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 trails 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

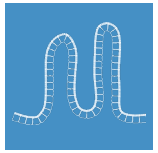
Lead in the development of a next generation biologic that inhibits CD122, addressing significant unmet needs in autoimmune and inflammatory diseases

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) 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

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

David Gryska

Barbera Finck, MD

Steve Doberstein, PhD

Steve Kornfeld

Shiv Kapoor

Rich Vincent

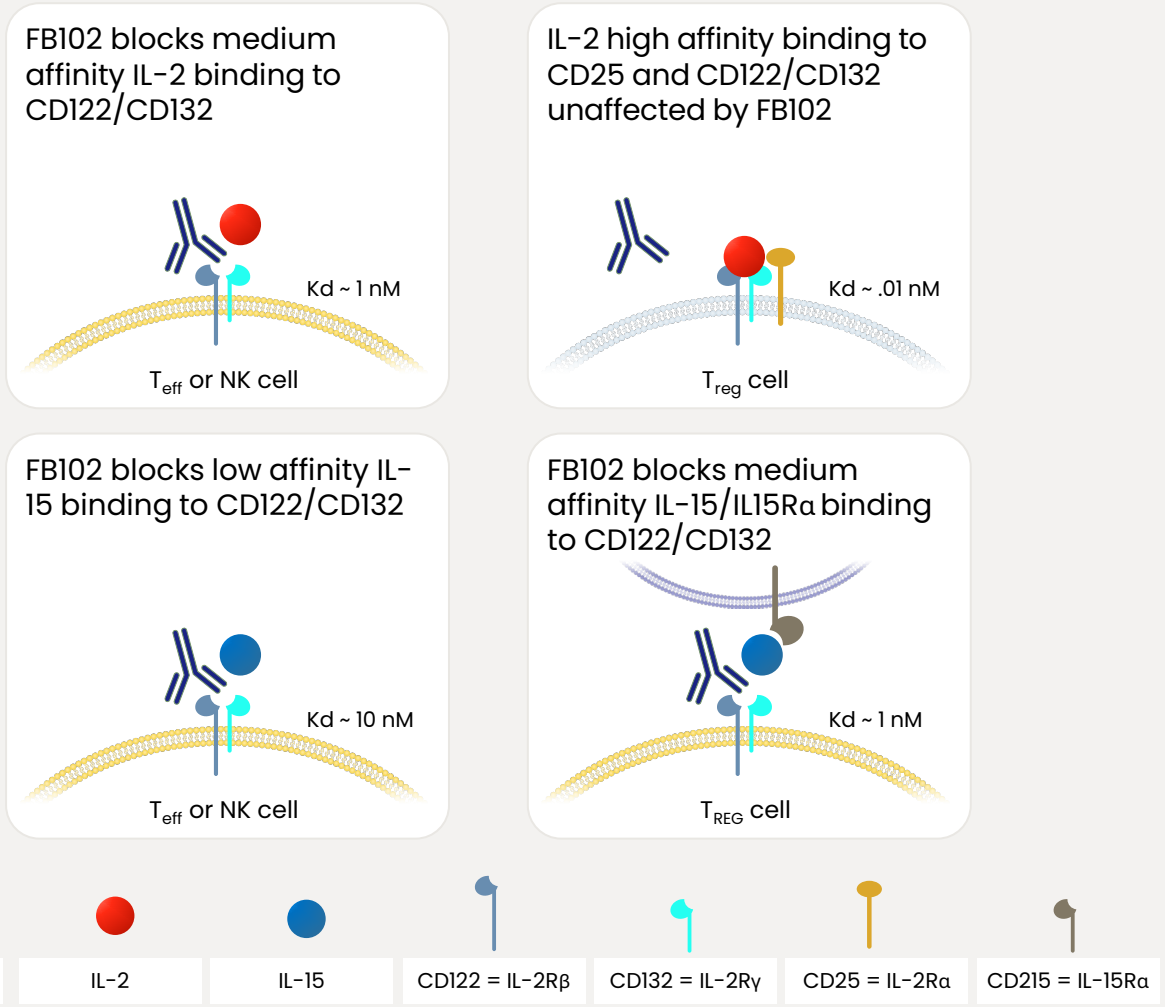
Why Target CD122?

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

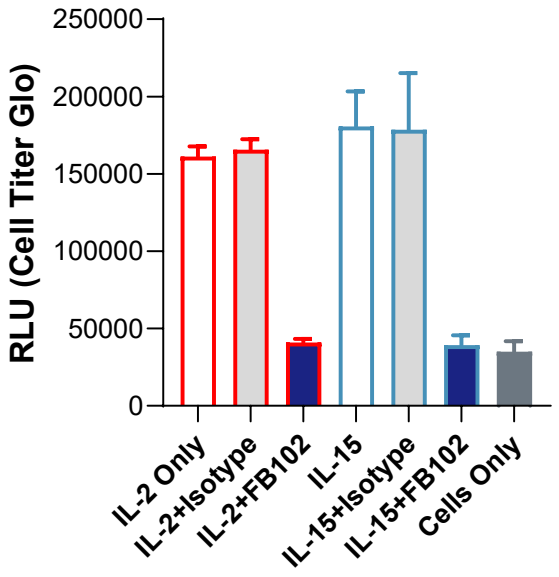
- Blocks IL-2 and IL-15 driven proliferation and activation of pathogenic T cells and NK cells
- Preserves IL-2 signaling in Tregs, maintaining immune regulation and limiting toxicity
- Designed for a differentiated therapeutic profile across autoimmune and inflammatory diseases



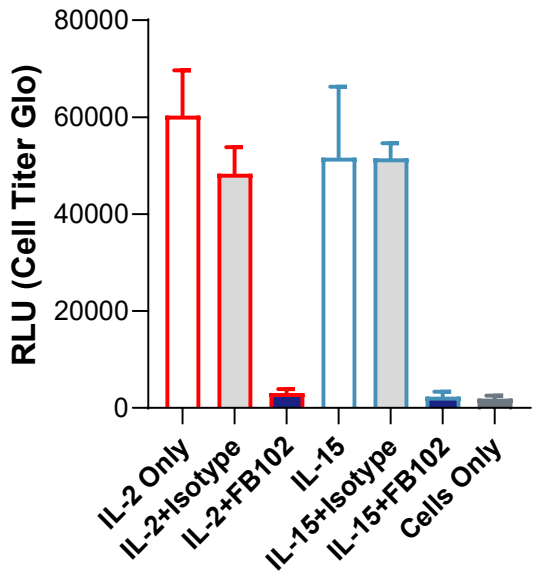
Lodolce 2002 Cytokine Growth Factor Rev. PMID 12401478
Ross 2018 Annu Rev Immunol. PMID 29677473

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

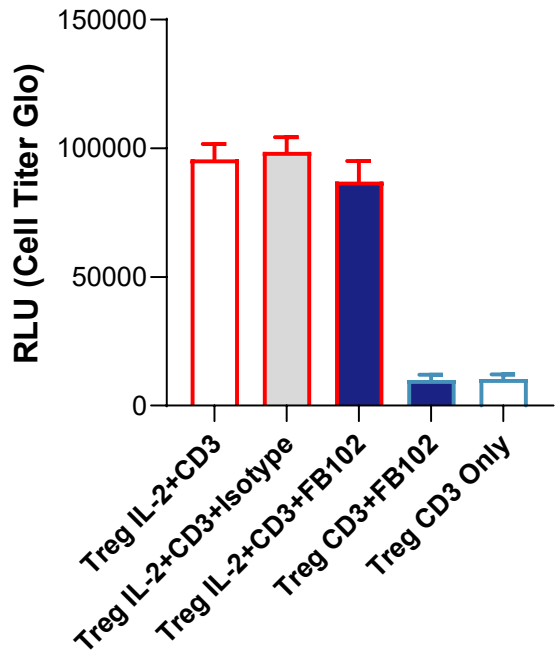
T cell proliferation assay
100 nM FB102 or isotype control



NK cell proliferation assay
100 nM FB102 or isotype control



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

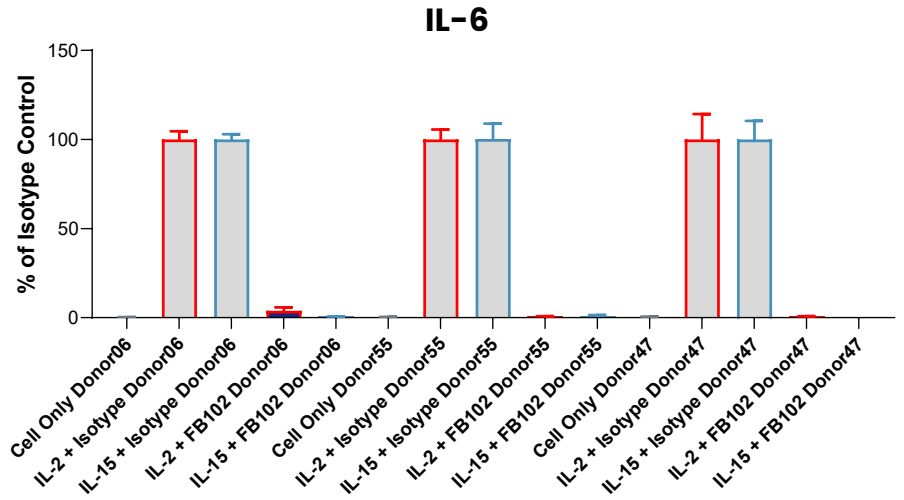


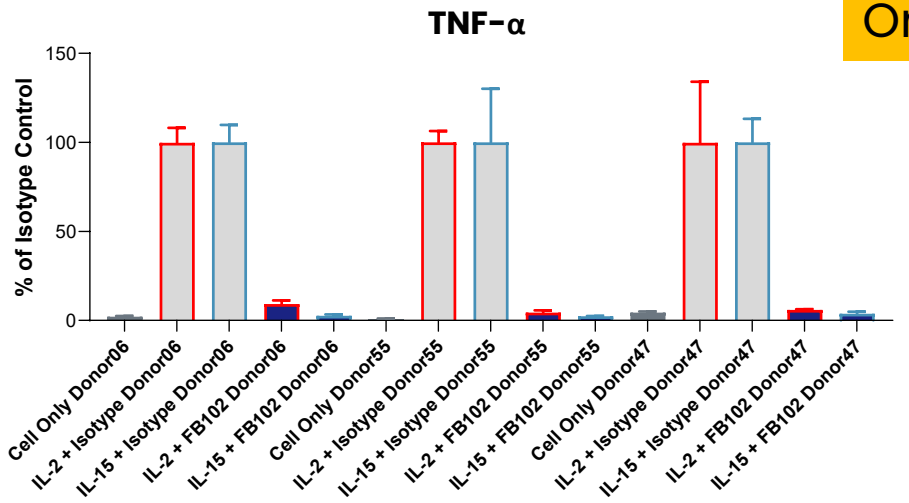
FB102 inhibits T cell and NK cell IL-2/IL-15 driven proliferation, while not inhibiting Treg proliferation

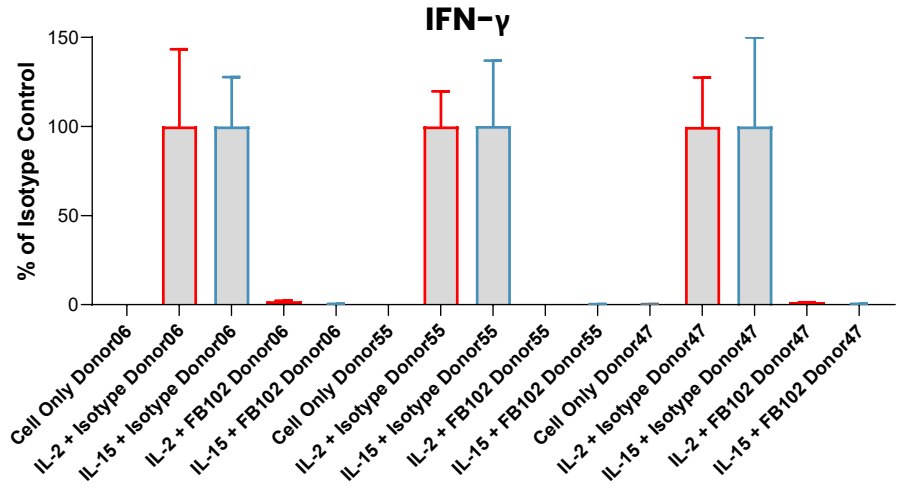
RLU= Relative Light Units, a measure of cell viability

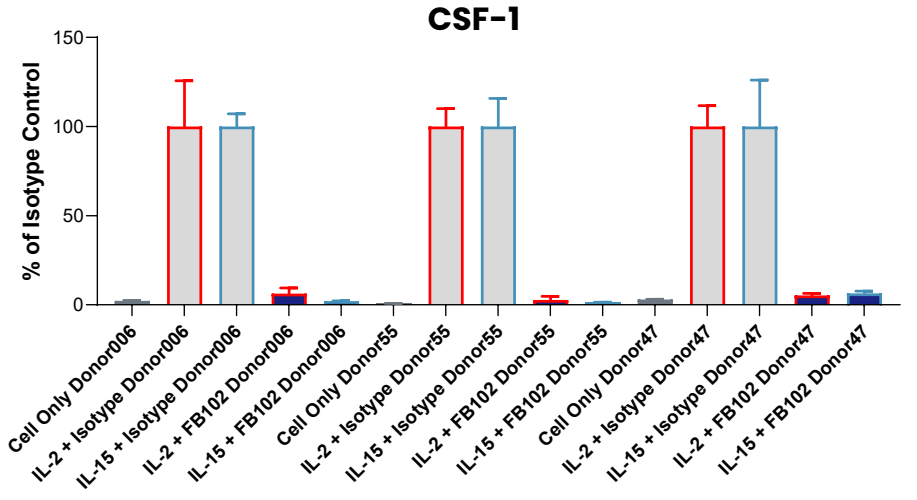
FB102 Inhibits Anti-inflammatory Cytokines Induced by IL-2 and IL-15

Original layout





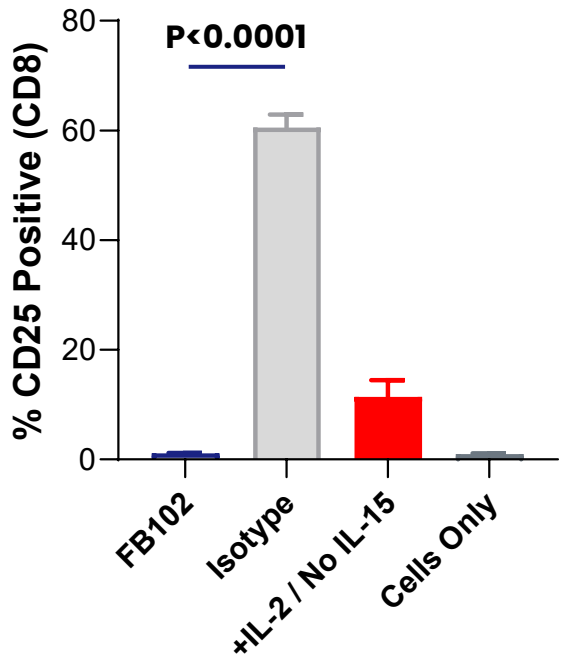




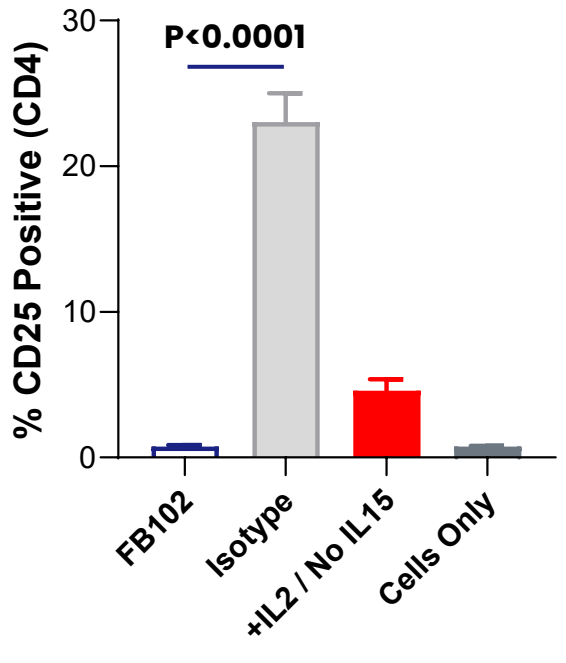
FB102 Inhibits IL-2/IL-15 CD4+ and CD8+ T Cell Activation in an In Vitro Disease Model

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

CD8+ T Cells



CD4+ T Cells



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

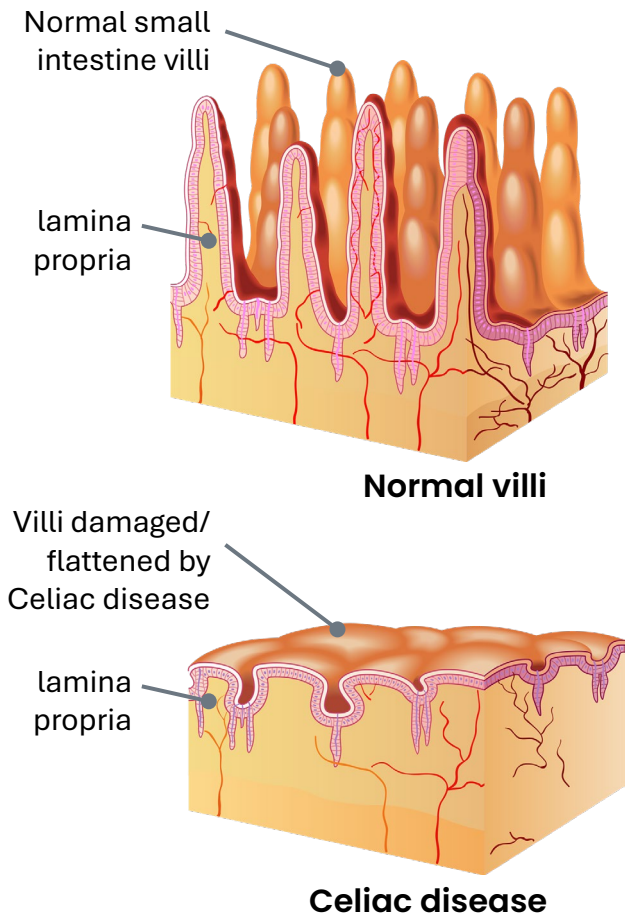
Celiac Disease: Large, Underserved Market Opportunity

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

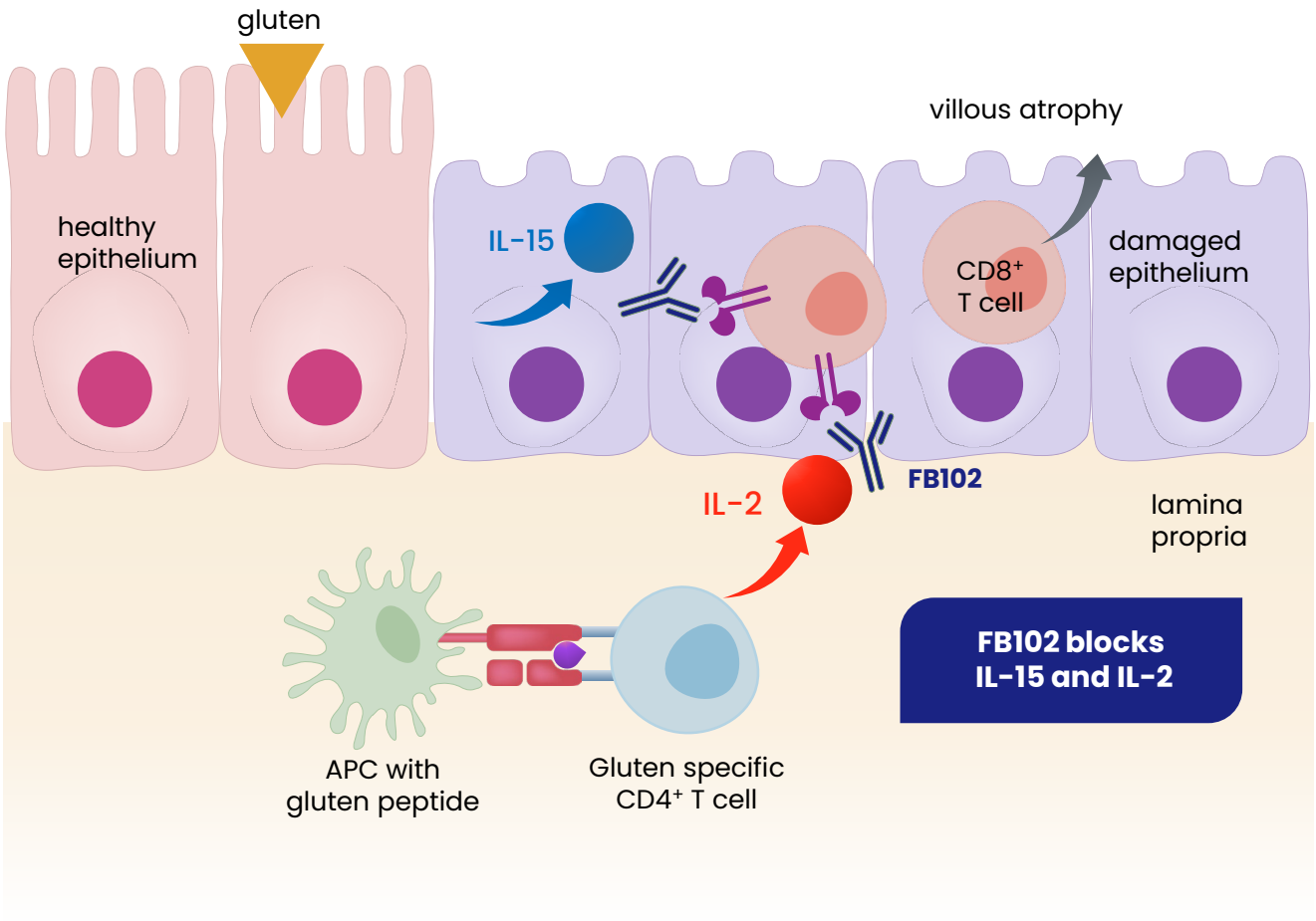
Unmet Need	• **No approved drug therapies**
	• **Gluten-free diet (GFD)** is the only treatment
	• **10–30%** fail to respond to GFD:
	  • **~50%** due to inadvertent gluten exposure (150-400 mg/day)
	  • **<10 mg/day** considered safe threshold
	  • **<5%** have true refractory disease



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

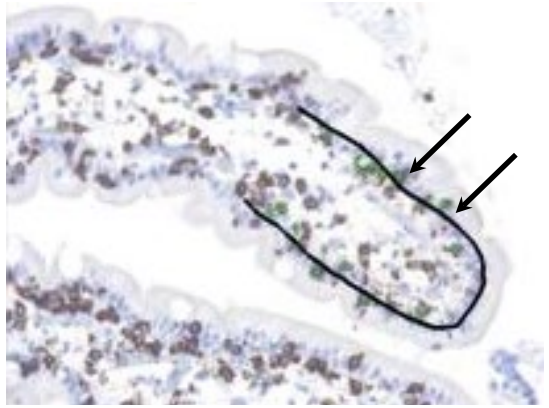
- Clear genetic basis for involvement in CeD
- Overexpressed in gut epithelium and immune cells upon gluten exposure
- Tissue levels correlate with degree of mucosal injury
- IL-15Rα overexpressed on IELs in CeD patients
- Stimulates IEL proliferation, IFN-γ and TNF-α release
- Activates pathogenic $CD8^+$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

Adapted from Levescot 2022 Gut PMID 35879049
IELs=Intra-epithelial lymphocytes

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			

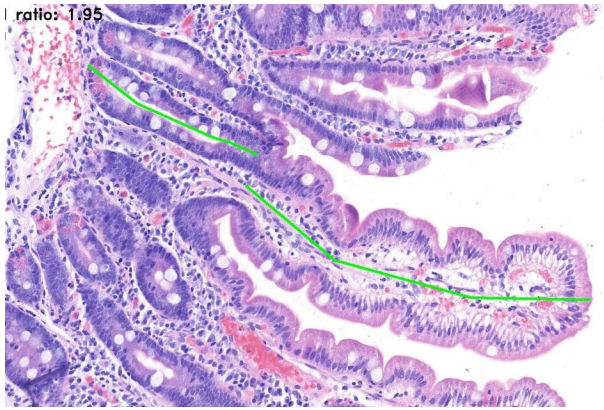
Key Morphologic and Inflammatory Measurements in Celiac Disease



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 Diet[3]

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

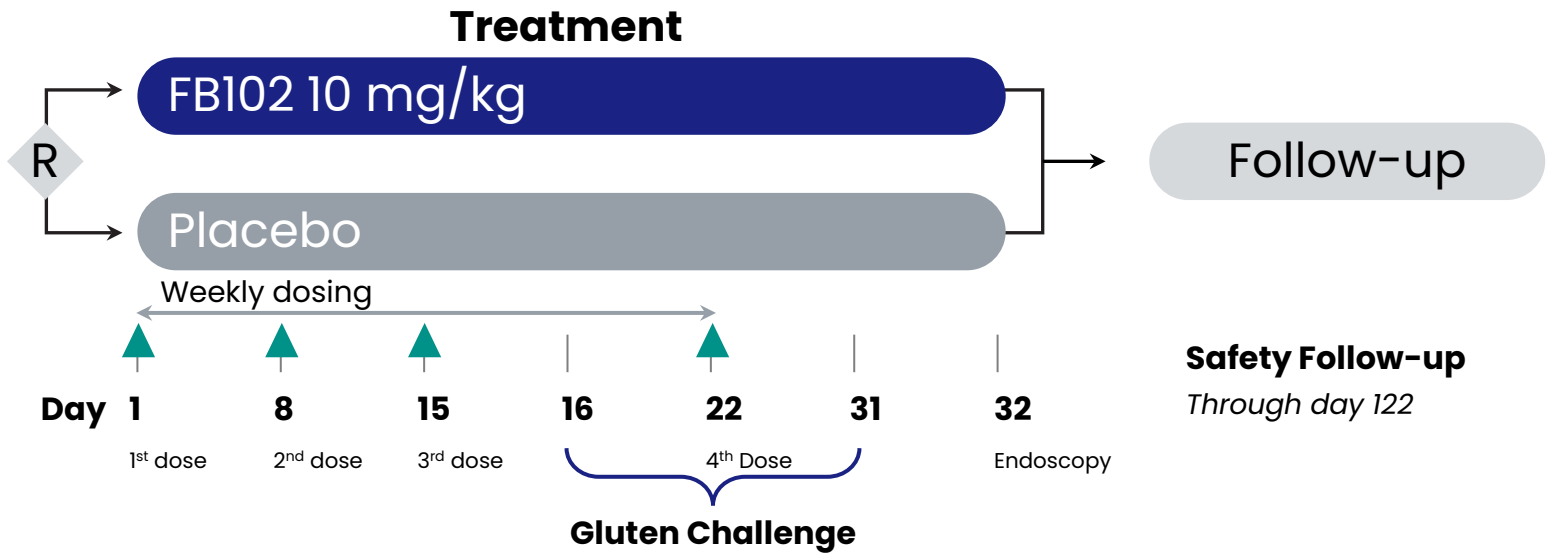
Proposed VCIEL composite scale comprising individual subject values for Vh:Cd and IEL equally weighted

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 Gastroenterolm Hepatol: 2024: 22: 1238-1244

FB102 Phase 1b Study in Celiac Disease



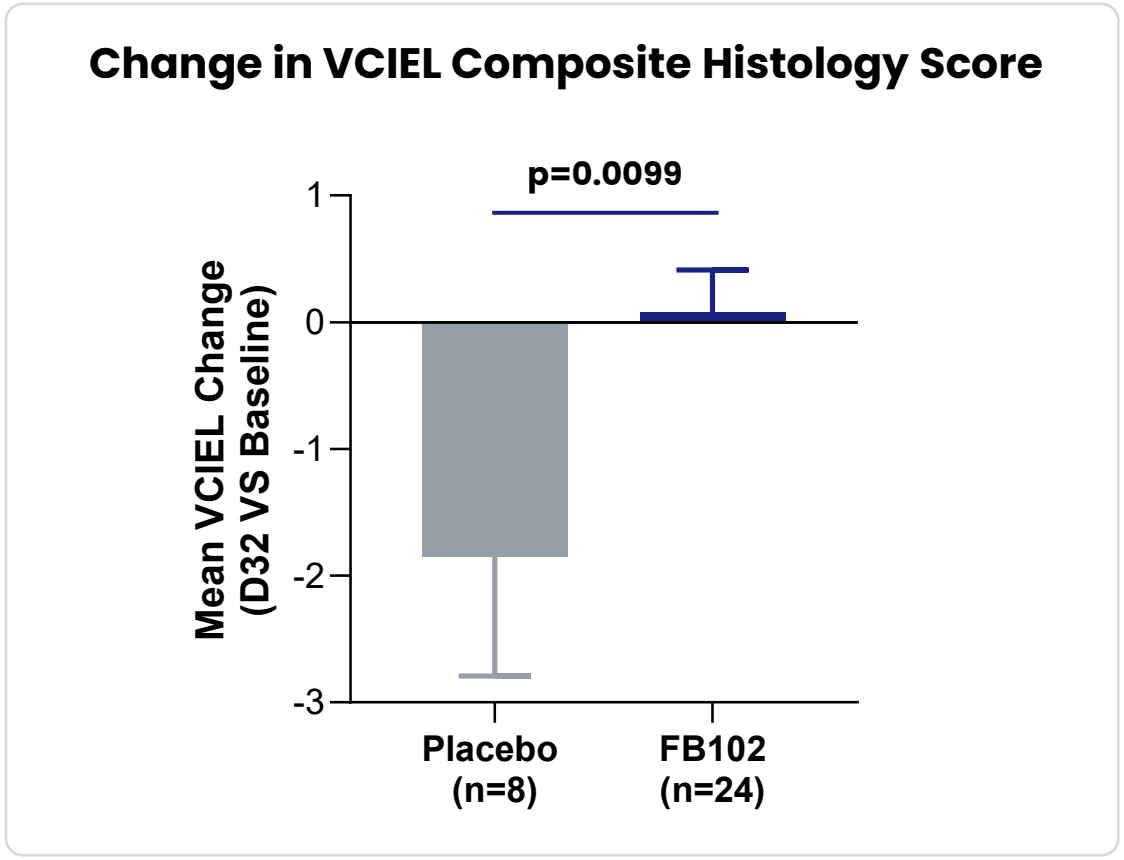
- 32 subjects, 9 sites
- Progressive Gluten Challenge (GC) with 2 g on D16, 4 g on D17 and 8g on Ds 18-31
- 3 of 4 weekly doses prior to 16-day gluten challenge followed by 4th dose on D22
- Safety and tolerability primary endpoint with endoscopy/biopsy at baseline and at end of gluten challenge (D32)
- 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

VCIEL= Villus Height to Crypt Depth with Intraepithelial Lymphocytes, IELs = Intraepithelial Lymphocytes, VH:Cd = Villus Height to Crypt Depth ratio

Baseline Demographics

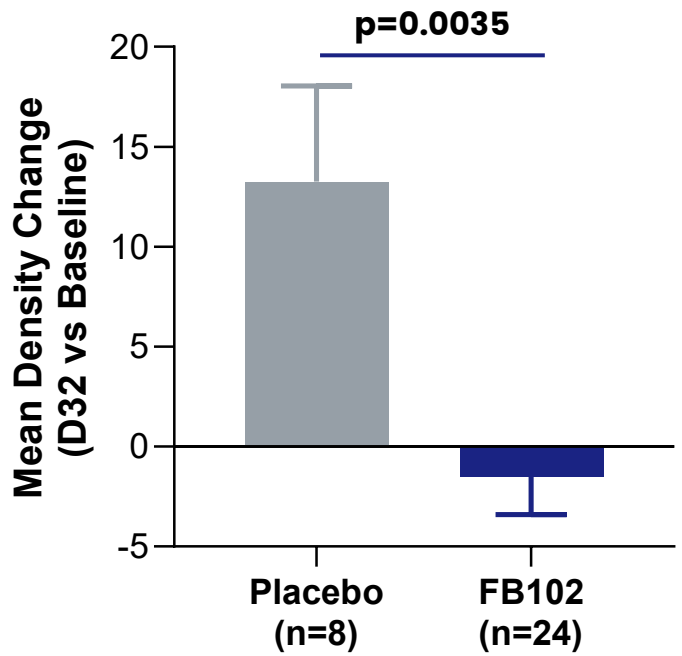
Parameter		Placebo N=8	FB102 N=24	Overall N=32
Age (Years)	Mean	38.3	40.8	40.1
Sex [n (%)]	Female	5 (62.5%)	19 (79.2%)	24 (75.0%)
	Male	3 (37.5%)	5 (20.8%)	8 (25.0%)
Ethnicity [n (%)]	Hispanic or Latino	0	0	0
	Not Hispanic or Latino	7 (87.5%)	23 (95.8%)	30 (93.8%)
	Not Reported	1 (12.5%)	1 (4.2%)	2 (6.3%)
	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



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

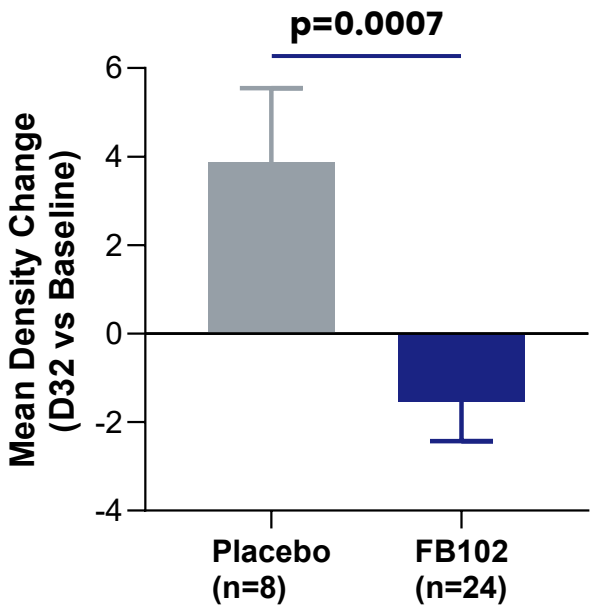
FB102 Demonstrates Statistically Significant Change in IEL Density Compared to Placebo



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

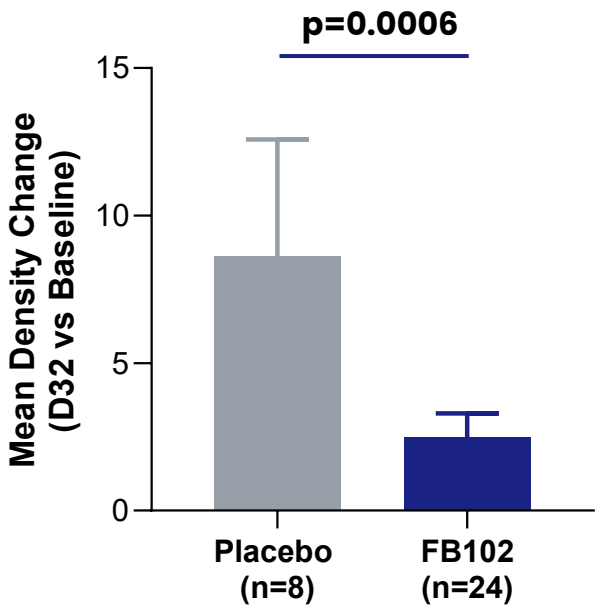
TCR γδ Positive IELs



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

Gluten interacts with receptors on TCR γδ IELs driving cytotoxic T cell expression and epithelial damage

Ki67 Positive IELs

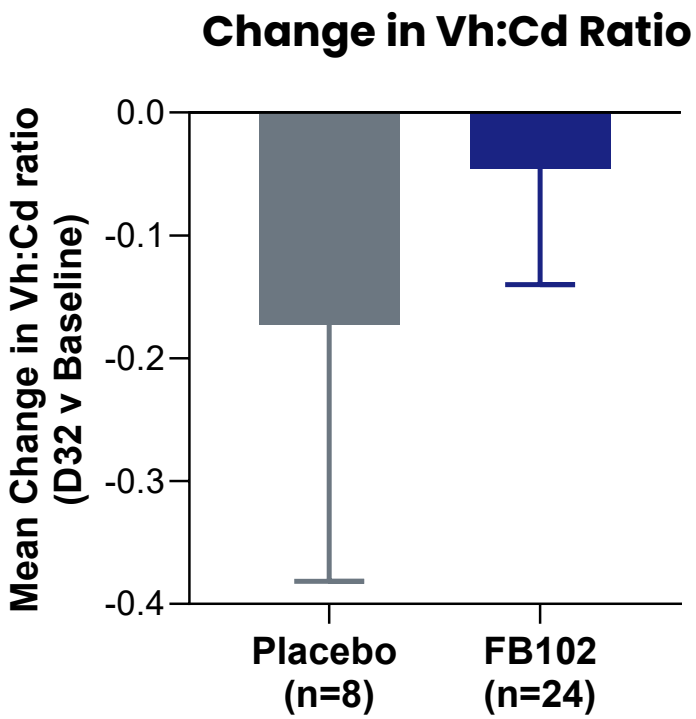


Increase of 8.6 for PBO compared to 2.5 for FB102 treated subjects

Ki67 is a marker of T cell proliferation (inflammation)

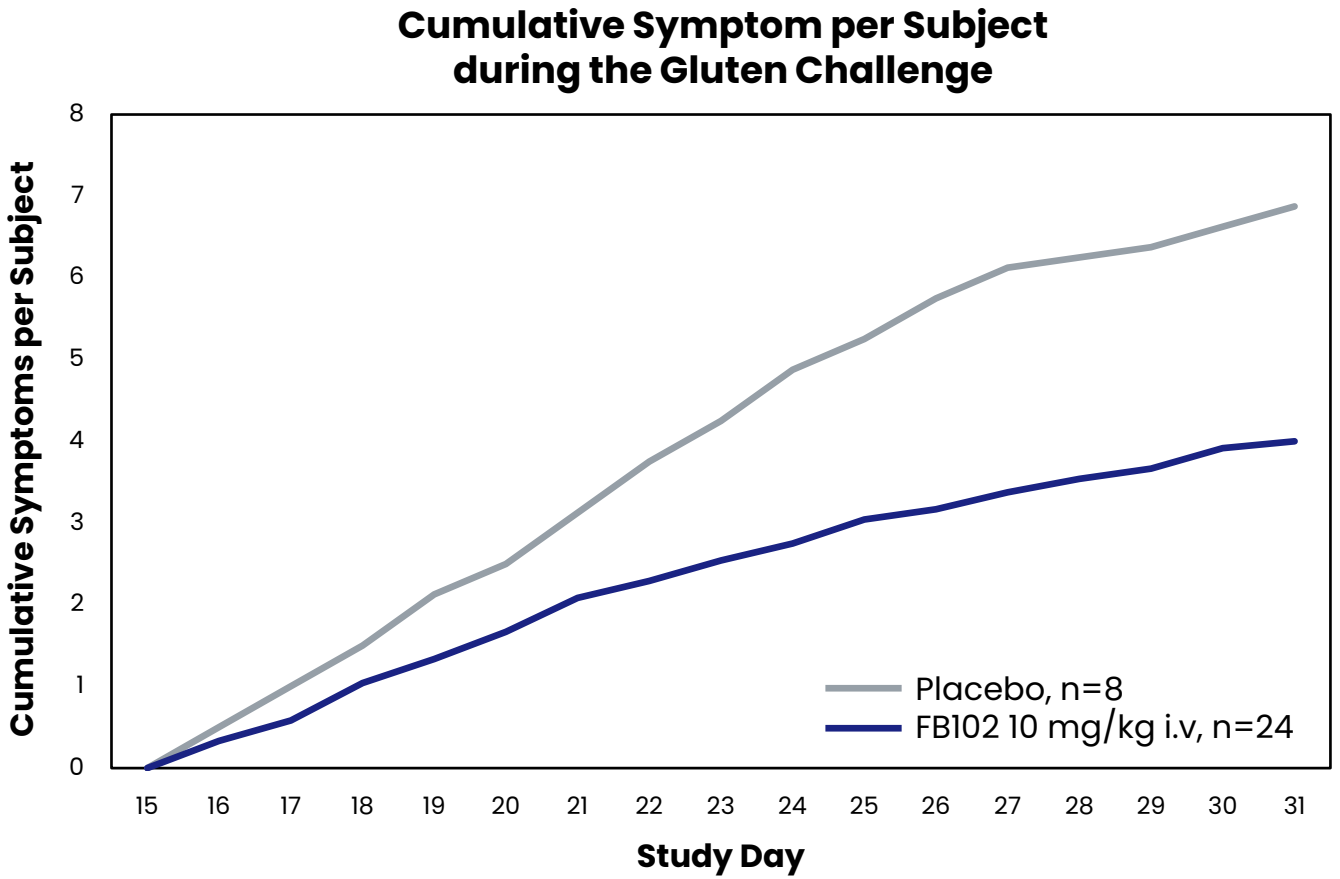
Based on analysis of covariance of changes from baseline to day 32, with baseline value as covariate, per 100 enterocytes

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



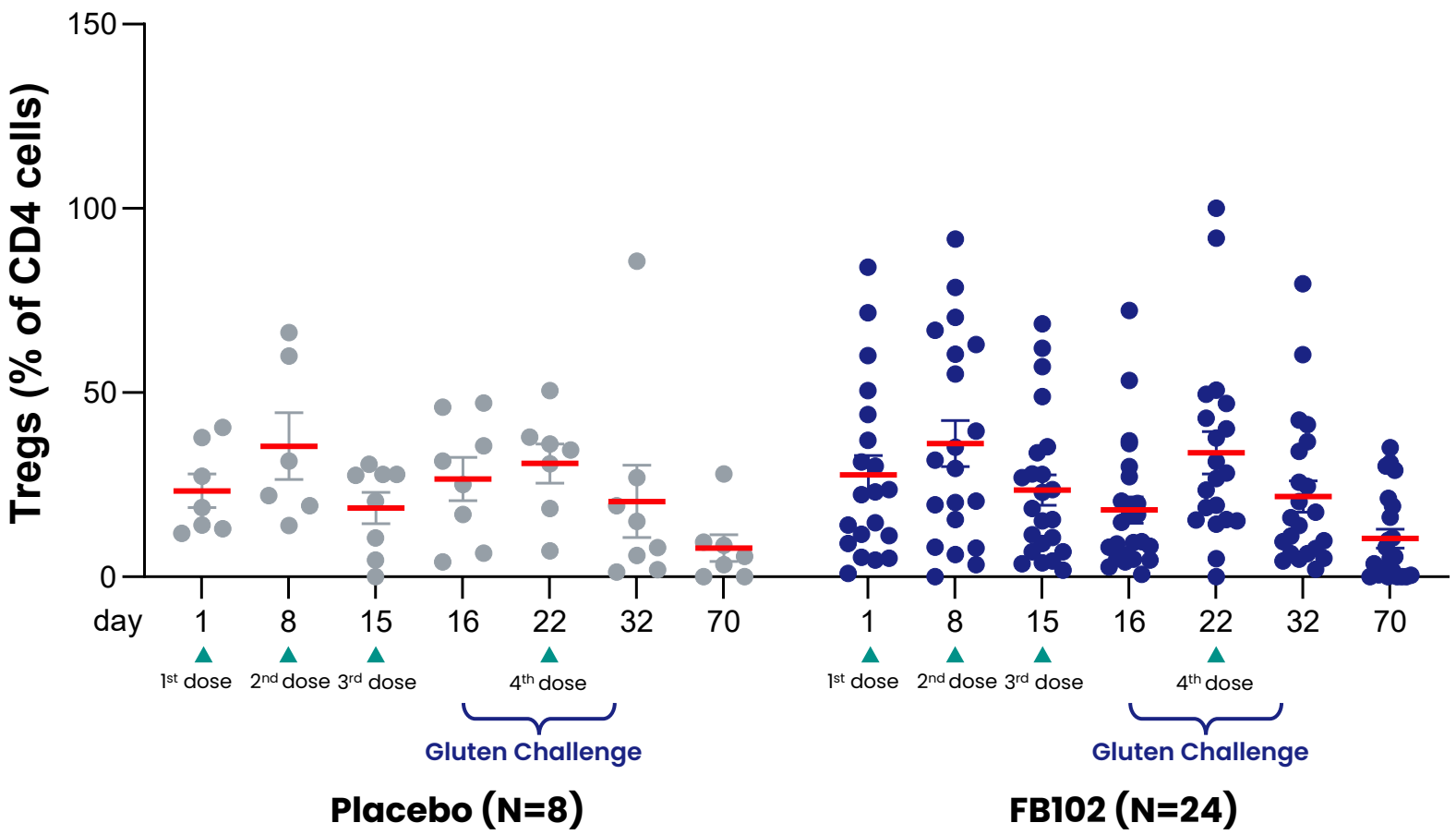
Symptoms monitored:
Nausea, diarrhea, vomiting, abdominal pain, abdominal bloating (patient diaries and AE collection)

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%

Subjects may be counted in more than one grade category

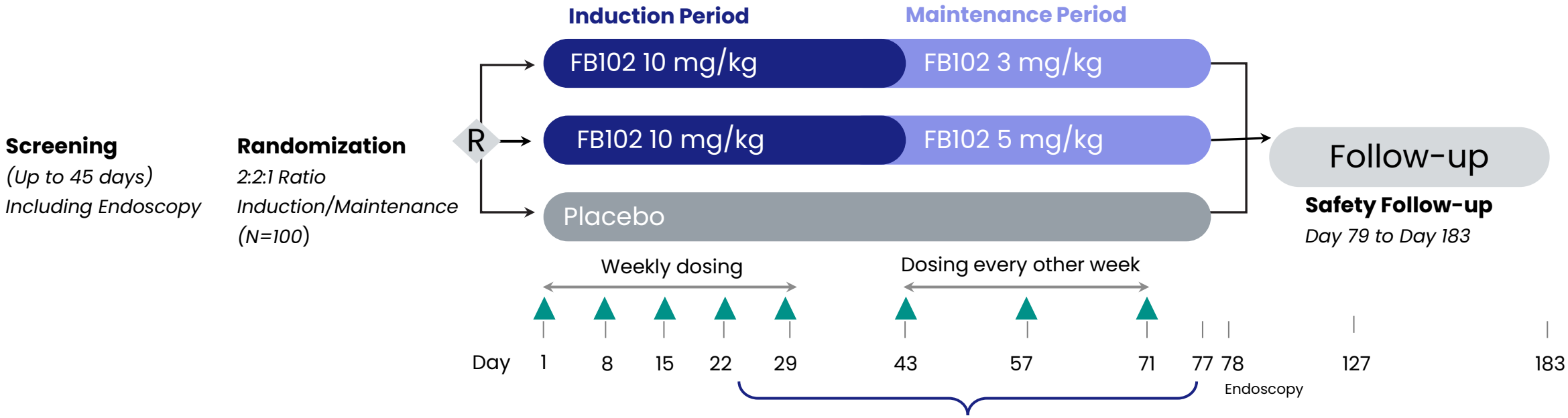
FB102 Generally Safe and Well Tolerated

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%

Subjects may be counted in more than one grade category

Celiac Disease Phase 2 Design FB102-301



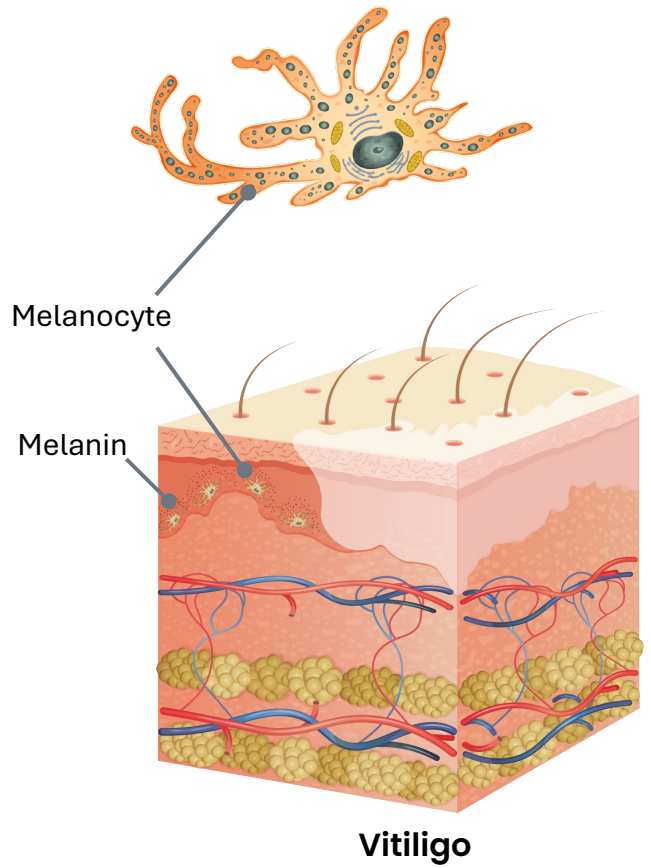
- 14 sites AUS/NZ initially
- Initiation in 2H25
- US IND expected late 2025/early 2026
- Men and women aged 18 to 70 years
- Has documented diagnosis of CeD
- Body mass index (BMI) 16.0 - 40.0 kg/m^2, inclusive
- Self-reported to be on a GFD for at least 12 months prior to Screening
- Primary Endpoint: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

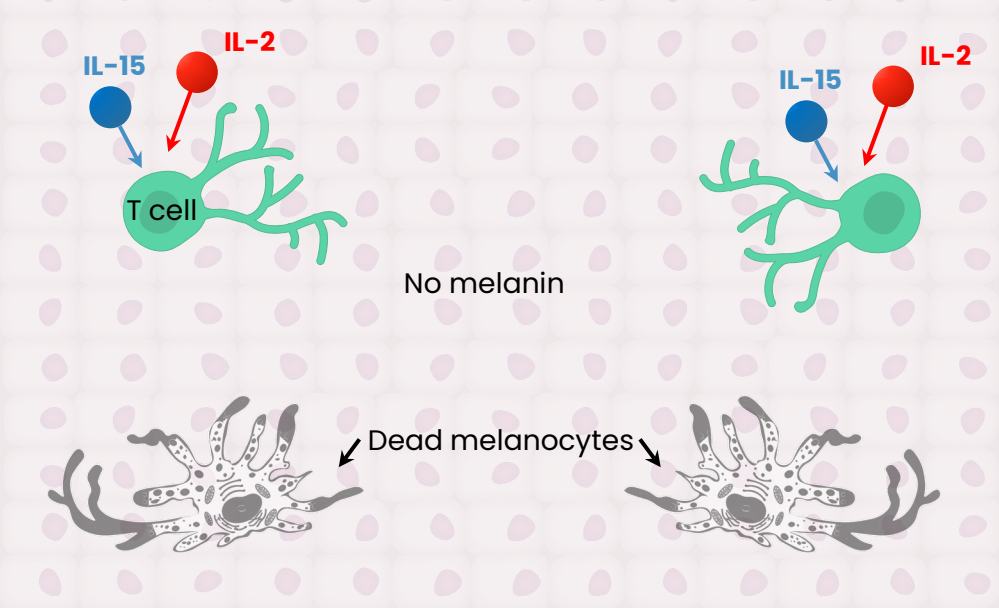
Disease Overview	• Autoimmune condition in which pathogenic T cells destroy melanocytes resulting in depigmented (white) skin patches • Non-Segmental vitiligo: 85-90% of cases, bilateral • Segmental vitiligo: 10-15% of cases, unilateral
Epidemiology	• Estimated global prevalence: 0.5-2% globally, including 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



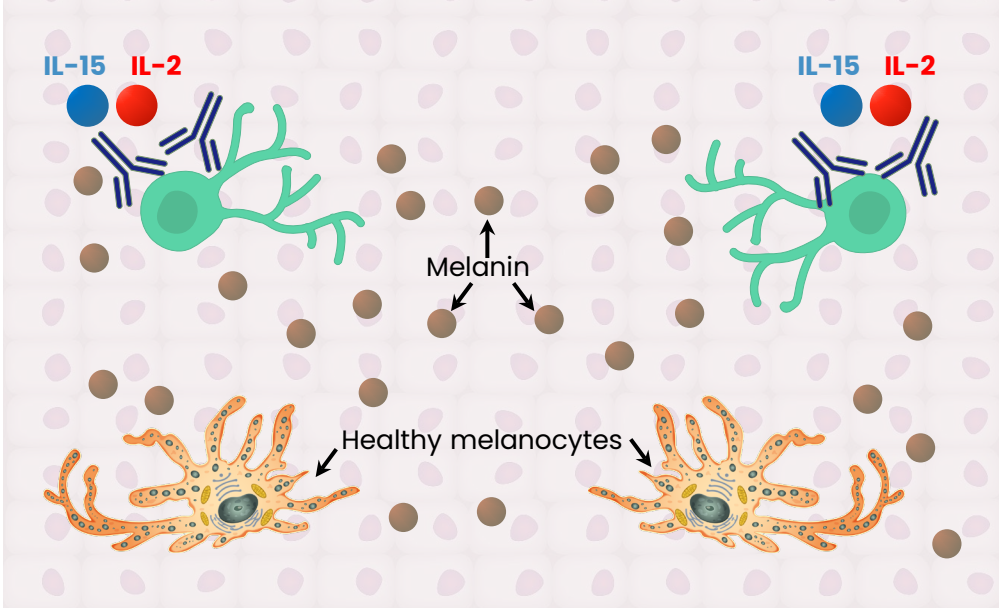
https://my.clevelandclinic.org/health/diseases/12419-vitiligo
MACE: Major adverse cardiovascular events including stroke, heart attack, and cardiovascular death

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



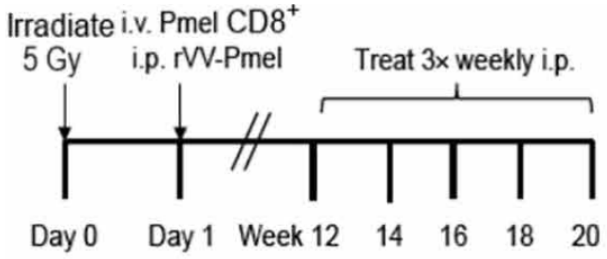
Vitiligo has been observed in responders to high-dose IL-2 in melanoma, with multiple studies linking this to robust immune activation, reinforcing IL-2's role in driving vitiligo[1]

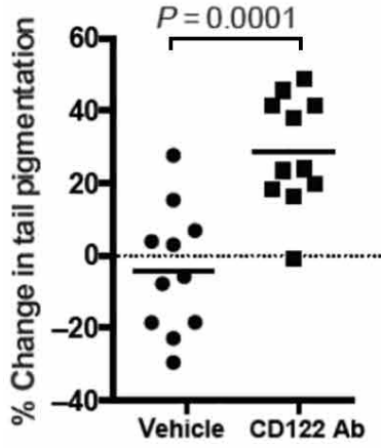
Adapted from Tokura, et al. Front Immunol. 2021 PMID: 33633737

[1.] Phan, et al. J Clin Oncol. 2001 PMID: 11481353

Anti-CD122 Antibody Reverses Disease in a Vitiligo Mouse Model

Repigmentation study





Melanocyte reactive T cells eliminate tail pigmentation

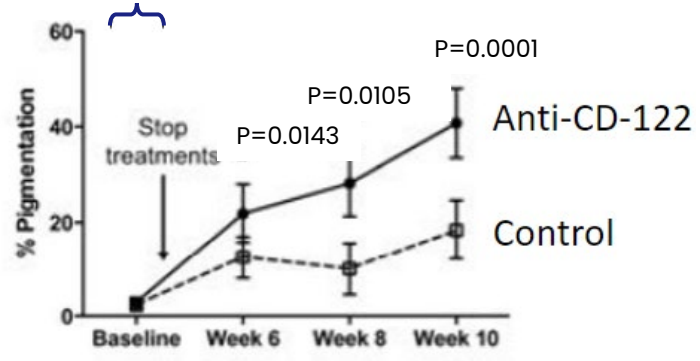
Anti-CD122 treatment restores pigmentation

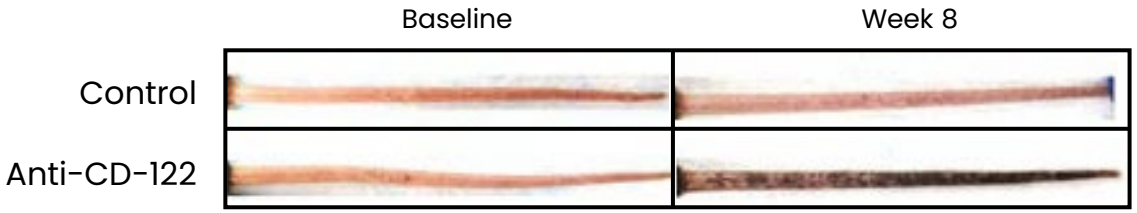
i.p. = intraperitoneally
Richmond, et al. Sci Transl Med. 2018 PMID 30021889
Note: Vitiligo induced in C57BL/6J (B6) mice via adoptive transfer of anti melanocyte antigen T cells with anti-mouse CD122 (surrogate molecule) antibody used

Short Course Anti-CD122 Demonstrates Durable Response

Systemic

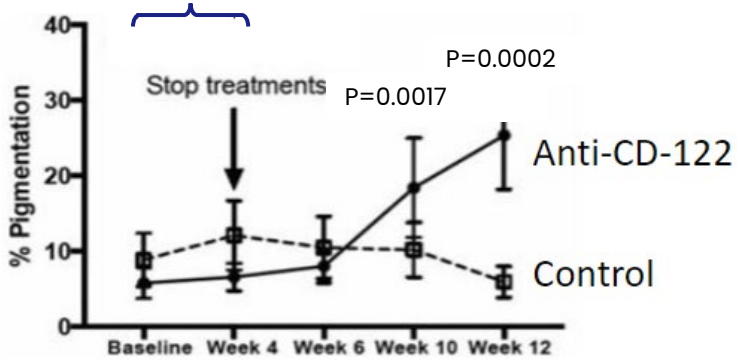
3x weekly i.p. x 2 weeks

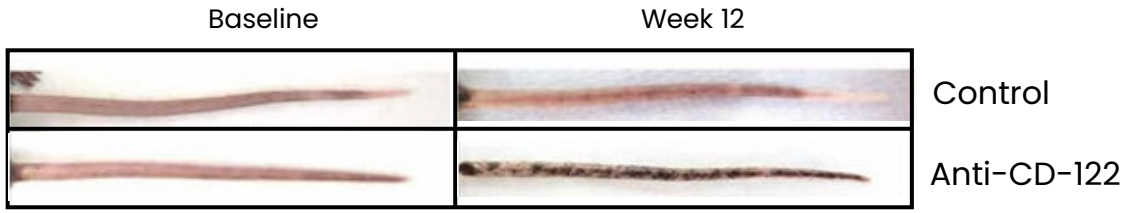




Local

3x weekly i.d. x 4 weeks





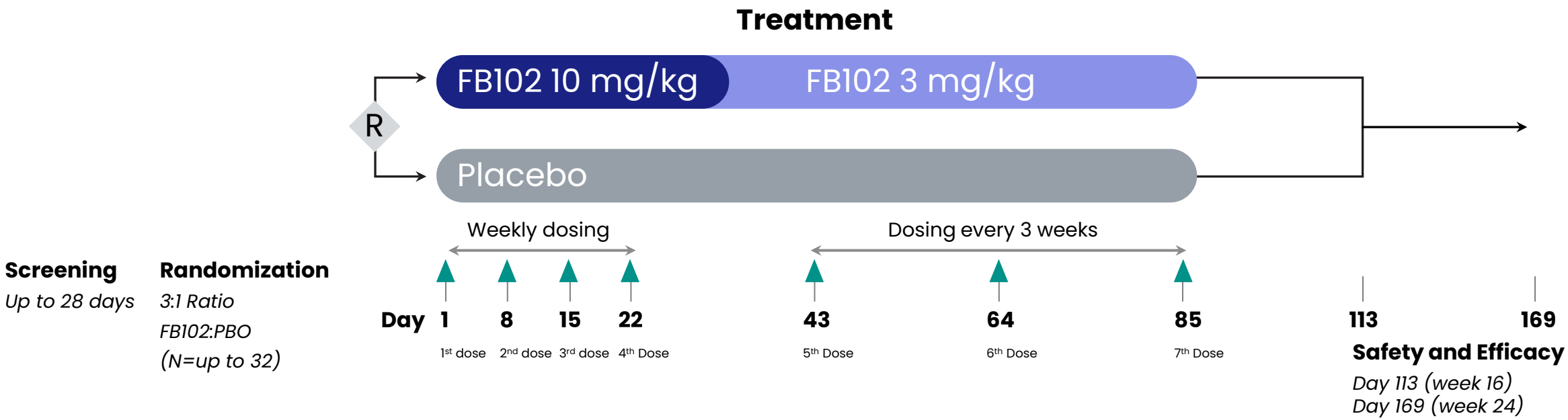
Comparable effect with systemic or local treatment
Lower injection burden may improve patient adherence

i.d. = intradermal
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

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) Pfizer	IL-17A → aixekizumab (Taltz), Lilly	**CD122 (IL-2/IL-15) → FB102** FORTE BIOSCIENCES, INC
JAK 1/3 → Tofacitinib (ZELJANZ) Pfizer	IL-17A → secukinumab (COSENTYX) NOVARTIS	IL-12/23 → ustekinumab (Stelara) janssen
JAK1/2/3 TYK2 → Upadacitinib abbvie	IFNα → anifrolumab AstraZeneca	CD122 (IL-2/IL-15)→ INCA34460 Incyte
JAK1 → povorcitinib (Incyte INCB54707) Incyte	TNF-α → adalimumab (Humira), etanercept TNF-α, infliximab (Remicade), abbvie AMGEN janssen	

Vitiligo Phase 1b Design FB102-401



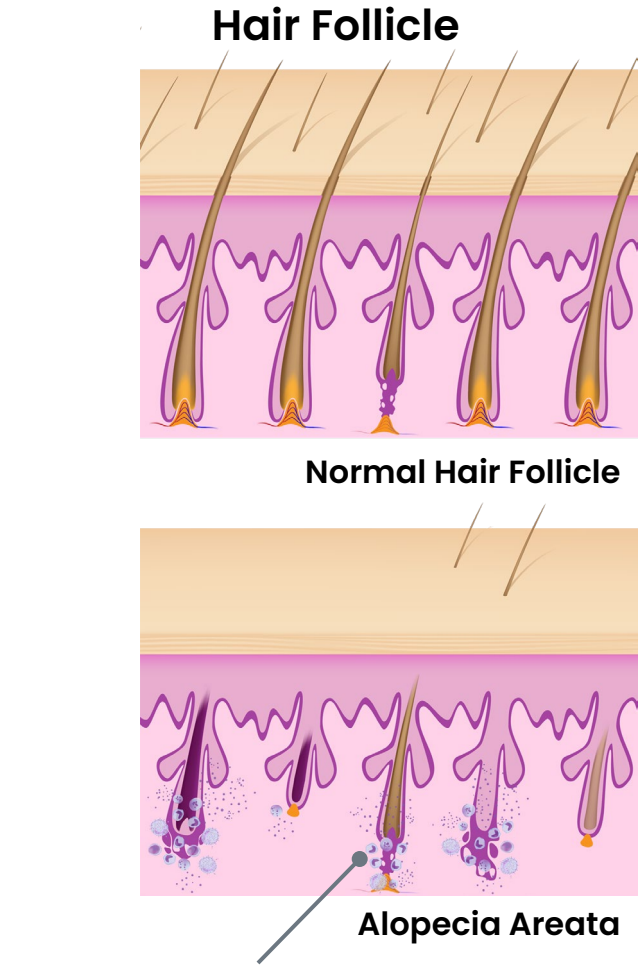
- Currently enrolling
- 10-12 sites in AUS/NZ
- Males and females aged 18 to 75 years
- Clinical diagnosis of non-segmental vitiligo
- Primary endpoints: F-VASI at 16 and 24 weeks

Topline data readout expected in 1H 2026

F-VASI: Facial Vitiligo Area Scoring Index

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

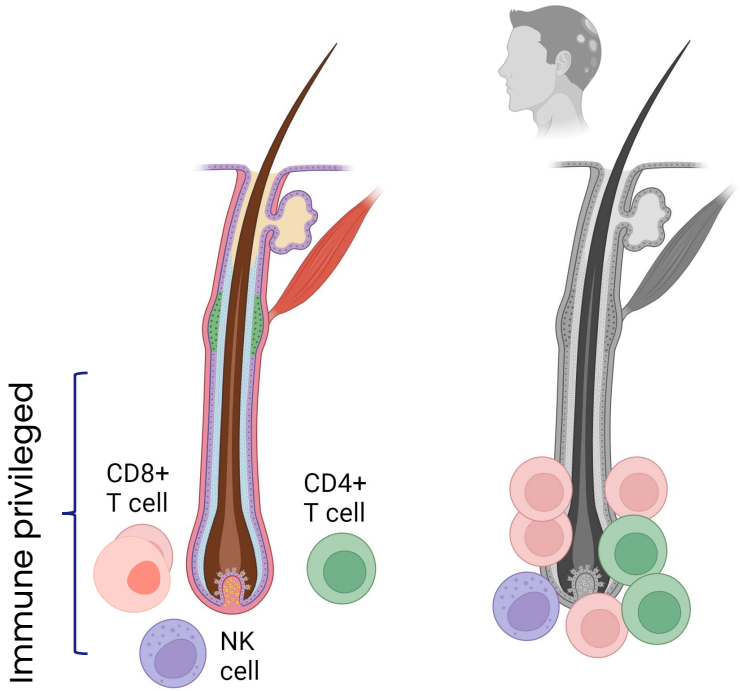
Disease Overview	• Common autoimmune disease that occurs when pathogenic T cells attack hair follicles causing hair loss • Sudden onset, affecting scalp, face and body
Epidemiology	• Prevalence: 2% of global population at some point in lifetime, including ~ US 6.5M, with 700K currently experiencing AA • 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



www.naaf.org/
www.niams.nih.gov/health-topics/alopecia-areata

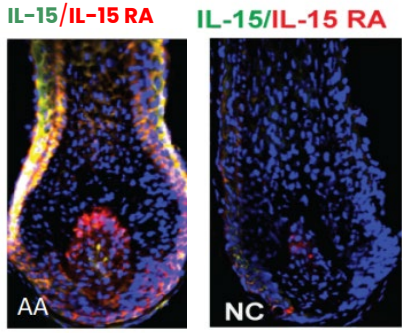
Alopecia Areta Driven by T Lymphocytes Invading Hair Follicles

- Healthy anagen (growth cycle) hair bulb is an immune privileged site – no MHC Class 1 expression and protected from immune attack[1]
- 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 cells[2]
- IL-2 is elevated in AA patients[3]; IL-15 is overexpressed in AA hair follicles[4]

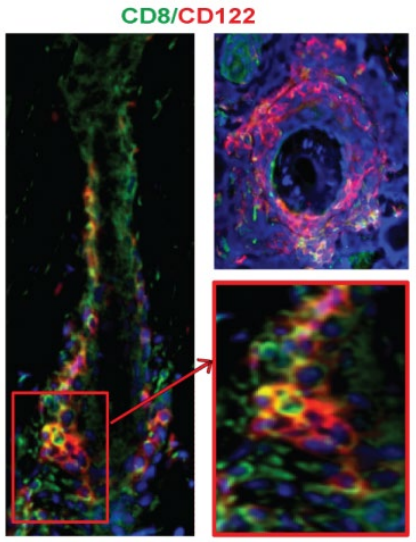


Healthy follicle
- Immune privileged space
- No MHC expression

Alopecia follicle
- Loss of immune privilege
- Invasion of T and NK cells
- Cytokines (e.g. IL-15, IL-2)
- MHC expression
- Hair loss



Co-expression of IL-15 and IL-15 RA in alopecia areata (AA) but not normal control (NC) [4]



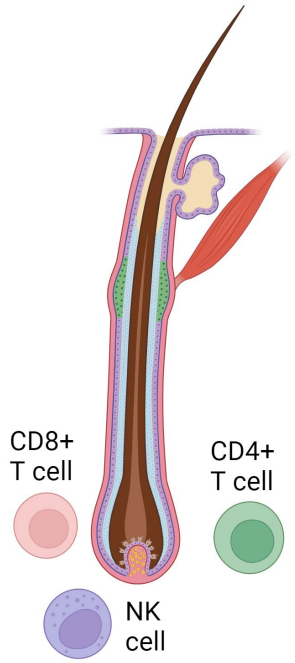
CD122+ CD8+ T cells in follicles from AA patients[4]

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

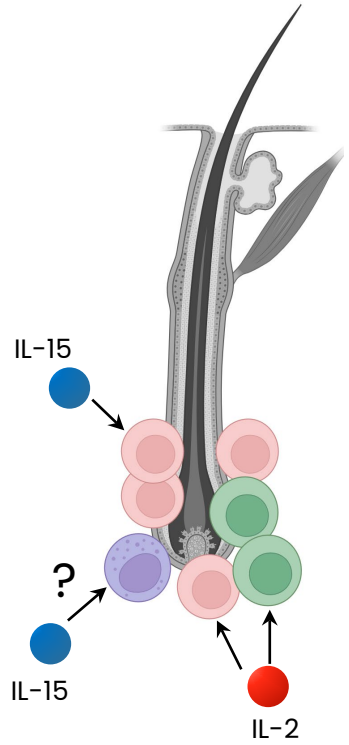
[1]Bertolini Exp Dermatol. 2020 PMID: 32682334
[2]Ito Clin Dev Immunol. 2013 PMID: 24151515
[3]Ito Exp Dermatol. 2014 PMID: 25040075
[4]Xing Nat Med. 2014 PMID: 25129481, Supplementary material

FB102 Anti-CD122 Antibody Potentially Restores Immune Privilege in AA

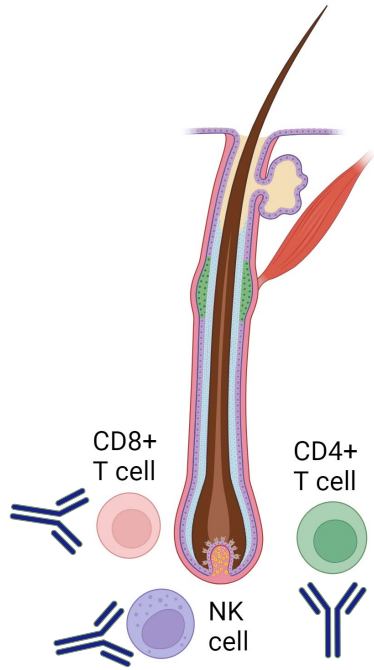
Immune privileged healthy cell



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



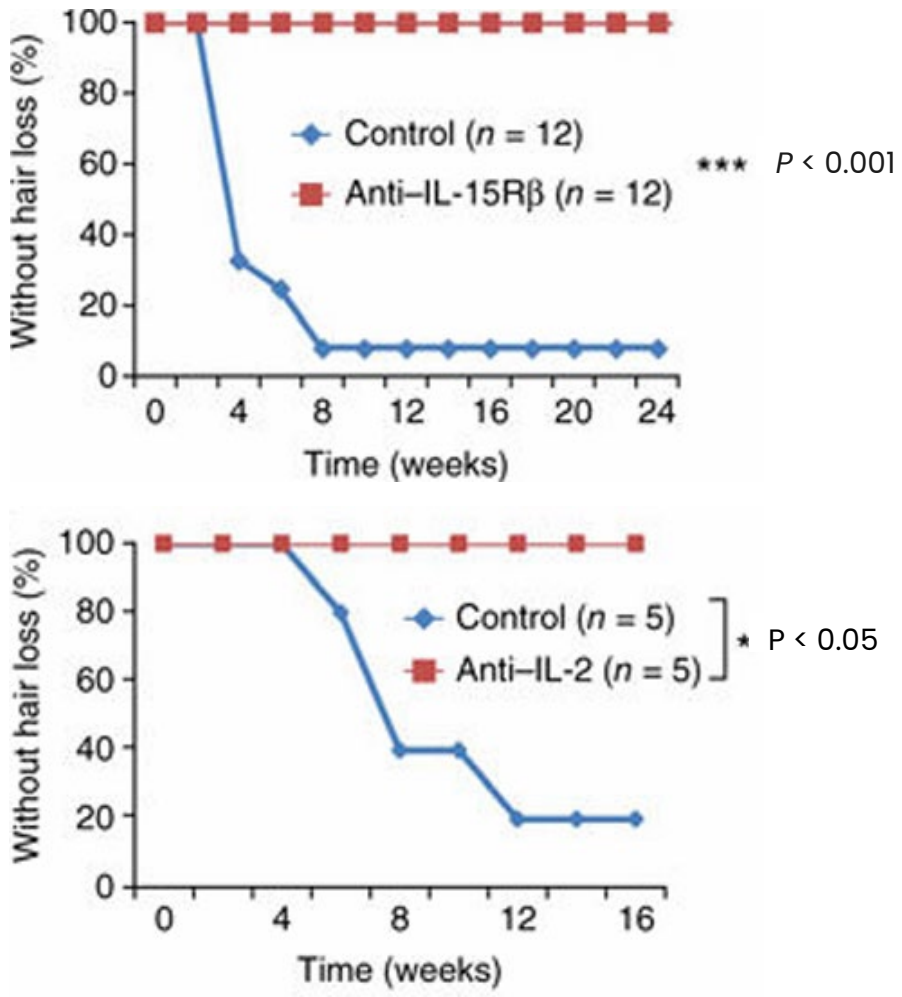
FB 102 blocks CD122 and prevents T and NK cells from attacking the hair follicle



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 days[1]
- Pathology resembles human disease
- Alopecia prevented with:
  - Anti-CD122 antibody, anti-IL-15Rβ, dosed 200 µg twice per week for 12 weeks
  - 2 combined anti-IL-2 antibodies, dosed 250 µg each three times per week[2]

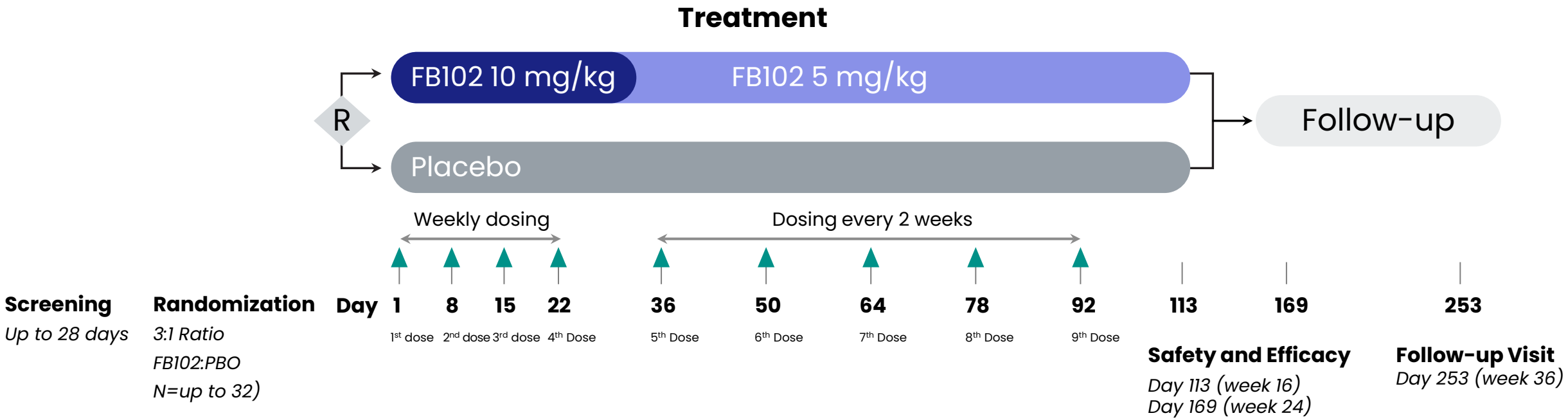


Clone TM-β1= anti-IL-15Rβ used
[1]McElwee J Invest Dermatol. 1998 PMID: 9804341
[2]Xing Nat Med. 2014 PMI 25129481

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)	S1P → etrasimod (VELSIPITY)	**CD122 (IL-2/IL-15) → FB102**
		FORTE BIOSCIENCES, INC
JAK 1/2 → baricitinib (Olumiant)	OX40 → IMG-007	IL-4/13 → dupilumab (Dupixent)
JAK 1/2 → deuruxolitinib (LEQSELVI)	IL-2 → Rezpegaldesleukin	IL7/TSLP → bempikibart
JAK1/2/3 TYK2 → Upadacitinib		IL-2/9/15 → EQ101

Alopecia Areata Phase 1b Design FB102-701



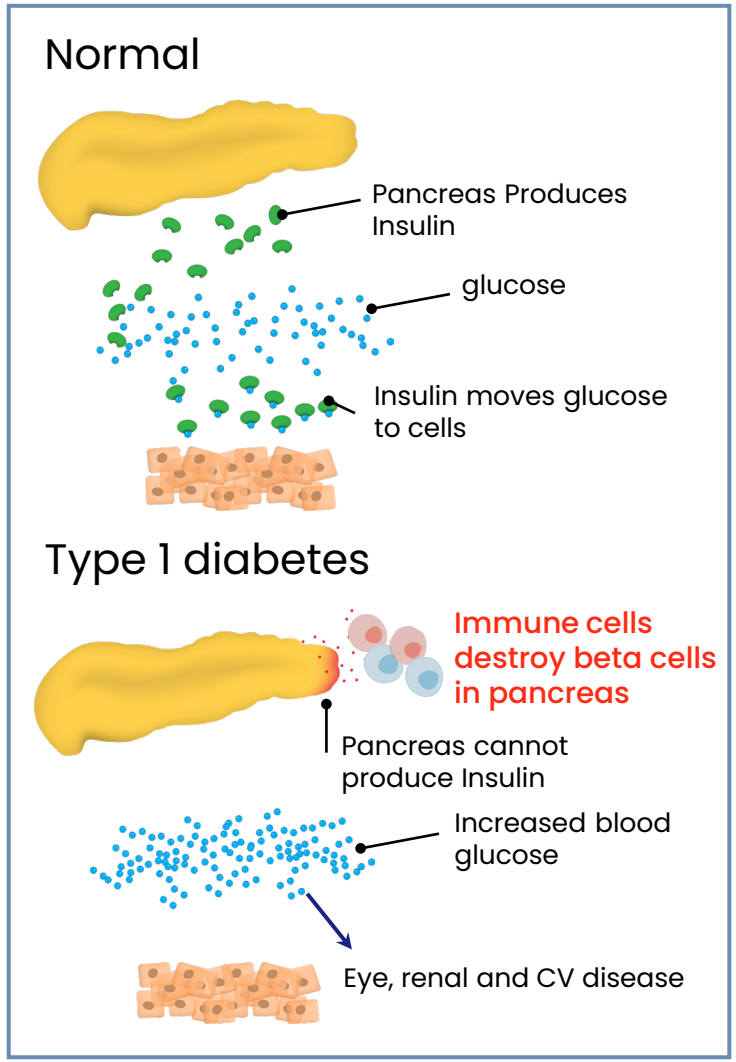
- 10-12 sites in AUS/NZ
- Primary endpoint: SALT at 16 and 24 weeks
- Males aged ≥18 to 60 and females aged ≥18 to 75 years
- Clinical diagnosis of severe to very severe AA with no other etiology for hair loss

Topline data readout expected in 2026

SALT: Severity of Alopecia Tool

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 annually[1]
Consequences	• Eye, renal and cardiovascular disease
Current Treatments	2 therapies approved to delay onset of TID 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



https://beyondtype1.org/type-1-diabetes-statistics/

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:

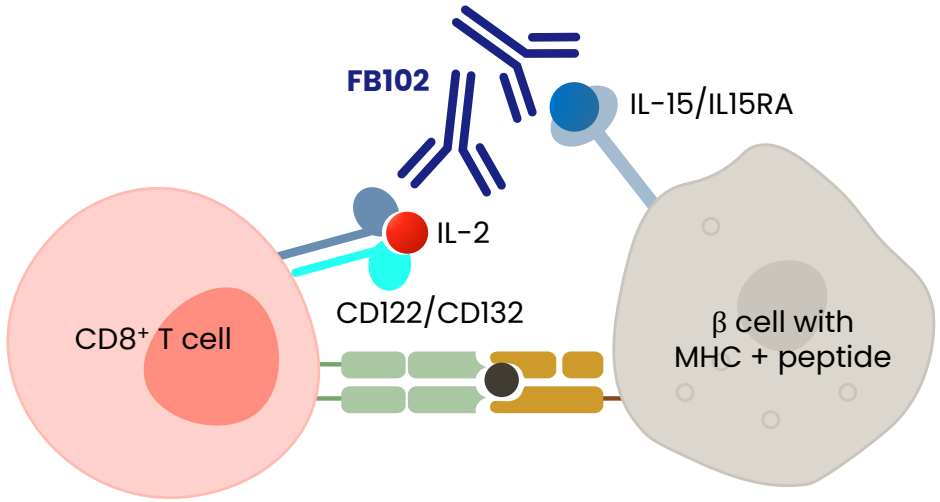
- cytokine release syndrome (2% vs 0%)
- serious infections‖ (9% vs 0%)
- hypersensitivity reactions and serum sickness (2% vs 0%)
- lymphopenia (73% vs 6%)
- neutropenia (7% vs 3%)

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

www.tzieldhcp.com/safety-data/
LANTIDRA PI

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

FB102 blocks IL-2/IL-15 binding

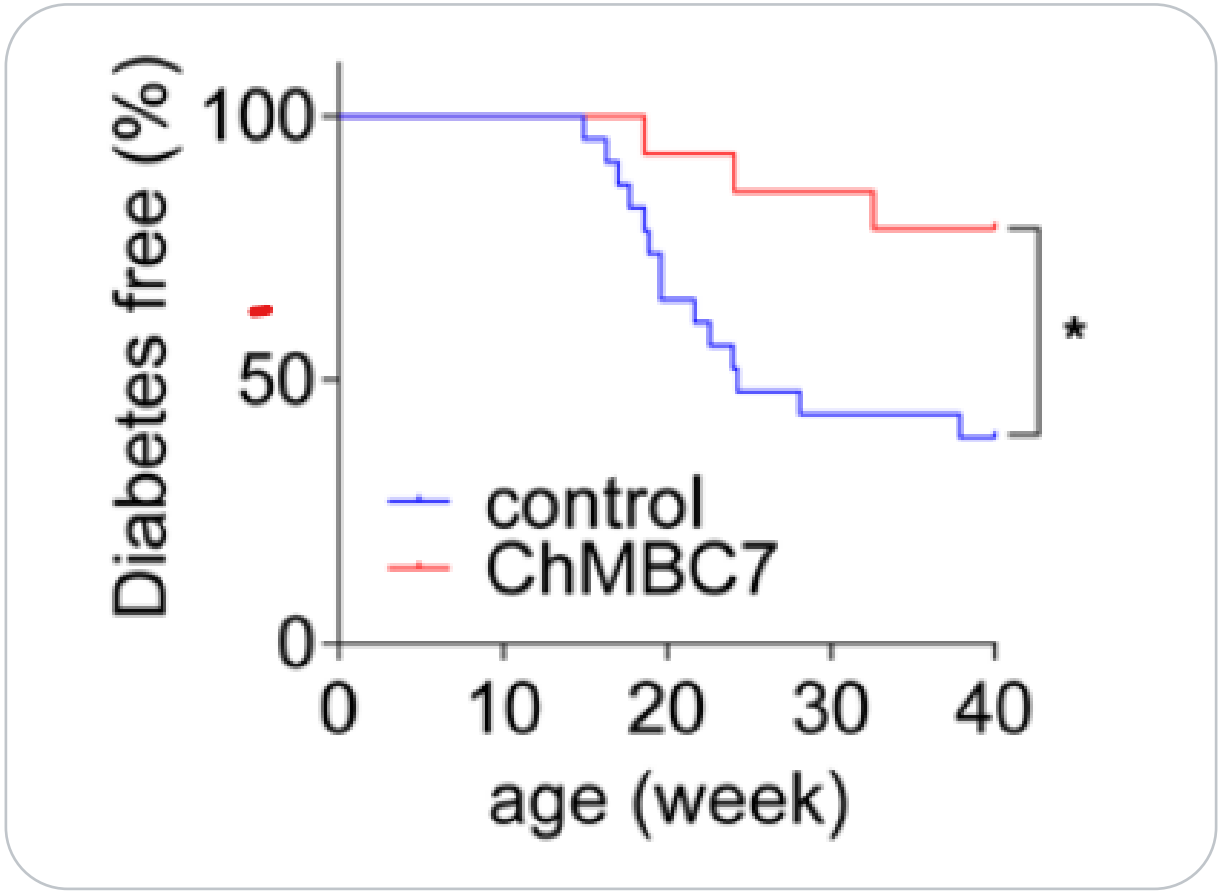


CD8+ T cells with receptors recognizing beta cell specific peptides are enriched in pancreatic islets of T1D patients

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

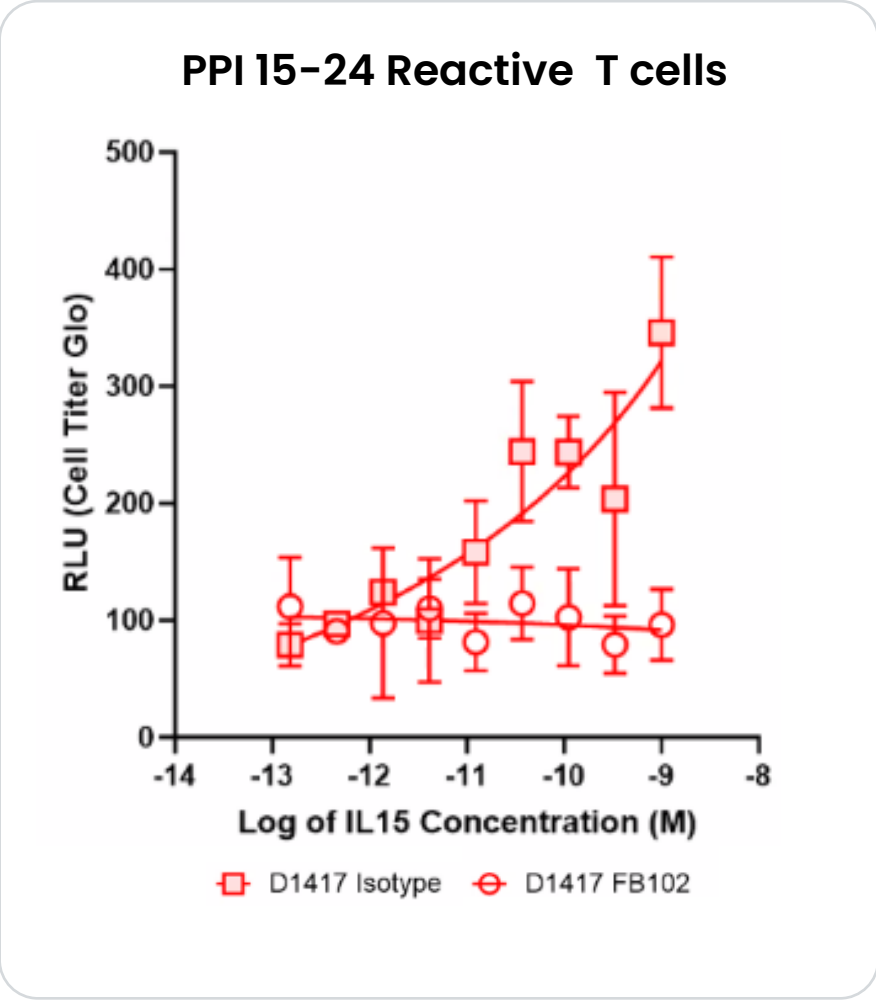
Herold 2024 Nat Rev Immunol. PMID 38308004

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

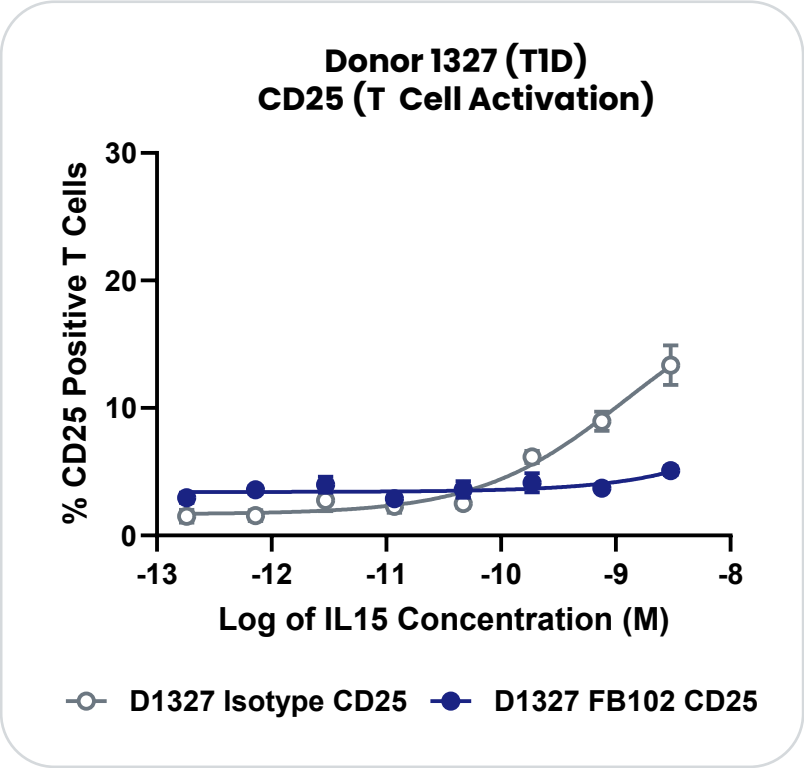


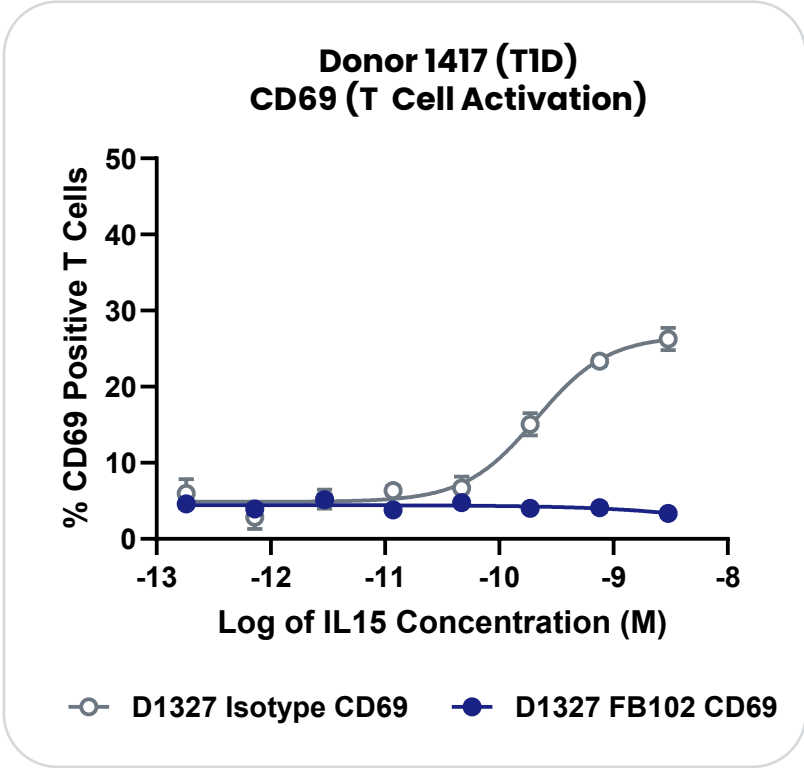
Autoreactive T cells obtained from Type 1 Diabetes patients co-cultured with IL-15 with and without FB102

FB102 inhibits beta cell induced proliferation of T1D T cells

PPI 15–24 refers to amino acids 15 to 24 of the preproinsulin protein sequence, which act as β-cell autoantigens recognized by T cells in T1D.

FB102 Inhibits T1D T Cell Activation



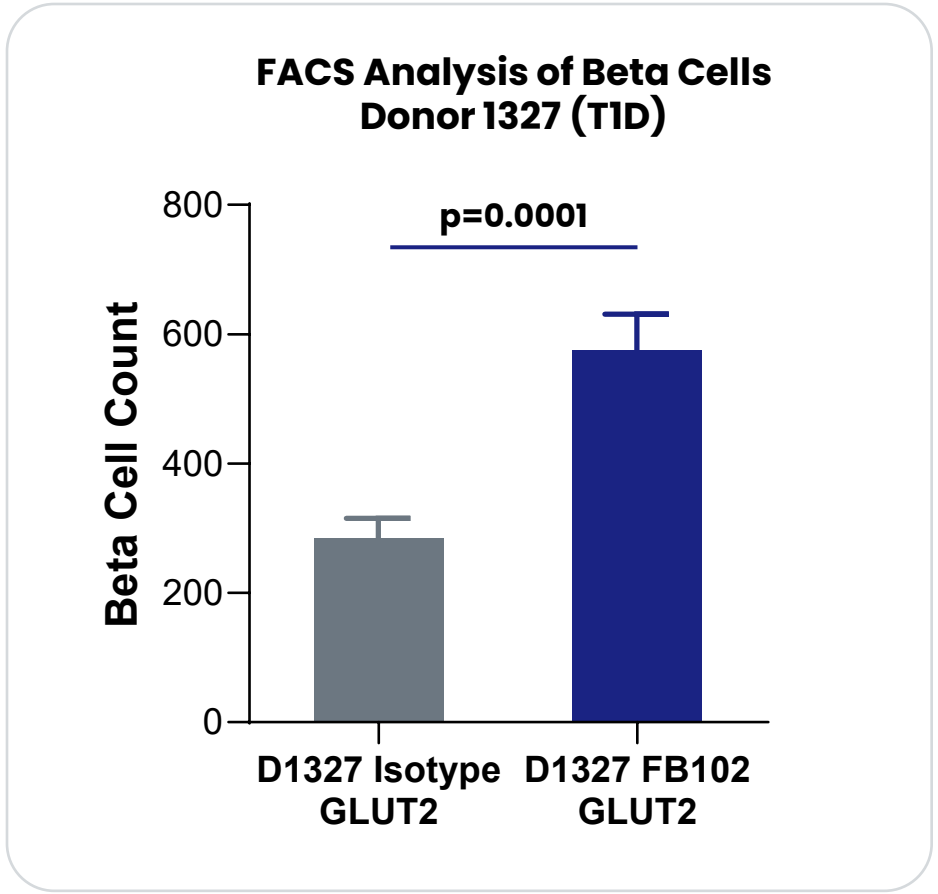


Activated TID T cells are measured by an increase in % CD69 and CD25 T cells

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

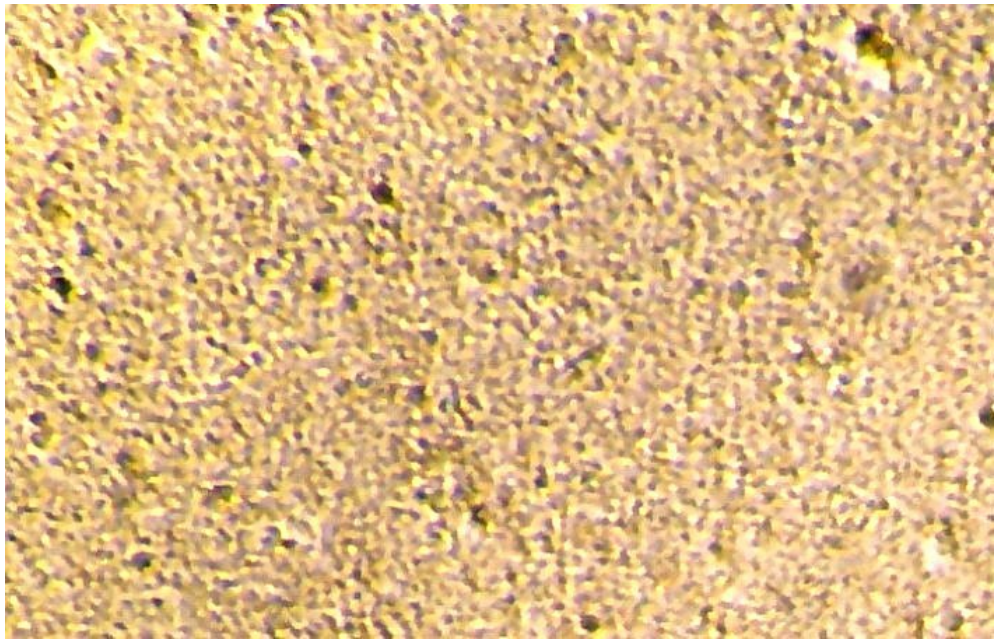


Beta cell killing by T1D reactive T cells is inhibited by FB102 vs isotype control

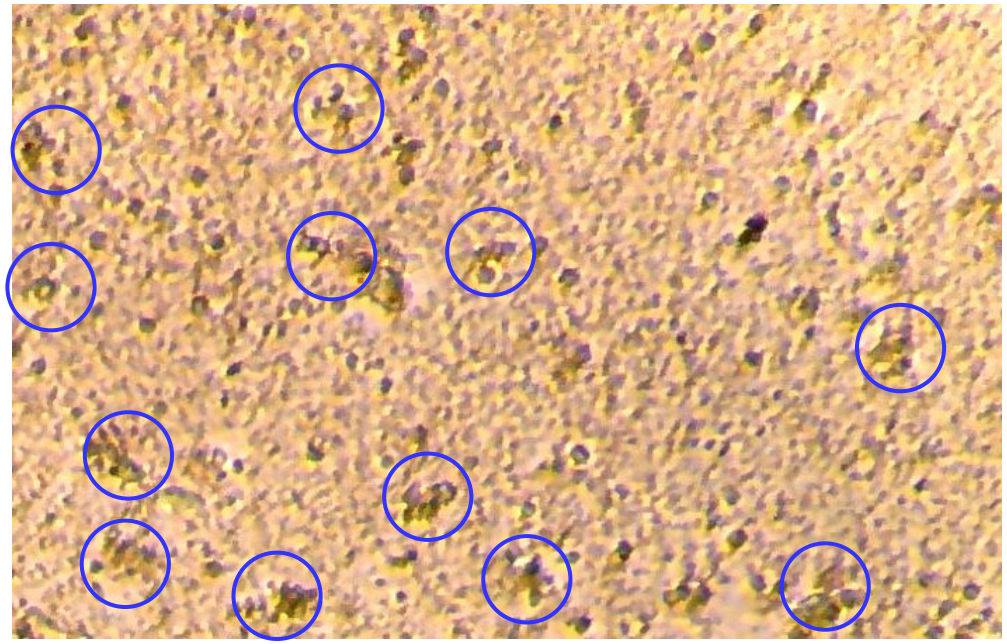
FACS (Fluorecence-Activated Cell Sorting) used to analyze beta cell survival

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

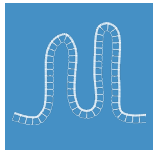
Program	Development	Phase 1	Phase 2	Phase 3	Upcoming Milestones
Celiac Disease					Phase 1b Topline data readout June 2025 Phase 2 Initiated 2H25 Phase 2 Topline data expected in 2026
Vitiligo					Phase 1b Initiated 1H25 Topline data expected in 1H26
Alopecia Areata					Phase 1b initiated in 2H25 Topline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