

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): November 10, 2014

**Corbus Pharmaceuticals Holdings, Inc.**

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(Exact name of registrant as specified in its charter)

Delaware  
(State or other jurisdiction of incorporation)

333-198563  
(Commission File Number)

46-4348039  
(IRS Employer  
Identification No.)

**100 River Ridge Drive Norwood, MA 02062**

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(Address of Principal Executive Offices) (Zip Code)

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

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 (17CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13-e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

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**Item 7.01. Regulation FD Disclosure.**

Corbus Pharmaceuticals Holdings, Inc. (the “Company”) is using the slides attached hereto as Exhibit 99.1 in connection with management presentations to describe its business.

The information in this Current Report on Form 8-K, including the information contained in Exhibit 99.1, is being furnished to the Securities and Exchange Commission, and shall not be deemed to be “filed” for the purposes of Section 18 of the Securities Exchange Act of 1934 or otherwise subject to the liabilities of that section, and shall not be deemed to be incorporated by reference into any filing under the Securities Act of 1933 or the Securities Exchange Act of 1934, except as shall be expressly set forth by a specific reference in such filing.

**Item 9.01. Financial Statements and Exhibits.**

(d) Exhibits

99.1 Investor Presentation.

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**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, CORP.

Date: November 10, 2014

By: /s/ Yuval Cohen

Yuval Cohen  
Chief Executive Officer

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## EXHIBIT INDEX

<u>Exhibit No.</u>	<u>Description</u>
99.1	Investor Presentation.

**CORBUS**  
PHARMACEUTICALS



OTCQB: CRBP

[www.CorbusPharma.com](http://www.CorbusPharma.com)

***Developing Breakthrough Therapies for  
Rare Inflammatory Diseases***



# Forward-Looking Statement

*This presentation contains certain forward-looking statements, including those relating to the Company's 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. Additional written and oral forward-looking statements may be made by the Company from time to time in filings with the Securities and Exchange Commission (SEC) or otherwise. The Private Securities Litigation Reform Act of 1995 provides a safe-harbor for forward-looking statements.*

*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," "will" and similar expressions and the negatives of those terms. These statements 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. Prospective investors are cautioned not to place undue reliance on such forward-looking statements, which speak only as of the date of this presentation. The Company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise.*



# Overview

- Corbus Pharma is focusing on rare, life-threatening, chronic inflammatory diseases
- Lead drug *Resunab*™: a first-in-class oral anti-inflammatory/fibrosis small molecule
- Acts to trigger inflammatory resolution: the *new kid* for chronic inflammation
- Proven safe in Phase 1 + promising pre-clinical potency in multiple animal models
- Phase 2 clinical trials to commence 2015:
  - Cystic Fibrosis (CF)
  - Diffuse Systemic Sclerosis (SSC) also known as “Scleroderma”
- Successful \$10.3m private financing round (May 2014)
- Obtained \$1.3m in NIH grants
- IP protection until 2033 and potentially longer
- Commenced trading on OTC.QB in October 2014

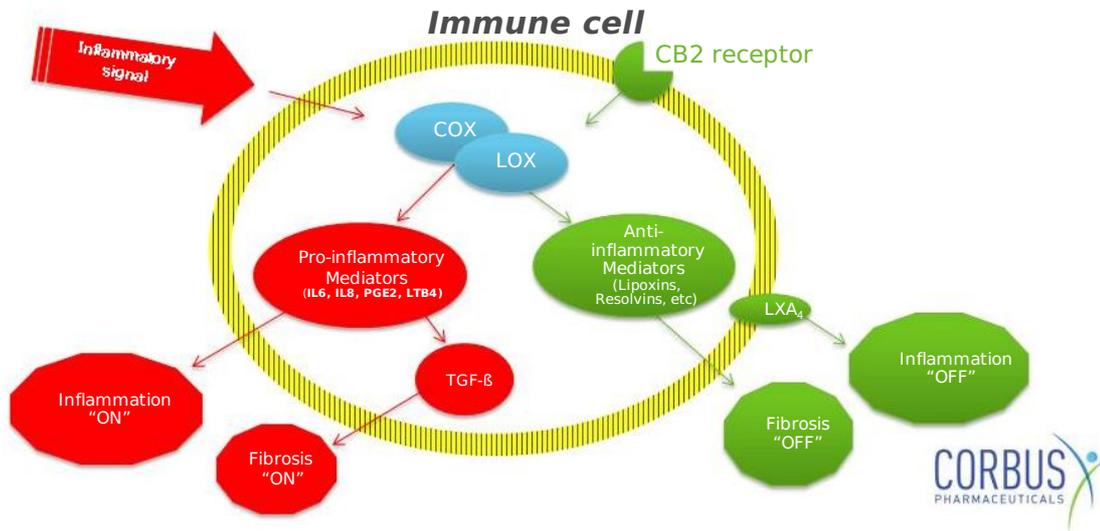


# Our Target Indications: Current & Future

Indication	Patient numbers (USA)	Estimated Market size	Current therapies for inflammation	Drawbacks to current therapies
<b>Current lead indications:</b>				
<b>Cystic Fibrosis</b>	30,000	>\$3B	Steroids, ibuprofen	Considerable side effects
<b>Diffuse Systemic Sclerosis (Scleroderma)</b>	50,000	>\$2B	Steroids, methotrexate	Side effects, poor efficacy
<b>Potential future indications:</b>				
<b>Dermatomyositis</b>	13,000	>\$1B	Steroids, mAbs	Side effects, poor efficacy
<b>Marfan Syndrome</b>	5,000	>\$1B	N/A	
<b>Lupus (SLE)</b>	500,000-1.5MM	>\$3B	Steroids, mAbs	Side effects, poor efficacy
<b>Idiopathic Pulmonary Fibrosis (IPF)</b>	70,000	>\$1B	Pirfenidone	Limited efficacy InterMune bought by Roche for \$8.5B (2014)

# CB2 Receptor: Turns inflammation "off"

- CB2 receptor is present on immune cells and activated by endogenous lipid mediators
- Activation of CB2 turns inflammation off ("inflammatory resolution")
- Resunab expected to be first CB2-binding anti-inflammatory drug to reach market
- Upstream of other approaches: potential for better safety and potency



EDITORIAL

Eicosanoids in Scleroderma: Lung Disease Hangs in the Balance

Bruce D. Levy

Lung disease has been recognized as a complication of scleroderma, and lung involvement is detectable by high-resolution

### Mechanisms of Disease: leukotrienes and lipoxins in scleroderma lung disease—insights and potential therapeutic implications

Otylia Kowal-Bielecka\*, Krzysztof Kowal, Oliver Distler and Steffen Gay

SUMMARY

Scleroderma interstitial lung disease (ILD) is a leading cause of morbidity and mortality in patients with systemic sclerosis. Although the pathogenesis of ILD is not clear, excessive fibroblast and inflammatory cell infiltration are the main histologic features of this disorder. Leukotrienes and lipoxins are two functionally different classes of lipoprotein-derived eicosanoids. Leukotrienes are potent proinflammatory mediators and directly and indirectly stimulate fibroblast chemotaxis, proliferation, and collagen synthesis. Lipoxins, on the other hand, regulate the proinflammatory actions of leukotrienes and create resolution of the inflammatory response. In addition, lipoxins inhibit growth factor-induced fibroblast proliferation and collagen synthesis. Studies indicate that inhibition of leukotriene synthesis

INTRODUCTION

Scleroderma interstitial lung disease (ILD) is a frequent complication, and the leading cause of death, in systemic sclerosis. Histologically, ILD is characterized by infiltration of inflammatory cells and excessive fibrosis of the lung parenchyma and alveoli, which leads to impaired gas exchange, restrictive ventilatory defects, and respiratory failure. Although the pathogenesis of interstitial lung disease is not fully understood, studies over the past 10 years point to early

EXTENDED REPORT

### The 12/15-lipoxygenase pathway counteracts fibroblast activation and experimental fibrosis

Gerhard Krönke,<sup>1,2</sup> Nicole Reich,<sup>1</sup> Carina Schöttysek,<sup>1,2</sup> Alfya Akhmetshina,<sup>1</sup> Stefan Uderhardt,<sup>1,2</sup> Pascal Zarr,<sup>1</sup> Kattin Palurko,<sup>1</sup> Veronika Lang,<sup>1</sup> Clara Dees,<sup>1</sup> Oliver Distler,<sup>2</sup> Georg Schett,<sup>1</sup> Jörg H W Distler<sup>1</sup>

ABSTRACT

Background: Idiopathic and inflammation-dependent fibrotic diseases such as systemic sclerosis (SSc) impose a major burden on modern societies. Understanding

ECM. However, the molecular mechanisms of fibroblast activation and potential counter-regulatory mechanisms, which limit the inflammatory reaction and the consecutive ECM accumulation,

that these leukotrienes contribute to increased irregular thickening of a thickening of KEYWORDS: asthma, CHEST



### CHEST Translating Basic Research into Clinical Practice

#### Eicosanoid Lipid Mediators in Fibrotic Lung Diseases\* Ready for Prime Time?

Steven K Huang, MD, and Marc Peters-Golden, MD

Recognition of a pivotal role for eicosanoids in both normal and pathologic fibroproliferation is long overdue. These lipid mediators have the ability to regulate all cell types and nearly all pathways relevant to fibrotic lung disorders. Abnormal fibroproliferation is characterized by an excess of proinflammatory leukotrienes and a deficiency of antiinflammatory prostaglandins. The relevance of an eicosanoid imbalance in patients to diseases involving the parenchymal, airway, and vascular compartments of the lung, and is supported by studies conducted both in humans and animal models. Given the lack of effective alternatives, and the existing and emerging options for therapeutic targeting of eicosanoids, such treatments are ready for prime time. (CHEST 2008; 133:1442-1450)

Key words: airway remodeling; leukotrienes; prostaglandins; pulmonary fibrosis

Abbreviations: cAMP = cyclic adenosine monophosphate; cPLA<sub>2</sub> = cytosolic phospholipase A<sub>2</sub>; COX = cyclooxygenase; cPLA<sub>2</sub> = cytosolic phospholipase A<sub>2</sub>; EP = E<sub>2</sub> prostaglandin synthase; IL = interleukin; IP = I<sub>2</sub> prostaglandin synthase; IPF = idiopathic pulmonary fibrosis; SLD = S<sub>2</sub>-lipoprotein; LT = leukotriene; PG = prostaglandin; TGF = transforming growth factor; T<sub>1</sub> = T helper

As a result of both research advances and therapeutic developments over the past 20 years, favored concepts regarding the pathobiology of pulmonary fibrosis have shifted from a central focus on inflammation to one of abnormal fibroproliferation

apoptotic loss of alveolar epithelial cells, recruitment, expansion, and activation of mesenchymal cells, and deposition of excess matrix proteins such as collagen, particularly by  $\alpha$ -smooth muscle actin-positive myofibroblasts. These processes in turn are

### Defective lipoxin-mediated anti-inflammatory activity in the cystic fibrosis airway

Christopher L Karp,<sup>1</sup> Leah M Flick,<sup>1,2</sup> Kiwon W Park,<sup>1,2</sup> Samir Sofri,<sup>1,2</sup> Todd M Greer,<sup>1</sup> Raquid Keldjian,<sup>1</sup> Rong Yang,<sup>1</sup> Jasim Uddin,<sup>1</sup> William B Guggino,<sup>1</sup> Sowan F Atabani,<sup>1</sup> Yasmine Belkaid,<sup>1</sup> Yan Xu,<sup>1</sup> Jeffrey A Whitsett,<sup>1</sup> Frank J Accurso,<sup>6</sup> Marsha Wills-Karp,<sup>3</sup> & Nicos A Petasis<sup>3</sup>

ORIGINAL ARTICLE  
CYSTIC FIBROSIS

### Reduced 15-lipoxygenase 2 and lipoxin A<sub>4</sub>/leukotriene B<sub>4</sub> ratio in children with cystic fibrosis

Fiona C. Ringholz,<sup>1</sup> Paul J. Buchanan,<sup>1</sup> Donna T. Clarke,<sup>1</sup> Roisin G. Millar,<sup>1</sup> Michael McDermott,<sup>2</sup> Barry Linnane,<sup>1,3</sup> Brian J. Harvey,<sup>4</sup> Paul McNally,<sup>1,2</sup> and Valerie Urbach<sup>1,4</sup>

Affiliations: <sup>1</sup>National Children's Research Centre, Crumlin, Dublin, Ireland; <sup>2</sup>Our Lady's Children's Hospital, Crumlin, Dublin, Ireland; <sup>3</sup>Midwestern Regional Hospital, Limerick, Ireland; <sup>4</sup>Centre for Interventions in Infection, Inflammation and Immunity (i3), Graduate Entry Medical School, University of Limerick, Limerick, Ireland; <sup>5</sup>Molecular Medicine Laboratories, Royal College of Surgeons in Ireland, Beaumont Hospital, Dublin, Ireland; <sup>6</sup>Insitut National de la Santé et de la Recherche Médicale, UMR5, Faculté de Médecine Paris Descartes, Paris, France.

Correspondence: Valerie Urbach, National Children's Research Centre, Crumlin, Dublin 12, Ireland. E-mail: valerie.urbach@nrc.ie

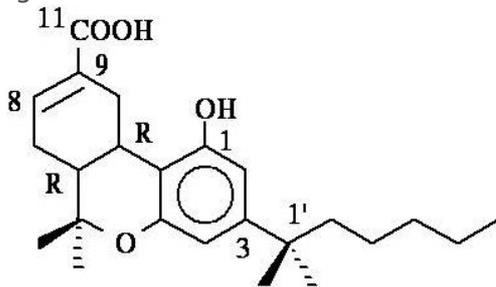
ABSTRACT: Airway disease in cystic fibrosis (CF) is characterized by impaired mucociliary clearance, persistent bacterial infection and neutrophilic inflammation. Lipoxin A<sub>4</sub> (LXA<sub>4</sub>) initiates the active resolution of inflammation and promotes airway surface hydration in CF models. 15-Lipoxygenase (LO)

© 2008 Nature Publishing Group

nature immunology

# Resunab

- Resunab: synthetic oral CB2 agonist small-molecule
- Designed to trigger the resolution of chronic inflammation
- Full manufacturing, drug supply, non-clinical safety & pharmacology package for Phase 2 programs
- Excellent clinical safety profile to date: two prior Phase 1 clinical trials (n=121)
  - Lacks CNS side effects of other CB2-binding class members
  - Lacks GI side effects of NSAID's (e.g. Aspirin™, ibuprofen, Celebrex™)
  - Lacks metabolic side effects of corticosteroids
- Preparing to launch two Phase 2 clinical studies in 2015



## Resunab: Only CB2-Agonist Targeting Inflammation

Company	Indication	Brain penetration	Status	Affects CNS
Corbus Pharma	Inflammation	Minimal	Entering Phase 2	No
AbbVie	Pain	Full	Phase 1	Yes
Glenmark	Pain	Full	Phase 1	Yes
Eli Lilly	Knee pain	Full	Phase 2	Yes
AstraZeneca	Post operative pain	Full	Phase 2	Yes

Resunab is the only CB2 drug that can be used to treat inflammation because it does not target the brain



# Cystic Fibrosis

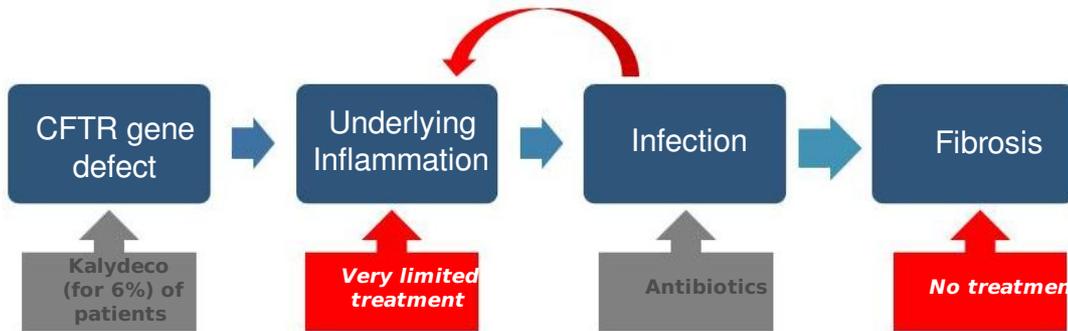
Targeting inflammation at the core of the disease



**CORBUS**  
PHARMACEUTICALS

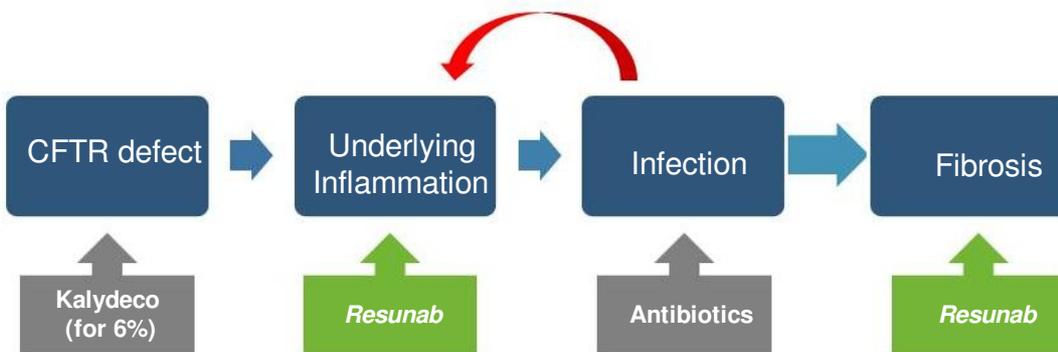
# Overview: Cystic Fibrosis

- Inflammatory orphan disease (30,000 patients in USA, 75,000 WW)
- Average life expectancy of CF patients is approximately 40 years
- Inflammation at core of disease's morbidity and mortality (pulmonary fibrosis)
- Very high doses of steroids/ibuprofen effective but rarely used due to toxicity
- Need for safe, chronic anti-inflammatory drug is unmet and universally recognized
- Pharmaco-economics support premium pricing (e.g. Kalydeco by Vertex priced at \$320,000/yr)

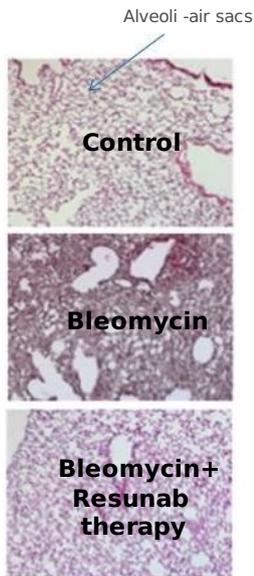


# Resunab targets key CF inflammatory players

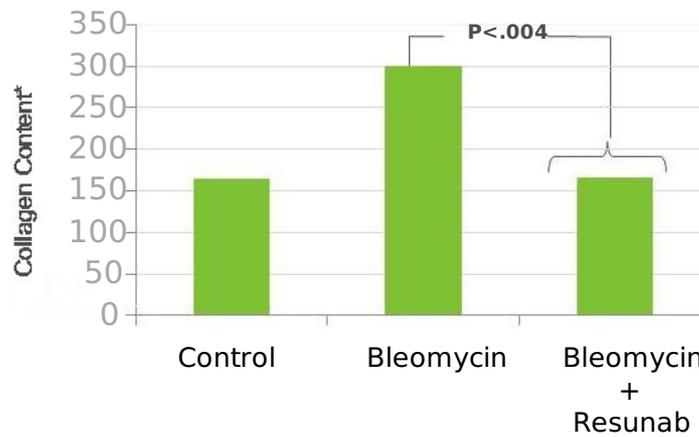
↓ TGF $\beta$	↑ Lipoxin-A4
<ul style="list-style-type: none"> <li>• Genetically linked to disease</li> <li>• Associated with worsening symptoms</li> </ul>	<ul style="list-style-type: none"> <li>• Absent in CF lungs</li> <li>• Replacement therapy effective in animal models</li> </ul>



# Resunab Reduces Pulmonary Fibrosis In Animal Models



Fibrosis-inducing agent (Bleomycin) administered to lungs day 1 followed by daily oral *Resunab* for 21 days



Gonzales et al., *Annals of Rheumatic Diseases*, 2012. 71:1545-51  
\* Measured by hydroxyproline

# Resunat Planned Cystic Fibrosis Phase 2 Trial

- Double blind placebo control study in the USA under IND from FDA
- **Primary endpoints** Safety/tolerability/PK
- **Secondary endpoints** Inflammatory biomarkers MOA + change in clinical outcome measures
- **Patient number** 70 adults with CF in 15-20 sites
- **Treatment duration** 6 months + 1 month follow-up
- **Dose response** 1mg/day, 5mg/day, 20mg/day and 20mg/2X/day

	Q1 2015	Q2 2015	Q3 2015	Q4 2015	Q1 2016	Q2 2016	Q3 2016	Q4 2016
Protocol filed with FDA	X							
Study launches		X						
First patient dosed		X						
Study duration		X	X	X	X	X	X	
Last patient dosed							X	
Study data released								X



# Diffuse Systemic Sclerosis (“Scleroderma”)

Relief for a disease with no effective long-term therapy

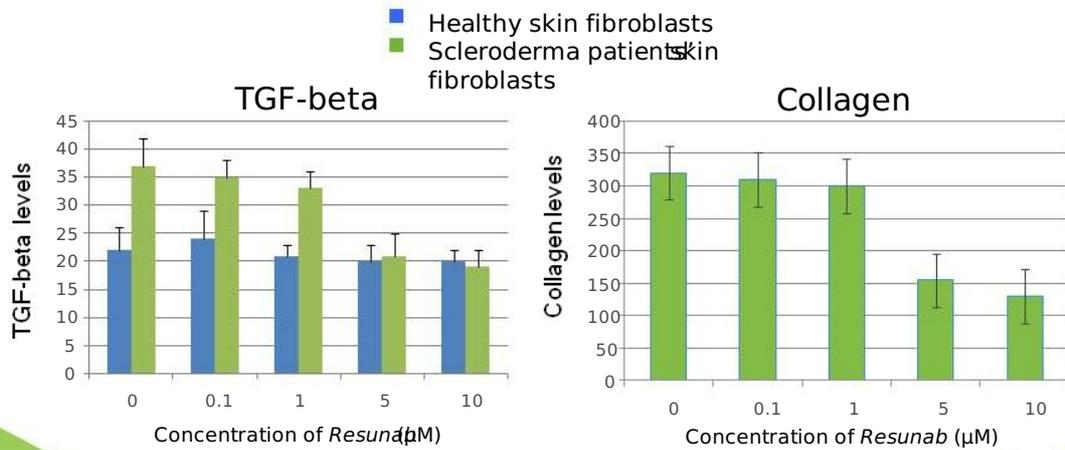


## Overview: Diffuse Systemic Sclerosis (Scleroderma)

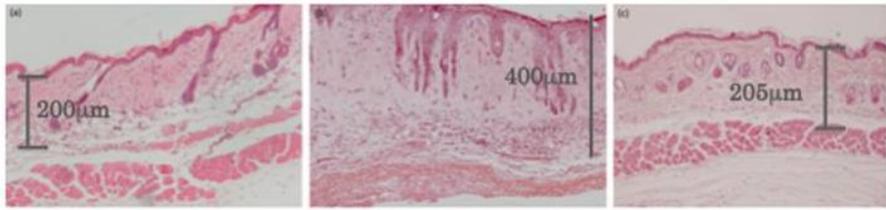
- Chronic inflammatory disease causing fibrosis of skin, joints and internal organs
- Orphan disease (50,000 patients in USA)
- 80% of patients are women in their 30's and 40's
- Common cause of death: lung fibrosis (50% mortality in 10 years)
- Early stage of disease responds to steroids/methotrexate but with serious side effects
- No effective and safe long-term therapy available
- Pipelines often target Idiopathic Pulmonary Fibrosis (IPF) in conjunction to SSC

# Resunab Inhibits Key Factors in SSC

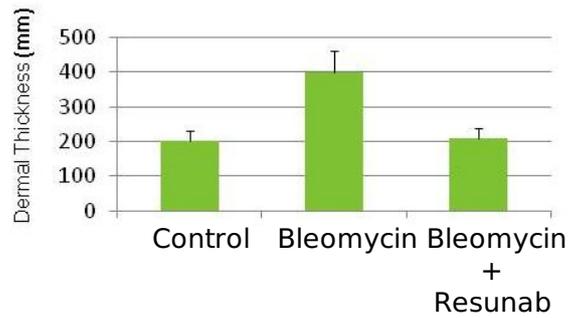
- TGF-beta plays key role in SSC progression (same in CF and IPF)
- Elevated TGF-beta levels associated with disease progression
- Strong Resunab efficacy data in animal models
- Resunab reduces TGF-beta and collagen in skin fibroblasts from SSC patients



# Resunab Inhibits Skin Thickening In Mouse SSC Model



Healthy skin

Thick skin induced by  
BleomycinNear normal skin after oral  
*Resunab* taken once daily  
for four weeks

Gonzales et al., *Annals of Rheumatic Diseases*, April 4, 2012

# Resunab Planned SSC Phase 2 Clinical Trial

- Double blind placebo control study in USA under IND from FDA
- **Primary endpoints** Safety/tolerability PK+ Change in clinical outcomes
- **Secondary endpoints** Inflammatory biomarkers + quality of life (QOL)
- **Patient number** 25 adults with SSC with 8-10 sites
- **Treatment duration** 3 months + 1 month follow-up
- **Dose response** 5mg/day 20mg/day and 20mg/2X/day

	Q1 2015	Q2 2015	Q3 2015	Q4 2015	Q1 2016	Q2 2016	Q3 2016	Q4 2016
Protocol filed with FDA	X							
Study launches	X							
First patient dosed		X						
Study duration		X	X	X	X	X	X	
Last patient dosed							X	
Study data released								X

# Management Team

## **Yuval Cohen, Ph.D.- Chief Executive Officer**

- Co-founder and former President of Celsus Therapeutics (CLTX)
- Expertise in developing anti-inflammatory drugs including for CF

## **Mark Tepper, Ph.D.- President & Chief Scientific Officer**

- Former VP USA Research & Operations, EMD Serono; Sr. Investigator, Bristol-Myers Squibb
- Key member of project teams which developed the following marketed drugs: Taxol® (Ovarian Cancer, 2000 peak sales of \$1.6B), Orenia® (RA, 2013 sales of \$1.4B), Rebif® (MS, 2013 sales of \$2.59B), Gonal-F® (Fertility, 2013 sales of \$815MM)

## **Sean Moran, C.P.A. M.B.A.- Chief Financial Officer**

- Former CFO: InVivo (NVIV), Celsion (CLSN), Transport Pharma, Echo Therapeutics (ECTE) & Anika Therapeutics (ANIK)

## **Barbara White, M.D.- Chief Medical Officer**

- Former VP Clinical & Medical Director at Amgen, UCB and Medimmune. Expert rheumatologist in scleroderma with decades of experience in clinical trial development.



# Board of Directors

## **Yuval Cohen, Ph.D.- Chief Executive Officer**

### **Amb. Alan Holmer - Chairman of the Board**

- Former CEO of PhRMA (1996-2005)
- Over two decades of public service in Washington, D.C. including Special Envoy to China (2007-2009)
- Former board member Inspire Pharma (sold to Merck for \$430m in 2011)
- Chairman of the Board of the Metropolitan Washington, D.C. Chapter of the Cystic Fibrosis Foundation

### **David Hochmann**

- Managing Partner of Orchestra Medical Ventures
- Over 17 years of venture capital and investment banking experience
- Former Managing Director of Spencer Trask Ventures, Inc. securing over \$420 million in equity capital

### **Renu Gupta, MD**

- 25 years of development, regulatory and senior management experience in the biopharm industry
- Former CMO of Inmeda, a specialty CF company and current advisor to the CEO
- Former Vice President and Head of US Clinical Research and Development at Novartis (2003-2006)

### **Avery W. (Chip ) Caitlin**

- CFO Celldex Therapeutics (CLDX) since 2000
- Raised over \$415MM financing
- 20 years experience in industry: Repligen (CFO) and Endogen (CFO)



# World Class Scientific Advisors

**Sumner Burstein, Ph.D. - UMass Medical School**

Professor of Biochemistry and Pharmacology; inventor of Resunab

**Michael Knowles, M.D., Ph.D. - UNC Chapel Hill**

Professor of Pulmonary and Critical Care Medicine

**James Chmiel, M.D. - Case Western Reserve Medical School**

Professor Medicine, National PI on largest ever anti-inflammatory CF study

**Robert Simms, M.D. - Boston University School of Medicine**

Chairman of International Clinical Scleroderma Consortium

**Daniel Furst, M.D. - UCLA School of Medicine**

Director of UCLA Scleroderma Program

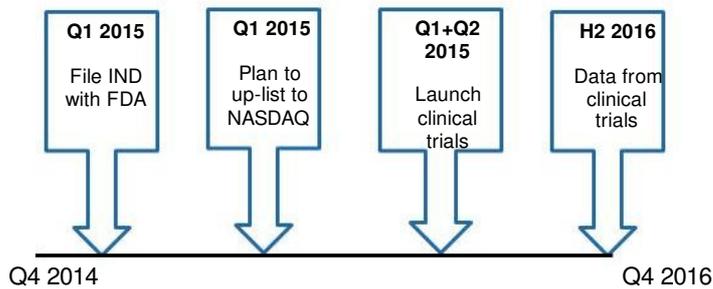
**Robert Zurier, M.D. - UMass Medical School**

Ex-Chair of Rheumatology

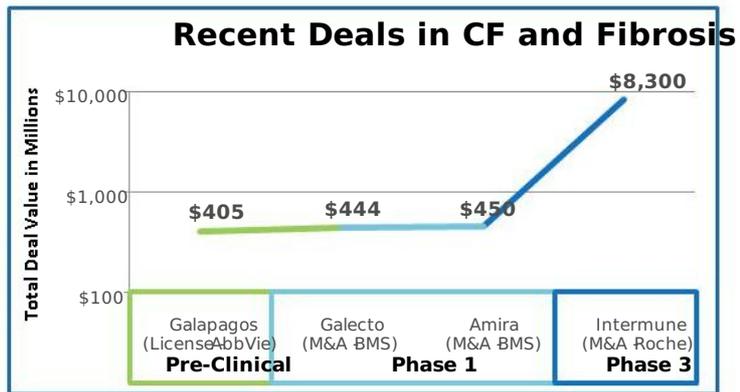


# Financial Profile

<b>Stock Ticker:</b>	CRBP:OTCQB
<b>\$77,400,000</b>	Market capitalization as of November 2014
<b>\$10,300,000</b>	Raise from successful private placement (Q2 2014) from institutional and retail base
<b>25,800,000</b>	Common shares outstanding
<b>41,500,000</b>	Fully diluted shares outstanding (including warrants and stock options)
<b>\$11,400,000</b>	Available from exercise of callable warrants
<b>NASDAQ</b>	Up-listing to NASDAQ planned by Q-1 2015



# Corbus Poised for Significant Upside

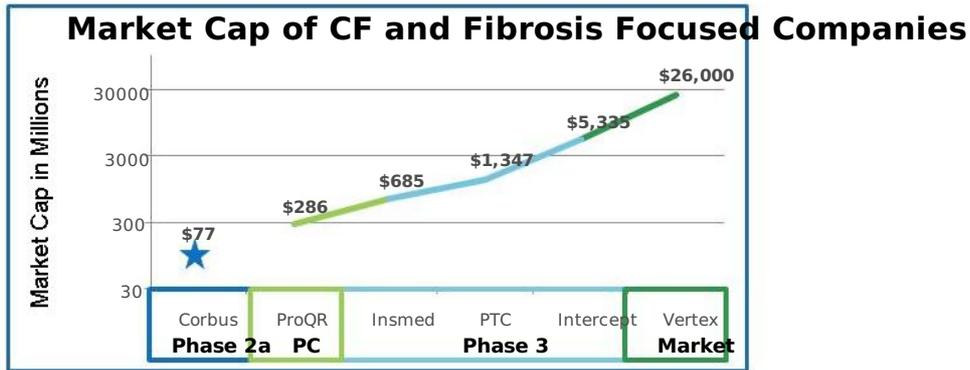


Recent Deals								
Date	Company	Partner	Type	Drug	Indication	Stage	Up-Front	Deal Total
11/14	Galecto	BMS	Option to acquire	TD139	Idiopathic pulmonary fibrosis	Phase 1	NA	\$444M*
8/14	InterMune	Roche	Acquisition	Esbriet	Idiopathic pulmonary fibrosis	Approved	NA	\$8.3B*
9/2013	Galapagos	AbbVie	License	GLPG1837	Mutations in CF patients, including F508del and G551D	Pre-clinical	\$45M*	\$405M*
7/2011	Amira	BMS	Acquisition	AM152	Idiopathic pulmonary fibrosis and systemic sclerosis	Phase 1	\$325M*	\$475M*

\* Figures from company press releases



# Potential Value Indicators



Recent IPO					
Date	Company	Lead Compound	Indication	Stage	Market Cap
9/2014	ProQR	QR-010	Cystic Fibrosis RNA repair	Pre-clinical	\$284.11M

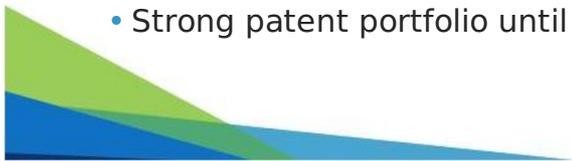
Approved Products				
Company	Drug	Indication	Cost per Year	2018 Sales Est.
Vertex	Kalydeco	Cystic Fibrosis mutations of CFTR gene: G551D, G1244E, G1349D, G178R, G551S, S1251N, S1255P, S549N, or S549R	\$294,000	\$1.2B**

\* Figures from company press releases  
\*\* Leerink analyst report



# Conclusions

- Lead Product *Resunab* a novel, safe and promisingly potent clinical stage anti-inflammatory/anti-fibrotic drug which acts to resolve inflammation
- Targets multiple rare inflammatory indications
- Proven safe in two Phase 1 trials
- Promising potency in multiple pre-clinical models
- Launch two Phase 2 trials in 2015 (Cystic Fibrosis and Scleroderma)
- Completion of studies in 2016
- Strong patent portfolio until 2033





100 River Ridge Drive  
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