



FORTE BIOSCIENCES, INC

CAUTIONARY NOTE REGARDING FORWARD-LOOKING STATEMENTS

Certain statements contained in this presentation regarding matters that are not historical facts, are forward-looking statements within the meaning of Section 21E of the Securities and Exchange Act of 1934, as amended, and the Private Securities Litigation Act of 1995, known as the PSLRA. These include statements regarding management's intention, plans, beliefs, expectations or forecasts for the future, and, therefore, you are cautioned not to place undue reliance on them. No forward-looking statement can be guaranteed, and actual results may differ materially from those projected. Forte Biosciences, Inc. ("we", the "Company" or "Forte") undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise, except to the extent required by law. We use words such as "anticipates," "believes," "plans," "expects," "projects," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions to identify these forward-looking statements that are intended to be covered by the safe-harbor provisions of the PSLRA.

Such forward-looking statements are based on our expectations and involve risks and uncertainties; consequently, actual results may differ materially from those expressed or implied in the statements due to a number of factors, including, but not limited to, risks relating to the business and prospects of the Company; Forte's plans to develop and potentially commercialize its product candidates, including FB102; the risk that results from preclinical studies and early-clinical trials completed by Forte and third parties may not be predictive of results from later-stage clinical trials; the timing of initiation of Forte's planned clinical trials, including Forte's planned Phase 2 celiac study and other future Phase 2 studies; the timing of the availability of data from Forte's clinical trials, including Forte's planned Phase 2 celiac study and Phase 1b vitiligo study; the timing of any planned investigational new drug application or new drug application; Forte's plans to research, develop and commercialize its current and future product candidates; Forte's projections of the size of the market in certain indications for FB102; the clinical utility, potential benefits and market acceptance of Forte's product candidates; Forte's commercialization, marketing and manufacturing capabilities and strategy; developments and projections relating to Forte's competitors and its industry; the impact of government laws and regulations; Forte's ability to protect its intellectual property position; Forte's estimates regarding future revenue, expenses, capital requirements and need for additional financing; and the impact of global events on the Company, the Company's industry or the economy generally.

We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs, and these statements represent our views as of the date of this presentation. We may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified. Information regarding certain risks, uncertainties and assumptions may be found in our filings with the Securities and Exchange Commission, including under the caption "Risk Factors" and elsewhere in our Quarterly Report on Form 10-Q for the period ending March 31, 2025, and other filings with the Securities and Exchange Commission. New risk factors emerge from time to time and it is not possible for our management team to predict all risk factors or assess the impact of all factors on the business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. While we may elect to update these forward-looking statements at some point in the future, we specifically disclaim any obligation to do so. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation.

Mission

Advance precision immunotherapies that target key drivers of autoimmune disease, delivering transformative outcomes for patients and long-term value for shareholders

Strategy

Leverage robust preclinical evidence and proprietary clinical study results to develop disease modifying therapies for multiple indications driven by IL-2/IL-15 axis dependent immune pathways

Vision

Lead in the development of a next generation biologic that inhibits CD122, addressing significant unmet needs in autoimmune and inflammatory diseases

Investment Highlights



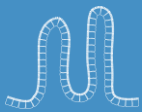
CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and certain subsets of T cells and is a subunit of the high affinity IL-2 receptor expressed on Tregs



FB102 (Forte's anti-CD122 antibody) is designed to mediate both IL-2 and IL-15 induced proliferation and activation of pathogenic NK and T cells while sparing beneficial Tregs



Potential "pipeline in a product"



Positive Phase 1b clinical data in celiac disease (CED). Phase 2 trial initiated 2H2025 with topline data expected 2026



Phase 1b trial in vitiligo enrolling with topline results expected 1H2026



Phase 1b trial in alopecia areata (AA) enrolling with topline results expected 2026



Experienced, focused team determined to make a difference

Leadership

Management

Paul Wagner, Ph.D.
Chief Executive Officer



Tony Riley
Chief Financial Officer



Chris Roenfeldt, PMP
Chief Operating Officer



Barbara Finck, MD
Senior Medical Clinician



Board of Directors

Scott Brun, MD
abbvie



Barbera Finck, MD
Coherus, PDL BioPharma, immunex

Steve Doberstein, PhD
NEKTAR

Steve Kornfeld
FRANKLIN-Templeton INVESTMENTS, CASTLE PEAK HOLDINGS



Rich Vincent
Deloitte.

Why Target CD122?

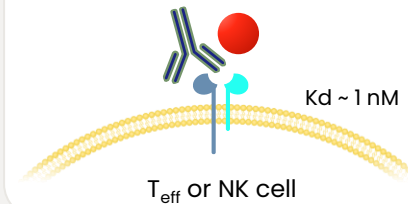
CD122 Biology

- Subunit of the intermediate affinity IL-2/IL-15 receptor expressed on pathogenic T cell subsets and NK cells
- Subunit of the high affinity IL-2 receptor critical for regulatory T cells (Tregs)

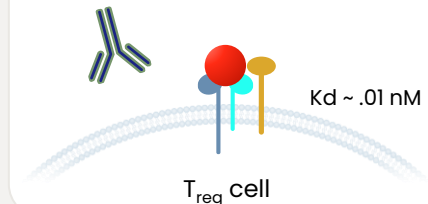
FB102: Forte's anti-CD122 antibody

- Blocks IL-2 and IL-15 driven proliferation and activation of pathogenic T cells and NK cells
- Preserves IL-2 signaling in Tregs, maintaining immune regulation and limiting toxicity
- Designed for a differentiated therapeutic profile across autoimmune and inflammatory diseases

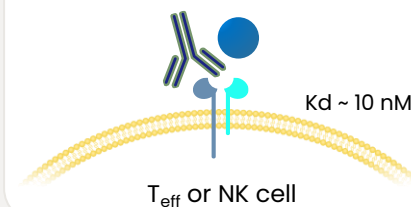
FB102 blocks medium affinity IL-2 binding to CD122/CD132



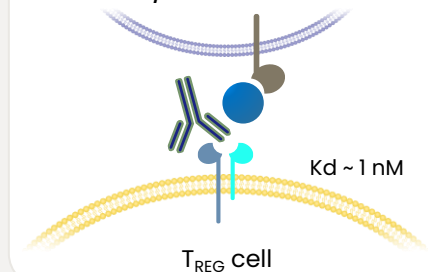
IL-2 high affinity binding to CD25 and CD122/CD132 unaffected by FB102



FB102 blocks low affinity IL-15 binding to CD122/CD132



FB102 blocks medium affinity IL-15/IL15Rα binding to CD122/CD132



FB102



IL-2



IL-15



CD122 = IL-2R β



CD132 = IL-2R γ



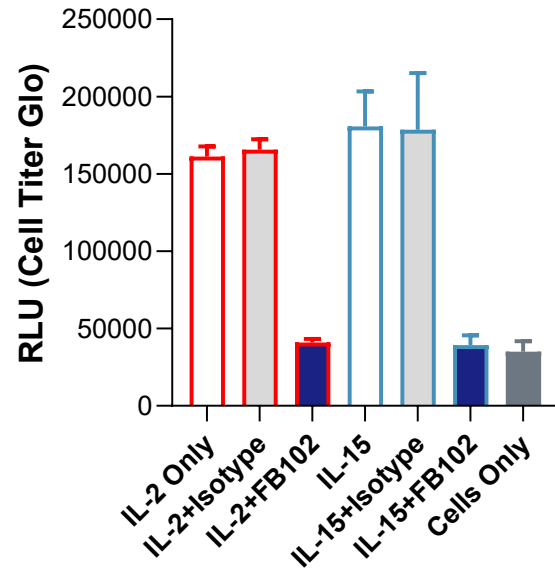
CD25 = IL-2R α



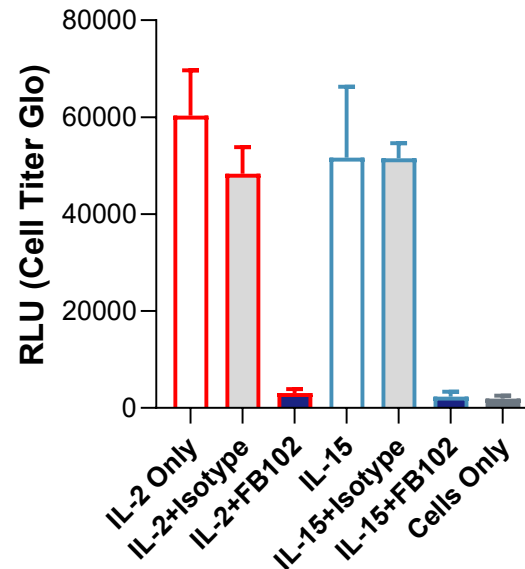
CD215 = IL-15R α

FB102 Selectively Shuts Down IL-2/IL-15 Driven T cell and NK Cell Proliferation While Sparing Regulatory T cells

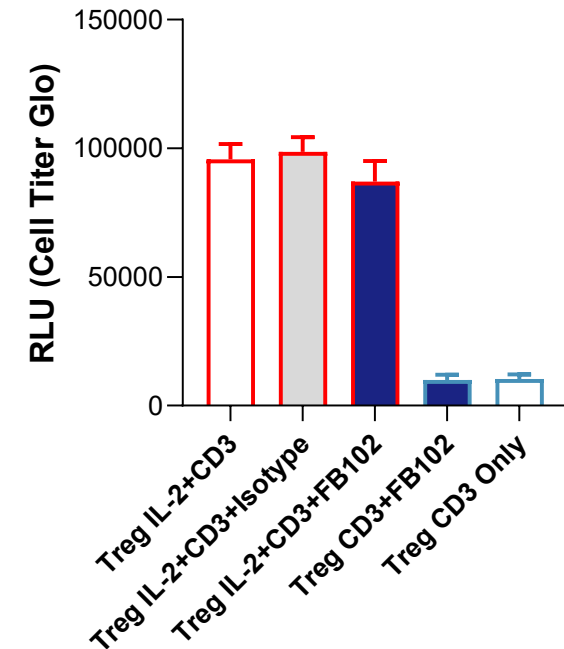
T cell proliferation assay
100 nM FB102 or isotype control



NK cell proliferation assay
100 nM FB102 or isotype control

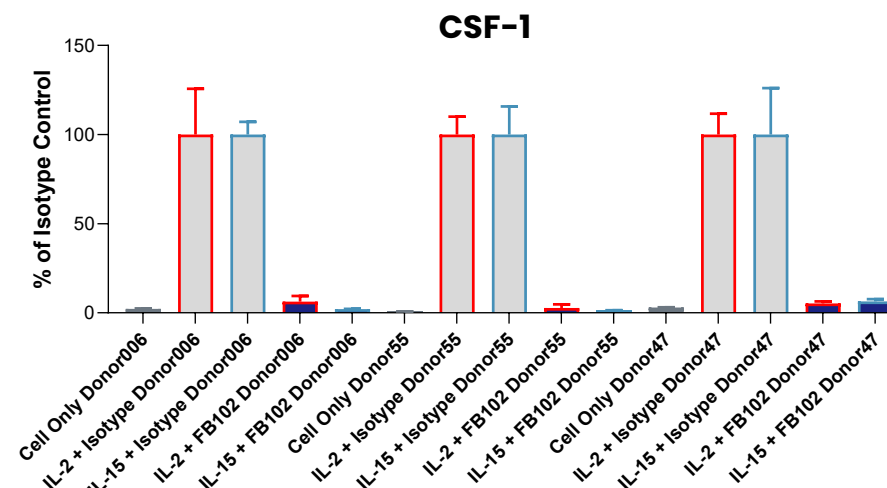
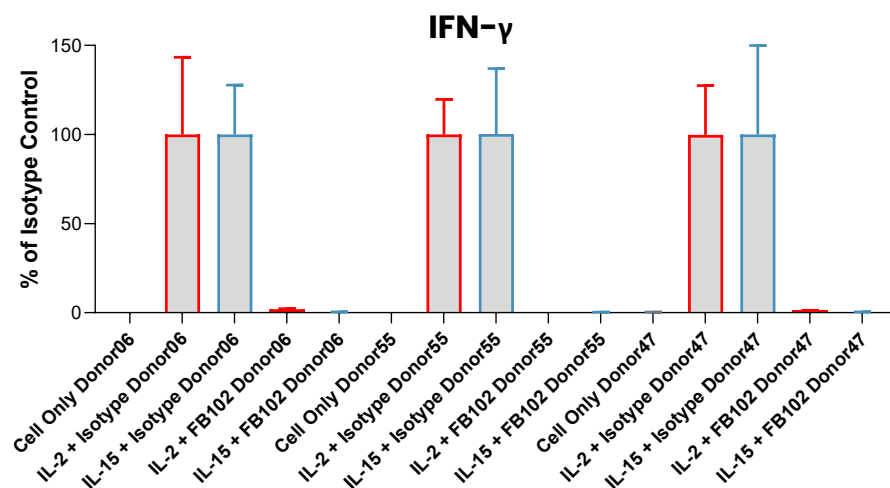
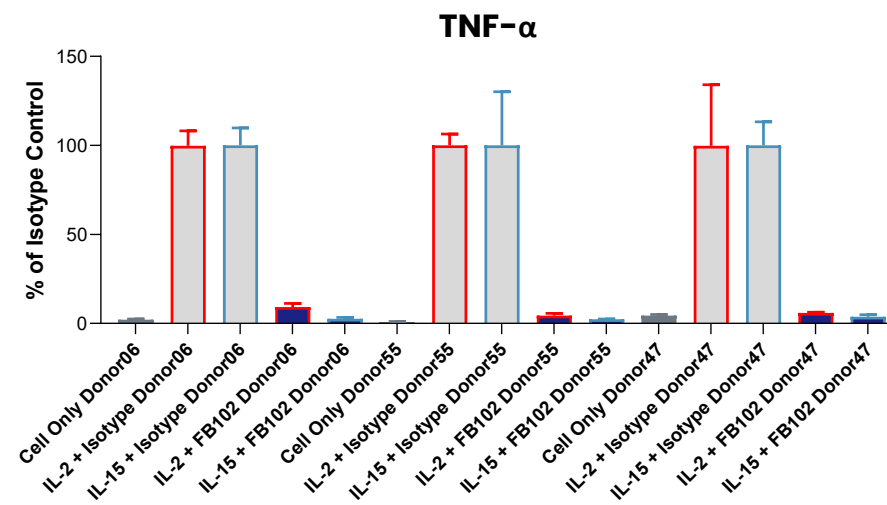
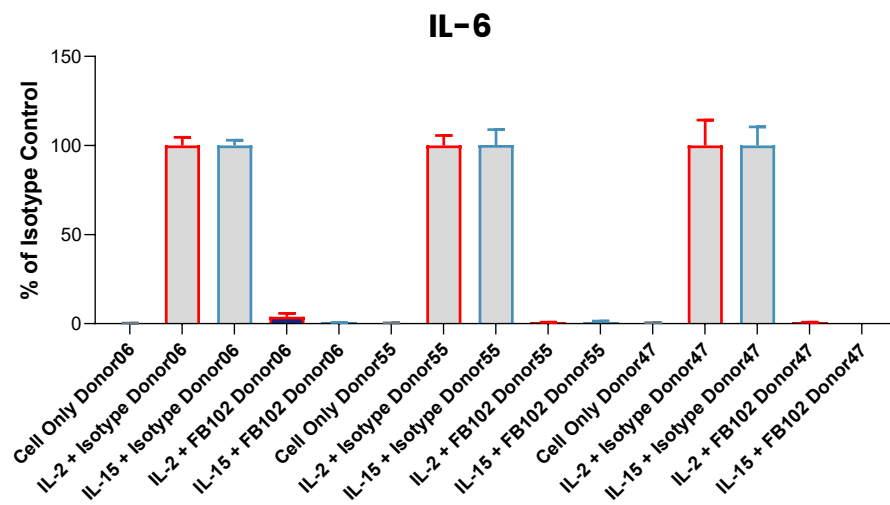


T regulatory cells in proliferation assay with CD3 and IL2 +/- FB102



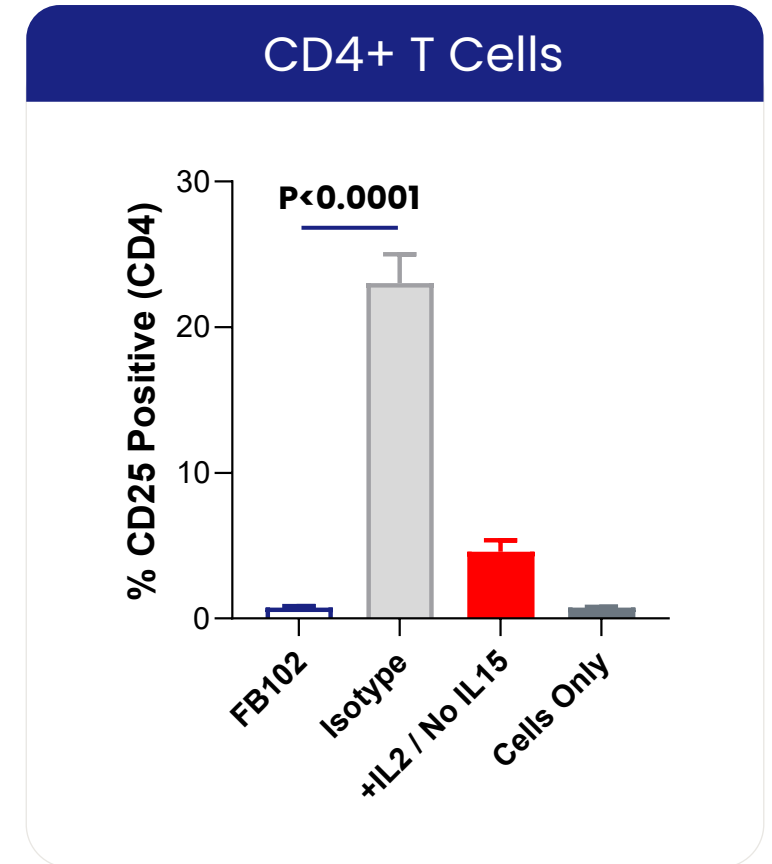
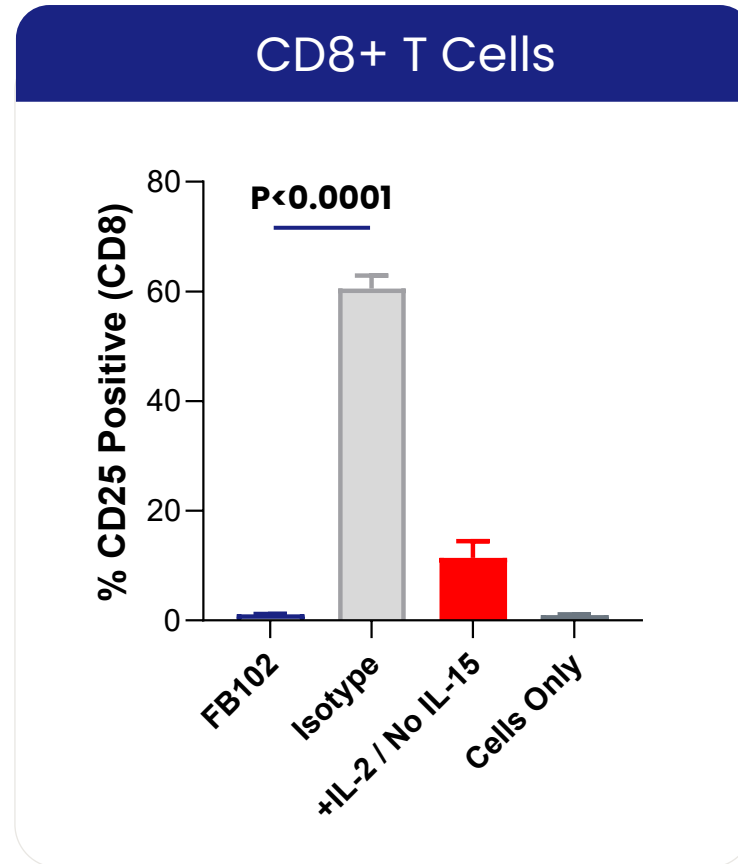
FB102 inhibits T cell and NK cell IL-2/IL-15 driven proliferation, while not inhibiting Treg proliferation

FB102 Inhibits Anti-inflammatory Cytokines Induced by IL-2 and IL-15



FB102 Inhibits IL-2/IL-15 CD4+ and CD8+ T Cell Activation in an In Vitro Disease Model

CD4+ and CD8+ T cells were treated with IL-2 for 24 hours then with IL-15 for 3 days, simulating disease activity in the presence or absence of FB102



FB102 inhibits T cell activation

FB102 Proposed 12 Month Clinical Development

Program	Development	Phase 1	Phase 2	Phase 3	Upcoming Milestones
Celiac Disease					Phase 1b Topline data readout June 2025 Phase 2 Initiated 2H25 Phase 2 Topline data expected in 2026
Vitiligo					Phase 1b Initiated 1H25 Topline data expected in 1H26
Alopecia Areata					Phase 1b initiated in 2H25 Topline data expected in 2026
Type 1 Diabetes					Phase 1b in development

FB102 “Pipeline in a Product” Preclinical Validation

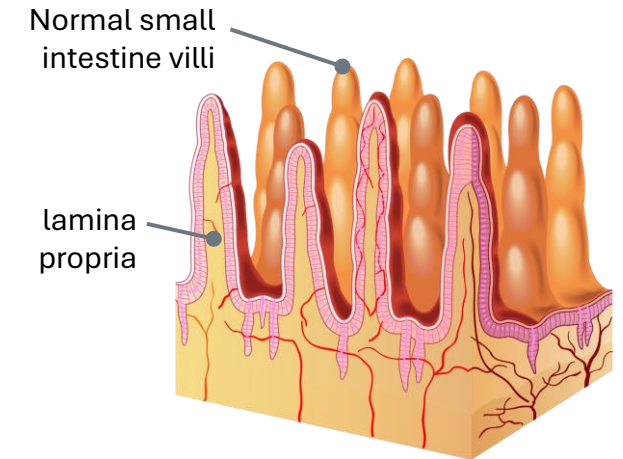
Disease	Species	Outcome	Reference
Celiac disease	Mouse	Improved IL-15-induced mucosal damage	PNAS, 2009
Vitiligo	Mouse	Enhanced repigmentation	Sci Transl Med, 2018
Alopecia areata	Mouse	Prevented fur loss	Nature Med, 2014
Type 1 diabetes	Mouse	Delayed disease onset	JCI Insight, 2018

Yokoyama et al PNAS; 2009 (106) 15849-15854
 Richmond et al Sci Transl Med. 2018 July 18; 10(450)
 Xing et al Nat Med 2014 Sep; 20 (9): 1043-9
 Yuan et al JCI Insight. 2018;3(2):e96600

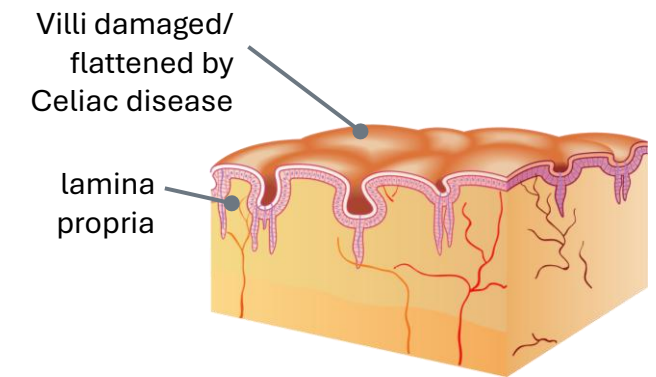
Celiac Disease: Large, Underserved Market Opportunity

Disease Overview	- Autoimmune disorder triggered by gluten ingestion - Causes immune-mediated small intestinal damage
Epidemiology	- Prevalence of ~1% of global population including 2.5M US Bimodal onset: - Children: 6–8 years median age - Adults: 40–50 years median age Female: Male = 2.5:1 (likely underdiagnosed in males)
Symptoms	GI: Diarrhea, nausea, abdominal pain Systemic: Fatigue, anemia, headaches Skin: Dermatitis herpetiformis (itchy rash)
Consequences of Untreated CD	- Malnutrition, osteoporosis, infertility - Increased risk of lymphoma and other autoimmune diseases
Unmet Need	No approved drug therapies Gluten-free diet (GFD) is the only treatment 10–30% fail to respond to GFD: ~50% due to inadvertent gluten exposure (150–400 mg/day) <10 mg/day considered safe threshold <5% have true refractory disease

Lining of the small intestine



Normal villi

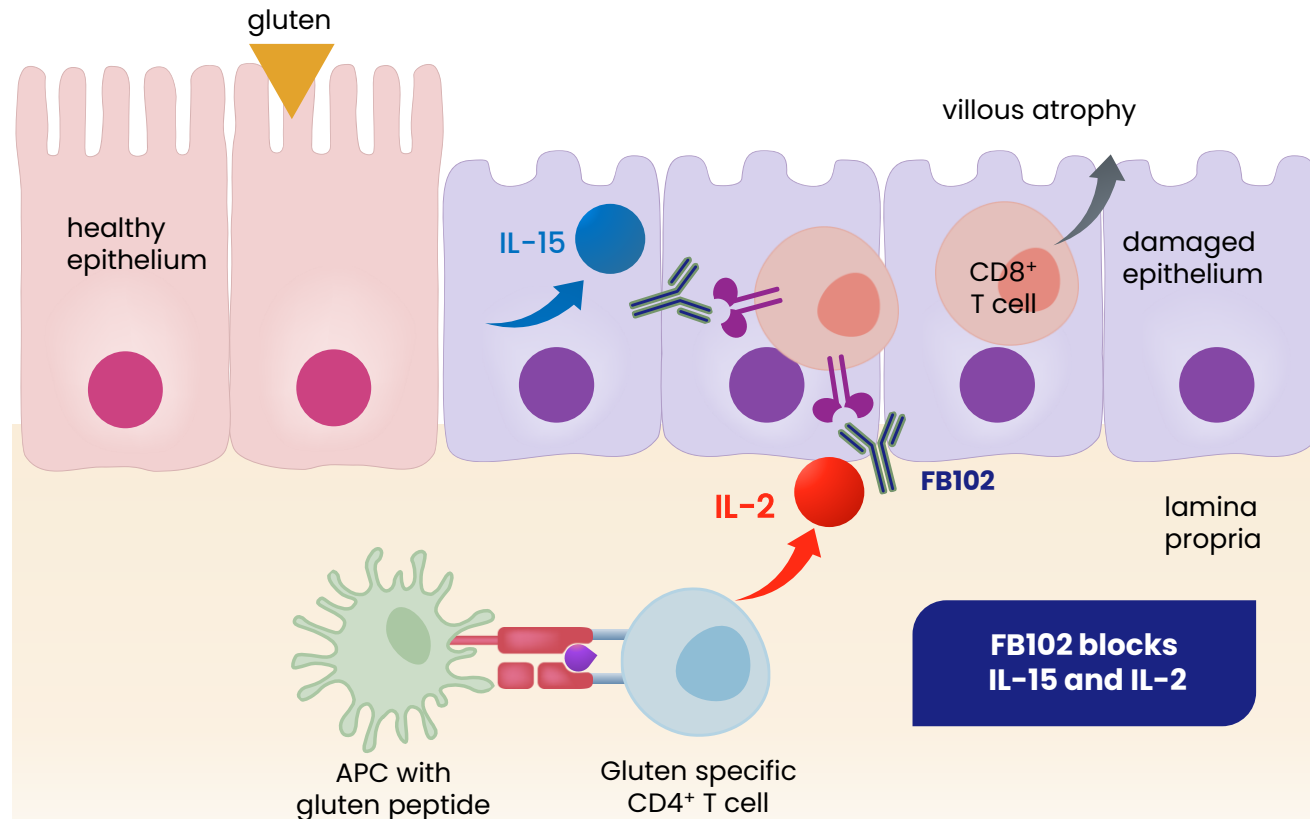


Celiac disease

FB102 Blocks Gluten-induced Intestinal Damage by inhibiting IL-2 and IL-15 Proliferating and Activation of T Cells and NK Cells

IL-2

- Clear genetic basis for involvement in CeD
- Rapid serum peak within ~4h after gluten exposure
- Correlates with symptom severity
- Drives early activation/expansion of IELs (mainly CD8⁺ T cells)
- Promotes Th1-type cytokines (e.g., IFN- γ)



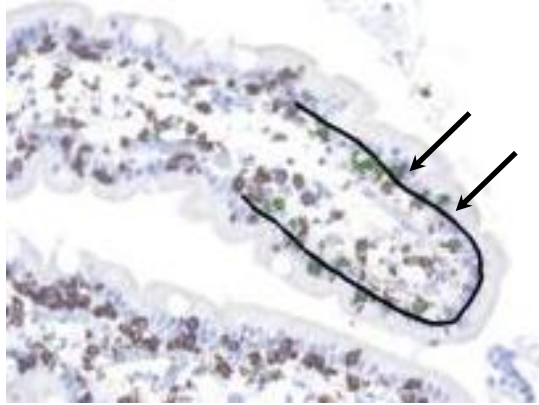
IL-15

- Clear genetic basis for involvement in CeD
- Overexpressed in gut epithelium and immune cells upon gluten exposure
- Tissue levels correlate with degree of mucosal injury
- IL-15R α overexpressed on IELs in CeD patients
- Stimulates IEL proliferation, IFN- γ and TNF- α release
- Activates pathogenic CD8⁺ T cells
- Impairs Treg function and disrupts TGF- β -mediated mucosal protection

FB102 blocks both IL-2 and IL-15, Providing Potential Advantages Over Other Investigational Drugs in Celiac Disease

Gluten Modification	Immunotolerance	Gut Healing	Immunomodulators	
			Single targeting	Multi targeting
IMGX-003 	CNP-101/TAK-101 	IMU-856 	IL-15 → CALY-002 	CD122 (IL-2/IL-15)→FB102 FORTE BIOSCIENCES, INC
TAK-062 	TMP502 		IL 15 → Ordesekimab 	IL15/IL-21→EQ-102 
E40 	KAN-101 		IL 15 → TEV-53408 	CD122 (IL-2/IL-15)→ANB033 
	TAK227/ZED1227 		OX40L amlitelimab 	
	DONQ52 			

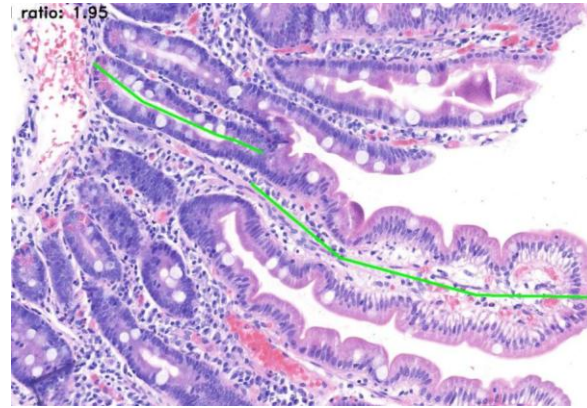
Key Morphologic and Inflammatory Measurements in Celiac Disease



In Celiac disease, IELs infiltrate the villus epithelium upon gluten exposure, driving villus atrophy

IELs are measured as the density of CD3+ T cells per 100 enterocytes

CeD patients typically have 20–30 IELs/100 enterocytes at baseline, which increases by >30%+ with gluten challenge^{1,2}



Villus height to crypt depth (Vh:Cd) ratio

Measures morphological damage due to IEL infiltration after gluten challenge in celiac disease

Vh:Cd ratio in patients with CeD is ~2.0–3.0 on Gluten Free Diet³

$$VCIEL = \left[\frac{Vh:Cd - \langle Vh:Cd \rangle}{\sigma_{Vh:Cd}} - \frac{IEL - \langle IEL \rangle}{\sigma_{IEL}} \right]^4$$

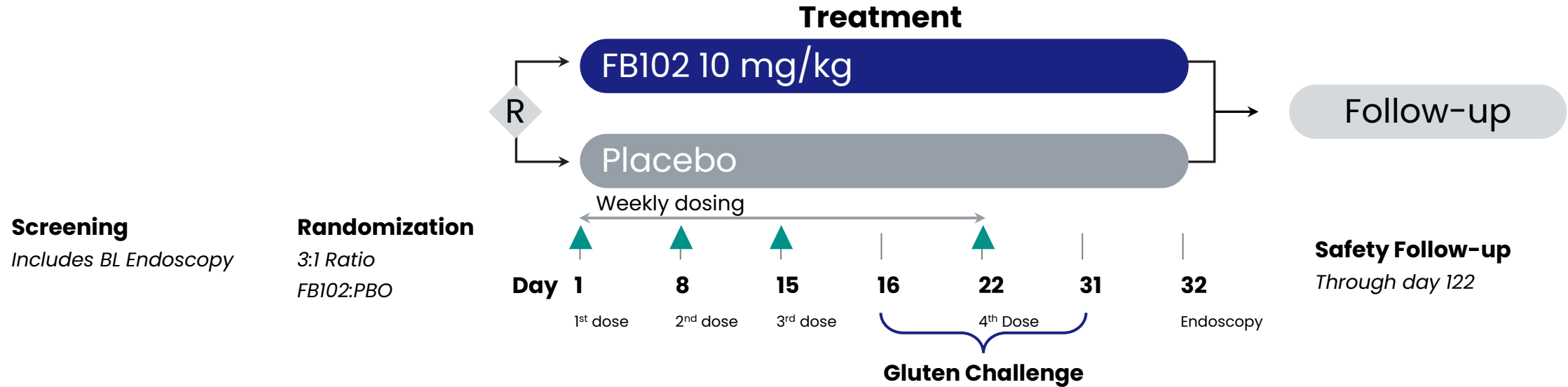
Proposed VCIEL composite scale comprising individual subject values for Vh:Cd and IEL equally weighted

Appears to offer better accuracy and statistical precision

Potentially a broader measure of mucosal health

1. Taavela J, PLoS One. PMID: 24146832; PMCID: PMC3795762.
 2. Rostami K Gut. 2017 Dec; PMID: 28893865; PMCID: PMC5749338.
 3. Adelman DC, Am J Gastroenterol. 2018 Mar;2018 Feb 20. PMID: 29460921
 4. Syage J, et al. Clin Gastroenterol Hepatol. 2024; 22: 1238–1244

FB102 Phase 1b Study in Celiac Disease



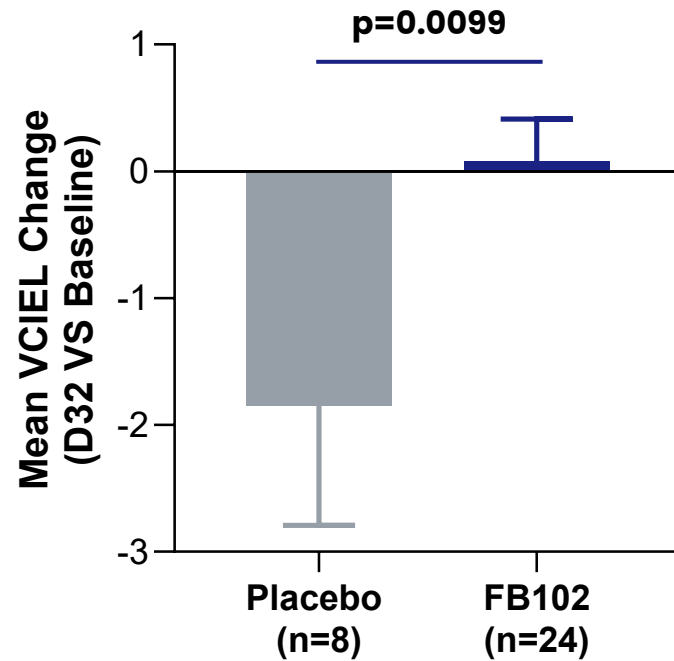
- 32 subjects, 9 sites
- Progressive Gluten Challenge (GC) with 2 g on D16, 4 g on D17 and 8g on Ds 18-31
- 3 of 4 weekly doses prior to 16-day gluten challenge followed by 4th dose on D22
- Safety and tolerability primary endpoint with endoscopy/biopsy at baseline and at end of gluten challenge (D32)
- Central review of histology endpoints VCIEL, IELs and Vh:Cd change from baseline
- Gluten challenge symptoms collected in patient diaries/AE reporting
- All subjects completed day 32 biopsy

Baseline Demographics

Parameter		Placebo N=8	FB102 N=24	Overall N=32
Age (Years)	Mean	38.3	40.8	40.1
Sex [n (%)]	Female	5 (62.5%)	19 (79.2%)	24 (75.0%)
	Male	3 (37.5%)	5 (20.8%)	8 (25.0%)
Ethnicity [n (%)]	Hispanic or Latino	0	0	0
	Not Hispanic or Latino	7 (87.5%)	23 (95.8%)	30 (93.8%)
	Not Reported	1 (12.5%)	1 (4.2%)	2 (6.3%)
	Unknown	0	0	0
Body Mass Index (kg/m ²) at Screening	Mean	25.61	24.8	25
Baseline Villus height to Crypt depth ratio	Mean	2.756	2.818	
	Standard error of mean	0.1398	0.1099	
Baseline CD3 positive IELs per 100 enterocyte	Mean	25.6	23.5	
	Standard error of mean	3.83	1.68	

FB102 Demonstrates Statistically Significant Composite Histology (VCIEL) Benefit Compared to Placebo

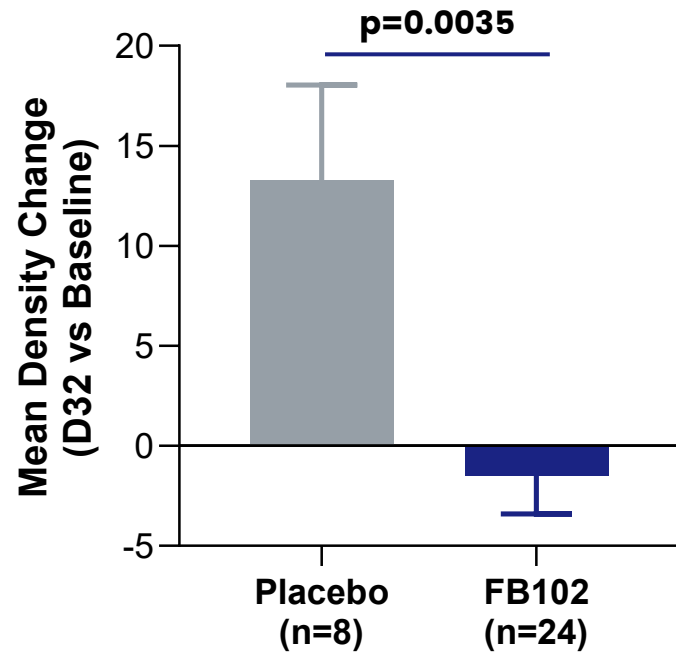
Change in VCIEL Composite Histology Score



Day 32 vs baseline VCIEL composite score - 1.849 for PBO compared to 0.079 for FB102 treated subjects

FB102 Demonstrates Statistically Significant Change in IEL Density Compared to Placebo

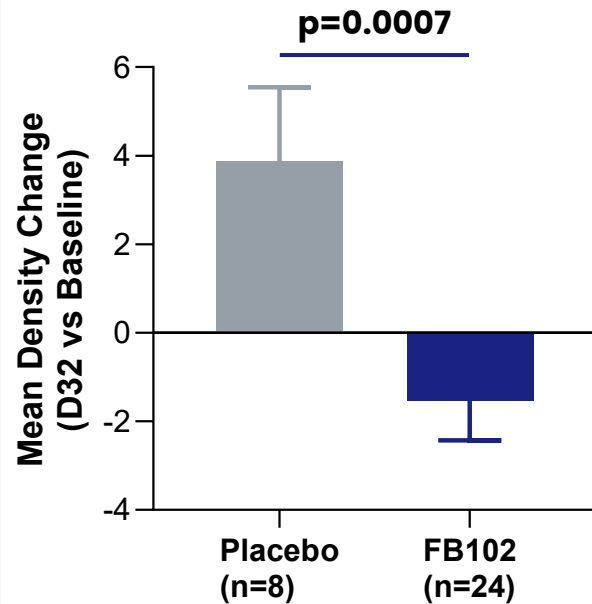
Change in IEL Density (Per 100 Enterocytes)



Day 32 vs baseline mean IEL density increase of 13.3 for PBO compared to a decrease of 1.5 for FB102 treated subjects

FB102 Demonstrates Statistically Significant Improvement in IEL Markers of Inflammation Compared to Placebo

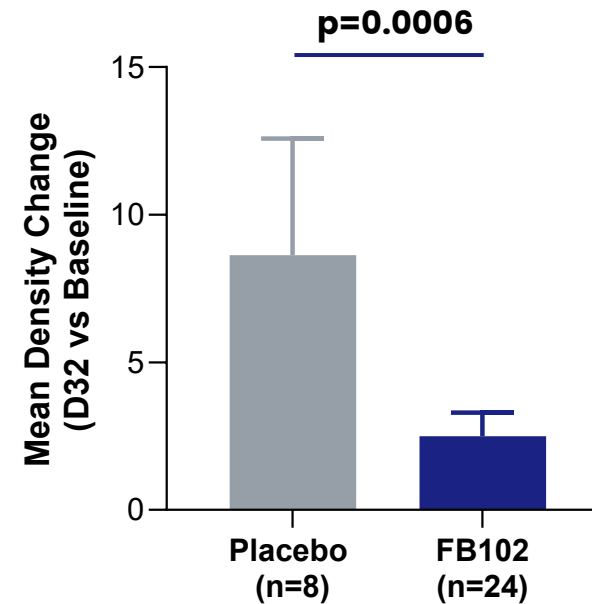
TCR $\gamma\delta$ Positive IELs



Increase of 3.9 for PBO compared to decrease of 1.5 for FB102 treated subjects

Gluten interacts with receptors on TCR $\gamma\delta$ IELs driving cytotoxic T cell expression and epithelial damage

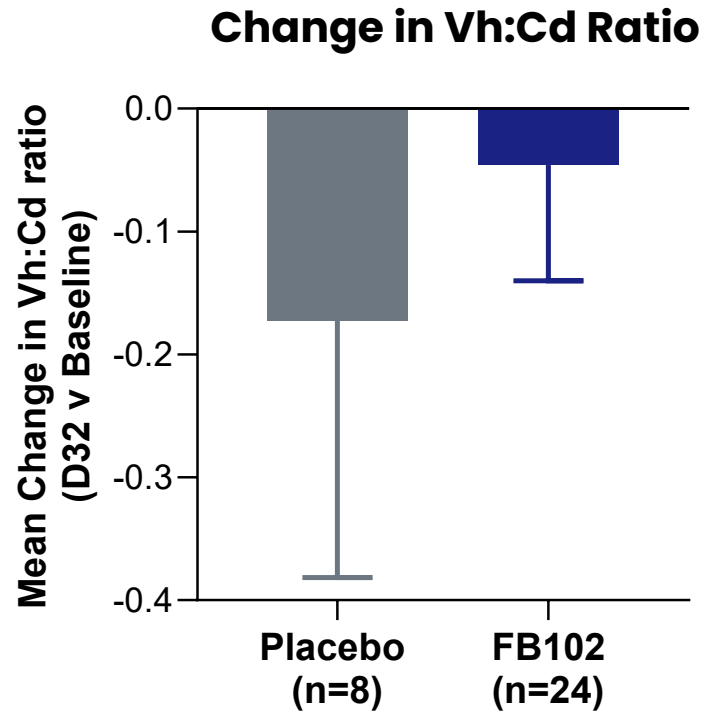
Ki67 Positive IELs



Increase of 8.6 for PBO compared to 2.5 for FB102 treated subjects

Ki67 is a marker of T cell proliferation (inflammation)

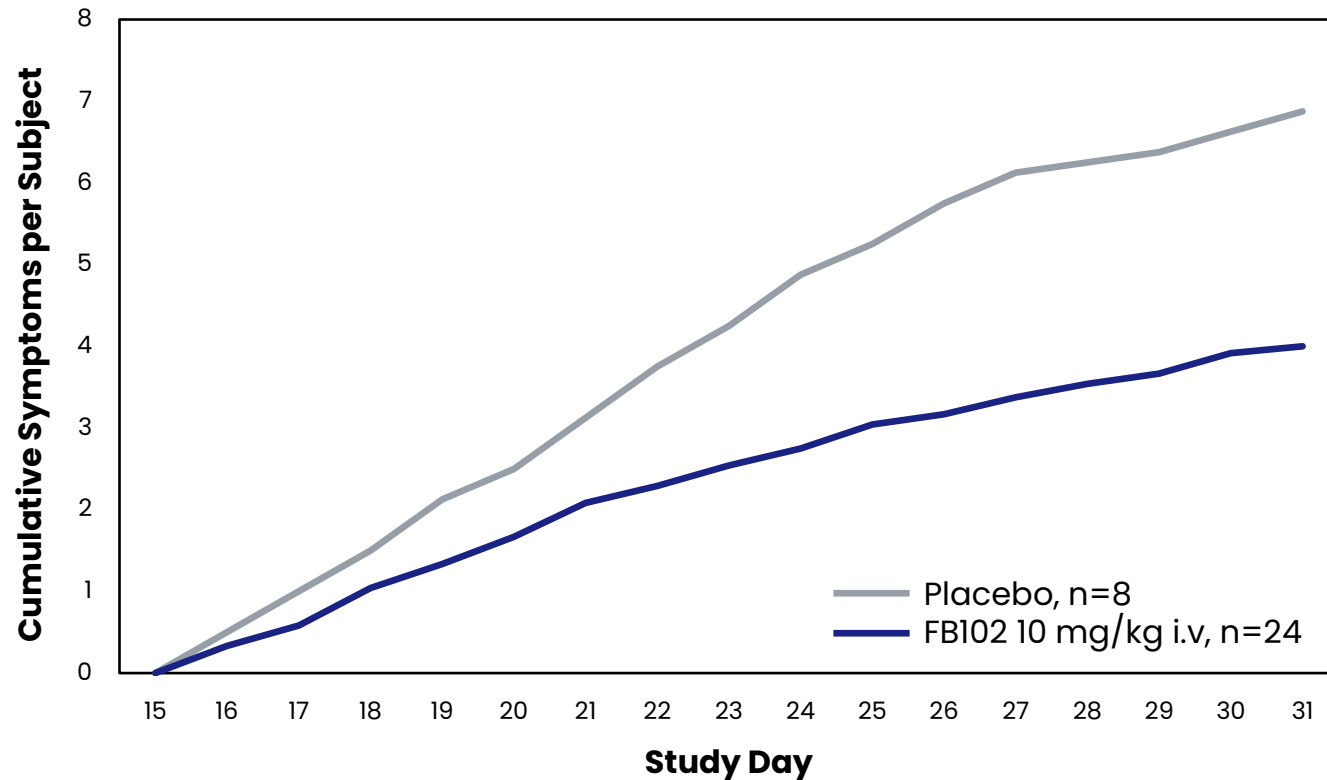
Vh:Cd Ratio Improvement Observed for FB102 vs Placebo



Day 32 vs baseline Vh:Cd ratio improvement of 73% for FB102 (-0.046) compared to PBO (-0.173)

FB102 Demonstrated Gluten Challenge Symptom Event Benefit Compared to Placebo

Cumulative Symptom per Subject during the Gluten Challenge



Symptoms monitored:

Nausea, diarrhea, vomiting, abdominal pain, abdominal bloating (patient diaries and AE collection)

Outcome:

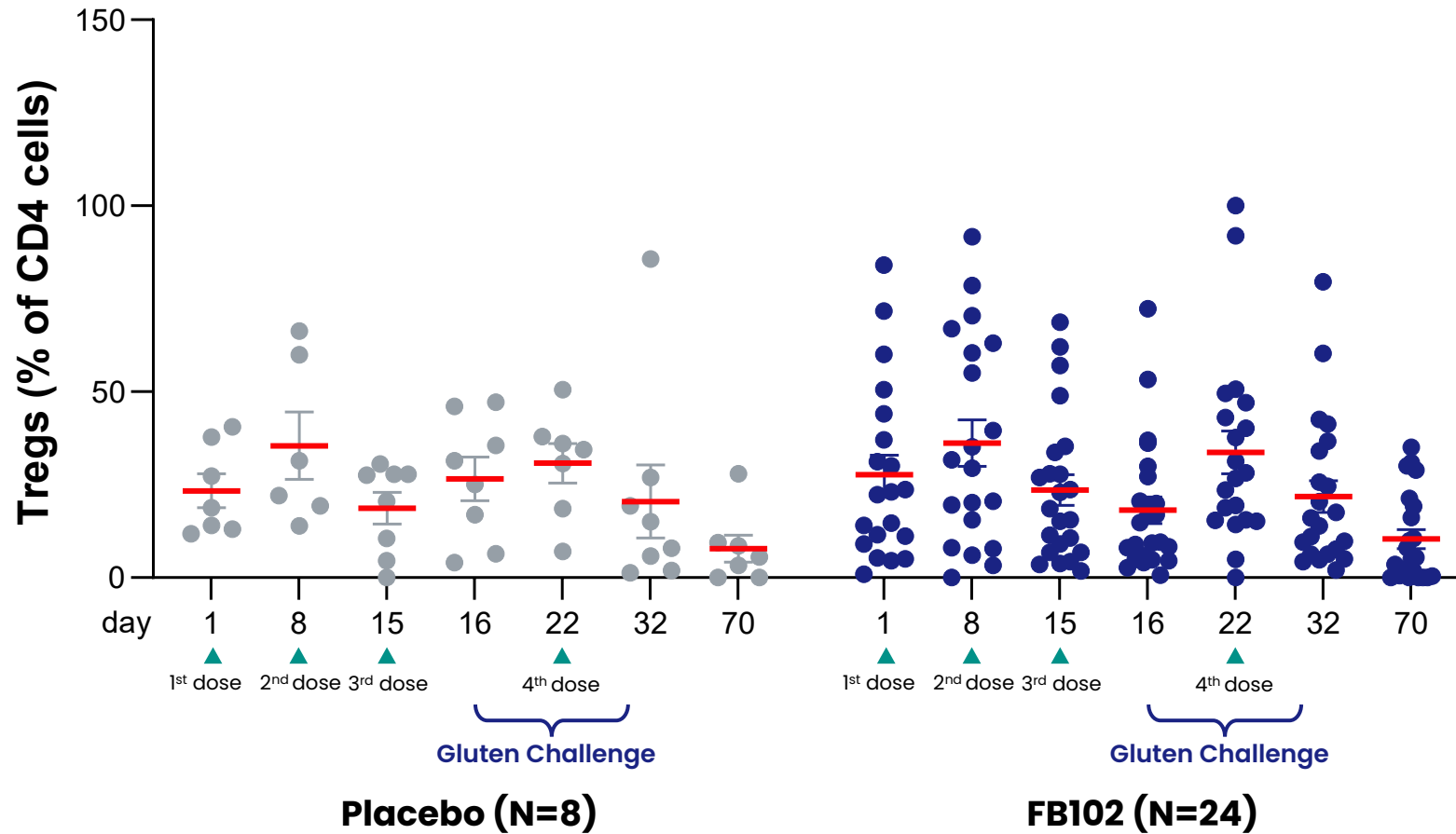
FB102 reduced cumulative symptom events per subject by end of challenge by 42%

- Placebo 6.9 events/subject
- FB102: 4.0 events/subject

Interpretation:

Symptom separation between FB102 and placebo emerged early and widened through the 16-day Gluten Challenge Period

No Inhibition of Tregs with FB102 Treatment



No statistically significant difference in Tregs with FB102 vs PBO at any timepoint

Preserves Treg driven immune tolerance

FB102 Generally Safe and Well Tolerated

Subjects Experiencing ≥ 1 Treatment Emergent Adverse Event by Grade

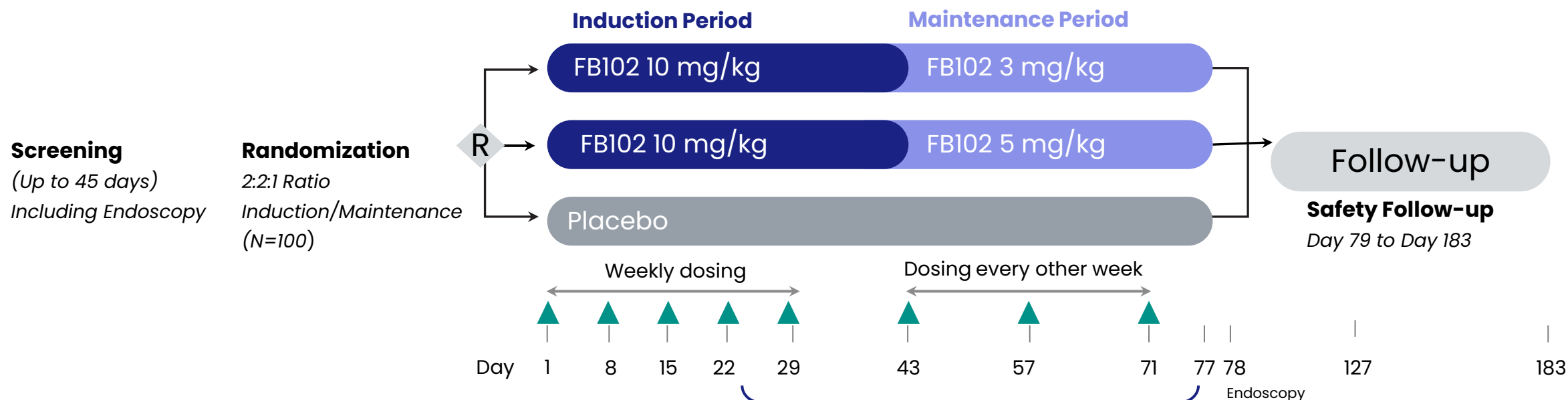
	Placebo N=8		FB102 N=24		Overall N=32	
	n	%	n	%	n	%
All Any Grade	8	100.0%	23	95.8%	31	96.9%
Grade 1 (Mild)	8	100.0%	22	91.7%	30	93.8%
Grade 2 (Moderate)	6	75.0%	9	37.5%	15	46.9%
Grade 3 (Severe)	1	12.5%	0	0.0%	1	3.1%

FB102 Generally Safe and Well Tolerated

Subjects Experiencing ≥1 Treatment Emergent Adverse Event by Organ Class

System Organ Class Summary	Placebo N=8		FB102 N=24		Overall N=32	
	n	%	n	%	n	%
Participants with at least one TEAE	8	100.0%	23	95.8%	31	96.9%
Gastrointestinal disorders	7	87.5%	21	87.5%	28	87.5%
Nervous system disorders	5	62.5%	10	41.7%	15	46.9%
General disorders and administration site conditions	2	25.0%	5	20.8%	7	21.9%
Infections and infestations	3	37.5%	4	16.7%	7	21.9%
Metabolism and nutrition disorders	2	25.0%	2	8.3%	4	12.5%
Musculoskeletal and connective tissue disorders	1	12.5%	2	8.3%	3	9.4%
Blood and lymphatic system disorders	2	25.0%	0	0.0%	2	6.3%
Psychiatric disorders	1	12.5%	1	4.2%	2	6.3%
Respiratory, thoracic and mediastinal disorders	2	25.0%	0	0.0%	2	6.3%
Ear and labyrinth disorders	0	0.0%	1	4.2%	1	3.1%
Vascular disorders	0	0.0%	1	4.2%	1	3.1%

Celiac Disease Phase 2 Design FB102-301



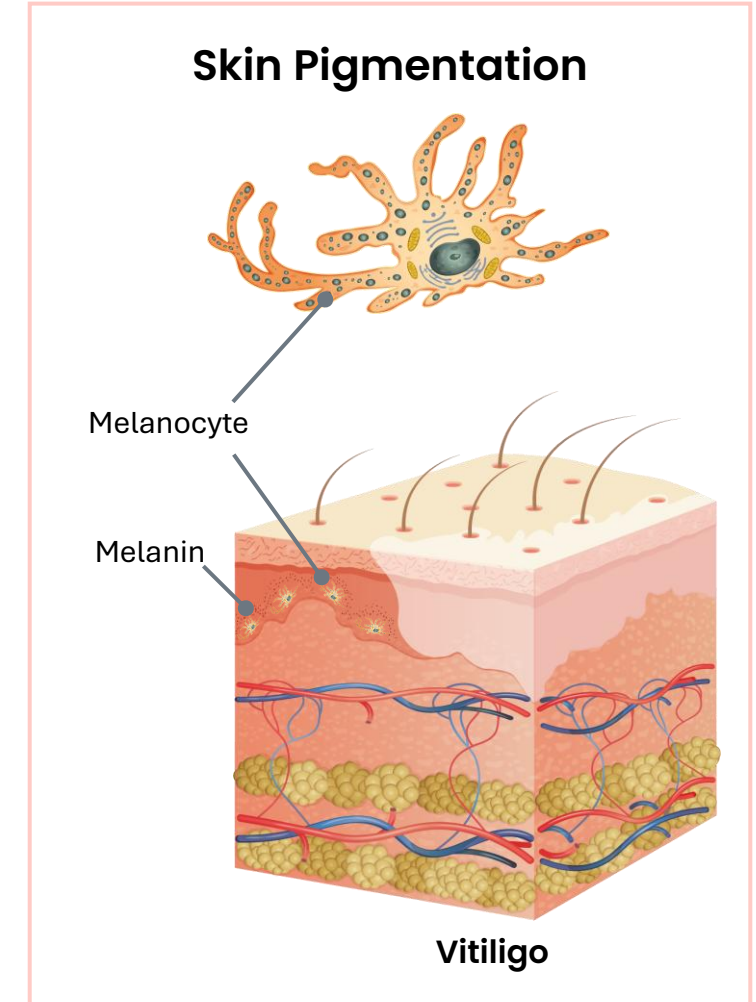
- 14 sites AUS/NZ initially
- Initiation in 2H25
- US IND expected late 2025/early 2026
- Men and women aged 18 to 70 years
- Has documented diagnosis of CeD
- Body mass index (BMI) 16.0 - 40.0 kg/m², inclusive
- Self-reported to be on a GFD for at least 12 months prior to Screening
- Primary Endpoint: VCIEL change from baseline at D78

Study Day	Amount of Gluten (grams per day)
Day 1 to 22	0
Day 23 to 36	8
Day 37 to 77	3
Day 78	Endoscopy

Topline data readout
expected in 2026

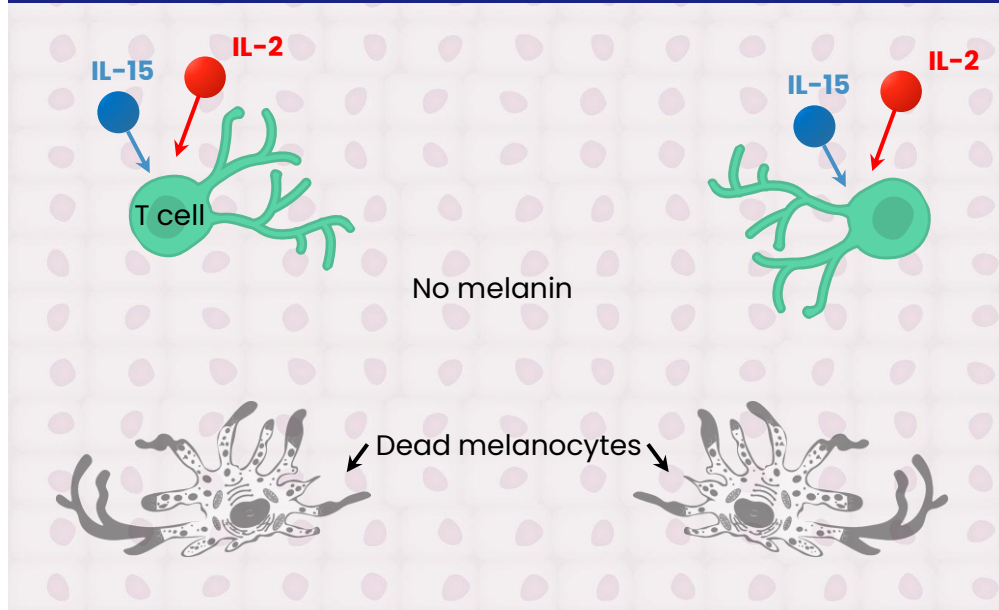
Vitiligo: Autoimmune Skin Disease with High Unmet Need

Disease Overview	- Autoimmune condition in which pathogenic T cells destroy melanocytes resulting in depigmented (white) skin patches - Non-Segmental vitiligo: 85–90% of cases, bilateral - Segmental vitiligo: 10–15% of cases, unilateral
Epidemiology	- Estimated global prevalence: 0.5–2% globally, including US 2M - Age of onset: 10–30 years old, 50% before age 20 - Female:Male equal
Symptoms and Consequences	- Not just cosmetic - Associated photosensitivity, ocular abnormalities, emotional burden and increased risk of other autoimmune - Psychosocial stress and reduced quality of life
Current Treatment	- Limited and variably effective (topical steroids, phototherapy) - Long term safety concerns, especially JAK inhibitors, include black box warning for serious infections, death, cancer, MACE and blood clots
Unmet Need	Disease modifying therapy that provides safe, accessible and durable response

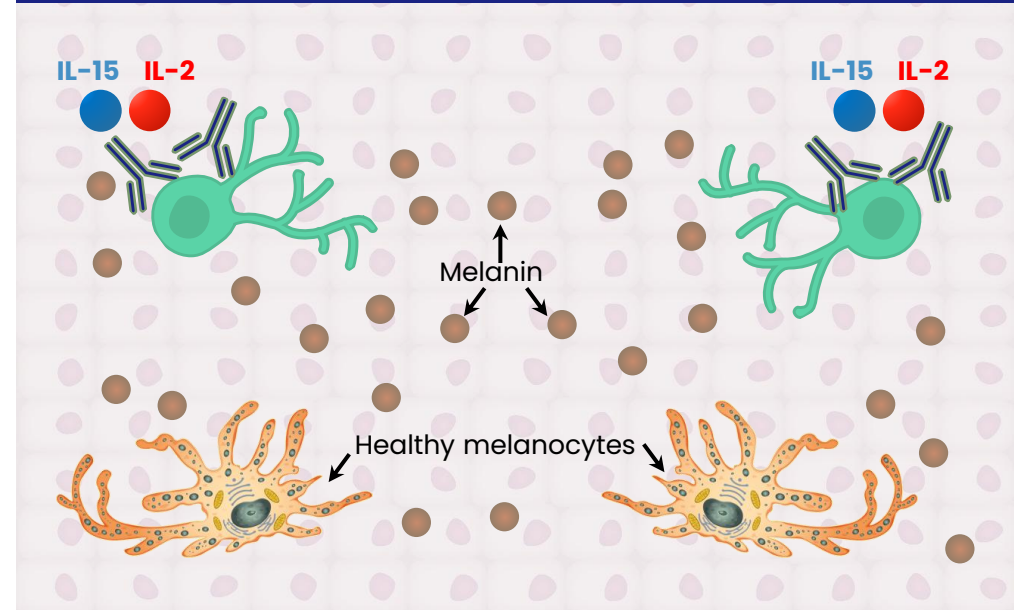


IL-15 and IL-2 Drive Vitiligo via Activation of Pathogenic T Cells

Activated pathogenic T cells kill melanocytes leading to loss of melanin and skin pigment



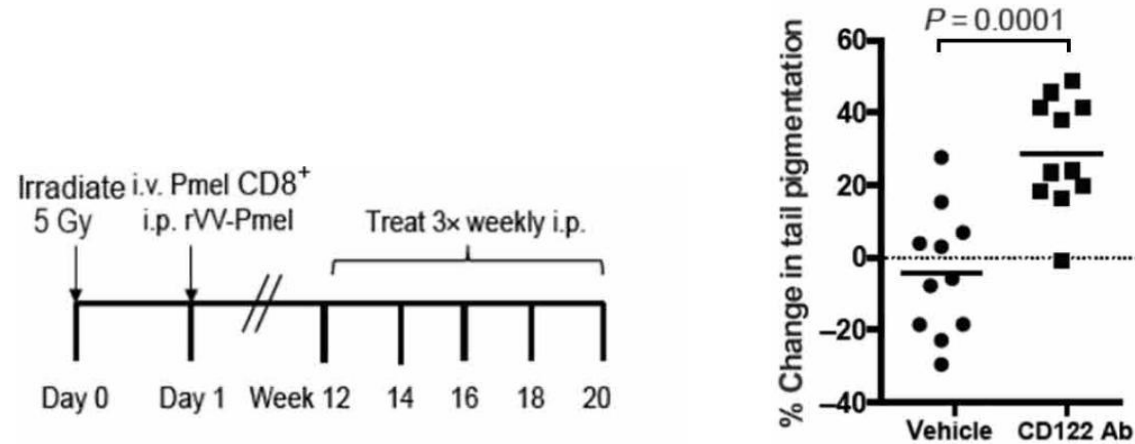
FB102 blocks activation of pathogenic T cells, restoring melanocyte health and skin pigmentation



Vitiligo has been observed in responders to high-dose IL-2 in melanoma, with multiple studies linking this to robust immune activation, reinforcing IL-2's role in driving vitiligo¹

Anti-CD122 Antibody Reverses Disease in a Vitiligo Mouse Model

Repigmentation study



Melanocyte reactive T cells eliminate tail pigmentation



Anti-CD122 treatment restores pigmentation

i.p. = intraperitoneally

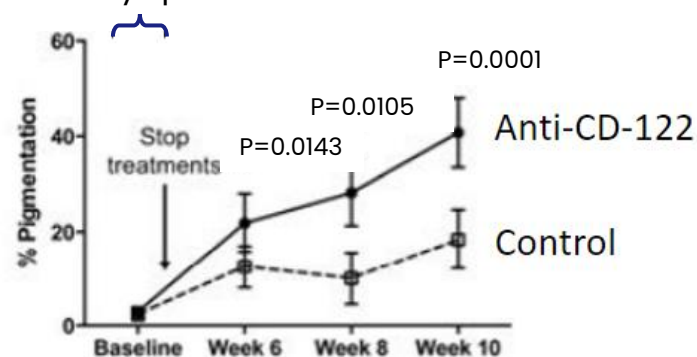
Richmond, et al. Sci Transl Med. 2018 PMID 30021889

Note: Vitiligo induced in C57BL/6J (B6) mice via adoptive transfer of anti melanocyte antigen T cells with anti-mouse CD122 (surrogate molecule) antibody used

Short Course Anti-CD122 Demonstrates Durable Response

Systemic

3x weekly i.p. x 2 weeks



Baseline

Week 8

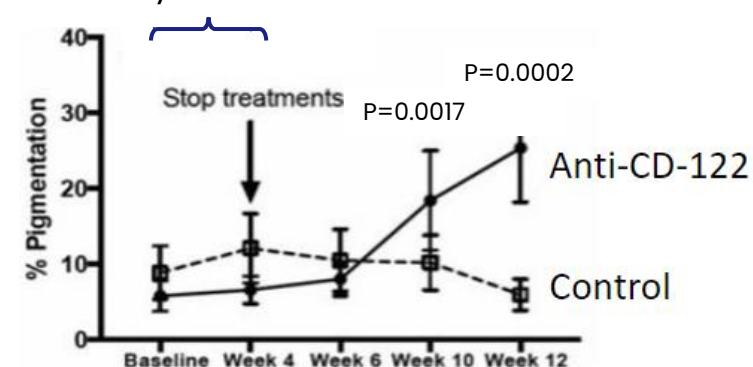
Control

Anti-CD-122



Local

3x weekly i.d. x 4 weeks



Baseline

Week 12

Control

Anti-CD-122



Comparable effect with systemic or local treatment
Lower injection burden may improve patient adherence

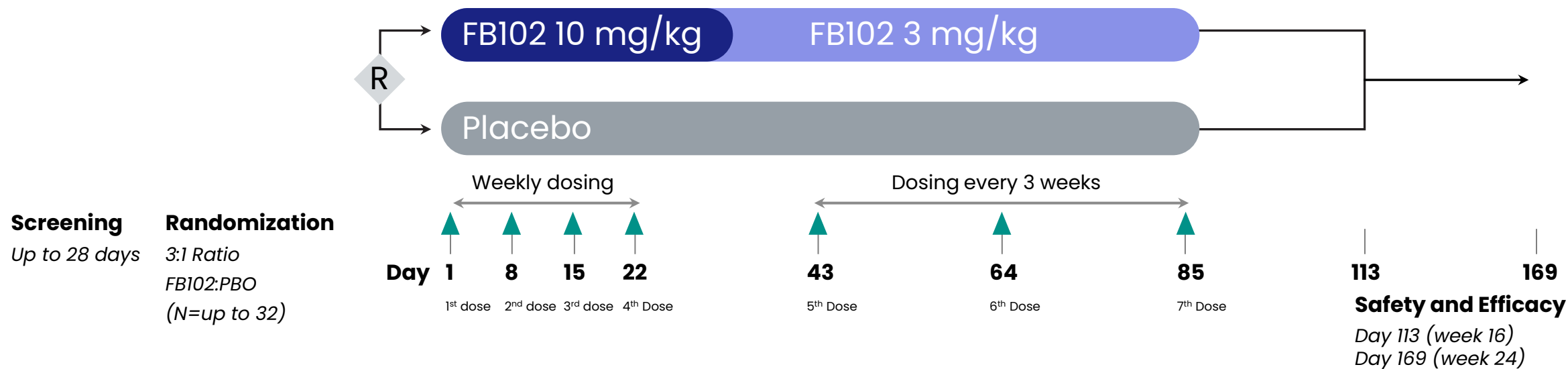
FB102: Dual IL-2/IL-15 Cytokine Inhibition

A Novel MOA to Restore Immune Privilege in Vitiligo

JAK based Carries Black Box Warning	Reducing Autoimmune Response	
	Single Cytokine	Multi Cytokine
JAK 3/TEC → ritlecitinib (LITFULO) 	IL-17A → aixekizumab (Taltz), 	CD122 (IL-2/IL-15) → FB102 FORTE BIOSCIENCES, INC
JAK 1/3 → Tofacitinib (ZELJANZ) 	IL-17A → secukinumab (COSENTYX) 	IL-12/23 → ustekinumab (Stelara) 
JAK1/2/3 TYK2 → Upadacitinib 	IFN α → anifrolumab 	CD122 (IL-2/IL-15) → INCA34460 
JAK1 → povorcitinib (Incyte INCB54707) 	TNF- α → adalimumab (Humira), etanercept TNF- α , infliximab (Remicade),   	

Vitiligo Phase 1b Design FB102-401

Treatment (16 weeks)

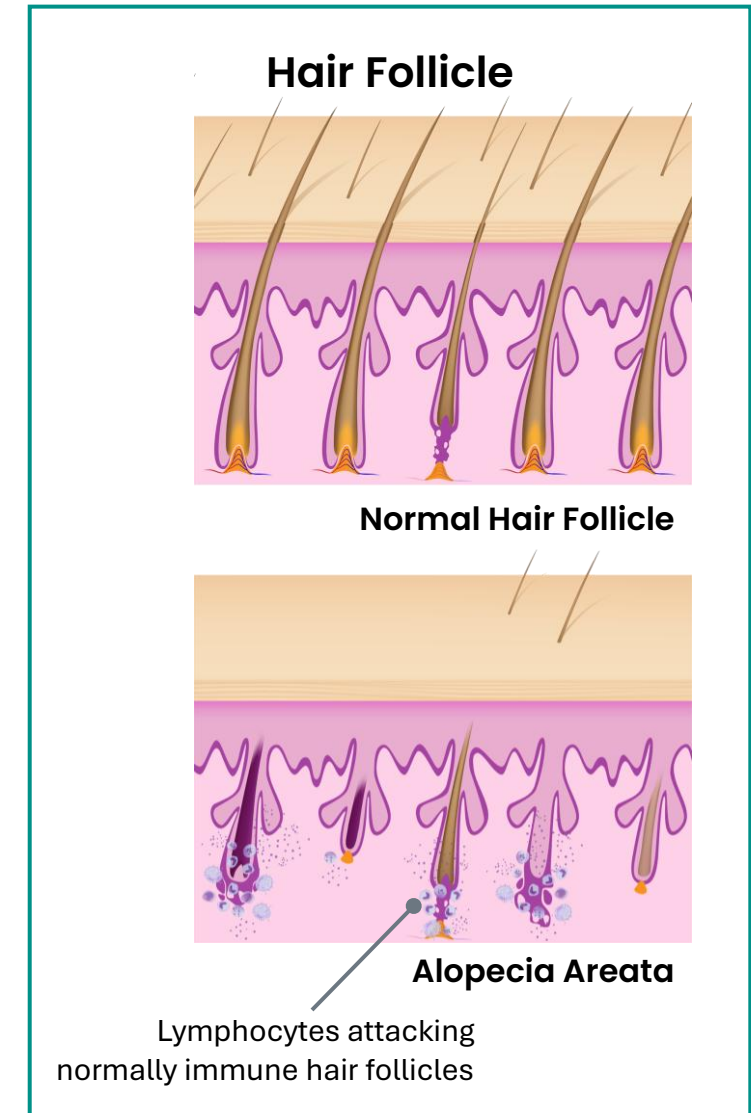


- Currently enrolling
- 10-12 sites in AUS/NZ
- Males and females aged 18 to 75 years
- Clinical diagnosis of non-segmental vitiligo
- Primary endpoints: F-VASI at 16 and 24 weeks

Topline data readout
expected in 1H 2026

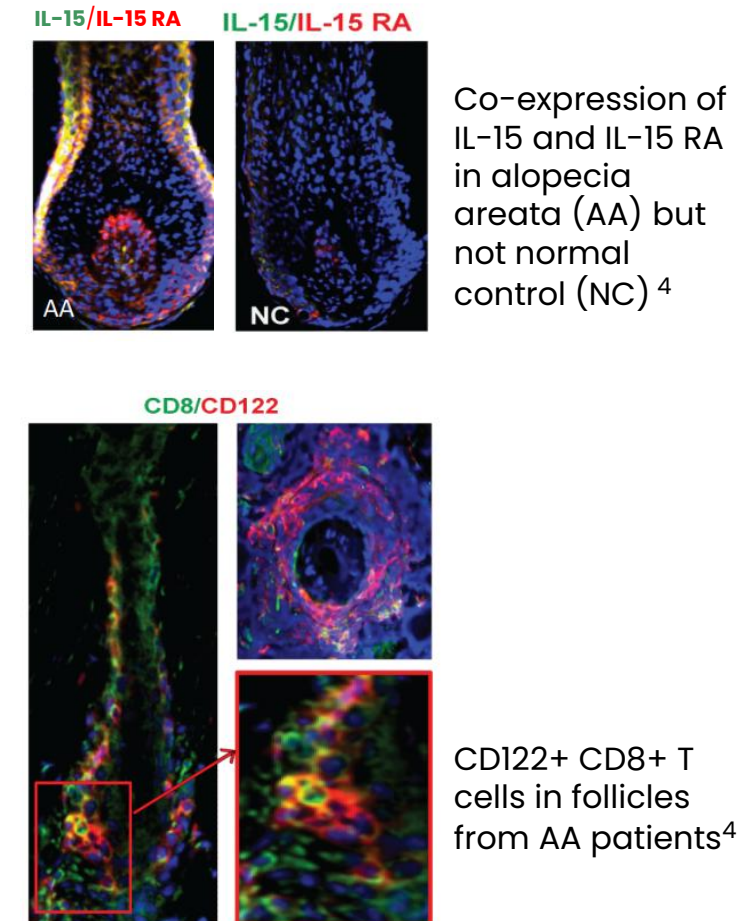
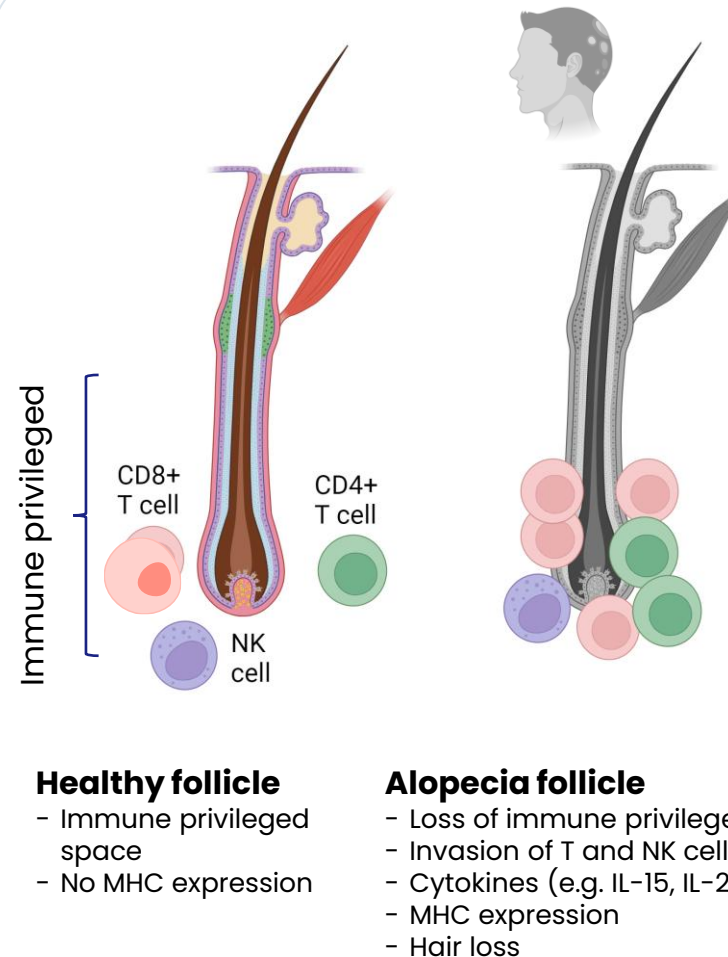
Alopecia Areata (AA): Auto Immune Disease with Large Unmet Need

Disease Overview	- Common autoimmune disease that occurs when pathogenic T cells attack hair follicles causing hair loss - Sudden onset, affecting scalp, face and body
Epidemiology	- Prevalence: 2% of global population at some point in lifetime, including ~ US 6.5M, with 700K currently experiencing AA - Age of onset: <40 years - Females:Males equal
Symptoms and Consequences	- Unpredictable course, often relapsing and remitting - Emotional and psychosocial impact incl depression, anxiety and social isolation - Associated with other auto immune conditions
Current Treatment	- No cure, current treatments suppress immune response (topical/intralesional steroids or off label immunosuppressants e.g. methotrexate) or stimulate hair growth - JAK inhibitors FDA approved systemic therapy with durable efficacy but black box warnings and long-term safety concerns including serious infections, death, cancer, MACE, and blood clots
Unmet Need	Safe effective and durable treatment across all severities



Alopecia Areta Driven by T Lymphocytes Invading Hair Follicles

- Healthy anagen (growth cycle) hair bulb is an immune privileged site – no MHC Class 1 expression and protected from immune attack¹
- Poorly understood environmental triggers cause immune privilege collapse
- Cytotoxic T cells infiltrate and attack follicular keratinocytes at the hair bulb base stopping hair growth
- IL-2 and IL-15 cytokines drive autoimmune response and hair loss
- Dense lymphocyte infiltration of 60–80% CD4+ and 20–40% CD8+ T cells²
- IL-2 is elevated in AA patients³; IL-15 is overexpressed in AA hair follicles⁴



MHC: Major Histocompatibility Complex (code for cell surface proteins responsible for presenting antigens to T cells)

¹Bertolini Exp Dermatol. 2020 PMID: 32682334

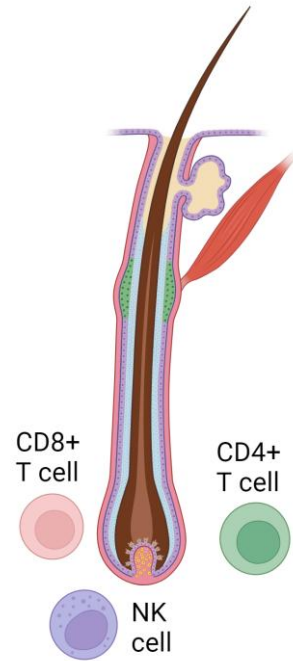
²Ito Clin Dev Immunol. 2013 PMID: 24151515

³Ito Exp Dermatol. 2014 PMID: 25040075

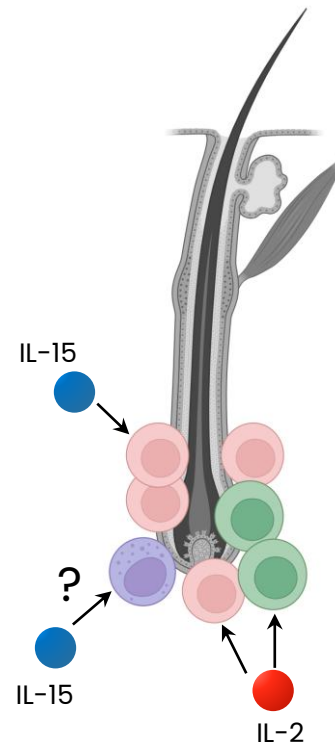
⁴Xing Nat Med. 2014 PMID: 25129481, Supplementary material

FB102 Anti-CD122 Antibody Potentially Restores Immune Privilege in AA

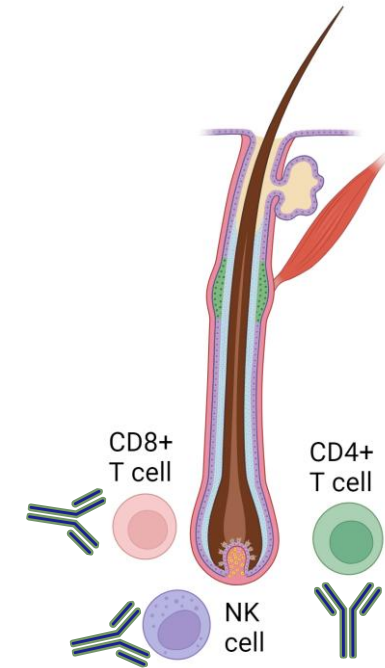
Immune privileged
healthy cell



Hair loss due to IL-2/IL-15
driven immune attack on hair
follicle



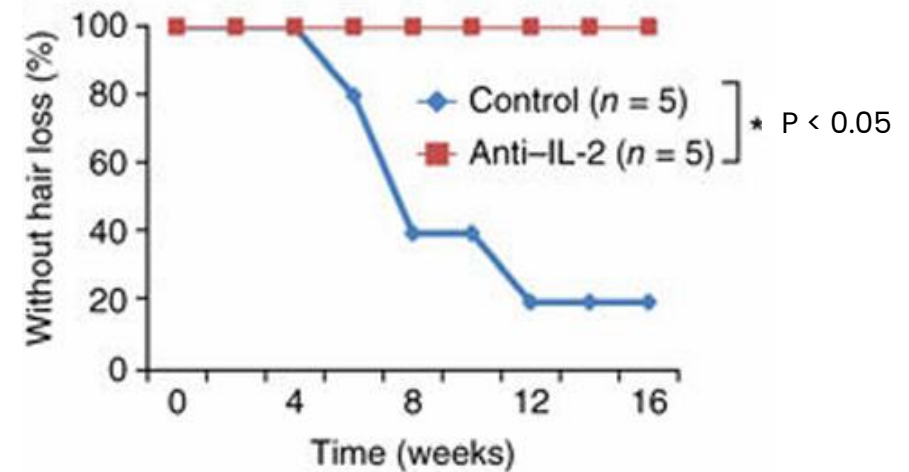
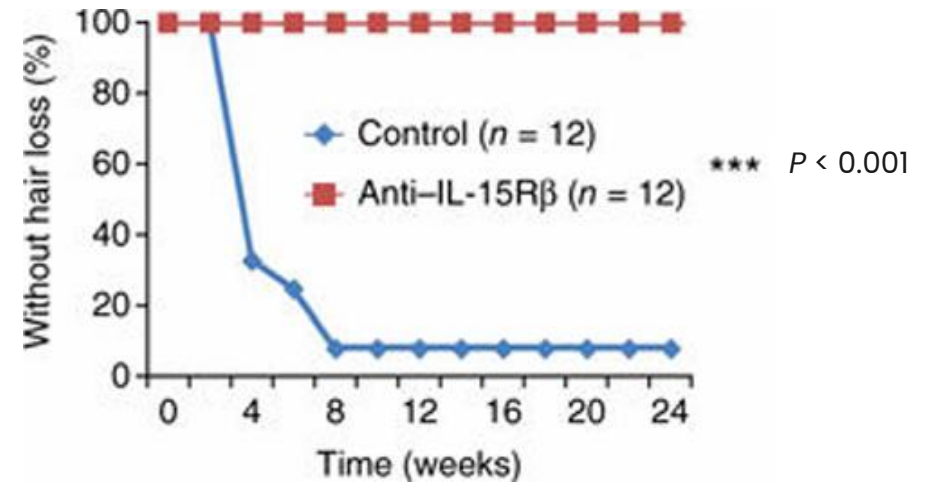
FB 102 blocks CD122 and
prevents T and NK cells from
attacking the hair follicle



Anti-CD122 and Anti-IL-2 Antibodies Prevent Hair Loss

C3H/HeJ Alopecia Areata Mouse Model

- Spontaneous alopecia with ~20% penetrance with age
- Grafting alopecic skin yields ~100% disease onset in successfully grafted recipient mice within 21-35 days¹
- Pathology resembles human disease
- Alopecia prevented with:
 - Anti-CD122 antibody, anti-IL-15R β , dosed 200 μ g twice per week for 12 weeks
 - 2 combined anti-IL-2 antibodies, dosed 250 μ g each three times per week²



Clone TM- β 1= anti-IL-15R β used

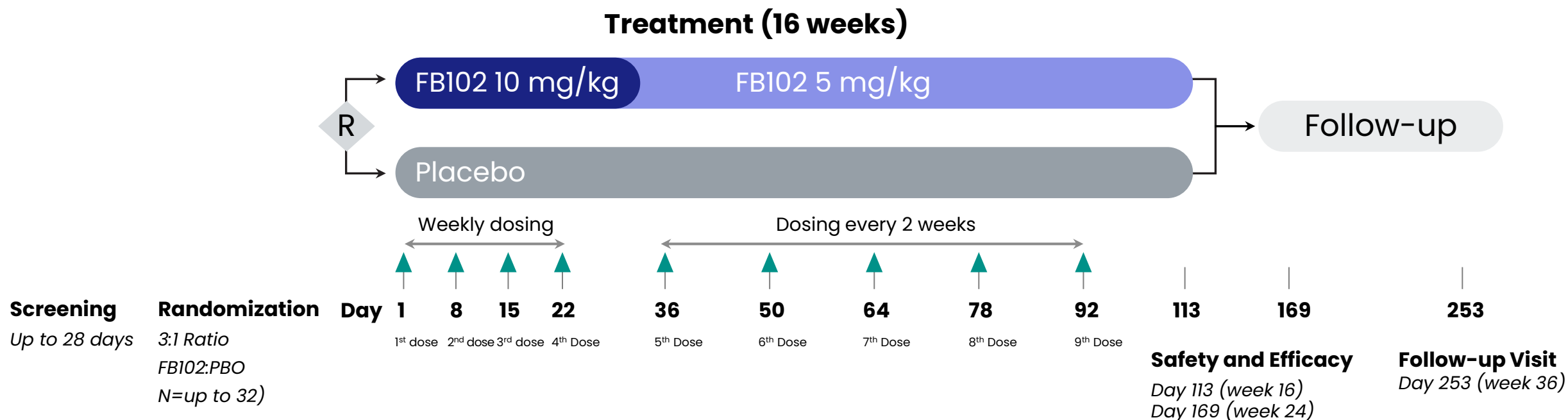
¹McElwee J Invest Dermatol. 1998 PMID: 9804341

²Xing Nat Med. 2014 PMI 25129481

FB102: CD122 Blockade Offers Superior Multi Target Coverage vs Single Target Agents Under Investigation in Alopecia Areata

JAK based Carries Black Box Warning	Reducing Autoimmune Response	
	Single Target	Multi Target
JAK 3/TEC → ritlecitinib (LITFULO) 	SIP → etrasimod (VELSIPITY) 	CD122 (IL-2/IL-15) → FB102 FORTE BIOSCIENCES, INC
JAK 1/2 → baricitinib (Olmiant) 	OX40 → IMG-007 	IL-4/13 → dupilumab (Dupixent)  
JAK 1/2 → deuruxolitinib (LEQSELVI) 	IL-2 → Repegaldesleukin 	IL7/TSLP → bempikibart 
JAK1/2/3 TYK2 → Upadacitinib 		IL-2/9/15 → EQ101 

Alopecia Areata Phase 1b Design FB102-701

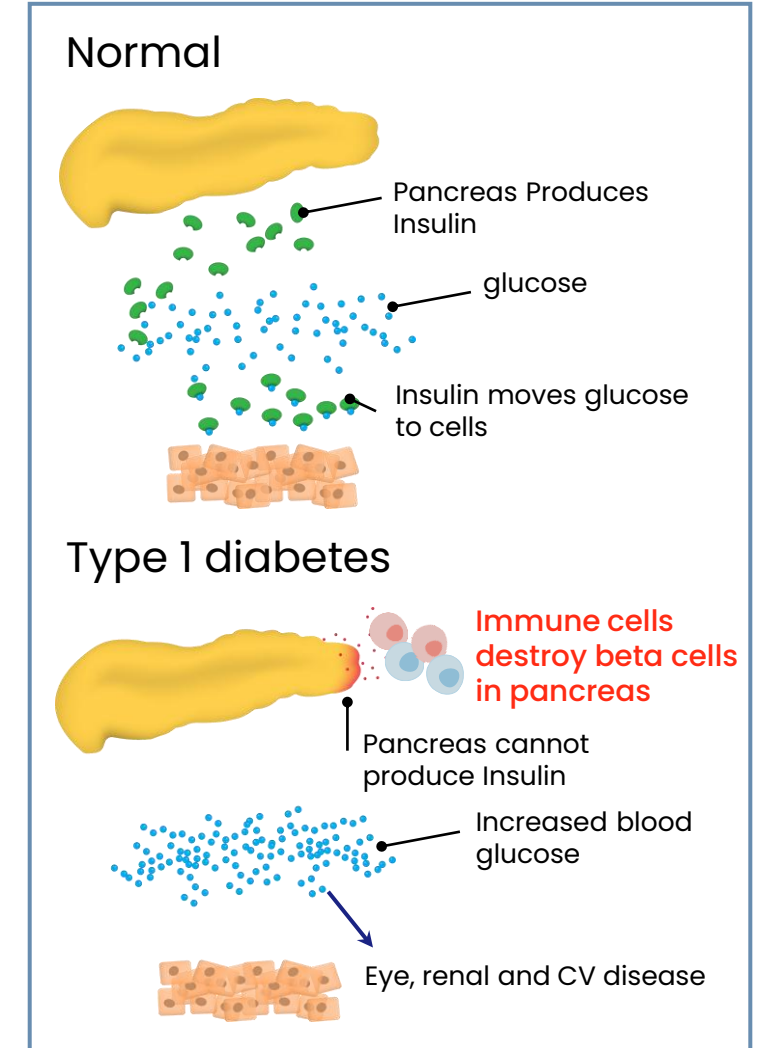


- 10-12 sites in AUS/NZ
- Primary endpoint: SALT at 16 and 24 weeks
- Males aged ≥ 18 to 60 and females aged ≥ 18 to 75 years
- Clinical diagnosis of severe to very severe AA with no other etiology for hair loss

Topline data readout
expected in 2026

Type 1 Diabetes, an Autoimmune Disease that Targets Insulin-producing Pancreatic Beta Cells

Disease Overview	- Autoimmune disease driven by genetic susceptibility and environmental triggers - Chronic disease characterized by autoreactive T cells that destroy insulin producing pancreatic beta cells
Epidemiology	- Prevalence 7.4M globally, includes ~2M US 64,000 people diagnosed with Type 1 diabetes annually¹
Consequences	Eye, renal and cardiovascular disease
Current Treatments	2 therapies approved to delay onset of T1D Lantidra™ - Islet cell transplant from deceased donors - Variable response with some pts achieving insulin independence - Requires lifelong immunosuppression Tzield® - Monoclonal AB vs CD3 T cells - Delays progression by 2 years - Safety issues including immunosuppression and infection
Unmet Need	Affordable, disease modifying therapy that cures or delays onset of disease



Type 1 Diabetes Clinical Competitive Space

The 2 approved products both carry significant challenges in their safety profile.

- LANTIDRA™
 - cell therapy that requires hospitalization for administration and cost \$300K/cycle
 - 90% of subjects experience serious AEs
- TZIELD®
 - IV infusion once daily for 14 consecutive days and Cost \$190K/cycle
 - Severe AEs including lymphopenia

Most drug candidates in development for Type 1 Diabetes work using indirect pathways

FB102 offer the advantage of directly impacting the T cells that cause the damage that leads to Type 1 Diabetes

From LANTIDRA™ PI

ADVERSE REACTIONS

Ninety percent (90%) of subjects had at least one serious adverse reaction. (6.1)
The major causes are attributed to:

- Infusion procedure
 - liver laceration/hematoma, hemorrhage, and intra-abdominal bleeding (13%)
 - elevation of portal pressure (7%)
- Immunosuppression
 - Infection (87%)
 - Malignancy (37%)

From TZIELD® PI

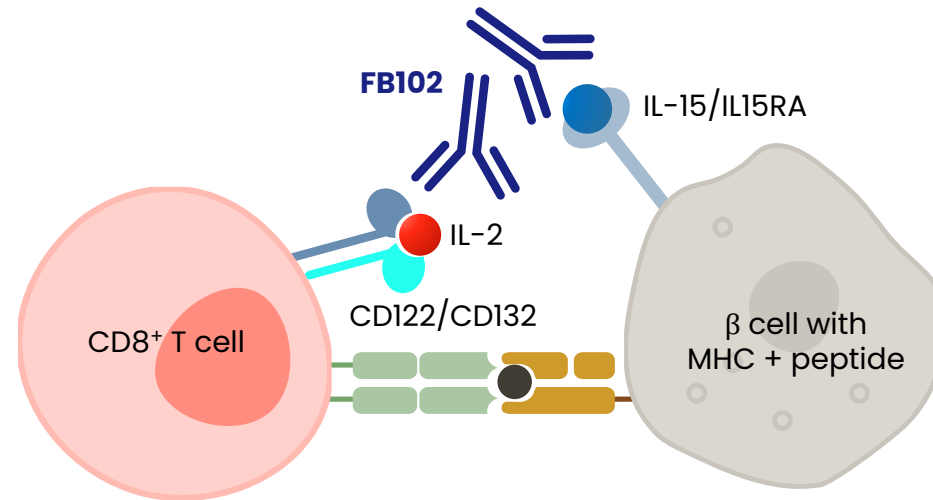
Throughout the study, greater incidences of these ARs were reported in TZIELD-treated patients vs placebo-treated patients:

- cytokine release syndrome (2% vs 0%)
- serious infections^{II} (9% vs 0%)
- hypersensitivity reactions and serum sickness (2% vs 0%)
- lymphopenia (73% vs 6%)
- neutropenia (7% vs 3%)

FB102 offers a unique MOA in potentially deactivating the autoreactive immune cells that lead to the destruction of the beta cells

FB102 Blocks IL-2/IL-15 Signaling to Prevent Beta Cell Destruction

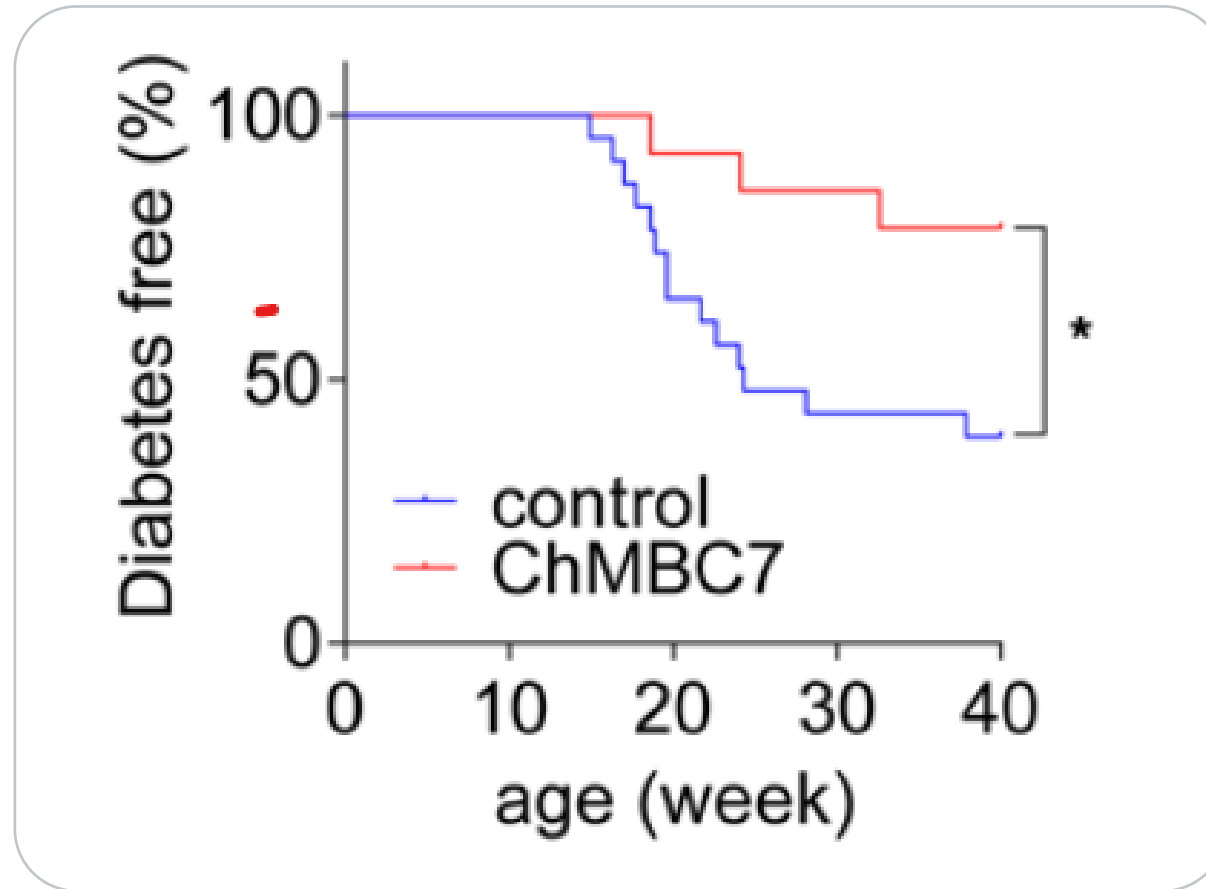
FB102 blocks IL-2/IL-15 binding



CD8⁺ T cells with receptors recognizing beta cell specific peptides are enriched in pancreatic islets of T1D patients

Environmental stress causes beta cells to upregulate MHC and to express IL-15 and IL-15RA

Anti-CD122 Antibody Delays Diabetes Progression



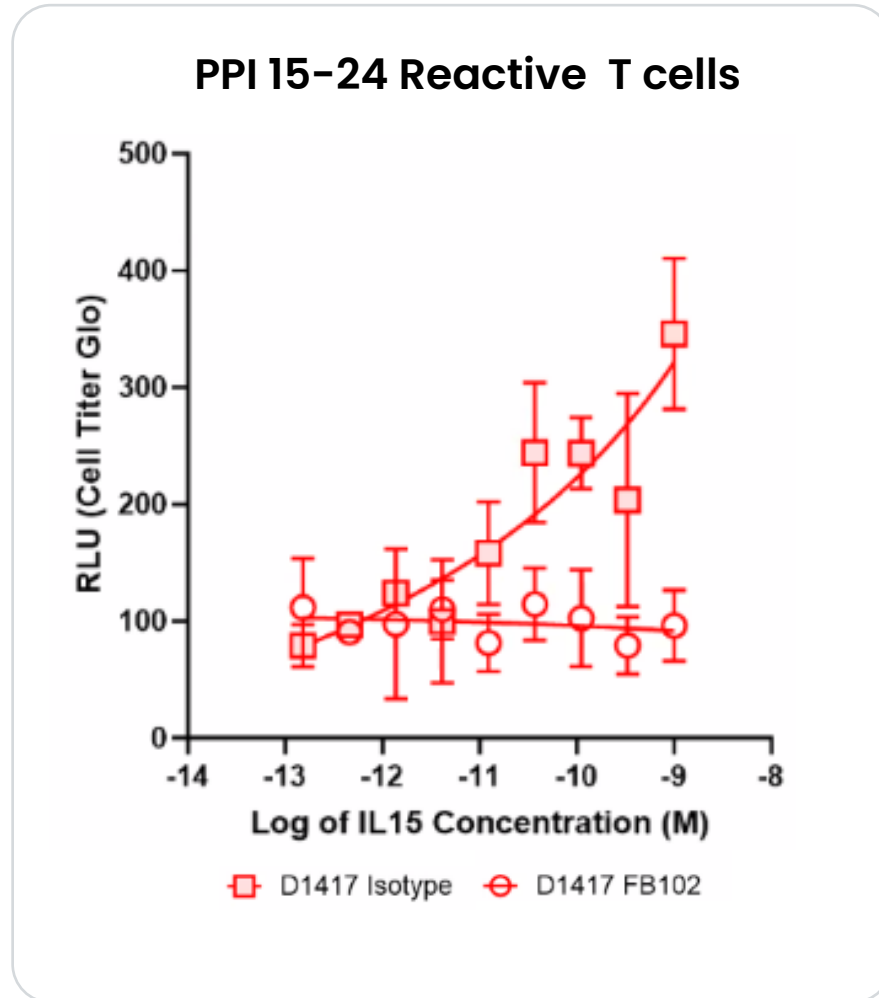
*P < 0.05

NOD: Non-obese Diabetic

ChMBC7 and TM-β1 are Anti-CD122 antibodies

Yuan 2018 JCI Insight PMID 29367461

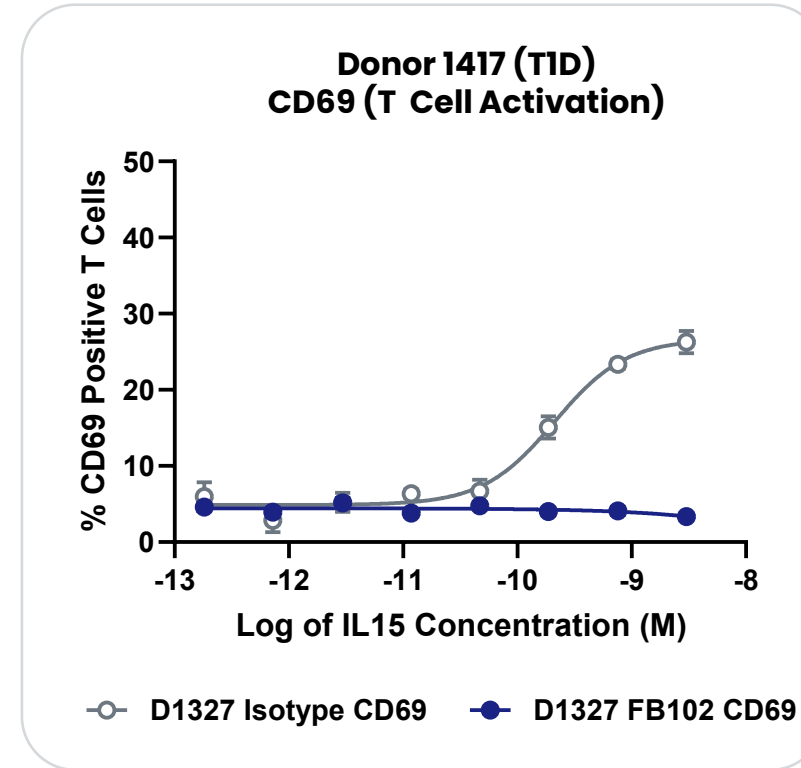
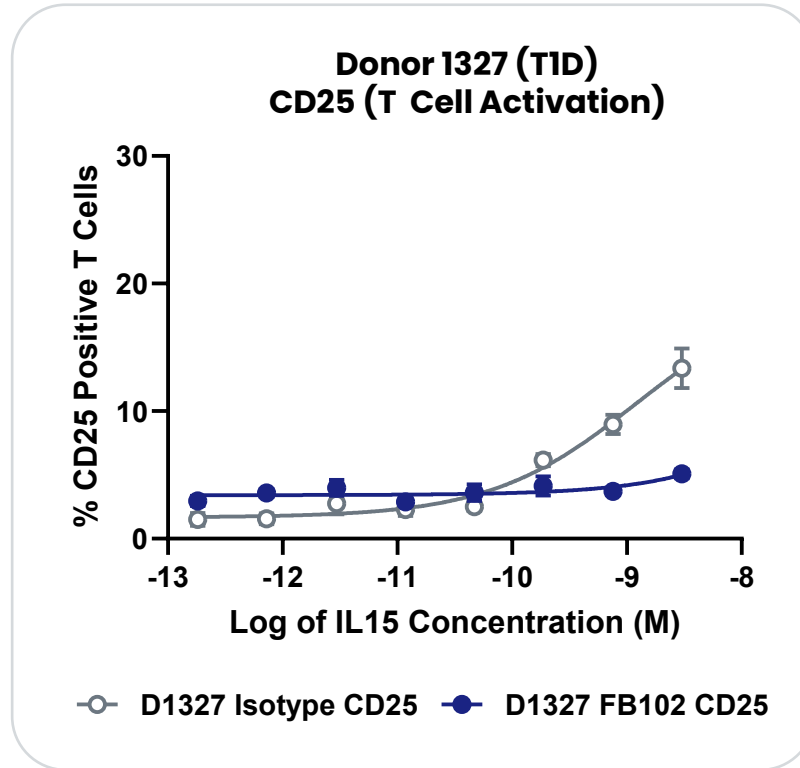
FB102 Inhibits Pathogenic T Cell Proliferation



Autoreactive T cells obtained from Type 1 Diabetes patients co-cultured with IL-15 with and without FB102

FB102 inhibits beta cell induced proliferation of T1D T cells

FB102 Inhibits T1D T Cell Activation

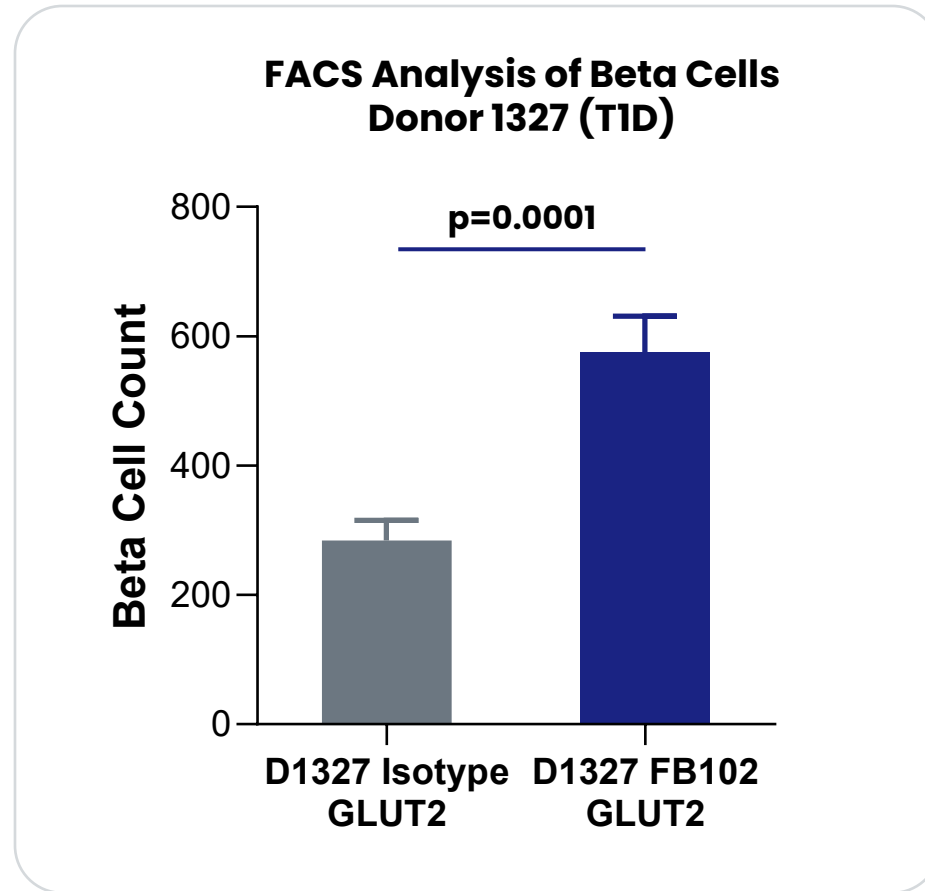


Activated T1D T cells are measured by an increase in % CD69 and CD25 T cells

Activation of T cells is inhibited by FB102
but not by the (negative) control antibody

FB102 Inhibits T1D T Cell Driven Killing of Beta Cells

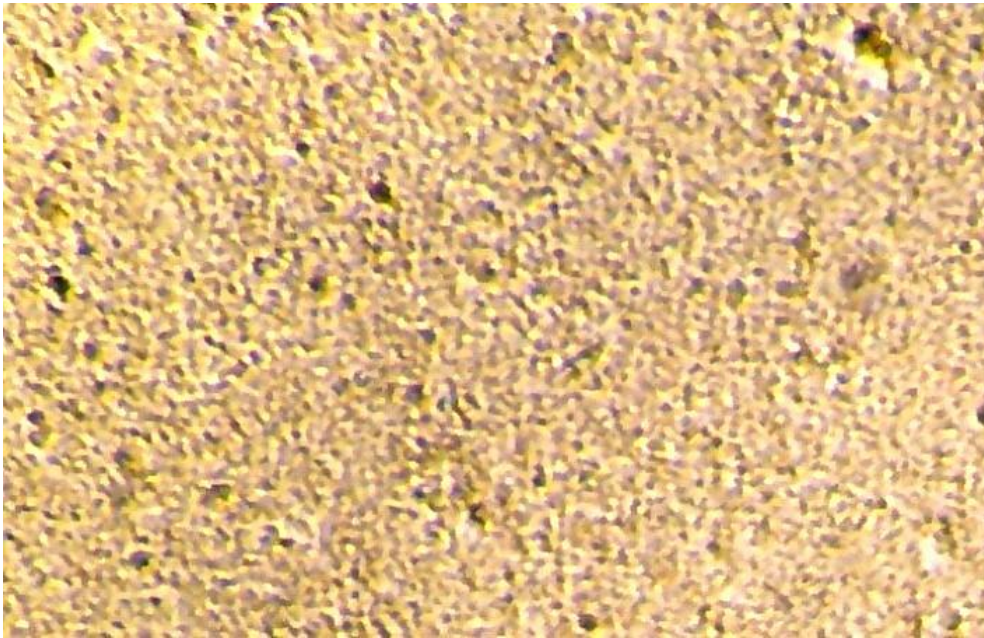
FB102 inhibits T1D donor T cell killing of beta cells



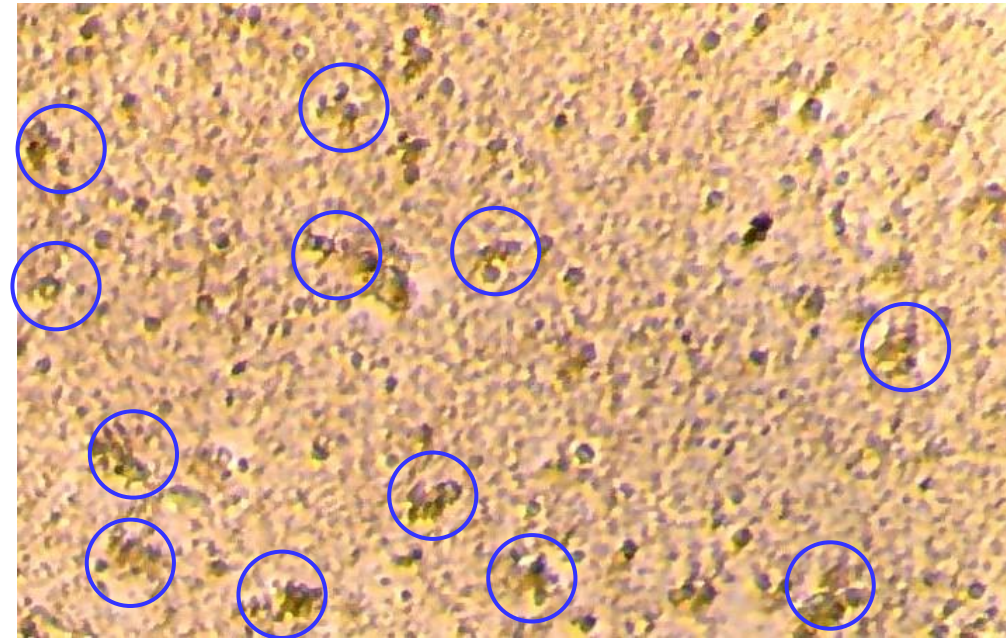
Beta cell killing by T1D reactive T cells is inhibited by FB102 vs isotype control

Microscopy Shows FB102 Induced Inhibition of T Cell Attack on Beta Cells

D1417 + Beta Cells with FB102
T cells are NOT clustering to Beta Cells



D1417 + Beta Cells with Isotype Control
T cells are clustering to Beta Cells



FB102 Proposed 12 Month Clinical Development

Program	Development	Phase 1	Phase 2	Phase 3	Upcoming Milestones
Celiac Disease					Phase 1b Topline data readout June 2025 Phase 2 Initiated 2H25 Phase 2 Topline data expected in 2026
Vitiligo					Phase 1b Initiated 1H25 Topline data expected in 1H26
Alopecia Areata					Phase 1b initiated in 2H25 Topline data expected in 2026
Type 1 Diabetes					Phase 1b in development

Investment Highlights



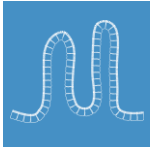
CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and certain subsets of T cells and is a subunit of the high affinity IL-2 receptor expressed on Tregs



FB102 (Forte's anti-CD122 antibody) is designed to mediate both IL-2 and IL-15 induced proliferation and activation of pathogenic NK and T cells while sparing beneficial Tregs



Potential "pipeline in a product"



Positive Phase 1b clinical data in celiac disease (CED) with Phase 2 trial initiated 2H2025 with topline data expected 2026



Phase 1b trial in vitiligo enrolling with topline results expected 1H2026



Phase 1b trial in alopecia areata (AA) enrolling with topline results expected 2026



Experienced, focused team determined to make a difference