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): May 25, 2021

CORBUS PHARMACEUTICALS HOLDINGS, INC.

(Exact name of registrant as specified in its charter)

Delaware	**001-37348**	**46-4348039**
(State or other jurisdiction of incorporation)	*(Commission File Number)*	*(IRS Employer Identification No.)*

500 River Ridge Drive, Norwood, MA	**02062**
(Address of principal executive offices)	*(Zip Code)*

Registrant's telephone number, including area code: **(617) 963-0100**

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	Name of Each Exchange on Which Registered
Common Stock, par value $0.0001 per share	CRBP	Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1 933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).

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 1.01. Entry into a Material Definitive Agreement.

Exclusive License Agreement with The Regents of the University of California

On May 26, 2021, Corbus Pharmaceuticals Holdings, Inc. (the "Company") entered into an Exclusive License Agreement (the "UCSF License Agreement") with The Regents of the University of California ('The Regents"), pursuant to which the Company received an exclusive license to certain patents relating to humanized antibodies against integrin αvβ8, one of which the Company is referring to as CRB-601, along with non-exclusive licenses to certain related know-how and materials.

In consideration for the license and other rights granted to the Company under the UCSF License Agreement, the Company paid The Regents a license issue fee of $1,500,000 and is obligated to pay an annual license maintenance fee, as well as up to $153,000,000 in potential milestone payments for the achievement of certain development, regulatory, and sales milestones. In addition, the Company is obligated to pay royalties in the low, single digits on sales of products falling within the scope of the licensed patents, which is subject to a minimum annual royalty obligation, and a percentage share of certain payments received by Company from sublicensees or in connection with the sale of the licensed program.

The UCSF License Agreement will remain in effect until the expiration or abandonment of the last patent rights licensed under the UCSF License Agreement. The UCSF License Agreement may be terminated by The Regents for the Company's failure to cure a default after notice, and the Company may terminate the UCSF License Agreement at any time upon 60 days' notice. In addition, the UCSF License Agreement will automatically terminate upon the filing of a petition for relief under the United States Bankruptcy Code by or against the Company or if the Company files a claim asserting that the patents licensed under the UCSF License Agreement are invalid or unenforceable.

The USCF License Agreement also contains customary representations, warranties and covenants, as well as customary provisions relating to indemnification,

confidentiality and other matters.

License Agreement with Milky Way BioPharma, LLC

On May 25, 2021, the Company entered into a License Agreement ("the Milky Way License Agreement") with Milky Way BioPharma, LLC ('Milky Way"), a subsidiary of Panorama Research Inc., pursuant to which the Company received an exclusive license, under certain patent rights and know-how owned or controlled by Milky Way, to develop, commercialize, and otherwise exploit products containing antibodies against integrin αvβ6 and/or integrin αvβ8 ("Licensed Products"), one of which the Company is referring to as CRB-602. Under the terms of the Milky Way License Agreement, the Company will have sole responsibility for research, development, and commercialization of any Licensed Products, and Company has agreed to use commercially reasonable efforts to perform these activities.

In consideration for the license and other rights granted to the Company under the Milky Way License Agreement, the Company paid Milky Way an upfront payment of $500,000 and will issue to Milky Way $250,000 of shares of its common stock, par value $0.0001 per share (the "Common Stock"), to be determined based on the average of the volume-weighted average price per share of Common Stock, to be issued on the later of (i) ninety (90) days following the date of the Milky Way License Agreement or (ii) five (5) business days following the date of approval by the stockholders of the Company of a proposal to increase the number of authorized shares of Common Stock in an amount equal to at least 300,000,000 shares of Common Stock. The Company is obligated to pay up to $53,000,000 in potential milestone payments for the achievement of certain development, regulatory, and sales milestones. At the Company's election, the Company may satisfy a portion of certain milestone payments by issuing shares of Common Stock. In addition, the Company is obligated to pay royalties in the low, single digits on sales of Licensed Products during the life of the applicable licensed patents on a country-by-county and product-by-product basis (the "Royalty Term"), which is subject to a minimum annual royalty obligation, as well as a percentage share of certain payments received by Company from sublicensees.

The Milky Way License Agreement will remain in effect, on a country-by-country and product-by-product basis, until the expiration of the Royalty Term in each country. The Milky Way License Agreement may be terminated by either party for the other party's uncured material breach or insolvency or bankruptcy, and the Company may terminate the agreement at any time upon 30 days' notice during the first year of the agreement or upon 180 days' notice thereafter.

The Milky Way License Agreement also contains customary representations, warranties and covenants, as well as customary provisions relating to indemnification, confidentiality and other matters.

Copies of the UCSF License Agreement and the Milky Way License Agreement referenced above will be filed as an exhibit in a subsequent periodic report to be filed under the Securities Exchange Act of 1934, as amended (the "Exchange Act").

Item 3.02. Unregistered Sales of Equity Securities

The information set forth above in Item 1.01 of this Current Report on Form 8-K under "License Agreement with Milky Way BioPharma, LLC" is incorporated into this Item 3.02 by reference. On May 26, 2021, the Company agreed to issue to Milky Way 147,875 shares of Common Stock (the "Shares") to be issued on the later of (i) ninety (90) days following the date of the Milky Way License Agreement or (ii) five (5) business days following the date of approval by the stockholders of the Company of a proposal to increase the number of authorized shares of the Common Stock in an amount equal to at least 300,000,000 shares of Common Stock. The Shares will be issued without registration under the Securities Act of 1933, as amended (the "Securities Act"), pursuant to an exemption provided by Section 4(a)(2) of the Securities Act. The Company will rely on this exemption from registration based in part on representations made by Milky Way.

Item 7.01. Regulation FD Disclosure.

On June 1, 2021, the Company issued a press release announcing the entry into the UCSF License Agreement and the Milky Way License Agreement. A copy of the press release is furnished as Exhibit 99.1 hereto and shall not be deemed "filed" for the purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Exchange Act or the Securities Act of 1933, as amended, except as shall be expressly set forth by specific reference in such a filing.

Item 8.01. Other Events

On June 1, 2021, the Company provided following business update:

As described above, on May 25, 2021, and May 26, 2021, we obtained license rights to two monoclonal antibodies ("mAbs"), compounds that we are referring to as CRB-601 and CRB-602, that target certain integrins to inhibit the activation of transforming growth factor β ("TGFβ"). TGFβ is a multifunctional cytokine involved in many cellular processes, including cell growth and differentiation, immune responses, wound healing, and tissue repair. Integrins are a family of membrane-bound αβ heterodimers that mediate interactions between cells and play key roles in cell signaling and homeostasis. The av family of integrins, such as avb6 and avb8, release TGFβ from its latent complex, allowing this cytokine to exert its biologic effects. These integrins are expressed by cancer cells and blocking them should inhibit multiple effects of TGFb in the tumor microenvironment that promote cancer growth and metastases. Integrin-mediated release of TGFb also plays a key role in fibrosis.

CRB-601

We obtained exclusive worldwide rights to CRB-601 from The Regents of the University of California. CRB-601 is an anti-avb8 mAb and potent at picomolar concentrations in inhibiting activation of TGFβ. TGFβ is thought to promote growth and metastases of established tumors, inducing immunosuppression, fibrosis, blood vessel growth, and changes in tumor cells themselves. Some tumor cells of human epithelial malignancies express αvβ8, leading to activation of TGFβ in the tumor microenvironment. Overexpression of αvβ8 by tumor cells and expression of TGFβ in tumors has been linked to poor clinical outcomes.

We plan to develop CRB-601 for treatment of solid tumors in combination with existing therapies, including checkpoint inhibitors. We expect to begin clinical trials with CRB-601 in 2022.

CRB-602

We obtained exclusive worldwide rights to CRB-602 from Milky Way BioPharma, LLC, a subsidiary of Panorama Research Inc. CRB-602 was developed to specifically inhibit both avb6 and avb8. The avb6 and avb8 integrins have been implicated in fibrotic diseases and in cancers of epithelial cell origin. We believe targeting these integrins at the same time is a rational approach to treating fibrotic diseases, including idiopathic pulmonary fibrosis, and carcinomas. We expect that CRB-602 may provide potential therapeutic benefit in fibrotic diseases and cancer. We expect to begin clinical trials with CRB-602 in 2022.

The Company is using the slides attached hereto as Exhibit 99.2 to this Current Report on Form 8-K in connection with management presentations to describe its business.

Forward- Looking Statements

This Current Report on Form 8-K contains certain forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934 and Private Securities Litigation Reform Act, as amended, including those relating to the Company's product development, clinical trials, clinical and regulatory timelines, market opportunity, competitive position, possible or assumed future results of operations, business strategies, potential growth opportunities and other statement that are predictive in nature. These forward-looking statements are based on current expectations, estimates, forecasts and projections about the industry and markets in which we operate and management's current beliefs and assumptions.

These statements may be identified by the use of forward-looking expressions, including, but not limited to, "expect," "anticipate," "intend," "plan," "believe," "estimate," "potential," "predict," "project," "should," "would" and similar expressions and the negatives of those terms. These statements relate to future events or our financial performance and involve known and unknown risks, uncertainties, and other factors which may cause actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Such factors include those set forth in the Company's filings with the Securities and Exchange Commission. Prospective investors are cautioned not to place undue reliance on such forward-looking statements, which speak only as of the date of this press release. The Company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibit No.	Description
99.1	Press Release issued by Corbus Pharmaceuticals Holdings, Inc. dated June 1, 2021
99.2	Investor Presentation
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.

CORBUS PHARMACEUTICALS HOLDINGS, INC.

Dated: June 1, 2021

By: */s/ Yuval Cohen*
Name: Yuval Cohen
Title: Chief Executive Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	Press Release issued by Corbus Pharmaceuticals Holdings, Inc. dated June 1, 2021
99.2	Investor Presentation
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Exhibit 99.1

Corbus Pharmaceuticals Licenses Two Integrin Targeting mAbs Further Expanding Pipeline into Cancer and Fibrotic Diseases

- Corbus diversifies pipeline with two new mAbs that target integrins that inhibit activation of TGFβ
- High potency anti- αvβ8 mAb licensed from University of California San Francisco and anti- αvβ6/ αvβ8 mAb licensed from Panorama Research Inc.
- Both mAbs expected to start Phase 1 testing in 2022
- Capital and resources in place to advance multiple programs into clinical development
- Company to host conference call and webcast today, Tuesday, June 1, 2021 at 8:30 a.m. ET

Norwood, MA, June 1, 2021 (GLOBE NEWSWIRE) — Corbus Pharmaceuticals Holdings, Inc. (NASDAQ: CRBP) ("Corbus" or the "Company"), today announced licensing deals for two new monoclonal antibodies (mAbs), CRB-601 and CRB-602, that target integrins to inhibit activation of transforming growth factor β (TGFβ). This new integrin program, in addition to the existing endocannabinoid system program, strengthens and diversifies Corbus' immunology pipeline for inflammatory, fibrotic, and metabolic diseases, and cancer. With these additions, Corbus expects to have four compounds other than lenabasum in Phase 1 testing in 2022.

Targeting integrins to inhibit TGFβ activation

TGFβ is a multifunctional cytokine involved in many cellular processes, including cell growth and differentiation, immune responses, wound healing, and tissue repair. TGFβ plays a key role in fibrosis and also promotes cancer growth and metastasis via its effects in the tumor microenvironment (TME). The integrins αvβ6 and αvβ8 are expressed by cancer cells, and αvβ6 is also expressed on epithelial cells in fibrotic diseases. These integrins enable TGFβ to exert its biologic effects by releasing it from its latent complex. The goal of blocking these integrins is to inhibit the deleterious effects of TGFβ. A number of other preclinical and early clinical stage programs are testing this approach of inhibiting αv integrins.

CRB-601 and CRB-602 are two novel and distinct anti-integrin mAbs:

- CRB-601 is an anti-αvβ8 mAb rationally designed by Dr. Stephen Nishimura and his colleagues at the University of California San Francisco and is potent at picomolar concentrations in inhibiting activation of TGFβ. C6D4, the parent mAb of CRB-601, has single agent activity as well as synergistic activity when combined with an anti-PD1 mAb in syngeneic mouse tumor models. Corbus plans to develop CRB-601 for treatment of solid tumors in combination with existing therapies, including checkpoint inhibitors. Phase 1 studies are expected to start in 2022.
- CRB-602 was developed by Panorama Research Inc. to specifically inhibit both αvβ6 and αvβ8. Both αvβ6 and αvβ8 have been implicated in fibrotic diseases and in cancers of epithelial cell origin. Corbus believes targeting both integrins at once is a rational approach to treating fibrotic diseases and carcinomas. Phase 1 studies are expected to start in 2022.

"We look forward to a strong partnership with Corbus and hope to see our mAb make a positive impact on the lives of cancer patients all over the world," said Anthony Francis, Executive Director of Technology Management at UCSF Innovation Ventures.

"Corbus is committed to developing new medicines to improve the lives of people who need them," stated Yuval Cohen, Ph.D., Chief Executive Officer. "We believe these two new integrin-targeting mAbs offer a promising approach to inhibiting TGFβ, fit well with our expertise in immunology, and diversify and expand our pipeline. We plan to advance up to four new programs into the clinic next year and have the capital and resources to do so."

The Company's $125 million of cash and investments on hand, as of March 31, 2021, is expected to fund operations into the first quarter of 2024, based on the current planned expenditures.

Transactions Terms and Conditions:

Under the combined terms of the two exclusive licensing agreements, Corbus will pay $2,000,000 upfront and will make potential development and sales milestone payments totaling up to $206,000,000 and pay low single-digit royalties on sales.

Conference Call and Webcast Information:

Corbus management will host a conference call and webcast presentation for investors, analysts, and other interested parties today, Tuesday, June 1, 2021, at 8:30 a.m. ET.

To participate on the call, please dial (877) 407-3978 (domestic) or (412) 902-0039 (international). The live webcast will be accessible on the Events page of the Investors section of the Corbus website, www.corbuspharma.com, and will be archived for 90 days.

About UCSF

The University of California, San Francisco (UCSF) is exclusively focused on the health sciences and is dedicated to promoting health worldwide through advanced biomedical research, graduate-level education in the life sciences and health professions, and excellence in patient care. It includes UCSF Health, which comprises three top-ranked hospitals, as well as affiliations throughout the Bay Area. Learn more at ucsf.edu, or see our Fact Sheet.

About Panorama Research Inc.

Panorama Research Inc. is a translational research lab and incubator with deep domain expertise in antibody engineering and preclinical drug development. Panorama focuses on identifying promising therapeutic targets and incubates novel, proprietary technologies from in-house research or through collaboration with leading academic institutions. Panorama is headquartered in Sunnyvale, CA.

About Corbus

Corbus is committed to leveraging our expertise in immunology to fulfill our purpose of developing innovative new medicines that improve the lives of people living with inflammatory, fibrotic, and metabolic diseases, and cancer. Corbus' current pipeline includes small molecules that activate or inhibit the endocannabinoid system and anti-integrin monoclonal antibodies that block activation of TGFβ. Corbus is headquartered in Norwood, Massachusetts. For more information on Corbus, visit corbuspharma.com. Connect with us on Twitter, LinkedIn and Facebook.

Forward-Looking Statements

This press release contains certain forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934 and Private Securities Litigation Reform Act, as amended, including those relating to the Company's restructuring, trial results, product development, clinical and regulatory timelines, market opportunity, competitive position, possible or assumed future results of operations, business strategies, potential growth opportunities and other statement that are predictive in nature. These forward-looking statements are based on current expectations, estimates, forecasts and projections about the industry and markets in which we operate and management's current beliefs and assumptions.

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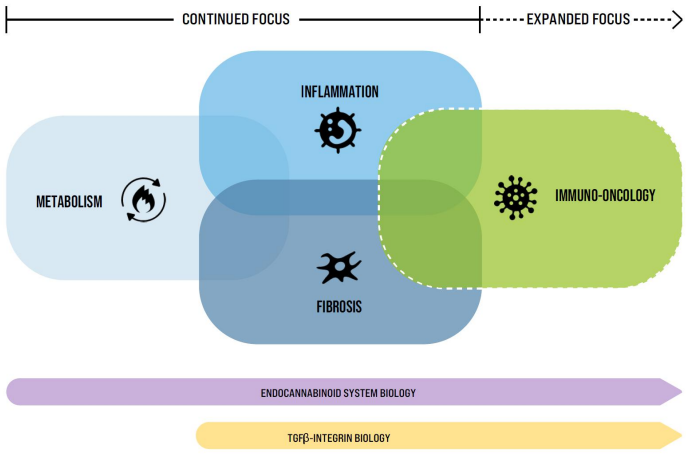
Exhibit 99.2

FORWARD-LOOKING STATEMENTS

This presentation contains certain forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934 and Private Securities Litigation Reform Act, as amended, including those relating to the Company's restructuring, trial results, product development, clinical and regulatory timelines, market opportunity, competitive position, possible or assumed future results of operations, business strategies, potential growth opportunities and other statements that are predictive in nature. These forward-looking statements are based on current expectations, estimates, forecasts and projections about the industry and markets in which we operate and management's current beliefs and assumptions. These statements may be identified by the use of forward-looking expressions, including, but not limited to, "expect," "anticipate," "intend," "plan," "believe," "estimate," "potential," "predict," "project," "should," "would" and similar expressions and the negatives of those terms. These statements relate to future events or our financial performance and involve known and unknown risks, uncertainties, and other factors, including the potential impact of the recent COVID-19 pandemic and the potential impact of sustained social distancing efforts, on our operations, clinical development plans and timelines, which may cause actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Such factors include those set forth in the Company's filings with the Securities and Exchange Commission. Prospective investors are cautioned not to place undue reliance on such forward-looking statements, which speak only as of the date of this press release. The Company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise.

Expanding Our Therapeutic Focus



- Expertise across all phases of drug development
- Track record of executing complex global studies on time and on budget
- Funded through Q1 2024

A Diverse Pipeline with Multiple Shots on Goal

Compound	Therapeutic Areas / Indications	Preclinical	Phase 1	Phase 2	Phase 3
TARGETING THE ENDOCANNABINOID SYSTEM					
Lenabasum	Dermatomyositis				
	Lupus				
CB1 Inverse Agonists	Metabolism				
CB2 Agonists	Solid Tumors				
TARGETING THE TGFβ ACTIVATING INTEGRINS					
Anti-αvβ8 mAb	Solid Tumors				
Anti-αvβ6/αvβ8 mAb	Fibrosis				

CB1 = cannabinoid receptor type 1; CB2 = cannabinoid receptor type 2

PROGRAM 1:

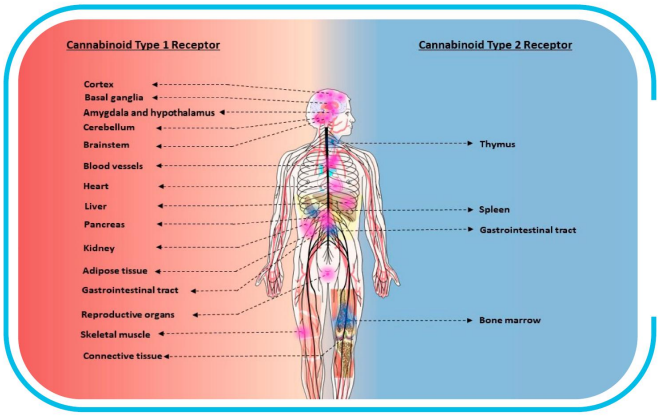
TARGETING THE ENDOCANNABINOID SYSTEM

Endocannabinoid System

2 GPCRs:
CB1 and CB2

2 endogenous agonists:
anandamide & 2-AG

Metabolic enzymes
FAAH and MAGL



Haspula, et al. *Cannabinoid Receptors: An Update on Cell Signaling, Pathophysiological Roles and Therapeutic Opportunities in Neurological, Cardiovascular, and Inflammatory Diseases.* International Journal of Molecular Sciences. 2020;21:5.

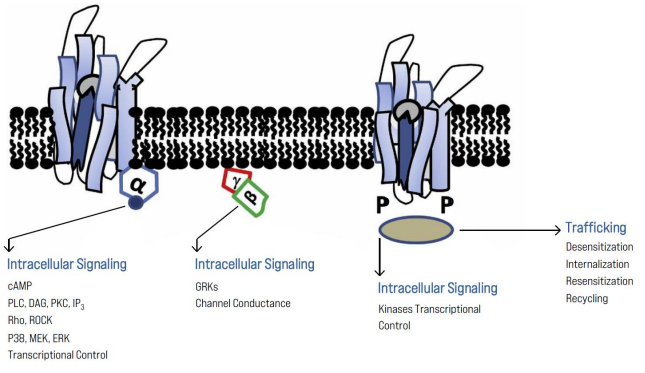
LENABASUM:
A LATE-STAGE CB2 AGONIST FOR AUTOIMMUNE DISEASES

Lenabasum: CB2 Agonist In Late-Stage Development

PURPOSE
Inflammatory and fibrotic diseases

INNOVATION
- First-in-class
- Targets activated immune cells
- Non-immunosuppressive
- Add-on to current therapies

MOA
- Activates resolution of inflammation
- Reduces levels of inflammatory mediators
- Inhibits fibrotic processes



Howlett, et al. *CB 1 and CB 2 Receptor Pharmacology.* Advances in Pharmacology. 2017;80:192.

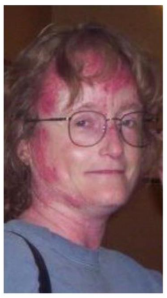
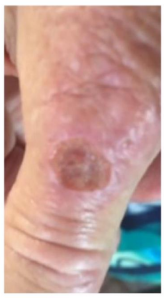
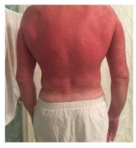
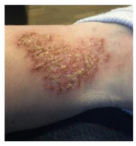
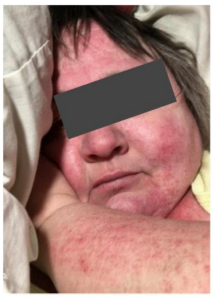
Key Lenabasum Phase 2 and 3 Studies

Target (Program)	Phase	Trial Size (n)	Status
Dermatomyositis	3	176	• Topline data expected Q2 2021
SLE	2	100	• Ongoing, topline data expected second half of 2021
Systemic Sclerosis	3	363	• Primary efficacy endpoint (ACR CRISS score) not met • FVC changes seen in sub-population in post-hoc analysis • Acceptable safety profile • Waiting for dermatomyositis data before deciding next steps
Cystic Fibrosis	2b	425	• Primary efficacy endpoint (pulmonary exacerbation rate) was not met • Acceptable safety profile • Not planning additional studies in CF

ACCEPTABLE SAFETY PROFILE IN STUDIES TO DATE, ~1,300 SUBJECTS HAVE RECEIVED LENABASUM

Most common adverse events related to lenabasum: Dizziness, headache, fatigue

What is Dermatomyositis?

DERMATOMYOSITIS:

der·mat·o·my·o·si·tis

A rare and life-threatening autoimmune disease characterized by skin and muscle inflammation.

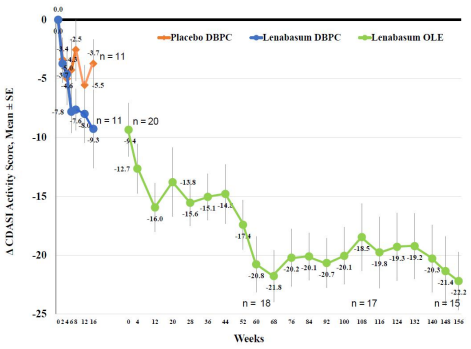
Images provided by Myositis Support and Understanding and The Myositis Association;

[1]Health Advances, LLC Analysis | [2]Schiopu et al, 2012

Affects **~80,000 PEOPLE** in North America, EU, & Japan[1,2]

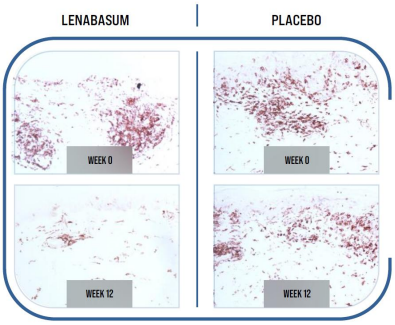
30% Mortality in 5 years [2]

Phase 2 DM Study · Improvement in Skin Activity and Biomarkers



Week 0 DBPC CDASI activity score mean (SD) = 33.3 (9.74) for lenabasum and 35.8 (7.77) for placebo. P* = 0.09, p = 0.05, p = 0.28, p = 0.04, for lenabasum vs. placebo at Weeks 4, 8, 12, and 16, respectively, of DBPC Part A of study, MMRM, 2-sided.

Lenabasum treatment was associated with reduction in CD4+ T cell infiltration in skin biopsies in DM



Reductions in CB2, interferon-1β and interferon-γ mRNAs and proteins were also observed

Dermatomyositis: Topline Phase 3 Study Results Expected Q2 2021

Trial in Adults with Active Classic or Amyopathic Dermatomyositis

PRIMARY ENDPOINT IN U.S. & EU:

- ACR & EULAR 2016 Total Improvement Score (TIS) in Adult Dermatomyositis and Polymyositis at Week 28, lenabasum BID vs. placebo (n = 176)

KEY SECONDARY ENDPOINTS:

- Definition of Improvement
- Investigator Global Assessment scale of skin activity
- TIS in subjects receiving any immunosuppressant medication for > 1 year at Baseline
- CDASI activity score

ORPHAN DRUG DESIGNATION FROM

FDA

EUROPEAN MEDICINES AGENCY
SCIENCE MEDICINES HEALTH

Systemic Lupus Erythematosus (SLE): Topline Phase 2 Study Results Expected 2H 2021

NIH

STUDY FUNDED AND MANAGED BY THE AUTOIMMUNITY CENTERS OF EXCELLENCE AT THE NATIONAL INSTITUTES OF HEALTH

PRIMARY ENDPOINT:

- Mean 7-day average maximum daily pain numerical rating score (NRS) at 12 weeks, n = 101

KEY SECONDARY ENDPOINTS:

- SLE Responder Index
- SELENA SLEDAI
- BILAG 2004

SLE AFFECTS **+200,000 PEOPLE** *IN THE U.S.*

Izmirly, et al. *Prevalence of Systemic Lupus Erythematosus in the United States: Estimates from a Meta-Analysis of the Centers for Disease Control and Prevention National Lupus Registries.* Arthritis & Rheumatology, 2021; Epub ahead of print.

2ND GENERATION CB1 INVERSE AGONISTS FOR METABOLIC AND FIBROTIC DISEASES

Obesity is a Growing Global Health Crisis

Obesity is a Growing Health Crisis in the U.S.

Currently **33%** of the U.S. population is obese and it is estimated to rise to **~50% by the end of the decade**[1]

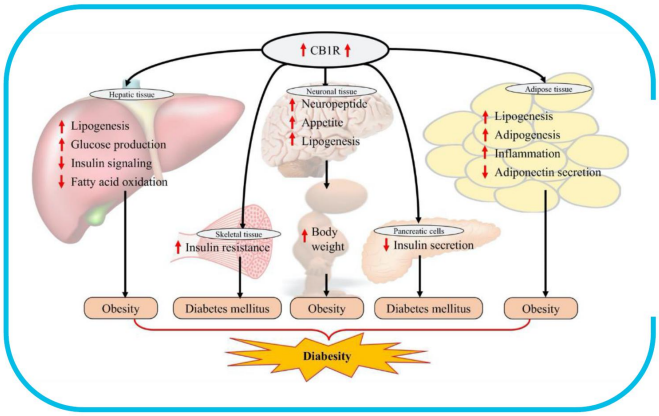
Obese patients are **2 X MORE LIKELY** to develop hypertension, dementia and certain cancers [2]

Obese patients are over **3 X MORE LIKELY** to develop diabetes, osteoarthritis and end stage renal disease [2]

~ $480B of annual direct medical costs in the US are attributed to obesity [2]

[1]Ward et al, Projected U.S. State-Level Prevalence of Adult Obesity and Severe Obesity, NEJM, Dec. 2019 | [2]Milkin Institute October 2018

CB1 Activation Contributes To "Diabesity"



CB1 ACTIVATION INCREASES:
- Appetite
- Food intake
- Fat production and storage
- Insulin resistance
- Inflammation
- Fibrosis

CB1 ACTIVATION DECREASES:
- Insulin secretion
- Fat oxidation
- Energy expenditure

Deeba, et al. *Targeting the endocannabinoid system in diabesity: Fact or fiction?*. Drug Discovery Today. 2021;in press:2.

CB1 is a Validated Target in Metabolic Diseases

SEVERAL FIRST GENERATION CB1 INVERSE AGONISTS ADVANCED TO LATE-STAGE CLINICAL DEVELOPMENT

SANOFI
Pfizer Otenabant
Bristol Myers Squibb Ibipinabant
MERCK Taranabant

ACOMPLIA Rimonabant

Rimonabant

- Approved in EU and launched in 2006 for treatment of obesity and other metabolic-related parameters
- Withdrawn in 2008 for depression and suicidality related to rimonabant binding to CB1 in the brain
- Drug class abandoned

SECOND GENERATION OF CB1 INVERSE AGONISTS AIM TO MAINTAIN POTENCY BUT MINIMIZE CB1 BINDING IN BRAIN

*Pfizer, Bristol Myers Squibb, and Merck were in clinical trials. Trials were terminated due to rimonabant withdrawal.

CB1 Inverse Agonist Program

PURPOSE

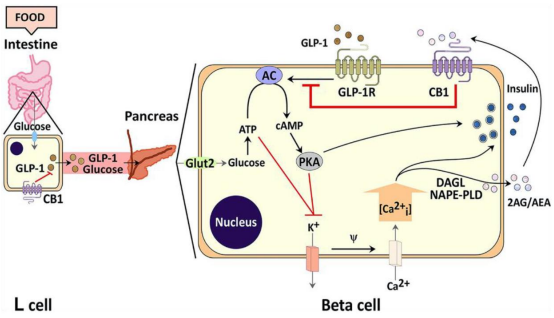
- Obesity, diabetes/diabetic nephropathy, NASH

INNOVATION

- Second generation small molecule with limited brain levels to increase safety
- Potential to augment effects of GLP-1R agonists in diabetes and obesity
- Potential to preserve renal function

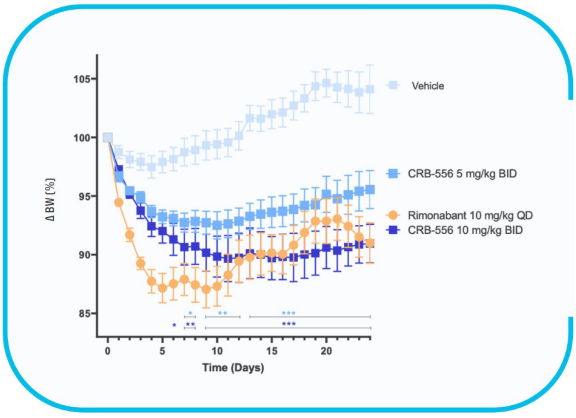
MOA

- Reduces appetite, food intake, lipogenesis, dyslipidemia, inflammation
- Increases insulin sensitivity and secretion



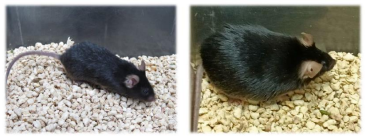
González-Mariscal, et al. *Blockade of cannabinoid 1 receptor improves GLP-1R mediated insulin secretion in mice.* Molecular and Cellular Endocrinology. 2016;422:1-10.
Neurological, Cardiovascular, and Inflammatory Diseases. International Journal of Molecular Sciences. 2020;21:5.

DIO Model: CRB-556 Induces Weight Loss in Obese Mice



Mice received a high-fat diet for 14 weeks to induce obesity and glucose intolerance prior to testing, then continued to receive high-fat diet while receiving test compounds. Vehicle is CRB-556 control. Day 0 is start of dosing with test compounds. N = 10 mice per time point per dose of compound.

- CB1 inverse agonist CRB-556 induced dose-dependent weight loss in mice with diet-induced obesity
- Effect similar to rimonabant

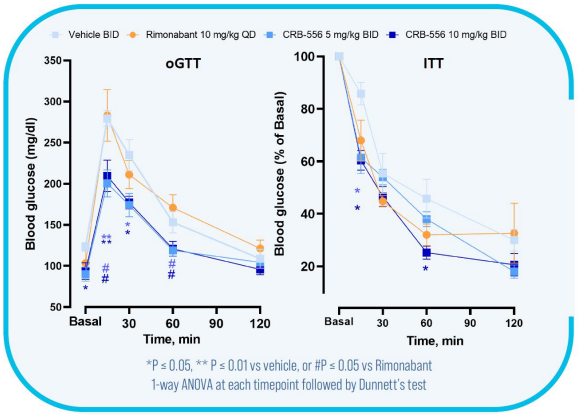


Chow diet | High fat diet

Note, mice pictured were not treated with CRB-556.
Photos are courtesy of GVK-Aragen.

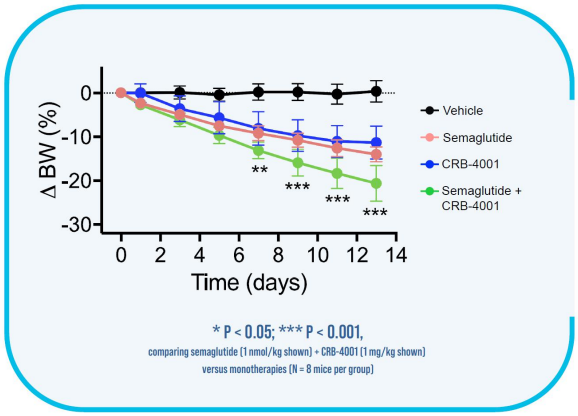
DIO Model: CRB-556 Improves Glucose Tolerance and Insulin Sensitivity in Obese Mice

CRB-556 improved glucose tolerance and insulin sensitivity in mice with diet-induced obesity, similar to rimonabant



Mice received a high-fat diet for 14 weeks to induce obesity and glucose intolerance prior to testing, then continued to receive high-fat diet while receiving test compounds for 28 days. Vehicle is CRB-556 control. Day 0 is start of dosing with test compounds. Oral glucose challenge (2 g/kg, post overnight fast) and insulin sensitivity testing (2 U/kg Humulin IP, post 6 hour fast) were done on Day 25, 1 hr 45 min after morning dose. N = 5 mice per time point per dose of compound

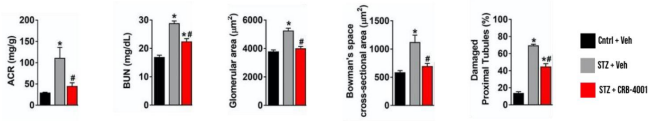
CRB-4001 Augments Weight Loss Provided by Semaglutide in Obese Mice



CB1 inverse agonist CRB-4001 augmented semaglutide-induced weight loss in obese mice and insulin sensitization (improved basal glucose levels and glucose disappearance rate)

Tizzari, et al. CB1 and GLP-1 Receptors Cross Talk Provides New Therapies for Obesity. Diabetes. 2021;70:421.

CRB-4001 Improves Renal Structure Function in a Model of Diabetic Nephropathy



#P < 0.05 relative to the corresponding streptozotocin (STZ) group treated with vehicle (Veh)

C57Bl/6 mice with STZ-induced diabetic nephropathy were treated for 16 weeks with 3 mg/kg CRB-4001. Compared to the STZ group treated with vehicle.

CRB-4001 INCREASED:

- Preservation of renal structure

CRB-4001 DECREASED:

- GLUT2 expression and translocation to basal membrane
- Blood urea nitrogen (BUN)
- Albumin to creatinine ratio (ACR)
- Inflammatory mediators
- Tubulo-interstitial fibrosis

Hinden, et al. Modulation of Renal GLUT2 by the Cannabinoid-1 Receptor: Implications for the Treatment of Diabetic Nephropathy. Journal of the American Society of Nephrology. 2018;29:436–446.

Select Corbus CB1 Inverse Agonists Have Low Brain Levels and Receptor Occupancy With Repeated Dosing in Mice

Compound	C_{max} Brain: Plasma (Ratio, Range)	$AUC_{0\text{-}24}$ Brain: Plasma (Ratio, Range)	CB1 Receptor Occupancy in Brain, Chronic Dosing, 10-20 mg/kg	Comment
Rimonabant (Sanofi)	0.90 - > 1	-	Upper limit of quantification	Single IP dose[1]
CRB-4001*	0.06-0.07	0.49-0.83	Lower limit of quantification[2]	Accumulates in brain with repeated dosing
CRB-556**	0.01-0.03	0.04-0.07	Lower limit of quantification	Minimal accumulation
CRB-545**	0.01-0.04	0.03-0.08	Lower limit of quantification	No accumulation
CRB-625**	0.00-0.002	0.00-0.01	Not determined	No accumulation

*We are not continuing development of CRB-4001. **Current candidates under evaluation for potential clinical development

1. Han, et al. *A novel peripheral cannabinoid 1 receptor antagonist, AJ5012, improves metabolic outcomes and suppresses adipose tissue inflammation in obese mice.* FASEB J. 2019; 33:4314-4326.
2. Tam, et. al. *Peripheral cannabinoid-1 receptor inverse agonism reduces obesity by reversing leptin resistance.* Cell Metab. 2012; 16:167-79

NEW DRUG CLASS:
CANNABINOID RECEPTOR AGONISTS FOR SOLID TUMORS

CB2 Agonist Immuno-oncology Program

PURPOSE

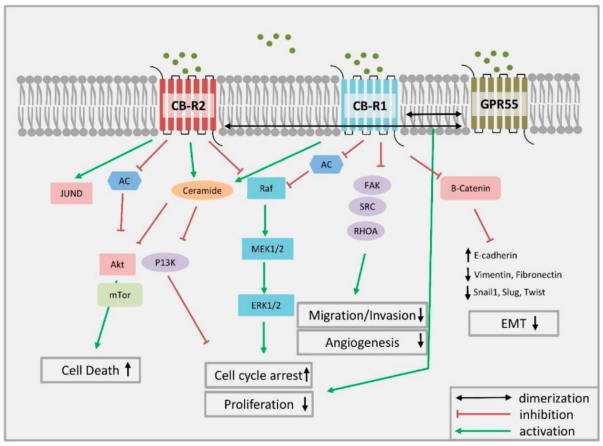
Solid tumors in combination with standard treatments including checkpoint inhibitors

INNOVATION

- First-in-class
- Affects both tumor cells and immune system
- Potential to augment effects of CPIs

MOA

- Inhibits cell cycle progression
- Inhibits cell proliferation
- Induces apoptosis
- Reduces angiogenesis
- Reduces epithelial-mesenchymal transformation
- Inhibits cell cycle progression

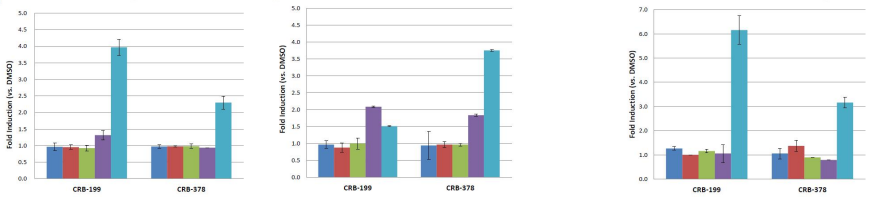


Kiskova, et al. *Future Aspects for Cannabinoids in Breast Cancer Therapy.* International Journal of Molecular Sciences. 2019;20:5.

Corbus CB2 Agonists Reduce Cancer Cell Viability by Promoting Apoptosis

Compound	Inhibition of cancer cell viability, IC50 (μM)									
	HER2+ Breast Cancer			Triple Negative Breast Cancer			ER+ Breast Cancer		Glioblastoma	
	BT474	HCC1954	SKBR3	MDA-MB-468	MDA-MB-231	MDA-MB-436	T-47D	MCF7	SF126	U251
CRB-199*	4.5	3.5	5.2	3.1	5.4	4.0	5.4	>10	9.0	3.8
CRB-378*	8.0	6.4	10.5	3.6	8.4	4.0	10.4	13.8	18.5	3.5

*Current candidates under evaluation for potential clinical development

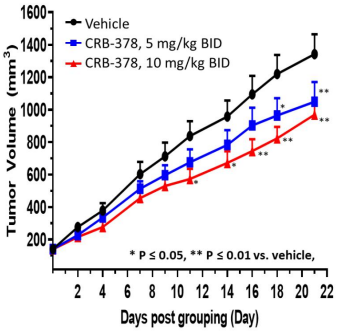


Fold-induction of caspases 3 and 7 vs. vehicle (non-cytotoxic concentrations)

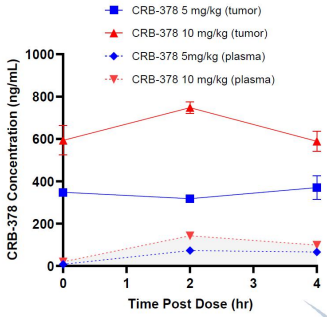
Concentrations (μM) 20.0 6.7 2.2 0.7 0.2

CRB-378 Has Single Agent Activity in HCC1954 Her2+ Breast Cancer Xenograft Model

CRB- 378 REDUCES TUMOR VOLUME



CRB- 378 IS CONCENTRATED IN THE TUMOR

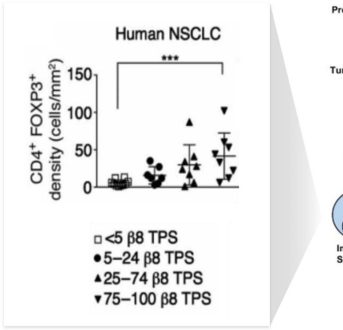


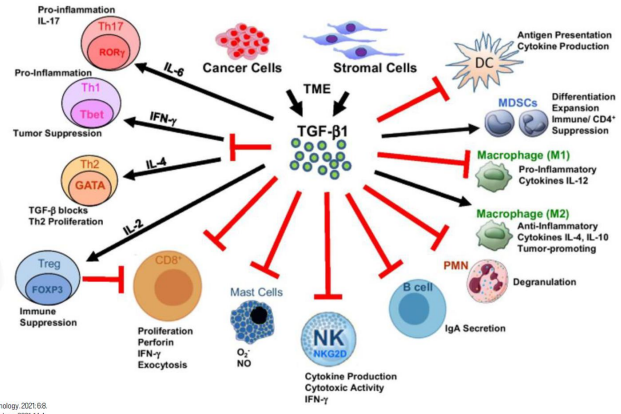
Female Balb/c mice nude mice were injected in the flank with HCC1954 Her2+ breast cancer cells. Pharmacological treatments for 21 days were started when tumors reached 90-180 mm3 were cultured with vehicle or CRB-378 for different times, with compound BKM-129 (PI3 kinase inhibitor) serving as the positive control. Tumor size was measured using a caliper and tumor volume was calculated. Concentration of CRB-378 in plasma and tumors was determined on Day 21 by LC-MS/MS.

PROGRAM 2:

TARGETING TGFβ ACTIVATING INTEGRINS

Immune Evasion is Mediated by TGFβ in Late-Stage Tumors



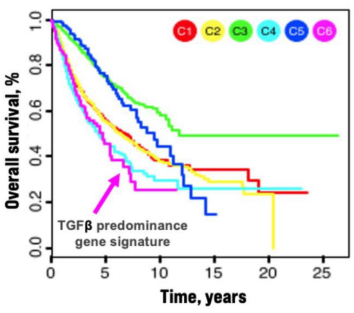


Seed, et al. *A tumor-specific mechanism of Treg enrichment mediated by the integrin αvβ8*, Science Immunology, 2021; 6:8.
Kim, et al. *Novel therapies emerging in oncology to target the TGF-β pathway*, Journal of Hematology & Oncology, 2021; 14:4.

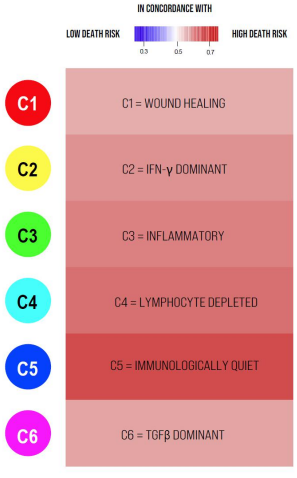
Treg numbers are increased in human non-small lung cell cancers in proportion to number of cancer cells expressing β8 (TPS)

Lower Survival in Patients with High TGFβ Tumor Gene Signature

N = 8,461 cancers, multiple cell types



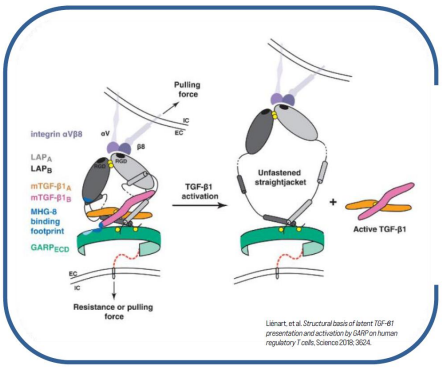
Strong concordance between TGFβ immune expression signature score and overall survival for each immune subtype of tumor



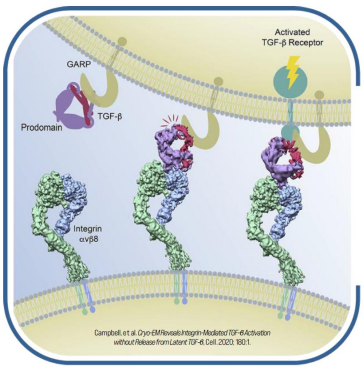
Thorsson, et al. *The Immune Landscape of Cancer*, Immunity, 2018; 48:817

αvβ8 on Tumor Cells Activates TGFβ

DIFFUSIBLE ACTIVE TGFβ



NON-DIFFUSIBLE ACTIVE TGFβ



Non-diffusible TGFβ may be most relevant form of active TGFβ in cancer

Drug Development of Inhibitors of TGFβ-Activating Integrins

MONOCLONAL ANTIBODY
SMALL MOLECULE

	ONCOLOGY		FIBROSIS	
	PHASE (DISCLOSED INDICATION)	TARGET	PHASE (DISCLOSED INDICATION)	TARGET
Pfizer	Phase 1 Solid tumors	αvβ8 Y	-	-
PLIANT	Preclinical	αvβ8 ●	Phase 2 IPF & PSC	αvβ6/1 ●
MORPHIC THERAPEUTIC	Preclinical	αvβ8 ●	Preclinical	αvβ1 ●
VENN THERAPEUTICS	Preclinical	αvβ8 Y	Preclinical	αvβ8/6 Y
AstraZeneca	-	-	Phase 1 CKD	αvβ8 Y
PLIANT / NOVARTIS	-	-	Phase 1 NASH	αvβ1 ●
abbvie / MORPHIC THERAPEUTIC	-	-	Preclinical	αvβ6 ●

CRB-601: Anti-αvβ8 mAb for Solid Tumors

PURPOSE

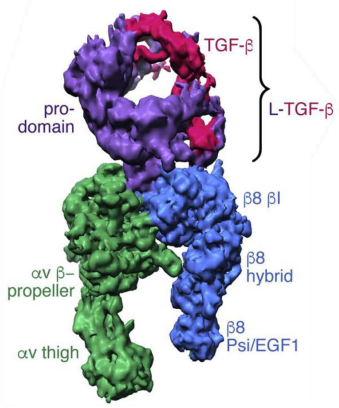
Treatment of solid tumors, in combination with standard treatments including checkpoint inhibitors

INNOVATION

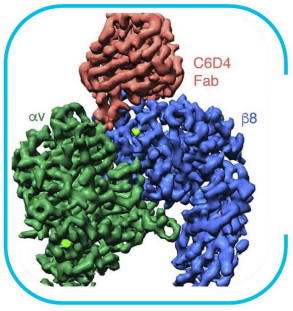
- Anti-αvβ8 mAb from Nishimura lab (UCSF)
- Genealogy: mC6D4 > hC6D4 > CRB-601
- Potential to augment effects of CPIs

MOA

- Binds with high affinity to block RGD-binding site of αvβ8
- Inhibits activation of diffusible and non-diffusible forms of TGFβ, at ~30-fold lower concentrations than 1st gen C6D4 mAb



TGFβ binding to αvβ8



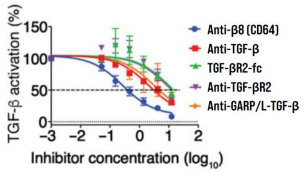
mC6D4 binding to αvβ8

Campbell, et al. *Cryo-EM Reveals Integrin-Mediated TGF-ß Activation without Release from Latent TGF-ß.* Cell. 2020; 180:490–493.

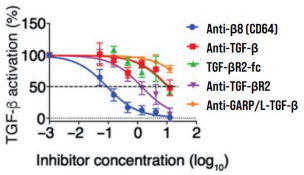
C6D4 Inhibits Activation of Both Diffusible and Non-Diffusible TGFβ

Inhibition of Activation of:

Diffusible TGFβ



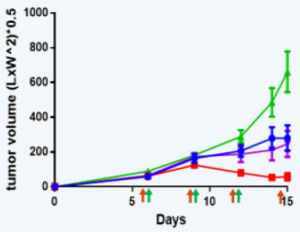
Non-Diffusible TGFβ

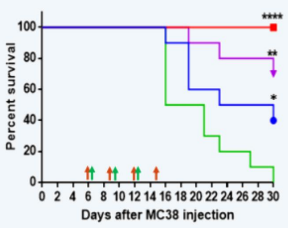


Seed, et al. *A tumor-specific mechanism of T reg enrichment mediated by the integrin αvβ8.* Science Immunology. 2021; 6:7.

Parental C6D4 Anti-αvβ8 mAb Augments Activity of Anti-PD-1 mAb

MC38 Syngeneic Mouse Colon Cancer Model



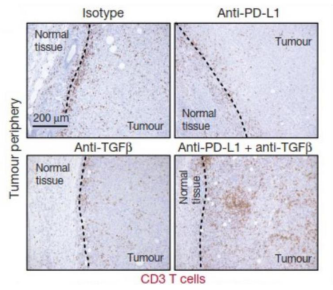


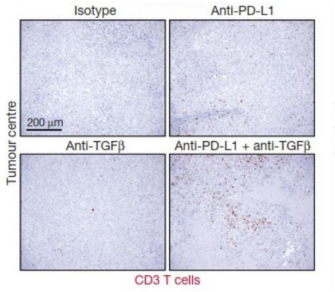
* P , 0.05, ** P < 0.01, **** P < 0.0001, vs. isotype control mAbs

C56Bl/6 mice were inoculated with MC38 cells, n = 10 per group. C6D4 (7 mg/kg) and anti-PD-1 (RMP1-14, 20 mg/kg) were administered on days 7, 10, 13, and 16.

Takasaka, et al. *Integrin αvβ8-expressing tumor cells evade host immunity by regulating TGF-ß activation in immune cells.* JCI Insight. 2018; 3:3.

Blocking Both TGFβ and PD-1 Augments T Cell Infiltration in Tumors

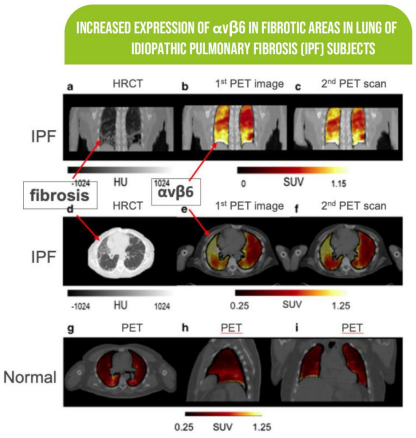




- Majority of infiltrating cells are CD8+ T cells
- Consistent with lessening immunosuppression
- CRB-601 + anti-PD-1 mAb have the potential to provide superior relief of immunosuppression

Mariathasan, et al. *TGFB attenuates tumour response to PD-L1 blockade by contributing to exclusion of T cells.* Nature. 2018; 554:547.

CRB-602: Anti-αvβ6/8 mAb for Fibrosis and Cancer



Lukey, et al. *Clinical quantification of the integrin avβ6 by [18 F]FB-A20FMDV2 positron emission tomography in healthy and fibrotic human lung (PETAL Study).* European Journal of Nuclear Medicine and Molecular Imaging. 2020; 47:974.

- Licensed from by Panorama Research Inc.
- αvβ6 integrin also activates TGFβ
- αvβ6 is expressed in high levels on tumors of epithelial origin (carcinomas)
- αvβ6 is also expressed on epithelial cells in fibrotic diseases and thought to play an important role in lung, liver, biliary, and kidney fibrosis
- αvβ6 is more highly expressed in fibrotic areas in lungs of IPF subject than in non-fibrotic areas or normal lungs
- Antibody that targets both αvβ6 and αvβ8 may be useful in treatment of certain carcinomas
- Corbus anti-αvβ6/8 mAb will be tested in animal models of cancer and fibrosis, with estimated Phase 1 start by end of 2022

Expected Clinical Milestones 2021-2022

PROGRAM	MILESTONE	Q2 2021	H2 2021	H1 2022	H2 2022
LENABASUM IN DERMATOMYOSITIS	PHASE 3 DATA	✓			
LENABASUM IN SYSTEMIC LUPUS ERYTHEMATOSUS	PHASE 2 DATA		✓		
CB1 INVERSE AGONISTS IN METABOLIC DISEASES	PHASE 1			✓	
CB2 AGONIST IN SOLID TUMORS	PHASE 1				✓
CRB-601 IN SOLID TUMORS (anti-αvβ8 mAb)	PHASE 1				✓
CRB-602 IN FIBROSIS (anti-αvβ6/8 mAb)	PHASE 1				✓

FINANCIAL PROFILE: CRBP (NASDAQ)

125M Common Shares Outstanding (140.1M Fully Diluted)

$125M Cash Balance as of 3.31.2021

A Team With a Proven Record of Execution

Yuval Cohen, PhD
Chief Executive Officer, Director
Executive leadership experience in inflammatory disease drug

Sean Moran, CPA, MBA
Chief Financial Officer
Senior financial experience with emerging biotechnology, drug delivery and medical device companies

Craig Millian, MBA
Chief Commercial Officer
Experience leading commercial organizations and building successful brands at multiple biopharma companies

Barbara White, MD
Chief Medical Officer and Head of Research
Previous academician with industry, clinical development, and medical affairs experience in inflammatory and autoimmune diseases

Ross Lobell
VP, Regulatory Affairs
Regulatory affairs experience with an extensive biopharmaceutical background in leading preclinical, clinical and nonclinical regulatory strategies

Dylan Wenke
Head, Business Development
Experience leading corporate development, partnerships, and collaborations at pharmaceutical and venture-backed startups

An Experienced and Engaged Board of Directors

Amb. Alan Holmer Ret.
Chairman of the Board
More than two decades of public service in Washington, D.C. including Special Envoy to China; Former CEO of PhRMA

Avery W. (Chip) Catlin
Director
More than 25 years of senior financial leadership experience in life science companies; Former CFO and Secretary of Celldex Therapeutics

Yuval Cohen, PhD
Chief Executive Officer, Director
More than 13 years of executive leadership experience in inflammatory disease drug development

Rachelle Jacques
Director
More than 25-year professional career, experience in U.S. and global biopharmaceutical commercial leadership, including multiple high-profile product launches in rare diseases; CEO of Enzyvant Therapeutics

John K. Jenkins, MD
Director
Distinguished 25-year career serving at the U.S. FDA, including 15 years of senior leadership in CDER and OND

Pete Salzmann, MD, MBA
Director
20 years of industry experience and currently serves as Chief Executive Officer of Immunovant (NASDAQ: IMVT), a biopharmaceutical company focused on developing therapies for patients with autoimmune diseases

RESOLVE-1 Phase 3 in Systemic Sclerosis

Largest ever study in diffuse cutaneous systemic sclerosis (n=365, 52-weeks, 76 global sites)

First in a group of studies to allow patients to remain on background immunosuppressant therapy (IST)

RESULTS
Study did not meet primary endpoint

KEY LEARNINGS
Under-appreciated benefit from IST (especially in newly diagnosed patients) led to much higher improvement in the control group than anticipated

PRIMARY EFFICACY ENDPOINT: MEDIAN ACR CRISS SCORES AT WEEK 52			
Visit 11 (Week 52)	Lenabasum 20 mg BID - N = 120	Lenabasum 5 mg BID - N = 120	Placebo N = 123
n	100	113	115
Mean (SD)	0.598 (0.432)	0.575 (0.423)	0.636 (0.422)
Median (Q1, Q3)	0.888 (0.061, 0.997)	0.827 (0.070, 0.988)	0.887 (0.071, 0.999)
p-value (Ranked Score, MMRM)	0.497	0.349	-

There were also no significant differences among treatment groups for the secondary efficacy outcomes.

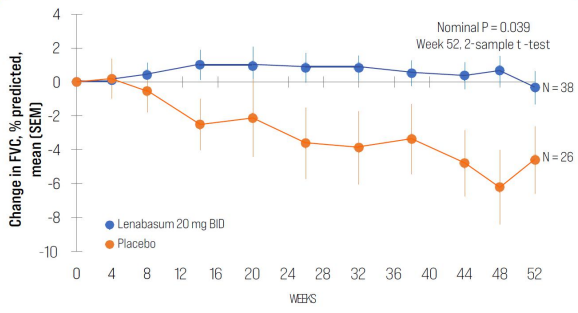
mITT population, primary efficacy analysis. MMRM with imputed values for missing core items, except LOCF for core items missing because of COVID-19.

NEXT STEPS: PREPARING THE RESOLVE-1 STUDY DATA FOR PUBLICATION AND WILL DECIDE ON THE NEXT STEPS IN THE DEVELOPMENT PROCESS PENDING THE OUTCOME OF THE DETERMINE STUDY.

PHASE 3 · Subjects Treated With Lenabasum 20 mg BID Added to Established IST (> 2 Year Duration) Had Stable FVC % Predicted

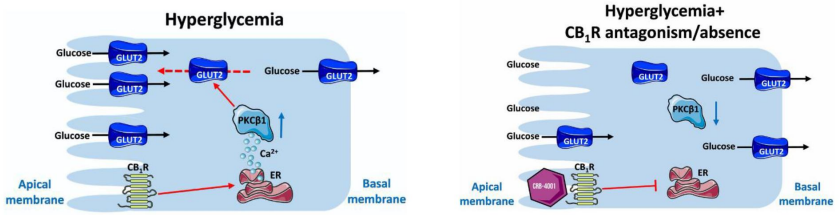
POST-HOC ANALYSES

Subjects treated with lenabasum 20 mg BID added to established immunosuppressant therapies (IST) had stable FVC % predicted over 1 year



IST = immunosuppressant therapies. Post-hoc analyses, per protocol population of subjects who completed study drug and Week 52. LOCF for any missing values. Subjects were receiving at least 1 background IST for greater than 2 years treatment duration at baseline, and any MMF treatment must be > 2 years duration

CRB-4001 Acts In a Variety of Key Metabolic Pathways



Hinden, et al. *Modulation of Renal GLUT2 by the Cannabinoid-1 Receptor: Implications for the Treatment of Diabetic Nephropathy.* Journal of the American Society of Nephrology. 2018;29:436-446.