UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 23, 2025

FORTE BIOSCIENCES, INC.
(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-38052**	**26-1243872**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

3060 Pegasus Park Dr. **Building 6** **Dallas, Texas**	**75247**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (310) 618-6994

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value	FBRX	The NASDAQ Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure.

On June 23, 2025, Forte Biosciences, Inc. (the "Company") issued a press release announcing positive data from a Phase 1b trial in celiac disease for lead program FB102. As part of the press release, the Company announced that it would be hosting a conference call at 8:30 a.m. ET on June 23, 2025 to review the study results. A copy of the press release is furnished herewith as Exhibit 99.1 to this Current Report on Form 8-K.

All of the information furnished in this Item 7.01 (including Exhibit 99.1) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such a filing.

Item 8.01. Other Events.

In connection with the conference call to review the Phase 1b trial results, the Company will be reviewing the Celiac Disease Phase 1b Results Presentation attached hereto as Exhibit 99.2, which is incorporated herein by reference.

The Company is also filing an updated corporate deck in connection with the Phase 1b trial results, which is attached hereto as Exhibit 99.3 and incorporated herein by reference.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit	Description
99.1	Press Release, dated June 23, 2025.
99.2	Celiac Disease Phase 1b Results Presentation, June 2025.
99.3	Corporate Presentation, June 2025.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

FORTE BIOSCIENCES, INC.

Date: June 23, 2025

By: /s/ Antony Riley
Antony Riley
Chief Financial Officer

Exhibit 99.1

FORTE BIOSCIENCES, INC

FORTE BIOSCIENCES ANNOUNCES POSITIVE DATA IN FB102 CELIAC DISEASE PHASE 1B STUDY

Phase 2 Celiac Disease Trial Initiating with Topline Readout Expected in 2026
Forte will be Hosting a Conference Call to Discuss the Results at 8:30 am ET

DALLAS, TX – JUNE 23, 2025 – Forte Biosciences, Inc. (www.fortebiorx.com) (NASDAQ: FBRX), a clinical-stage biopharmaceutical company focused on autoimmune and autoimmune-related diseases, today announced positive data from a Phase 1b trial in celiac disease for lead program FB102(FB102-101).

The company will be hosting a conference call today at 8:30 am ET. Prof. Jason Tye-Din, Head of Celiac Research at the Walter and Eliza Hall Institute and principal investigator in the FB102-101 study will be participating in the call.

Please connect to the call using the following link: https://lifescievents.com/event/jgs927tsivn4w038/. The event and accompanying slides can also be accessed by visiting the investor relations section of the company's website at https://www.fortebiorx.com/investor-relations/default.aspx. An archived webcast will be available on the company's website following the event.

The FB102-101 Phase 1b celiac disease study enrolled 32 subjects 3:1 randomized (24 on FB102 and 8 on placebo). Subjects received 4 doses of FB102 (10 mg/kg) and underwent a 16 day gluten challenge. In addition to safety and tolerability, the study assessed morphologic and inflammatory endpoints along with gluten challenge (GC) induced symptoms.

FB102 demonstrated a statistically significant benefit on the composite histological VCIEL endpoint (change from baseline). The mean VCIEL change from baseline was -1.849 for placebo subjects compared to 0.079 for FB102 treated subjects (p=0.0099).

The change in the density of CD3-positive T cells, or IELs, from baseline was an increase of 13.3 for placebo subjects compared to a decline of 1.5 for FB102 treated subjects (p=0.0035). Baseline IEL density was 25.6 for the placebo subjects and 23.5 for the FB102 treated subjects.

The mean change in the Vh:Cd ratio from baseline was -0.173 (0.21) for placebo subjects compared to -0.046 (0.09), a 73% improvement for FB102 treated subjects compared to placebo.

Gluten challenge induced GI symptoms (nausea, vomiting, diarrhea, abdominal pain and abdominal bloating) reported during the 16 day gluten challenge from patient diaries/AE collection demonstrated a 42% benefit for FB102 treated subject (4.0 events per subject) compared to placebo (6.9 events per subject).

There were no dropouts in the study. Treatment emergent adverse events (TEAE) were primarily mild (grade 1) with no grade 3 or higher SAEs reported in the FB102 arm.

“We want to congratulate all of the investigators and researchers that supported this study. I also want to acknowledge the incredible dedication and hard work of the Forte team. Celiac disease is debilitating for many patients with even trace exposure to gluten. FB102 has taken a big step forward towards addressing this very large unmet need with the results from this study. The Phase 2 celiac disease study is initiating with a topline readout expected in 2026.” said Paul Wagner, Ph.D. CEO and Chairperson of Forte Biosciences. “These results are also very encouraging given the biology of the additional FB102 indications including vitiligo, alopecia areata and type 1 diabetes. We also look forward to reading out the topline results of the FB102 vitiligo study in the first half of 2026.”

About Forte

Forte Biosciences, Inc. is a clinical-stage biopharmaceutical company that is advancing FB102, which is a proprietary anti-CD122 monoclonal antibody therapeutic candidate with potentially broad autoimmune and autoimmune-related indications.

Forward-Looking Statements

Forte cautions you that statements included in this press release that are not a description of historical facts are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negatives of these terms or other similar expressions. These statements are based on the Forte’s current beliefs and expectations. Forward-looking statements include statements regarding Forte’s beliefs, goals, intentions and expectations regarding its product candidate, FB102 and the therapeutic and commercial market potential of FB102, the expected timeline for the Phase 2 celiac study and related readout, and the expected timing of topline results for the FB102 vitiligo study. Actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of various risks and uncertainties, which include, without limitation: risks related to Forte’s ability to obtain sufficient additional capital to continue to advance Forte’s product candidate, FB102; uncertainties associated with the clinical development and regulatory approval of Forte’s product candidate, FB102, including potential delays in the commencement, enrollment and completion of clinical trials; the risk that results from preclinical and the Phase 1b trials may not be predictive of future results from clinical trials; risks associated with the failure to realize any value from FB102 in light of inherent risks, expense and difficulties involved in successfully bringing product candidates to market; and additional risks, uncertainties, and other information affecting Forte’s business and operating results is contained in Forte’s Quarterly Report on Forms 10-Q filed on May 15, 2025, and in its other filings with the Securities and Exchange Commission. All forward-looking statements in this press release are current only as of the date hereof and, except as required by applicable law, Forte undertakes no obligation to revise or update any forward-looking statement, or to make any other forward-looking statements, whether as a result of new information, future events or otherwise. All forward-looking statements are qualified in their entirety by this cautionary statement. This caution is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

Contact:
LifeSci Advisors
Mike Moyer, Managing Director
mmoyer@lifesciadvisors.com

Forte Biosciences, Inc.
Paul Wagner, CEO
investors@fortebiorx.com

Exhibit 99.2

FORTE BIOSCIENCES

FB102

CELIAC DISEASE PHASE 1B RESULTS

JUNE 2025

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.

CLINICAL STAGE FB-102

- CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and 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 the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK and T cells
- Celiac disease (CeD) phase 1b trial completed and demonstrated positive histological and symptom data for FB102 treated subjects compared to placebo
- FB102 Phase 2 celiac disease initiating with data expected in 2026
- FB102 Phase 1b vitiligo trial enrolling with topline results expected in 1H26

ALIGNMENT OF DISEASE BIOLOGY AND MECHANISM FOR FB102 HIGHLIGHTS "PIPELINE-IN-A-PRODUCT" POTENTIAL FOR FB102 IN AUTOIMMUNE DISEASES WITH HIGH UNMET NEED

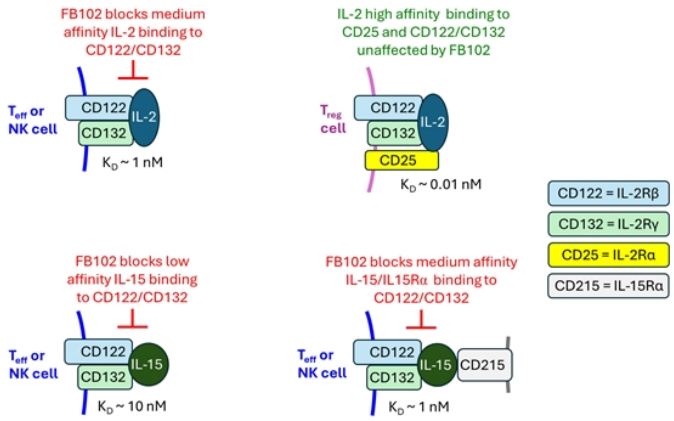
Disease	Species	Outcome	Reference
Celiac disease	Mouse	Improved IL-15-induced mucosal damage	PNAS, 2009
Vitiligo	Mouse	Enhanced repigmentation	Sci Transl Med, 2018
Alopecia areata	Mouse	Prevented fur loss	Nature Med, 2014
Type 1 diabetes	Mouse	Delayed disease onset	JCI Insight, 2018

FB102 CD122 ANTAGONIST MECHANISM

CLINICAL STAGE FB102 OVERVIEW

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

FB102 (Forte's anti-CD122 antibody) is designed to mediate <u>both</u> the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK cells, certain T cell subtypes without effecting the IL-2 biology of beneficial Tregs

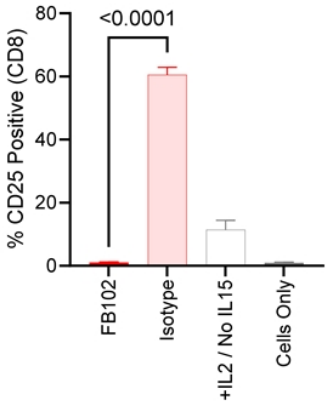


Lodolce 2002 Cytokine Growth Factor Rev. PMID 12401478
Ross 2018 Annu Rev Immunol. PMID 29677473

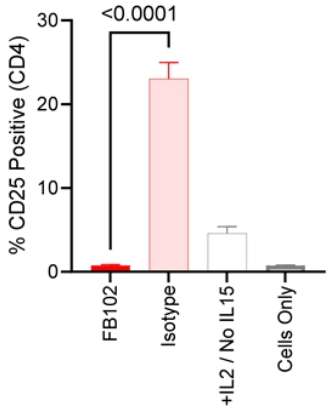
FB102 INHIBITS IL-2/IL-15 CD4+ AND CD8+ T CELL ACTIVATION IN IN VITRO DISEASE MODEL

CD4+ and CD8+ T cells were treated with IL2 for 24 hours then with IL15 for 24 hours, simulating disease activity in the presence or absence of FB102

FB102 provides nearly complete inhibition of T cell activation



CD8+ T Cells

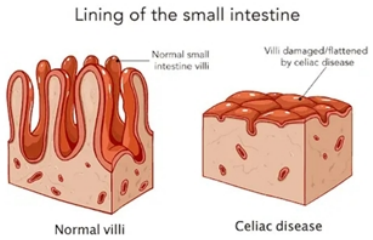


CD4+ T Cells

CELIAC DISEASE

NO APPROVED THERAPIES FOR CELIAC DISEASE; POTENTIAL FOR A SIGNIFICANT MARKET OPPORTUNITY

- Celiac disease is an autoimmune disease that's triggered by consuming gluten and results in damage to the small intestine
- Symptom include diarrhea, fatigue, headaches, anemia, nausea, dermatitis herpetiformis (an itchy skin rash)
- Significant patient population does not respond to gluten free diet
- Health consequence for not treating include malnourishment, cancer, other autoimmune conditions



- Market Opportunity
  - Estimated 1:133 in US (2.5 million people) with celiac disease (Fasano, Arch Intern Med. 2003 PMID: 12578508)
    - 0.3% to 0.5% of celiac disease patients are non-responsive (Malamut Gastroenterology. 2024 38556189)
  - No approved treatment options for celiac disease

IL-2 AND IL-15 IN CELIAC DISEASE (CED)

IL-2

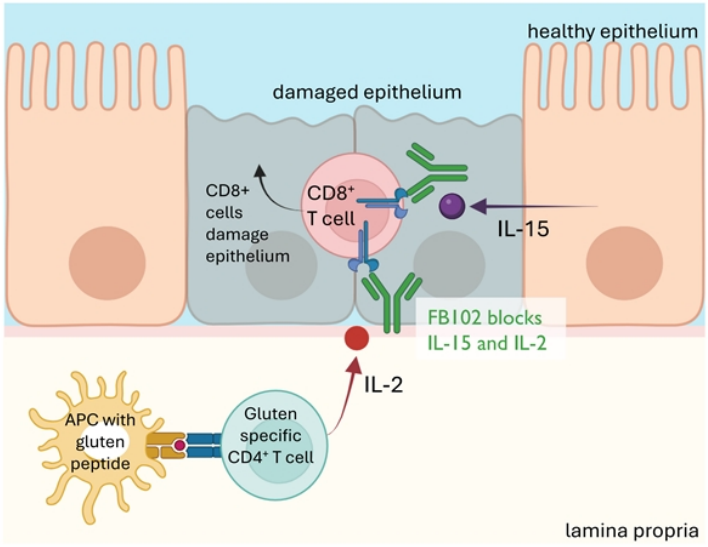
- Clear genetic basis for involvement of IL-2 in CeD
- Gluten-induced IL-2 production differentiates true CeD from non-gluten induced GI symptoms
- IL-2 strongly correlates with symptom severity and Serum IL-2 peaks within 4 hours after gluten exposure
- IL-2 production is followed by increases in Intraepithelial lymphocyte (IELs) and inflammatory Th-1 type cytokine IFN-γ

IL-15

- Clear genetic basis for involvement of IL-15 in CeD
- IL-15 levels in intestinal tissue correlate with intestinal damage
- IL-15 is overexpressed in gut epithelium and immune cells upon gluten exposure
- IL-15Ra is overexpressed in Intraepithelial lymphocyte (IELs) in patients with CeD
- IL-15 induces proliferation and activation of Intraepithelial lymphocyte (IELs) and inflammatory cytokines IFN- γ and TNF-α
- IL-15 activates intestinal cytotoxic CD8+ T cells that kill gut epithelium
- IL-15 impairs immunosuppressive and gut-protective activity of CD4+ Tregs and TGF-β

1 - van Heel 2007 Nat Genet. PMID 17558408

FB102 BLOCKS GLUTEN-INDUCED INTESTINAL DAMAGE IN CELIAC SUBJECT BY BLOCKING IL-2 AND IL-15 ACTIVATION OF CD8+ T CELLS



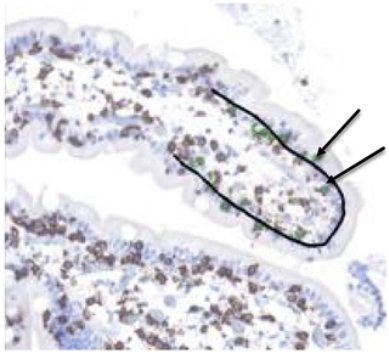
Levescot 2022 Gut PMID 35879049

FB102 BLOCKS BOTH IL-2 AND IL-15, PROVIDING POTENTIAL ADVANTAGES OVER OTHER DRUGS BEING CLINICALLY INVESTIGATED FOR CELIAC DISEASE

Gluten Modification	Immunotolerance	Gut Healing	Immunomodulators	
			Single targeting	Multi targeting
IMGX-003 (Entero Therapeutics)	CNP-101/TAK-101 (Takeda)	IMU-856 (Immunic Therapeutics)	IL-15 → CALY-002 (Calypso Biotech, A Novartis Company)	**CD122 (IL-2/IL-15)→FB102** (FORTE BIOSCIENCES, INC)
TAK-062 (Takeda)	TMP502 (TOPAS THERAPEUTICS)		IL 15 → Ordesekimab (sanofi AMGEN)	IL15/IL-21→EQ-102 (equillium)
E40 (nemysis)	KAN-101 (ANOKION)		IL 15 → TEV-53408 (teva)	
	TAK227/ZED1227 (Takeda)		OX40L amlitelimab (sanofi)	
	DONQ52 (CHUGAI)			

CELIAC DISEASE MORPHOLOGIC AND HISTOLOGICAL MEASUREMENTS IN CELIAC DISEASE

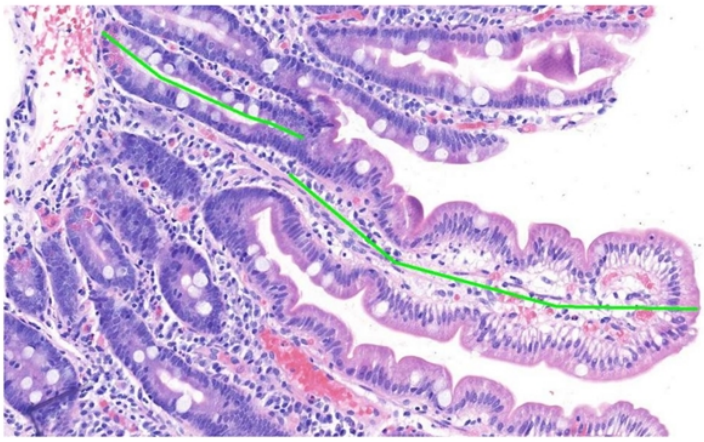
INTRAEPITHELIAL LYMPHOCYTES ARE MEDIATORS OF DAMAGE TO VILLI IN CELIAC DISEASE



- Intraepithelial lymphocyte (IELs) are CD3+T cells that infiltrate the villus upon gluten challenge in celiac disease leading to villus atrophy
- IEL count is measured as a density of CD3+ T cells per 100 enterocytes
- Subjects with celiac disease typically have IEL count density of 20-30 and can increase 30%+ on exposure to gluten

Taavela J, PLoS One. PMID: 24146832; PMCID: PMC3795762.
Rostami K Gut. 2017 Dec; PMID: 28893865; PMCID: PMC5749338.

VILLUS HEIGHT TO CRYPT DEPTH RATIO (VH:CD)



- Villus height to crypt depth (Vh:Cd) ratio measures morphological damage due to IEL infiltration after gluten exposure in celiac disease
- Vh:Cd ratio in patients with CeD is ~2.0-3.0 when the patients are on Gluten Free Diet

Adelman DC, Am J Gastroenterol. 2018 Mar;2018 Feb 20. PMID: 29460921.

COMPOSITE HISTOLOGICAL SCALE FOR CELIAC DISEASE

Clinical Gastroenterology and Hepatology 2024;22:1238–1244

A Composite Morphometric Duodenal Biopsy Mucosal Scale for Celiac Disease Encompassing Both Morphology and Inflammation

Jack A. Syage,[1] Markku Mäki,[2] Daniel A. Leffler,[3] Jocelyn A. Silvester,[3] Jennifer A. Sealey-Voyksner,[1] Tsung-Teh Wu,[4] and Joseph A. Murray[4]

[1]ImmunogenX, Inc, Newport Beach, California; [2]Faculty of Medicine and Health Technology, Tampere University, Tampere, Finland; [3]Celiac Disease Research Program, Harvard Medical School, Boston, Massachusetts; and [4]Mayo Clinic, Rochester, Minnesota

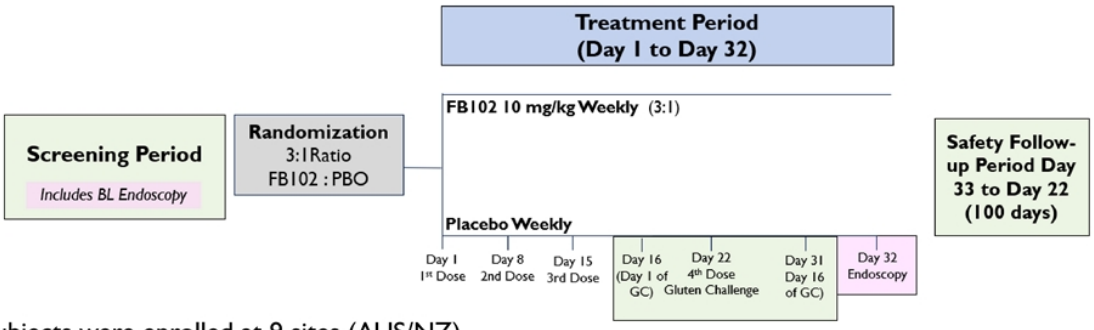
Findings:

A composite scale VCIEL comprising individual subject values for Vh:Cd and IEL with equal weighting appears to offer better accuracy and statistical precision, particularly for population analysis in clinical trials, as well as potentially offering a broader measure of mucosal health.

$$\mathrm{VCIEL} = \left[\frac{\mathrm{Vh{:}Cd} - <\mathrm{Vh{:}Cd}>}{\sigma_{\mathrm{Vh:Cd}}} - \frac{\mathrm{IEL} - <\mathrm{IEL}>}{\sigma_{\mathrm{IEL}}}\right]$$

FB102 PHASE 1B CELIAC DISEASE STUDY

CELIAC DISEASE PHASE 1B DESIGN



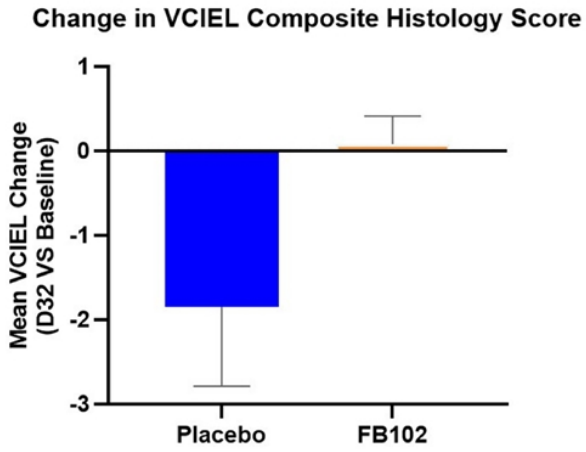
- 32 subjects were enrolled at 9 sites (AUS/NZ)
- Randomized 3:1 to FB102 vs PBO (24:8)
- Subjects received 3 of 4 doses of either FB102 (10mg/kg) or placebo then began 16 day gluten challenge (2g,4g, 8g for 14 days)
  - 4th dose of either FB102 or placebo on day 22
- Endoscopy/biopsy at baseline and at end of gluten challenge (central review of histology endpoints)
- 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/m2) 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	

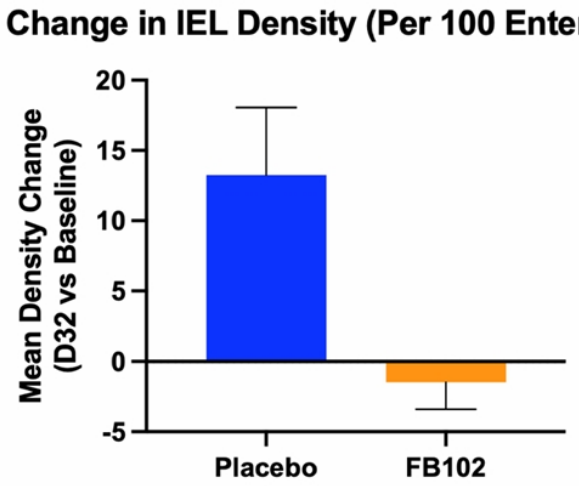
FB102 DEMONSTRATES SIGNIFICANT BENEFIT IN PHASE 1B CELIAC DISEASE STUDY

FB102 DEMONSTRATES STATISTICALLY SIGNIFICANT COMPOSITE HISTOLOGY (VCIEL) BENEFIT COMPARED TO PLACEBO



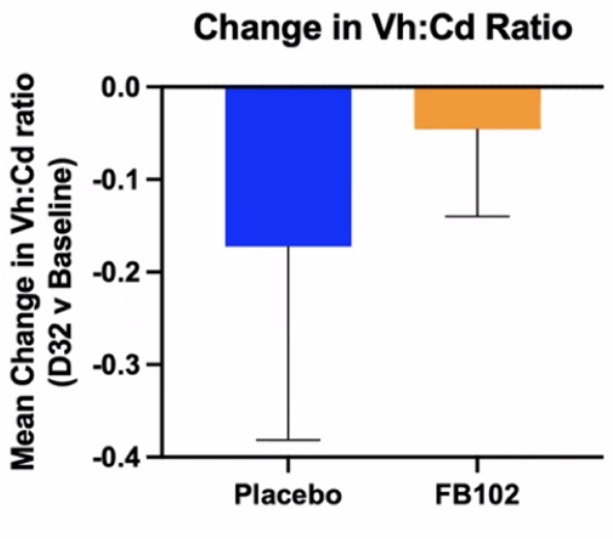
Day 32 vs baseline VCIEL composite score –1.849 for PBO compared to 0.079 for FB102 treated subjects (p=0.0099)

FB102 DEMONSTRATES STATISTICALLY SIGNIFICANT DIFFERENCE IN CHANGE IN IEL DENSITY COMPARED TO PLACEBO



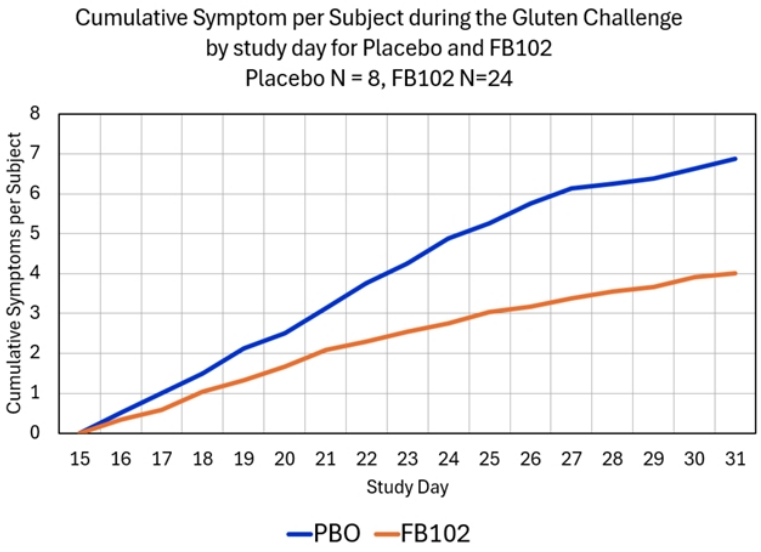
Day 32 vs baseline mean IEL density increase of 13.3 for PBO compared to a decrease of 1.5 for FB102 treated subjects (p=0.0035)

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 VS PLACEBO



Gluten challenge (GC) symptoms reported in patient diaries/AE collection

GC induced GI symptoms tracked: nausea, diarrhea, vomiting, abdominal pain, abdominal bloating

Through the 16 day gluten challenge FB102 demonstrated a 42% symptom benefit vs placebo (average of 6.9 events per subject on placebo compared to 4.0 events per subject on FB102)

FB102 PHASE 1B CELIAC DISEASE STUDY
SAFETY SUMMARY

FB102 GENERALLY SAFE AND WELL TOLERATED

Treatment Emergent Adverse Events By Grade

	Placebo (N=8)		FB102 (N=24)		Overall (N=32)	
	n (Participant Count)	%	n (Participant Count)	%	n (Participant Count)	%
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%

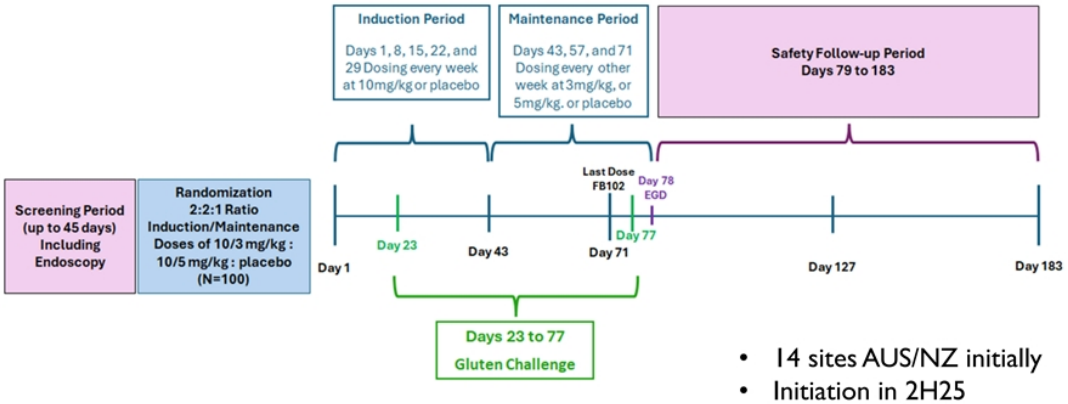
FB102 GENERALLY SAFE AND WELL TOLERATED

Treatment Emergent Adverse Events By Organ Class

	Placebo (N=8)		FB102 (N=24)		Overall (N=32)	
System Organ Class Summary	n (Participant Count)	%	n (Participant Count)	%	n (Participant Count)	%
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 FB102
PHASE 2 TRIAL OVERVIEW

CELIAC DISEASE PHASE 2 DESIGN



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

- 14 sites AUS/NZ initially
- Initiation in 2H25
- US IND expected late 2025/early 2026
- Topline data readout expected in 2026

DEVELOPMENT TIMELINES

FB102 PROPOSED 12 MONTH CLINICAL DEVELOPMENT

Celiac Disease

- Phase 1b
  - Initiation 3Q24
  - Positive Topline readout in June 2025
- Phase 2
  - Initiation 2H25
  - Topline data expected in 2026

Alopecia Areata

- Phase 2
  - Initiation 2026

Vitiligo

- Phase1b Study
  - Initiated 1H25
  - Topline data expected 1H26
- Phase 2
  - Initiation 2026

Type 1 Diabetes

- Evaluating study design and initiation

SUMMARY

CLINICAL STAGE FB-102

- CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and 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 the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK and T cells
- Celiac disease (CeD) phase 1b trial completed and demonstrated positive histological and symptom data for FB102 treated subjects compared to placebo
- FB102 Phase 2 celiac disease initiating with data expected in 2026
- FB102 Phase 1b vitiligo trial enrolling with topline results expected in 1H26

Exhibit 99.3

FORTE BIOSCIENCES

FORTE BIOSCIENCES

JUNE 2025

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.

CLINICAL STAGE FB-102

- CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and 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 the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK and T cells
- Celiac disease (CeD) phase 1b trial completed and demonstrated positive histological and symptom data for FB102 treated subjects compared to placebo
- FB102 Phase 2 celiac disease initiating with data expected in 2026
- FB102 Phase 1b vitiligo trial enrolling with topline results expected in 1H26

FORTE BIOSCIENCES OVERVIEW

- Strong Board of Directors comprised of leaders in industry including:
  - Scott Brun, MD – Former head of Abbvie product development
  - David Gryska – Former CFO of Incyte and Celgene
  - Barbera Finck, MD – Led Enbrel development at Immunex and Humira biosimilar development at Coherus
  - Steve Doberstein, PhD – Former Chief Scientific Officer of Nektar
  - Steve Kornfeld – Co-Managing Partner of Castle Peak Partners and former Healthcare Sector Team Leader and PM at Franklin Templeton
  - Shiv Kapoor – Co-Founder of Stonegate Healthcare and biotech veteran with over 25 years of experience in investments and therapeutics development
  - Rich Vincent – Multiple biotech CFO roles, Deloitte & Touche, CPA

EXPERIENCED MANAGEMENT

Forte's management has extensive experience in manufacturing, quality, regulatory and clinical development

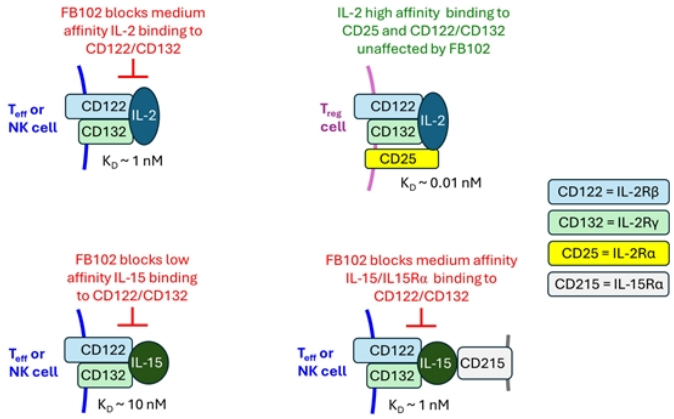
Paul Wagner, Ph.D. – CEO	CANbridge, PFEnex, Allianz Global Investors, PDL BioPharma, Lehman Brothers
Tony Riley – Chief Financial Officer	Krystal, AVANEX, ACLARA, KOSAN
Chris Roenfeldt, PMP – Chief Operating Officer	Halozyme, PFEnex, Genentech, A Member of the Roche Group, AMGEN
Barbara Finck, MD – Senior Medical Clinician	Coherus, PDL BioPharma, Immunex, Creating the Future of Medicine

FB102 CD122 ANTAGONIST MECHANISM

CLINICAL STAGE FB102 OVERVIEW

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

FB102 (Forte's anti-CD122 antibody) is designed to mediate <u>both</u> the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK cells, certain T cell subtypes without effecting the IL-2 biology of beneficial Tregs

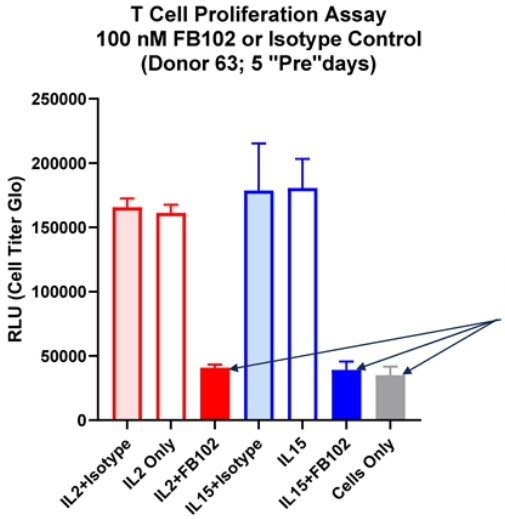


Lodolce 2002 Cytokine Growth Factor Rev. PMID 12401478
Ross 2018 Annu Rev Immunol. PMID 29677473

FB102 IN VITRO DATA

FB102 INHIBITS CD4+ AND CD8+ T CELL PROLIFERATION

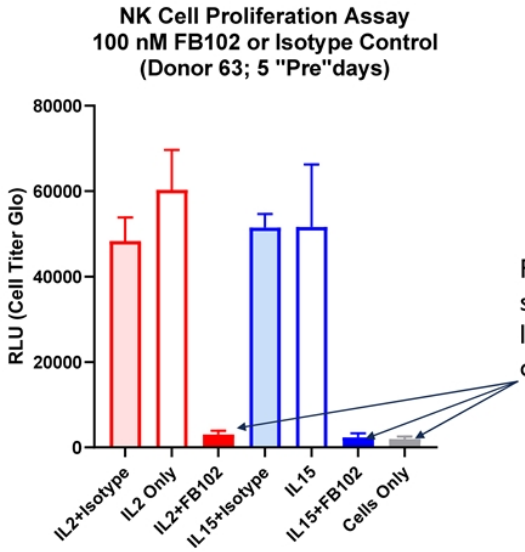
	Fold Reduction Isotype/FB102
IL-2	4.1
IL-15	4.6



FB102 inhibits IL-2/IL-15 signaling back to baseline levels of the unstimulated cell only

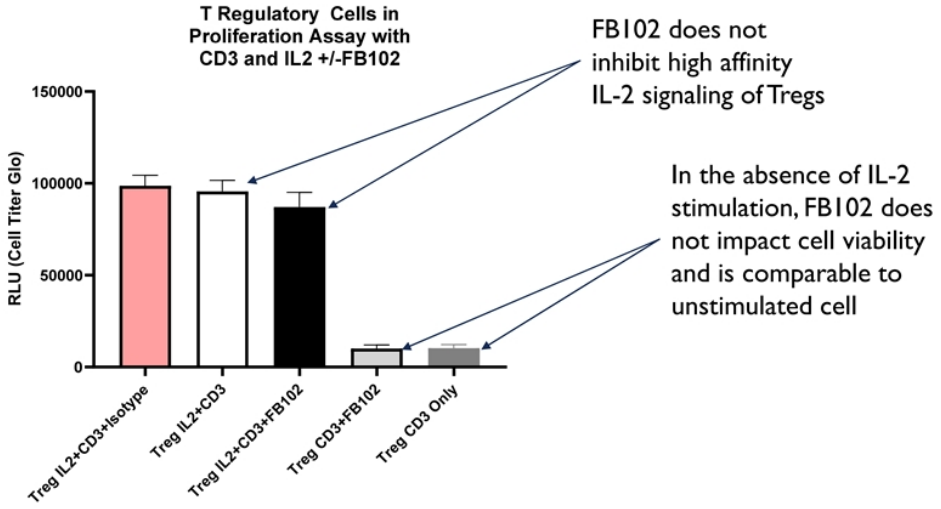
FB102 INHIBITS NK CELL PROLIFERATION BACK TO BASELINE LEVELS

	Fold Reduction Isotype/FB102
IL-2	5.6
IL-15	7.8

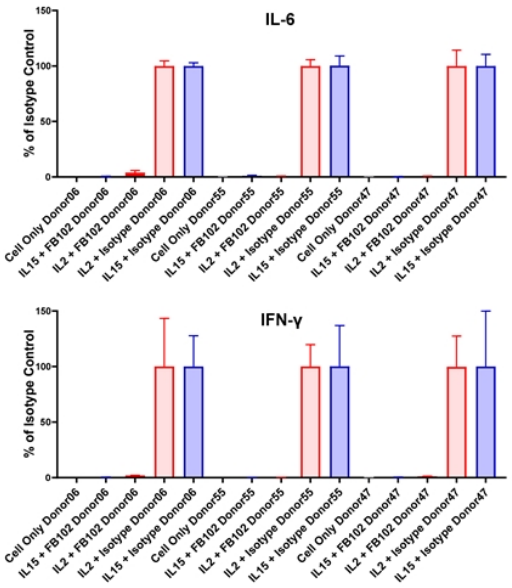


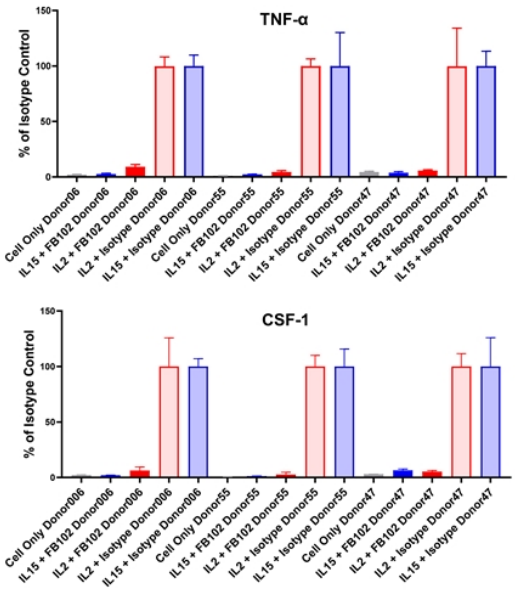
FB102 inhibits IL-2/IL-15 signaling back to baseline levels of the unstimulated cell only

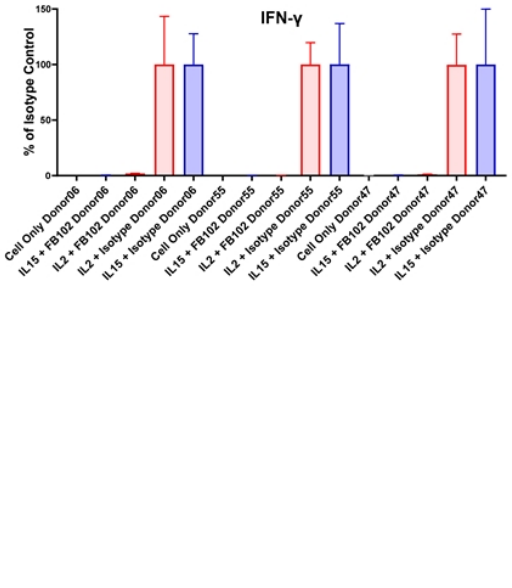
FB102 DOES **NOT** INHIBIT REGULATORY T CELL PROLIFERATION

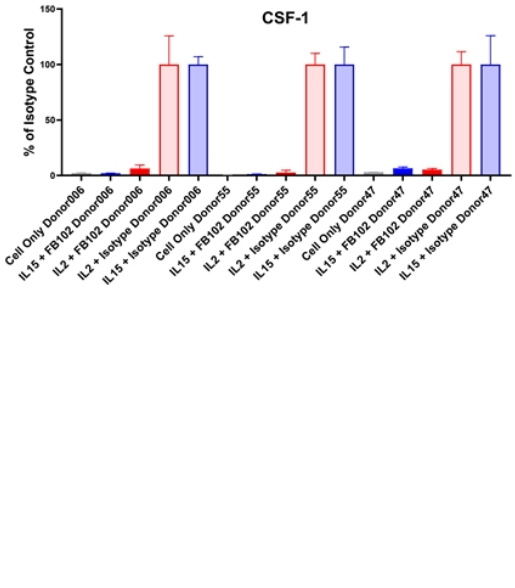


FB102 INHIBITS IL-2 AND IL-15 INDUCTION OF IL-6, TNF-ALPHA, IFN-GAMMA, AND CSF-1 WITH 3 DIFFERENT T CELL DONORS





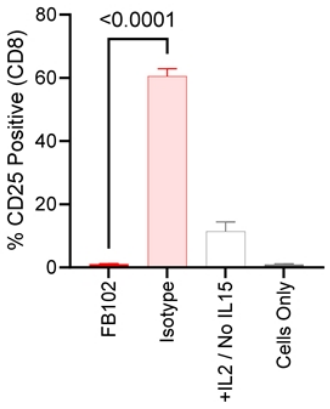




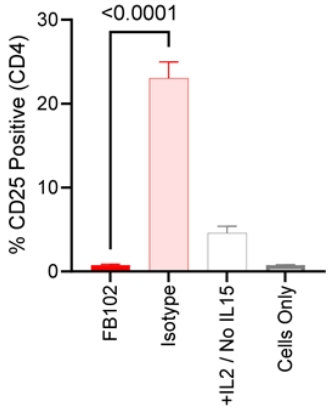
FB102 INHIBITS IL-2/IL-15 CD4+ AND CD8+ T CELL ACTIVATION IN IN VITRO DISEASE MODEL

CD4+ and CD8+ T cells were treated with IL2 for 24 hours then with IL15 for 24 hours, simulating disease activity in the presence or absence of FB102

FB102 provides nearly complete inhibition of T cell activation



CD8+ T Cells



CD4+ T Cells

FB102:
BROAD AUTOIMMUNE INDICATION POTENTIAL

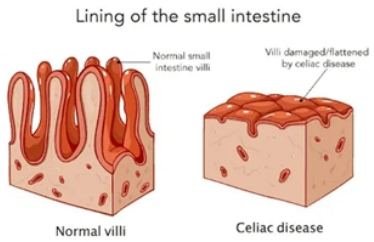
ALIGNMENT OF DISEASE BIOLOGY AND MECHANISM FOR FB102 HIGHLIGHTS "PIPELINE-IN-A-PRODUCT" POTENTIAL FOR FB102 IN AUTOIMMUNE DISEASES WITH HIGH UNMET NEED

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

CELIAC DISEASE

NO APPROVED THERAPIES FOR CELIAC DISEASE; POTENTIAL FOR A SIGNIFICANT MARKET OPPORTUNITY

- Celiac disease is an autoimmune disease that's triggered by consuming gluten and results in damage to the small intestine
- Symptom include diarrhea, fatigue, headaches, anemia, nausea, dermatitis herpetiformis (an itchy skin rash)
- Significant patient population does not respond to gluten free diet
- Health consequence for not treating include malnourishment, cancer, other autoimmune conditions



- Market Opportunity
  - Estimated 1:133 in US (2.5 million people) with celiac disease (Fasano, Arch Intern Med. 2003 PMID: 12578508)
    - 0.3% to 0.5% of celiac disease patients are non-responsive (Malamut Gastroenterology. 2024 38556189)
  - No approved treatment options for celiac disease

IL-2 AND IL-15 IN CELIAC DISEASE (CED)

IL-2

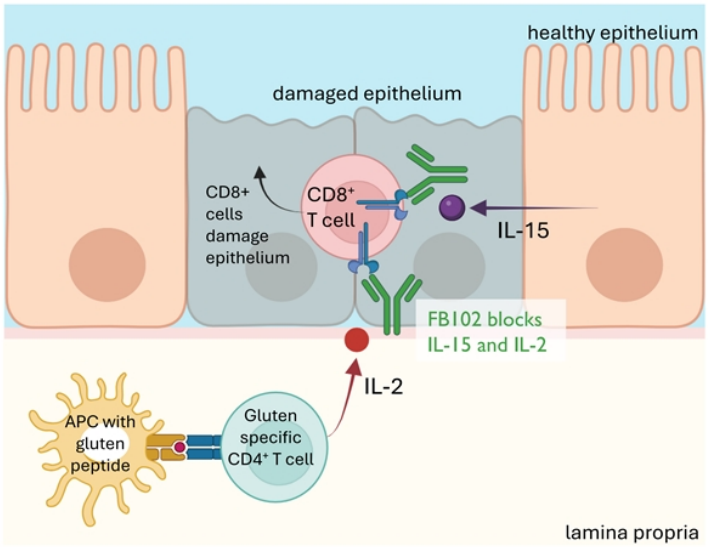
- Clear genetic basis for involvement of IL-2 in CeD
- Gluten-induced IL-2 production differentiates true CeD from non-gluten induced GI symptoms
- IL-2 strongly correlates with symptom severity and Serum IL-2 peaks within 4 hours after gluten exposure
- IL-2 production is followed by increases in Intraepithelial lymphocyte (IELs) and inflammatory Th-1 type cytokine IFN-γ

IL-15

- Clear genetic basis for involvement of IL-15 in CeD
- IL-15 levels in intestinal tissue correlate with intestinal damage
- IL-15 is overexpressed in gut epithelium and immune cells upon gluten exposure
- IL-15Ra is overexpressed in Intraepithelial lymphocyte (IELs) in patients with CeD
- IL-15 induces proliferation and activation of Intraepithelial lymphocyte (IELs) and inflammatory cytokines IFN- γ and TNF-α
- IL-15 activates intestinal cytotoxic CD8+ T cells that kill gut epithelium
- IL-15 impairs immunosuppressive and gut-protective activity of CD4+ Tregs and TGF-β

1 - van Heel 2007 Nat Genet. PMID 17558408

FB102 BLOCKS GLUTEN-INDUCED INTESTINAL DAMAGE IN CELIAC SUBJECT BY BLOCKING IL-2 AND IL-15 ACTIVATION OF CD8+ T CELLS



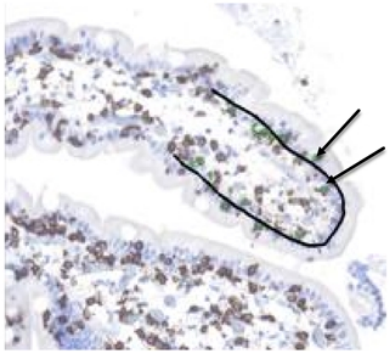
Levescot 2022 Gut PMID 35879049

FB102 BLOCKS BOTH IL-2 AND IL-15, PROVIDING POTENTIAL ADVANTAGES OVER OTHER DRUGS BEING CLINICALLY INVESTIGATED FOR CELIAC DISEASE

Gluten Modification	Immunotolerance	Gut Healing	Immunomodulators	
			Single targeting	Multi targeting
IMGX-003 (Entero Therapeutics)	CNP-101/TAK-101 (Takeda)	IMU-856 (Immunic Therapeutics)	IL-15 → CALY-002 (Calypso Biotech, A Novartis Company)	**CD122 (IL-2/IL-15)→FB102** (FORTE BIOSCIENCES, INC)
TAK-062 (Takeda)	TMP502 (TOPAS THERAPEUTICS)		IL 15 → Ordesekimab (sanofi AMGEN)	IL15/IL-21→EQ-102 (equillium)
E40 (nemysis)	KAN-101 (ANOKION)		IL 15 → TEV-53408 (teva)	
	TAK227/ZED1227 (Takeda)		OX40L amlitelimab (sanofi)	
	DONQ52 (CHUGAI)			

CELIAC DISEASE MORPHOLOGIC AND HISTOLOGICAL MEASUREMENTS IN CELIAC DISEASE

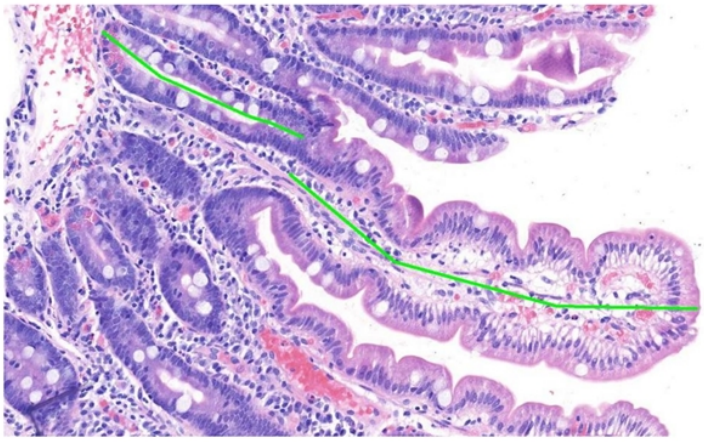
INTRAEPITHELIAL LYMPHOCYTES ARE MEDIATORS OF DAMAGE TO VILLI IN CELIAC DISEASE



- Intraepithelial lymphocyte (IELs) are CD3+T cells that infiltrate the villus upon gluten challenge in celiac disease leading to villus atrophy
- IEL count is measured as a density of CD3+ T cells per 100 enterocytes
- Subjects with celiac disease typically have IEL count density of 20-30 and can increase 30%+ on exposure to gluten

Taavela J, PLoS One. PMID: 24146832; PMCID: PMC3795762.
Rostami K Gut. 2017 Dec; PMID: 28893865; PMCID: PMC5749338.

VILLUS HEIGHT TO CRYPT DEPTH RATIO (VH:CD)



- Villus height to crypt depth (Vh:Cd) ratio measures morphological damage due to IEL infiltration after gluten exposure in celiac disease
- Vh:Cd ratio in patients with CeD is ~2.0-3.0 when the patients are on Gluten Free Diet

Adelman DC, Am J Gastroenterol. 2018 Mar;2018 Feb 20. PMID: 29460921.

COMPOSITE HISTOLOGICAL SCALE FOR CELIAC DISEASE

Clinical Gastroenterology and Hepatology 2024;22:1238–1244

A Composite Morphometric Duodenal Biopsy Mucosal Scale for Celiac Disease Encompassing Both Morphology and Inflammation

Jack A. Syage,[1] Markku Mäki,[2] Daniel A. Leffler,[3] Jocelyn A. Silvester,[3] Jennifer A. Sealey-Voyksner,[1] Tsung-Teh Wu,[4] and Joseph A. Murray[4]

[1]ImmunogenX, Inc, Newport Beach, California; [2]Faculty of Medicine and Health Technology, Tampere University, Tampere, Finland; [3]Celiac Disease Research Program, Harvard Medical School, Boston, Massachusetts; and [4]Mayo Clinic, Rochester, Minnesota

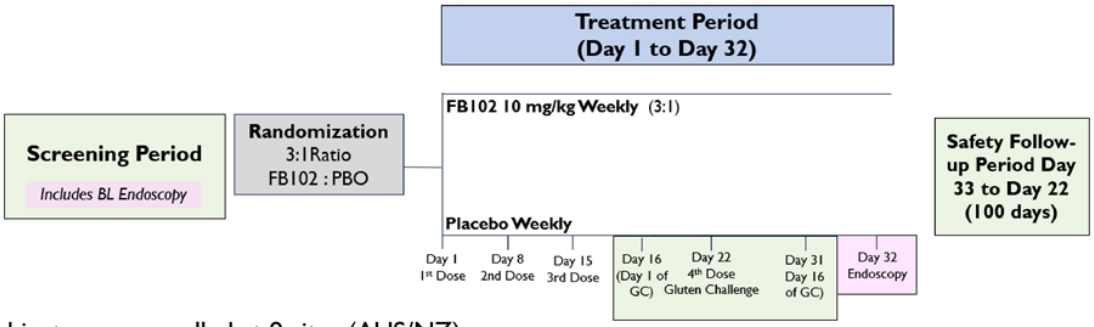
Findings:

A composite scale VCIEL comprising individual subject values for Vh:Cd and IEL with equal weighting appears to offer better accuracy and statistical precision, particularly for population analysis in clinical trials, as well as potentially offering a broader measure of mucosal health.

$$\mathrm{VCIEL} = \left[\frac{\mathrm{Vh{:}Cd} - <\mathrm{Vh{:}Cd}>}{\sigma_{\mathrm{Vh:Cd}}} - \frac{\mathrm{IEL} - <\mathrm{IEL}>}{\sigma_{\mathrm{IEL}}}\right]$$

FB102 PHASE 1B CELIAC DISEASE STUDY

CELIAC DISEASE PHASE 1B DESIGN



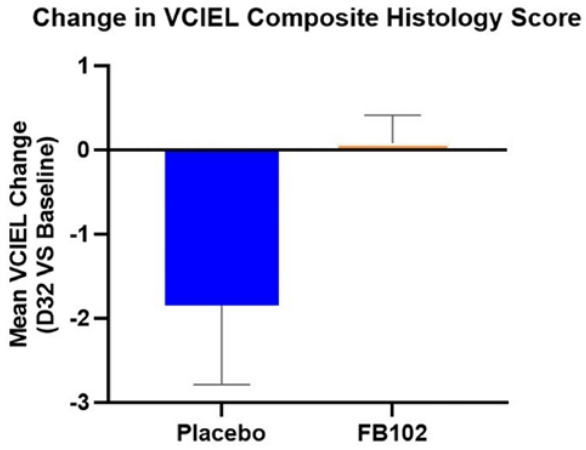
- 32 subjects were enrolled at 9 sites (AUS/NZ)
- Randomized 3:1 to FB102 vs PBO (24:8)
- Subjects received 3 of 4 doses of either FB102 (10mg/kg) or placebo then began 16 day gluten challenge (2g,4g, 8g for 14 days)
  - 4th dose of either FB102 or placebo on day 22
- Endoscopy/biopsy at baseline and at end of gluten challenge (central review of histology endpoints)
- 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/m2) 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	

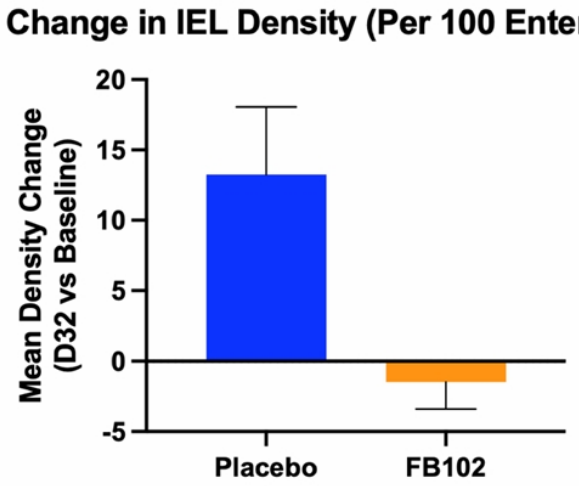
FB102 DEMONSTRATES SIGNIFICANT BENEFIT IN PHASE 1B CELIAC DISEASE STUDY

FB102 DEMONSTRATES STATISTICALLY SIGNIFICANT COMPOSITE HISTOLOGY (VCIEL) BENEFIT COMPARED TO PLACEBO



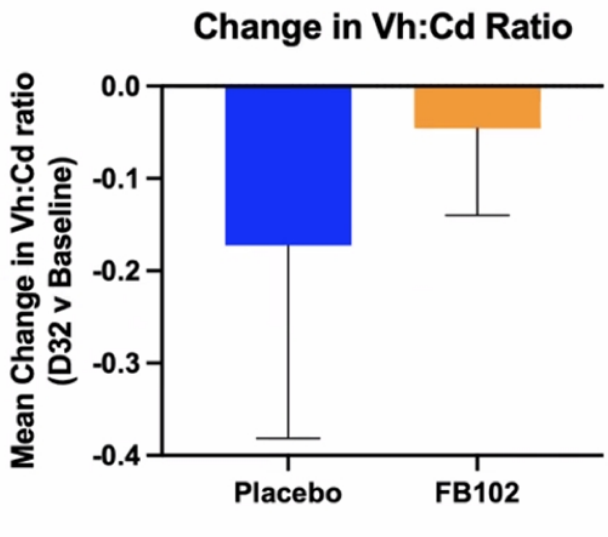
Day 32 vs baseline VCIEL composite score –1.849 for PBO compared to 0.079 for FB102 treated subjects (p=0.0099)

FB102 DEMONSTRATES STATISTICALLY SIGNIFICANT DIFFERENCE IN CHANGE IN IEL DENSITY COMPARED TO PLACEBO



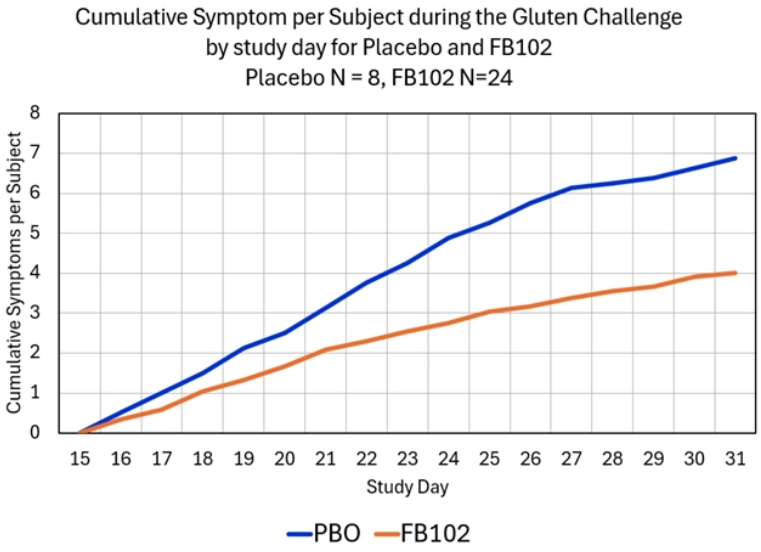
Day 32 vs baseline mean IEL density increase of 13.3 for PBO compared to a decrease of 1.5 for FB102 treated subjects (p=0.0035)

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 VS PLACEBO



Gluten challenge (GC) symptoms reported in patient diaries/AE collection

GC induced GI symptoms tracked: nausea, diarrhea, vomiting, abdominal pain, abdominal bloating

Through the 16 day gluten challenge FB102 demonstrated a 42% symptom benefit vs placebo (average of 6.9 events per subject on placebo compared to 4.0 events per subject on FB102)

FB102 PHASE 1B CELIAC DISEASE STUDY
SAFETY SUMMARY

FB102 GENERALLY SAFE AND WELL TOLERATED

Treatment Emergent Adverse Events By Grade

	Placebo (N=8)		FB102 (N=24)		Overall (N=32)	
	n (Participant Count)	%	n (Participant Count)	%	n (Participant Count)	%
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%

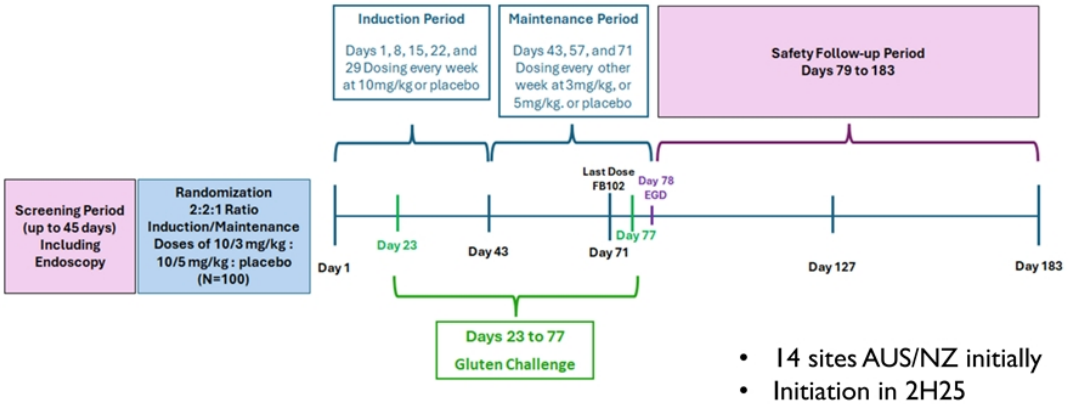
FB102 GENERALLY SAFE AND WELL TOLERATED

Treatment Emergent Adverse Events By Organ Class

	Placebo (N=8)		FB102 (N=24)		Overall (N=32)	
System Organ Class Summary	n (Participant Count)	%	n (Participant Count)	%	n (Participant Count)	%
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 FB102
PHASE 2 TRIAL OVERVIEW

CELIAC DISEASE PHASE 2 DESIGN

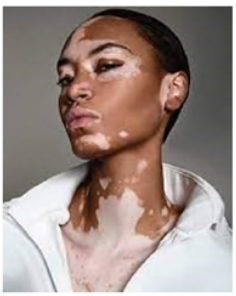


Study Day	Amount of Gluten (grams)
Day 1 to 22	0 grams of gluten per day
Days 23 to 36	8 grams of gluten per day
Days 37 to 77	3 grams of gluten per day
Day 78	EGD

- 14 sites AUS/NZ initially
- Initiation in 2H25
- US IND expected late 2025/early 2026
- Topline data readout expected in 2026

VITILIGO

LARGE UNMET NEED IN VITILIGO PRESENTS A SUBSTANTIAL MARKET OPPORTUNITY



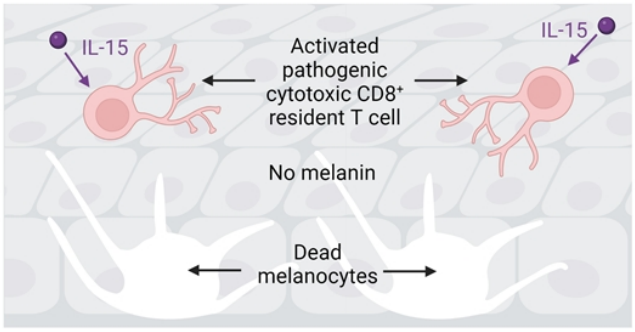
Amy Deanna / CoverGirl cosmetics

- Vitiligo is an autoimmune disease of the skin driven by pathogenic T cells that kill melanocytes and create white spots
- Vitiligo results in sensitive skin (increasing likelihood of sub burns), eye abnormalities, emotional challenges, and leads to a predisposition of other autoimmune conditions.
- Market Opportunity
  - Prevalent in 0.76% of population – 2 Million in US
  - While JAK inhibitors have demonstrated efficacy in vitiligo, regulatory scrutiny of the JAK class including black box warnings has dampened enthusiasm for this class and as a result there remains a significant unmet need for safe and effective therapies for treating AA and vitiligo

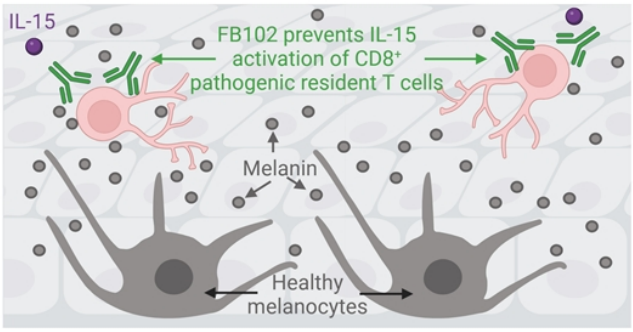
https://my.clevelandclinic.org/health/diseases/12419-vitiligo

IL-15 ACTIVATION OF PATHOGENIC CD8+ T CELLS IN SKIN

Vitiligo patients have unpigmented skin due activated pathogenic T cells killing melanocytes



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



Tokura Front Immunol. 2021 PMID 33633737

IL-2 THERAPY DRIVES VITILIGO

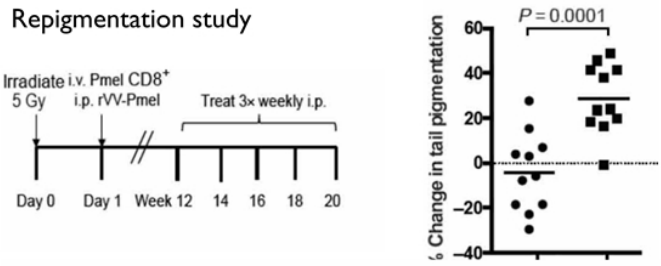
Enhanced Survival Associated with Vitiligo Expression during Maintenance Biotherapy for Metastatic Melanoma(1)

Peter D. Boasberg[1], Dave S.B. Hoon[2], Lawrence D. Piro[1], Maureen A. Martin[1], Akhide Fujimoto[2], Timothy S. Kristedja[1], Sandeep Bhachu[1], Xing Ye[2], Regina R. Deck[1] and Steven J. O'Day[1]

In a large retrospective analysis of 374 metastatic melanoma patients treated with high-dose IL-2, a total of 84 patients (22%) developed treatment-related vitiligo, although in patients with objective clinical responses the incidence of vitiligo was nearly 50% (2)

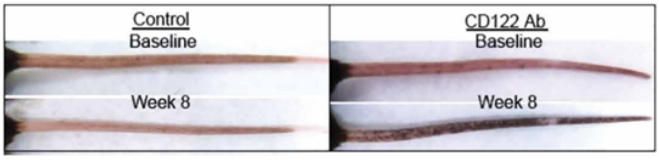
1) Journal of Investigative Derm. (2006) Vol 126
2) Journal of Clinical Oncology V19(15)

AN ANTI-CD122 ANTIBODY IS EFFECTIVE IN A MOUSE VITILIGO MODEL WITH ESTABLISHED DISEASE



Melanin-reactive T cells eliminated pigment in tail

Vehicle (control) treatment did not restore pigment

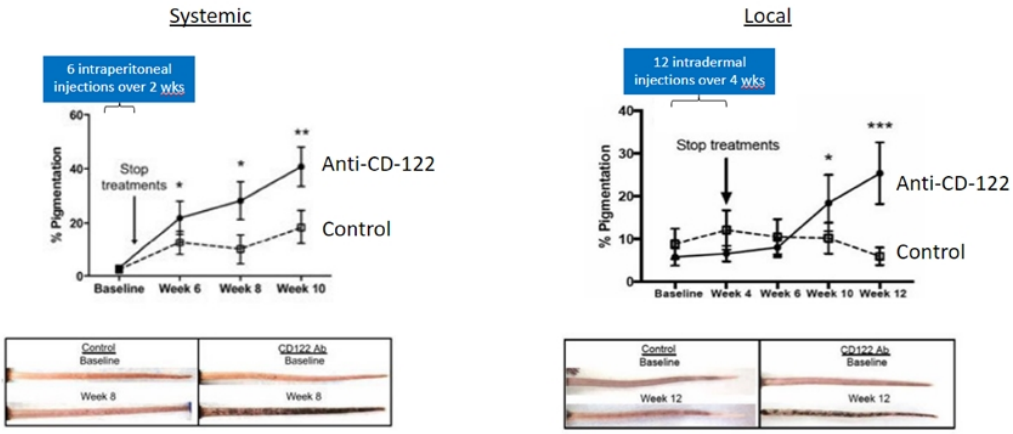


Melanin-reactive T cells eliminated pigment in tail

Anti-CD122 treatment restored pigmentation

Richmond, 2012. Sci Transl Med. 2018 PMID 30021889

ANTI-CD122 IN A MOUSE MODEL OF VITILIGO: POTENTIAL OF DURABLE RESPONSE WITH INFREQUENT DOSING REGIMEN



Richmond JM et al, Sci Transl Med. 2018;10(450): eaam7710.

Note: anti-mouse CD122 (surrogate molecule) was used in these studies.

FB102 HAS A UNIQUE MOA FOR TREATING VITILIGO BY INHIBITING TWO CYTOKINES TO REDUCE IMMUNE ATTACK OF MELANOCYTES

JAK based
Carries Black Box Warning

JAK 3/TEC → ritlecitinib (LITFULO)

Pfizer

JAK 1/3 → Tofacitinib (ZELJANZ)

Pfizer

JAK1/2/3 TYK2 → Upadacitinib

abbvie

JAK1 → povorcitinib (Incyte INCB54707)

Incyte

Reducing Autoimmune Response

Single Cytokine

IL-17A → aixekizumab (Taltz),

Lilly

IL-17A → secukinumab (COSENTYX)

NOVARTIS

IFNα → anifrolumab

AstraZeneca

TNF-α → adalimumab (Humira), ertanercept TNF-α, infliximab (Remicade),

abbvie AMGEN janssen

Multi Cytokine

CD122 (IL-2/IL-15) → FB102

FORTE BIOSCIENCES, INC

IL-12/23 → ustekinumab (Stelara)

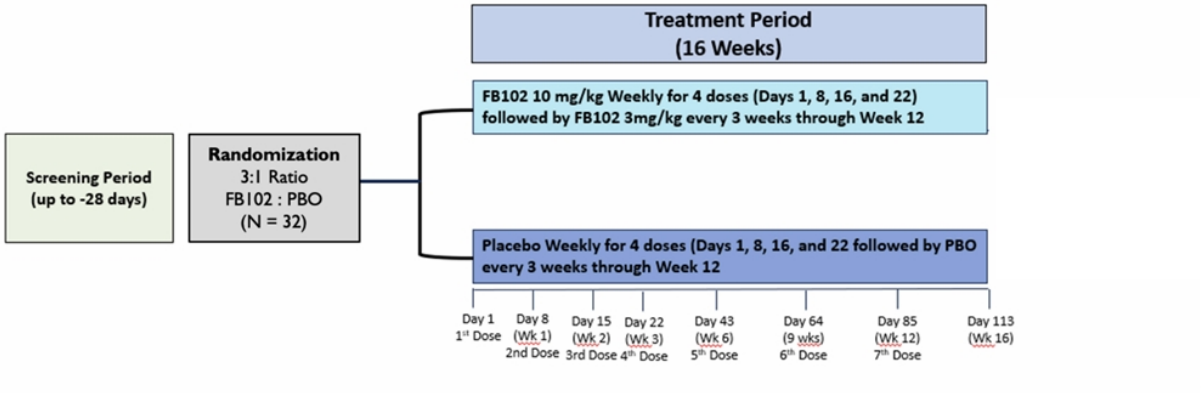
janssen

CD122 (IL-2/IL-15)→ INCA34460

Incyte

VITILIGO FB102 TRIAL OVERVIEW

VITILIGO PHASE 1B DESIGN

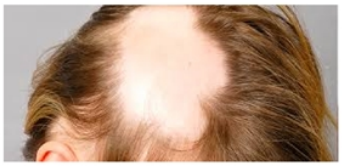


- 10-12 sites in AUS/NA
- Up to 32 subjects
- F-VASI endpoint (16/24 weeks)
- Topline data expected in 1H26

ALOPECIA AREATA

LARGE UNMET NEED IN ALOPECIA AREATA PRESENTS A SIGINIFICANT MARKET OPPORTUNITY

- Alopecia areata is a common autoimmune disease that occurs when pathogenic T cells attack hair follicles causing hair loss
- Alopecia areata affects both men and women and affects all racial and ethnic groups
- Market Opportunity
  - It is estimated that 2% of the global population will experience alopecia areata at some point in their lifetime
  - It is estimated that at 700K Americans are currently affected by alopecia areata
  - While JAK inhibitors have demonstrated efficacy, regulatory scrutiny of the JAK class including black box warnings has dampened enthusiasm for this class and as a result there remains a significant unmet need for safe and effective therapies for treating AA and vitiligo

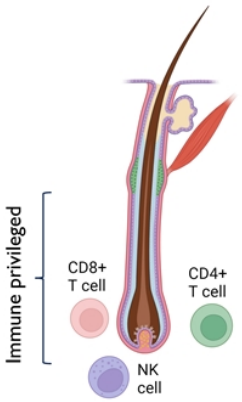


https://www.naaf.org/
https://www.niams.nih.gov/health-topics/alopecia-areata

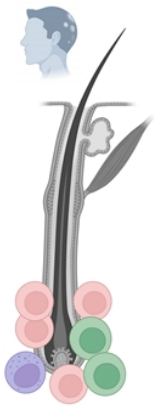
ALOPECIA ARETA (AA) IS DRIVEN BY T LYMPHOCYTES INVADING HAIR FOLLICLES

- A healthy hair bulb in anagen phase of the hair follicle cycle is surrounded by an immunosuppressive environment (an immune privileged space): includes no MHC class I expression
- Poorly understood environmental factors cause immune privilege collapse
- If the space collapses, immune cells attack pigment expressing cells in the hair bulb
  - MHC I is strongly overexpressed
  - Dense accumulation of lymphocytes at hair bulb: 60–80% CD4+ and 20–40% CD8+ T cells
- IL-2 is elevated in AA patients, and IL-15 is overexpressed in AA hair follicles

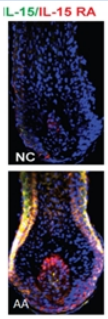
Bertolini Exp Dermatol. 2020 PMID: 32682334; Ito Clin Dev Immunol. 2013 PMID: 24151515; Ito 2014 Exp Dermatol. 2014 PMID: 25040075; Xing Nat Med. 2014 PMID: 25129481



Healthy follicle
- Immune privileged space (T and NK cells excluded)
- No MHC expression



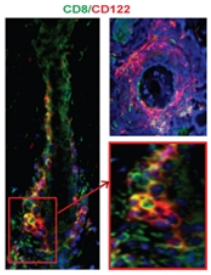
Allopecia follicle
- Loss of immune privilege
- Invasion of T and NK cells
- Cytokines (e.g. IL-15, IL-2)
- MHC expression
- Hair loss



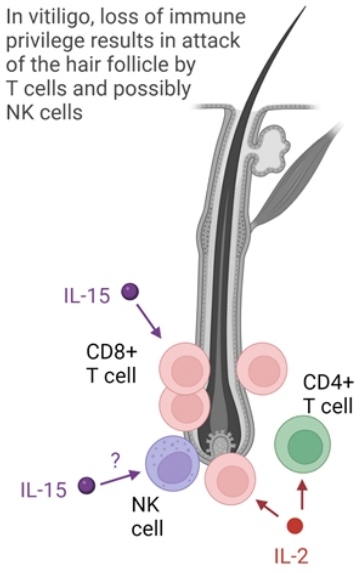
Left: Co-expression of IL-15 and IL-15RA in alopecia areata (AA) but not normal control (NC)

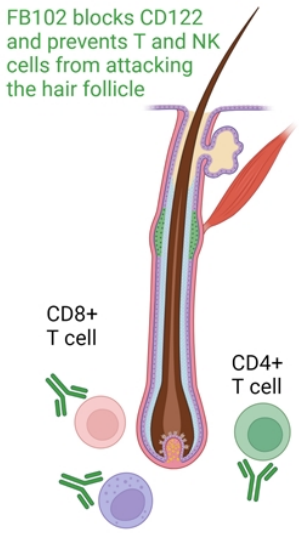
Below: CD122+ CD8+ T cells in follicles from AA patients

Xing Nat Med. 2014 PMID: 25129481



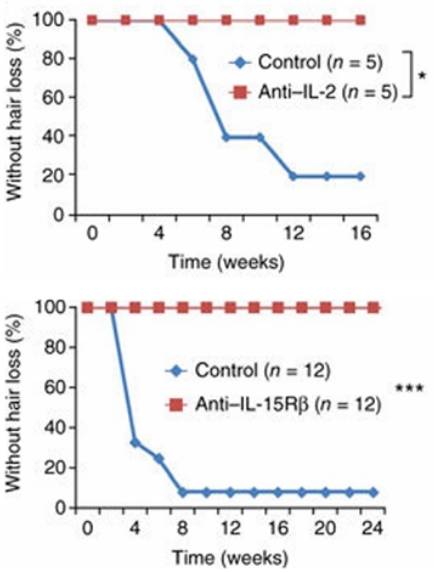
FB102 SHOULD PROTECT HAIR FOLLICLES FROM IMMUNE ATTACK AFTER LOSS OF IMMUNE PRIVILEGE





ANTI-CD122 OR ANTI-IL-2 ANTIBODIES PREVENT HAIR LOSS IN A MOUSE MODEL OF VITILIGO

- C3H/HeJ mice spontaneously alopecia with age with about 20% penetrance
- Grafting C3H/HeJ skin from a mouse with alopecia to mice that have not developed alopecia results in rapid disease onset (21 to 35 days) in 100% of successfully grafted recipient mice (McElwee J Invest Dermatol. 1998 PMID: 9804341)
- Pathologically resembles human disease
- Anti-CD122 antibody, dosed 200 µg twice per week for 12 weeks, or 2 combined anti-IL-2 antibodies, dosed 250 µg each three times per week, prevented vitiligo in this model (Xing Nat Med. 2014 PMID: 25129481)



COMPETITIVE PIPELINE

JAK based
Carries Black Box Warning

JAK 3/TEC → ritlecitinib (LITFULO)

Pfizer

JAK 1/2 → baricitinib (Olumiant)

Lilly

JAK 1/2 → deuruxolitinib (LEQSELVI)

SUN PHARMA

JAK1/2/3 TYK2 → Upadacitinib

abbvie

Reducing Autoimmune Response

Single Target

S1P → etrasimod (VELSIPITY)

Pfizer

OX40 → IMG-007

INMAGENE

IL-2 → Rezpegaldesleukin

NEKTAR

Multi Target

CD122 (IL-2/IL-15) → FB102

FORTE BIOSCIENCES, INC

IL-4/13 → dupilumab (Dupixent)

sanofi REGENERON

IL7/TSLP → bempikibart

32 BIO

IL-2/9/15 → EQ101

equillium

TYPE I DIABETES

TYPE 1 DIABETES IS CAUSED BY AUTOREACTIVE T CELLS DESTROYING INSULIN-PRODUCING PANCREATIC BETA CELLS

- Type 1 diabetes (T1D) is classified by three stages of progression
  - Stage 1: at least one diabetes-related autoantibody but has normal blood sugar and no symptoms
  - Stage 2: at least two diabetes-related autoantibodies and abnormal blood sugar levels but otherwise symptom free
  - Stage 3: significant beta cell loss has occurred; abnormal blood sugar levels; hemoglobin A1C >6.4%; excessive thirst and urination; blurry vision; fatigue; requires insulin for disease management
- Market Opportunity
  - 64,000 people diagnosed with Type 1 diabetes annually[1]
  - 2 approved treatment option to delay the onset of Type 1 Diabetes

FB102 may represent a safer way to delay the onset of Type 1 Diabetes

1 - 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 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[ll] (9% vs 0%)
- hypersensitivity reactions and serum sickness (2% vs 0%)
- lymphopenia (73% vs 6%)
- neutropenia (7% vs 3%)

From LANTIDRA PI

ADVERSE REACTIONS

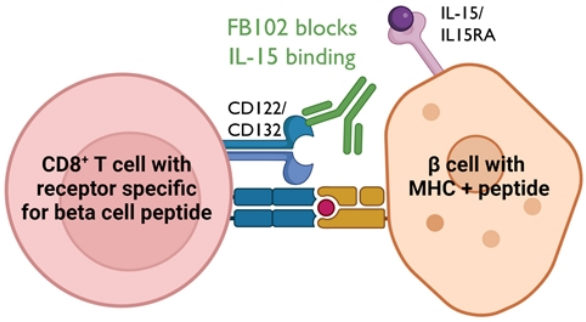
Ninety percent (90%) of subjects had at least one serious adverse reaction. (6.1)
The major causes are attributed to:

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

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

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

BASED ON THE MOA, FB102 INHIBITS IL-15/IL-2 CYTOKINE FROM ACTIVATING BETA-CELL SPECIFIC CD8+ T CELLS, PREVENTING BETA-CELL KILLING

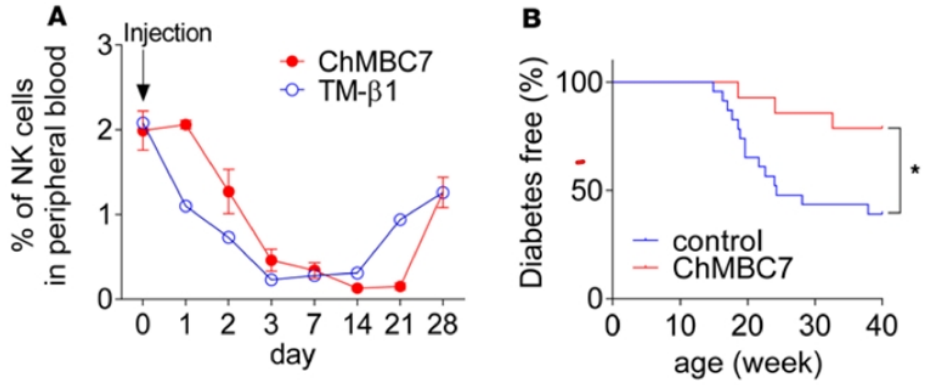


CD8+ T cells with receptors recognizing β-cell specific peptides are enriched in pancreatic islets of T1D patients

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

Herold 2024 Nat Rev Immunol. PMID 38308004

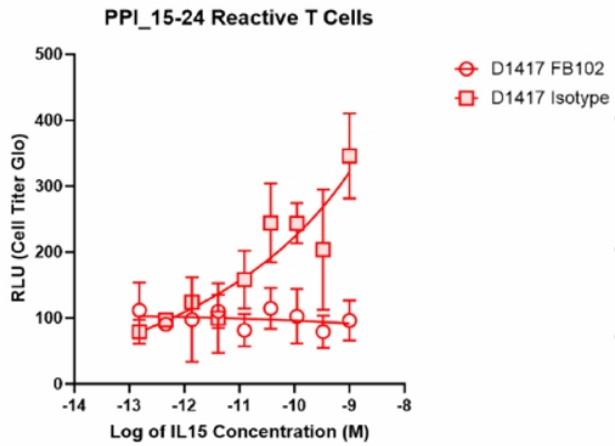
A SINGLE INJECTION OF ANTI-CD122 ANTIBODY DELAYS DIABETES ONSET IN NOD MICE AND ALSO KNOCKS DOWN NK CELLS



Yuan 2018 JCI Insight PMID 29367461

FB102 SHOWS SIGNIFICANT ACTIVITY: IN VITRO MODELS OF TYPE 1 DIABETES

FB102 PREVENTS REACTIVE T CELLS FROM PROLIFERATING AND THEREFORE PREVENTING THE KILLING OF THE BETA CELLS



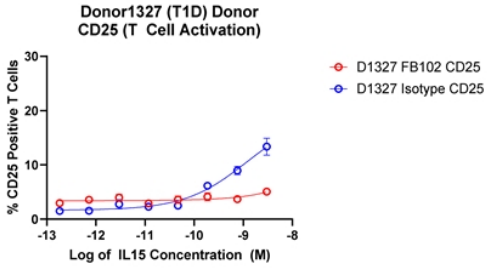
- T cells antigen specific for Beta cells and Beta cells, both collected from T1D patients
- T cells cultured with Beta cells in presence or absence of FB102
- FB102 inhibits the Beta cell induced activation and proliferation of T1D T cells

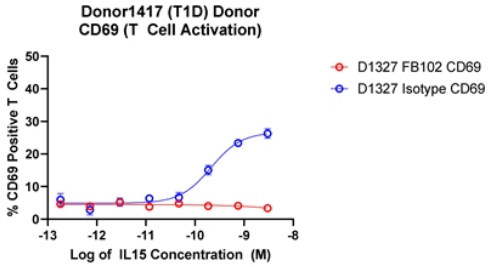
FB-102 INHIBITS TYPE 1 DIABETES T CELL ACTIVATION

FB102 inhibits T cells from T1D donor from being activated by beta cells.

T1D T cells activated in the presence of Beta Cells as measured by an increase in CD69 levels and CD25 levels

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



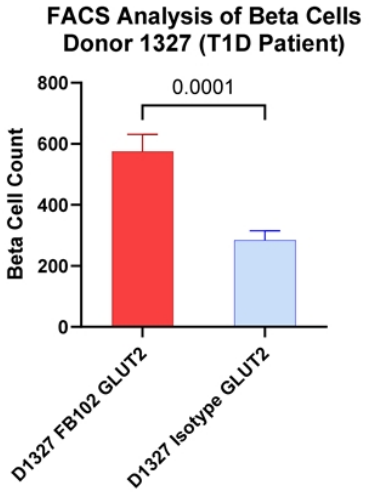


FB102 INHIBITS T1D T CELL KILLING OF BETA CELLS

FB102 inhibits T cells from T1D donor killing of beta cells.

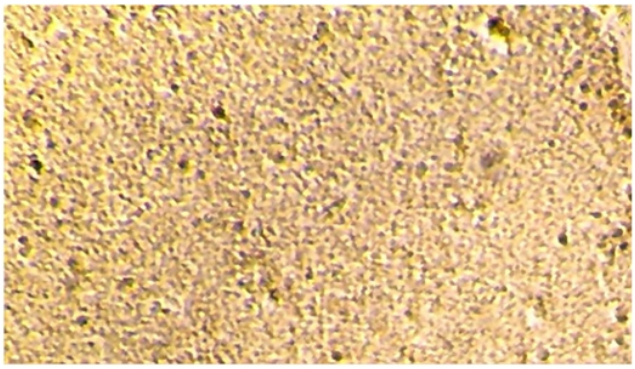
FACS Analysis of Beta Cells with FB102 or Isotype Using T Cells from Type 1 Diabetes Patient – Beta Cell Killing

Beta cell killing by T1D reactive T cells is inhibited by FB102 but not by the (negative) control antibody (p=0.0001)

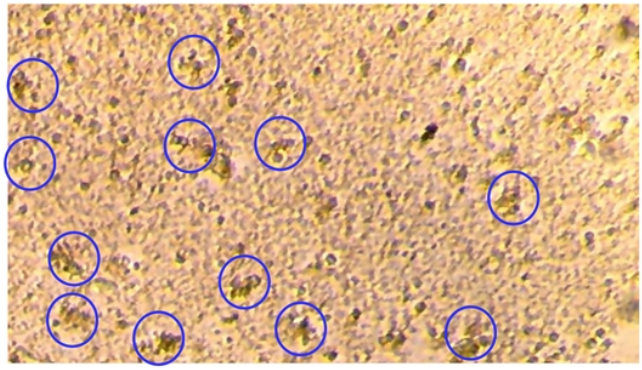


MICROSCOPY SHOWS FB102 INDUCED INHIBITION OF T CELLS ATTACK ON BETA CELLS

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



DEVELOPMENT TIMELINES

FB102 PROPOSED 12 MONTH CLINICAL DEVELOPMENT

Celiac Disease

- Phase 1b
  - Initiation 3Q24
  - Positive Topline readout in June 2025
- Phase 2
  - Initiation 2H25
  - Topline data expected in 2026

Alopecia Areata

- Phase 2
  - Initiation 2026

Vitiligo

- Phase1b Study
  - Initiated 1H25
  - Topline data expected 1H26
- Phase 2
  - Initiation 2026

Type 1 Diabetes

- Evaluating study design and initiation

SUMMARY

CLINICAL STAGE FB-102

- CD122 is a subunit of the intermediate affinity IL-2/IL-15 receptor expressed on NK and 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 the IL-2 and the IL-15 induced proliferation and activation of pathogenic NK and T cells
- Celiac disease (CeD) phase 1b trial completed and demonstrated positive histological and symptom data for FB102 treated subjects compared to placebo
- FB102 Phase 2 celiac disease initiating with data expected in 2026
- FB102 Phase 1b vitiligo trial enrolling with topline results expected in 1H26