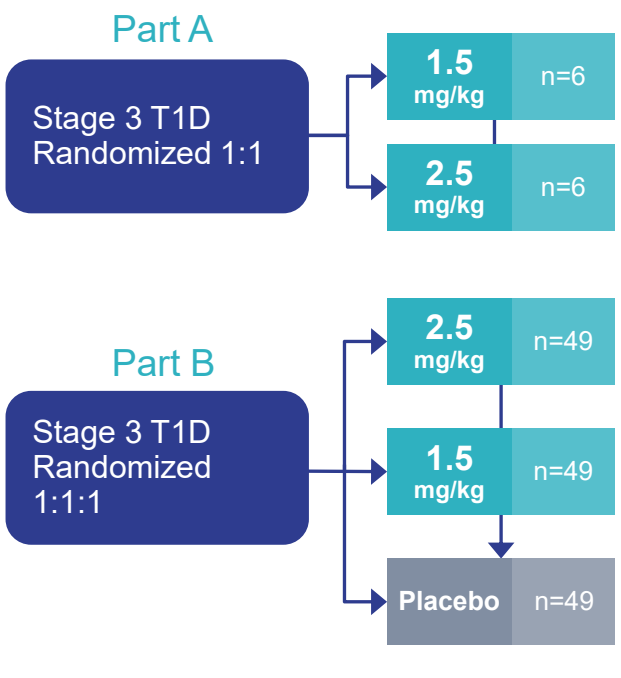
Europe (EMA)

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 Financing Fully Funds Phase 2b SAFEGUARD Study:

Raised $175 million in July 2025 with the potential for an additional $284 million if milestone-based warrants are exercised in full

T1D Clinical Development Partner:

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

Financial Snapshot:

Cash*
$144M

Cash Runway*
Through 2028

Key Clinical and Strategic Partners:

AUSTRALASIAN TYPE 1 DIABETES IMMUNOTHERAPY COLLABORATIVE

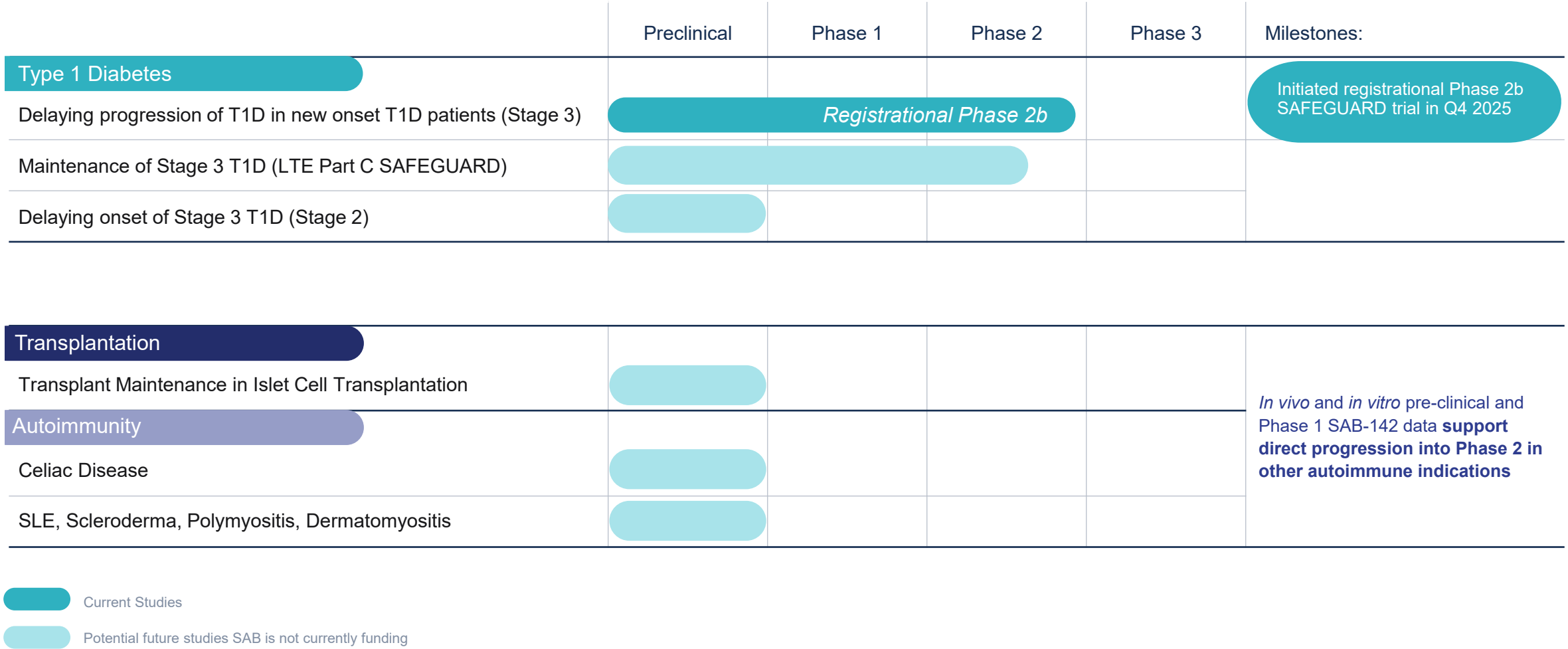
Breakthrough T1D™ Formerly JDRF

INNODIA

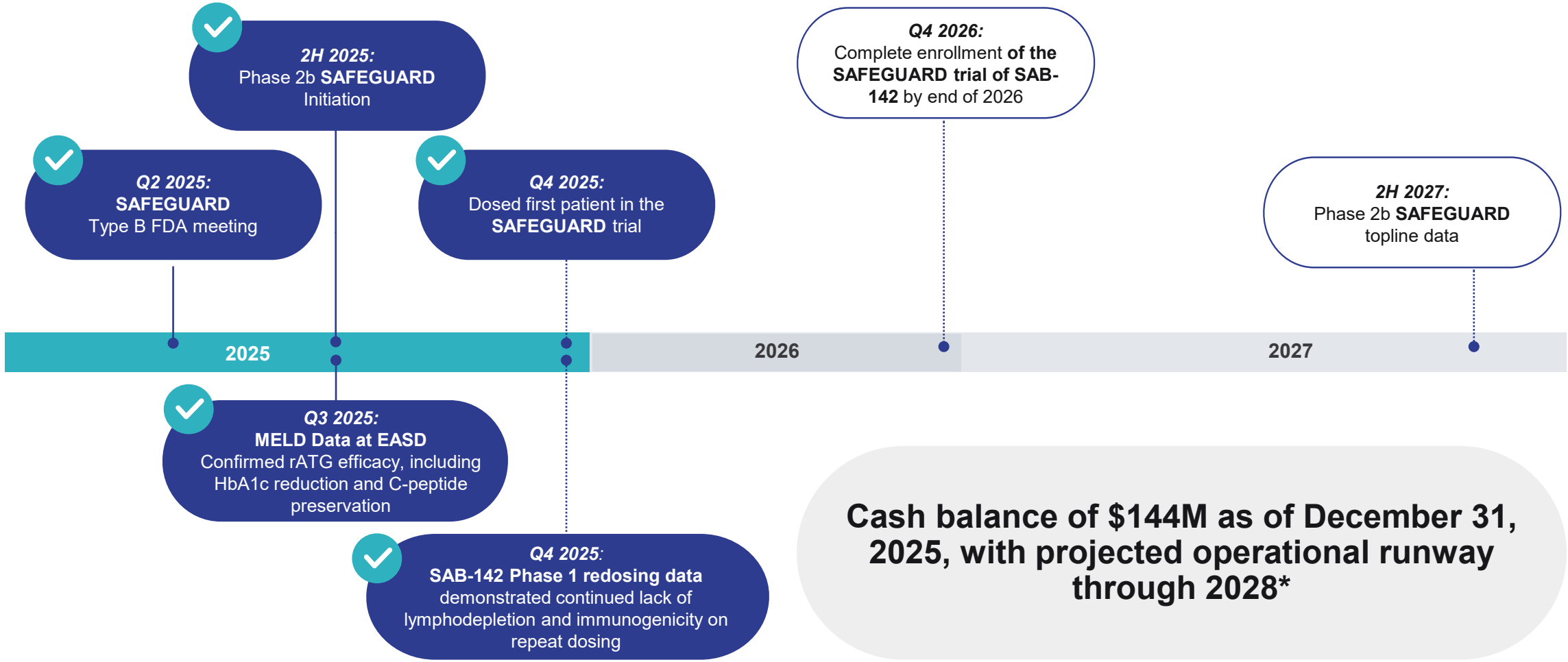
sanofi

*Note: Cash, cash equivalents, and available for sale securities as of December 31, 2025.

Advancing a Pipeline in Autoimmune Diseases Led by SAB-142



Strong 2025 Execution with Significant Catalysts Expected in 2026-2027



Cash balance of $144M as of December 31, 2025, with projected operational runway through 2028*

*Note: Cash, cash equivalents, and available for sale securities as of December 31, 2025.

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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

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Backed by a syndicate of life science specialist investors

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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

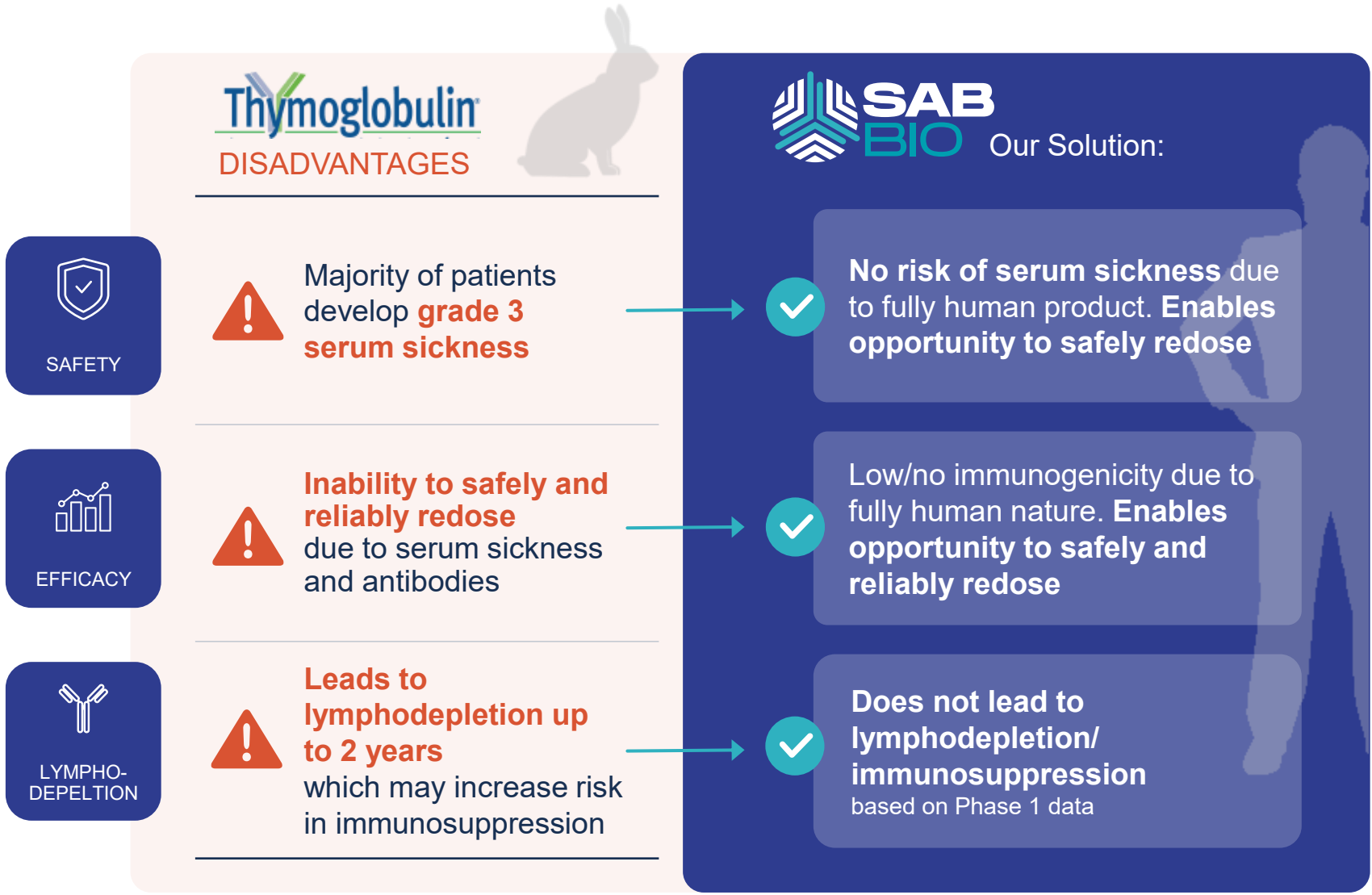
SAB BIO

Appendix

SAB-142 Offers Distinct Advantages Over rATG

SAB-142 / hATG Mechanism of Action:

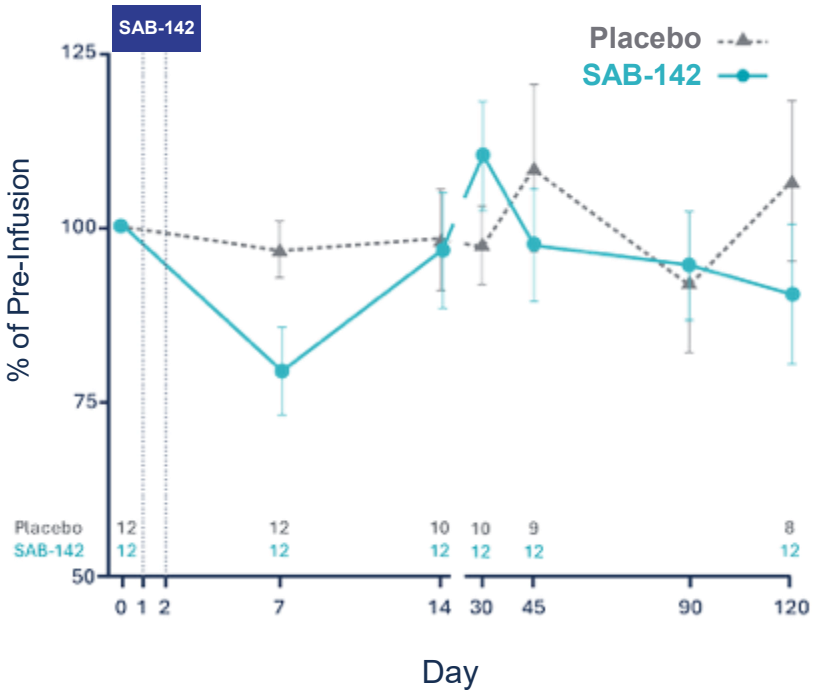
- **SAB-142 is a human alternative** to rATG (Thymoglobulin)
- SAB-142's mechanism of action is comparable to the PD profile of rATG shown to correlate with C-peptide preservation
- SAB-142, like rATG, modulates immune function resulting in sustained exhaustion in T cells likely involved in destroying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



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

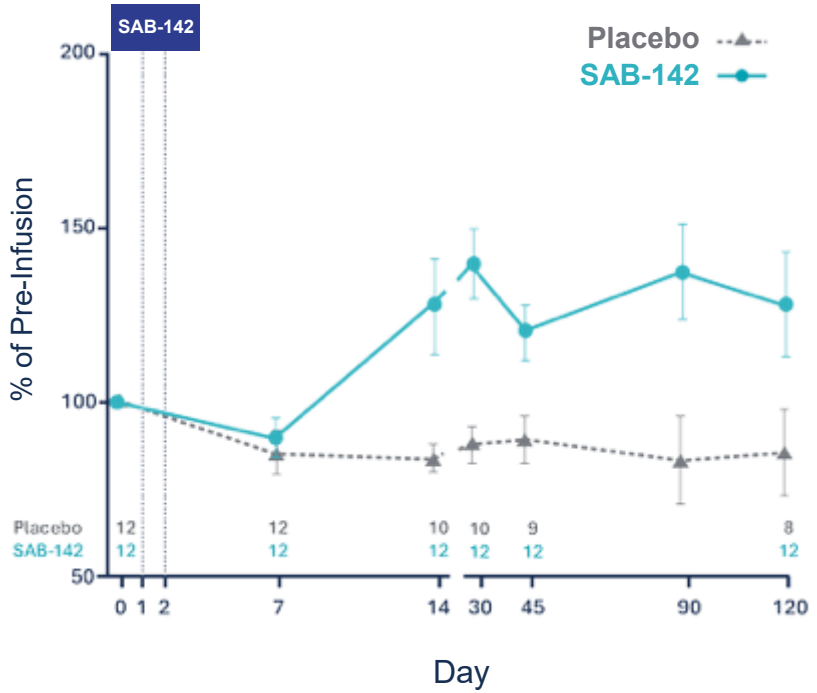
1 Treg preservation

Treg ($CD3^+$ $CD4^+$ $CD127^{lo}$ $CD25^+$ $FoxP3^+$) **± SEM**



2 Increase in CD4 exhaustion

Relative PD-1$^+$ Tconv Cells ± SEM



rATG Published Data:

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

JCI insight CLINICAL MEDICINE

Responders to low-dose ATG induce CD4$^+$ T cell exhaustion in type 1 diabetes

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

[1]Department of Pediatrics, College of Medicine, University of Florida, Gainesville, Florida, USA. [2]Department of Pathology, Immunology, and Laboratory Medicine, University of Florida Diabetes Institute, Gainesville, Florida, USA. [3]Benaroya Research Institute at Virginia Mason, Seattle, Washington, USA. [4]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$^+$FOXP3$^+$ 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$^+$ exhaustion phenotype (increased PD-1$^+$KLRG1$^+$CD57$^-$ on CD4$^+$ T cells [$P = 0.011$] and PD1$^+$CD4$^+$ 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$^+$ exhaustion transcript and cellular phenotyping profiles may be useful for identifying signatures of clinical response to ATG in T1D.

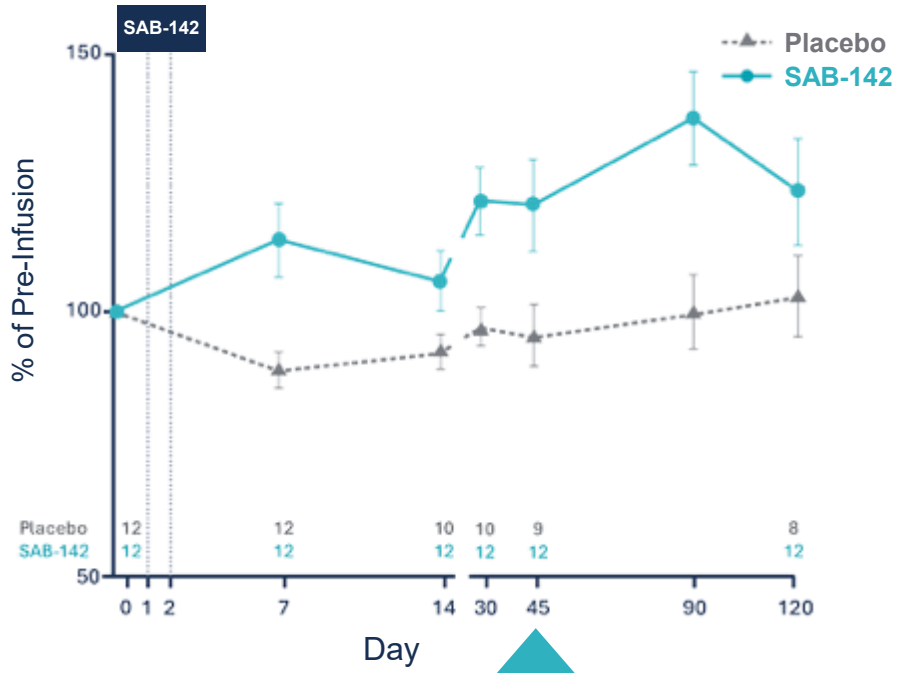
Note: SAB-142: combined 1.5mg/kg and 2.5mg/kg dosed cohorts.

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

SAB-142 Phase 1 Top-Line Data:

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

Relative TIGIT⁺ Tconv Cells ± 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

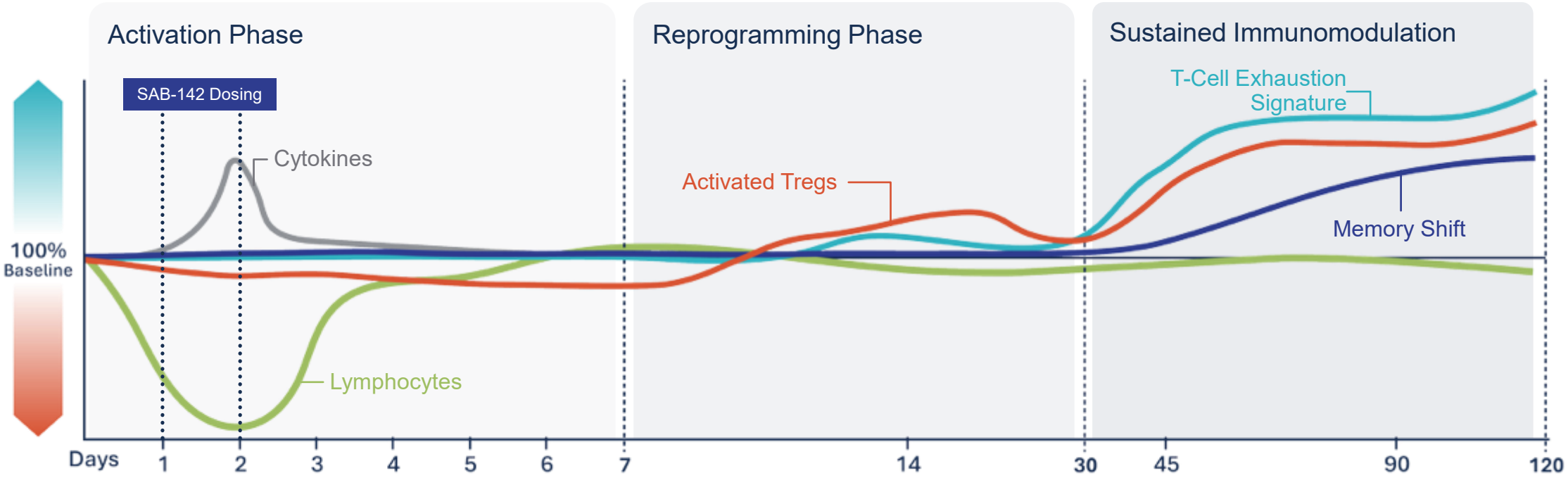
SAB-142 induced persistent expression of co-inhibitory receptors on CD4+ Tconv cells

Note: SAB-142: combined 1.5mg/kg and 2.5mg/kg dosed cohorts.

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

Rabbit ATG: De-Risked Mechanism of Action

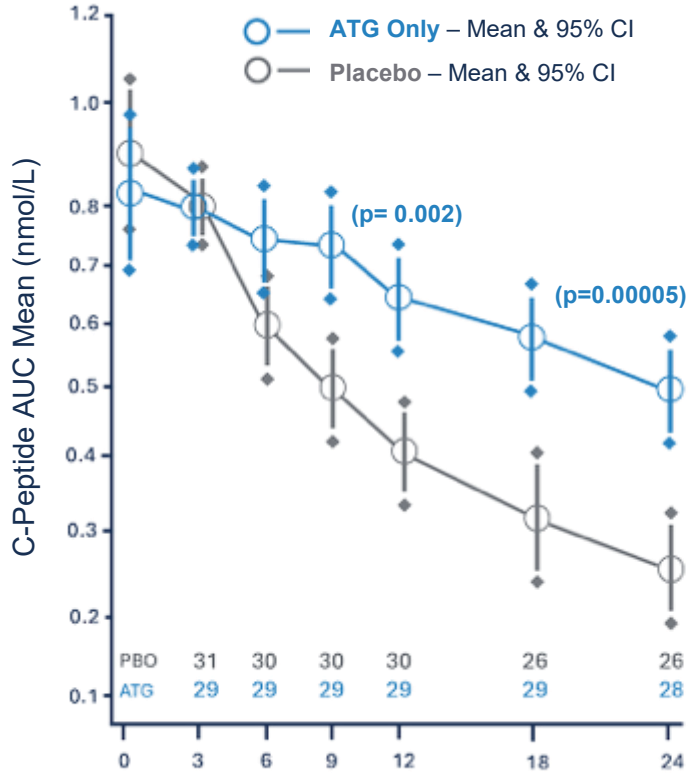
TN-19

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

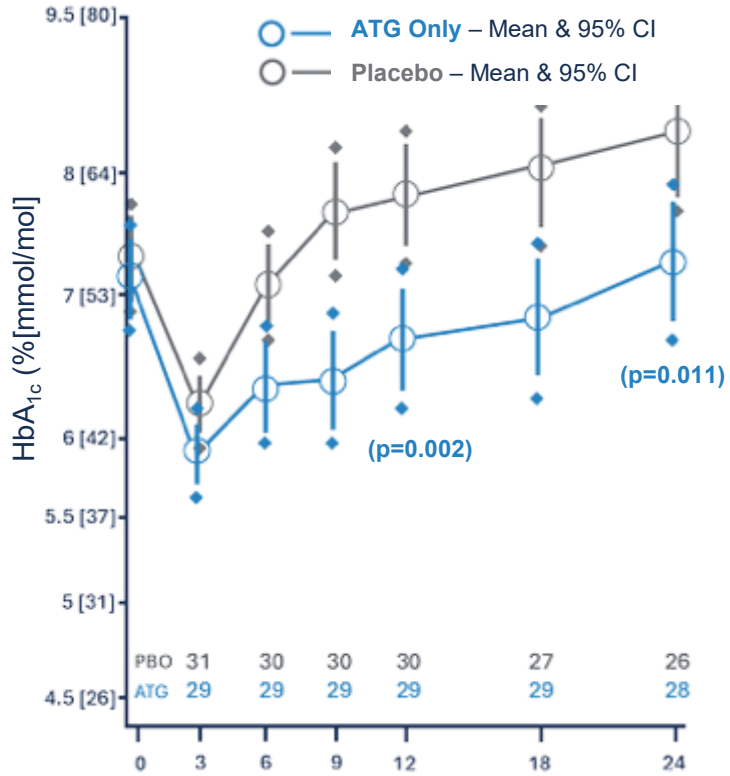
C-peptide, the cleavage product of proinsulin, released in equal amounts with insulin, reflects a person's ability to produce endogenous insulin and is the standard biomarker of pancreatic β-cell function

Thymoglobulin® Anti-thymocyte Globulin (Rabbit)

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



HbA_{1c} 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_{1c}, 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.

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 ≤2.5 mg/kg with statistically significant C-peptide preservation and glycemic control

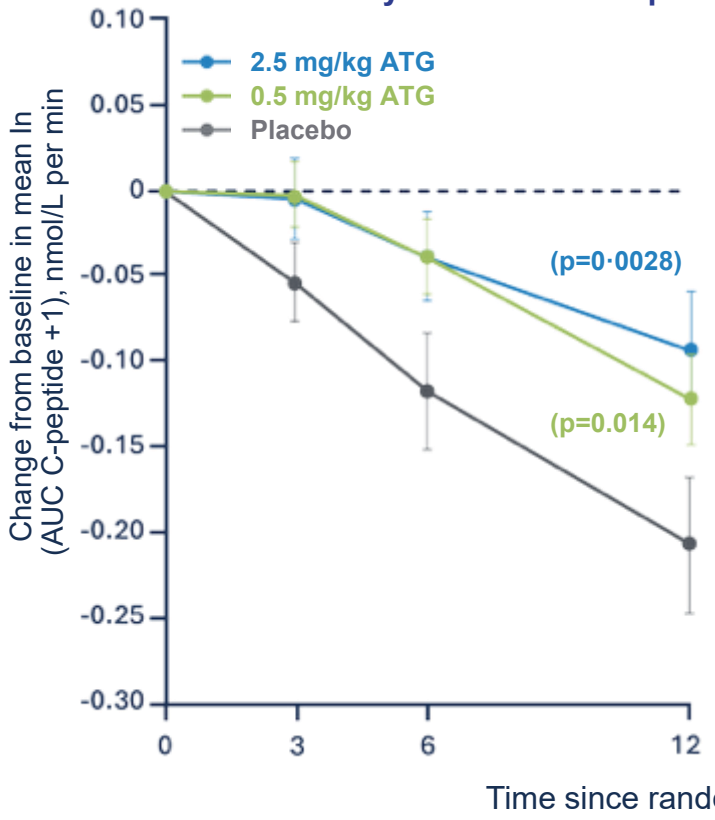
*Mathieu C, Wych J, Hendriks AEJ, Van Ryckeghem L, Tree T, Chmura P, Möller C, Casteels K, Danne T, Reschke F, Šmigoc Schweiger D, Battelino T, Johannesen J, Rami-Merhar B, Pieber T, De Block C, Evans M, Hilbrands R, Bosi E, Willemsen RH, Basu S, Pulkkinen MA, Knip M, Cnop M, Nitsche A, Schulte AM, Niemöller 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.

Thymoglobulin®
Anti-thymocyte Globulin (Rabbit)

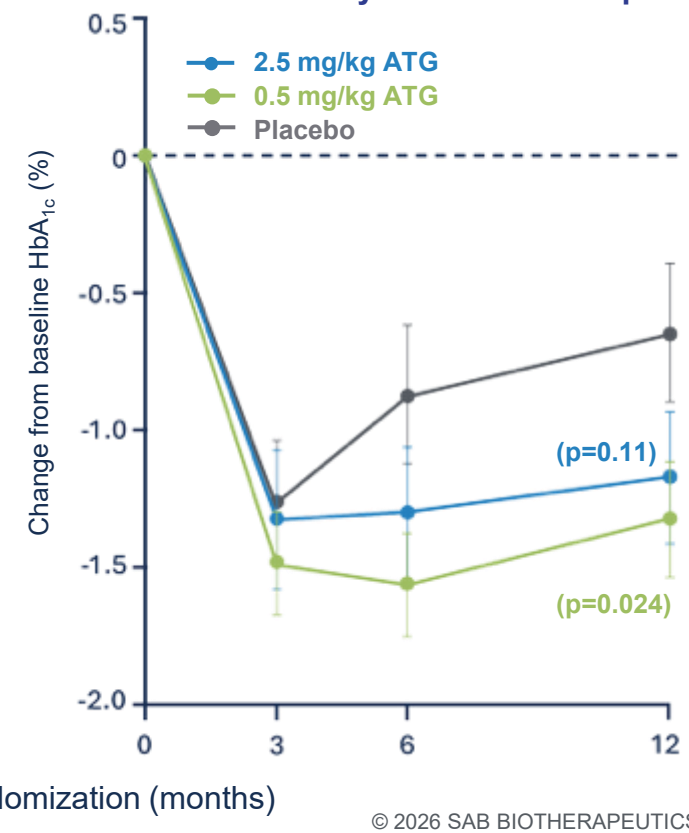
ATG is the only mechanism of action that has consistently reproduced clinical data demonstrating preservation of C-peptide and improvements in glycemic control

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

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



HbA1c Adjusted Mean Difference by Treatment Group



Time since randomization (months)

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

	Tzield PHASE 3 (PROTECT) DATA	Thymoglobulin PHASE 2 TN-19 and MELD DATA
C-peptide	Primary end point of C-peptide levels **met at Month 18**	Primary end point of C-peptide AUC **met at Month 12**
HbA1c	**Missed statistical significance**	**Statistically significant** at Month 12
Dosing	**Two courses of IV therapy:** **Each course is** 12 days of consecutive IV therapy administered at Randomization and at Month 6	**A single dose of IV** administered over 2 days
Patient Population	Children and adolescents **8-17 years**	Adolescents and adults **12-45 years (TN-19)** **5-25 years (MELD-ATG)**
Study Period	**18 months**	**12 months**
Sample Size n =	200 on Tzield vs. 100 on placebo	29-34 on Thymoglobulin (per dose level) vs. 29-31 on placebo

Thymoglobulin ADVANTAGES

- **Statistically significant on C-peptide** like Tzield
- **Statistically significant on HbA1C** where Tzield missed
- **More Convenient Dosing**
- Shown to work in **Broader range of patients**
- **Required one course and less time** to primary endpoint
- **Superior statistically significant efficacy** with smaller sample size

* Not head-to-head studies.