CORBUS PHARMACEUTICALS

DEVELOPING BREAKTHROUGH THERAPIES FOR RARE INFLAMMATORY DISEASES

NASDAQ:CRBP I CORBUSPHARMA.COM

FORWARD-LOOKING STATEMENTS

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" 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 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

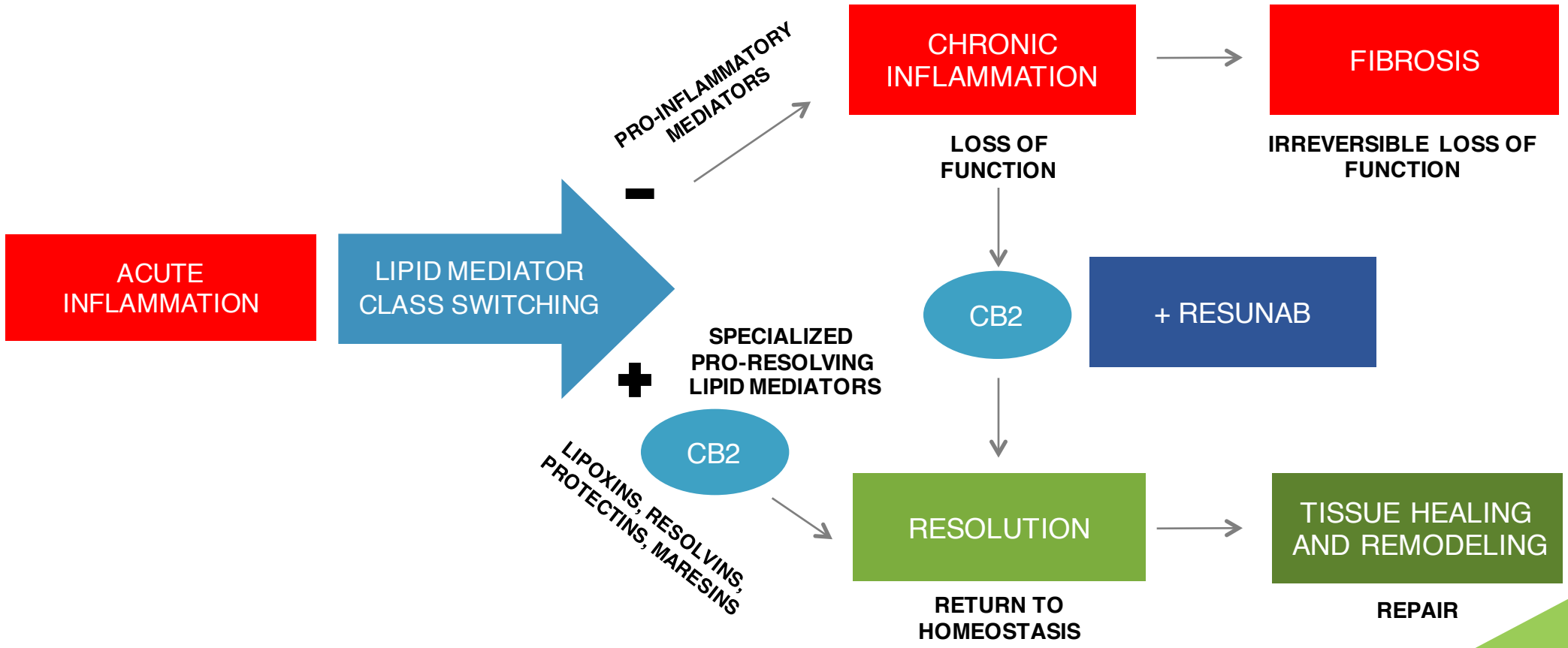
- Focus: rare, life-threatening, inflammatory/fibrotic diseases
- Resunab™: novel oral small molecule entering Phase 2 in
  - **Cystic Fibrosis (CF)**
  - **Diffuse Cutaneous Systemic Sclerosis ("Scleroderma")**
    - Fast Track Status granted by U.S. FDA
  - **Dermatomyositis (DM)**
- Data read out from Phase 2 studies in Q4 2016
- $5MM Development Award from Cystic Fibrosis Foundation
- IP portfolio → 2033
- NASDAQ: CRBP

THERAPEUTIC PIPELINE

INDICATION	PRE-CLINICAL	PHASE 1	PHASE 2	PIVOTAL STUDY
CYSTIC FIBROSIS (CF)	RESUNAB™			
SYSTEMIC SCLEROSIS (SCLERODERMA)	RESUNAB™			
DERMATOMYOSITIS (DM)	RESUNAB™			

POTENTIAL FUTURE INDICATIONS				
INDICATION	**Patient numbers**	**Estimated market**	**Current therapies**	**Drawbacks**
LUPUS (SLE)	500 000 – 1.5MM	> $3B	STEROIDS, mAbs	Side effects, poor efficacy
IDIOPATHIC PULMONARY FIBROSIS (IPF)	70 000	> 1B	PIRFENIDONE	Limited efficacy: Intermune bought by Roche for $8.5b(2014)
OTHER CHRONIC INFLAMMATORY DISEASES	10 000 – 100 000	> $10B	STEROIDS, mAbs	Side effects, poor efficacy

INNATE IMMUNE RESPONSES AND INFLAMMATION REQUIRE ACTIVE RESOLUTION TO RESTORE HOMEOSTASIS



FREIRE & VAN DYKE. Natural resolution of inflammation. Periodontology 2000, 2013;63:149–164

PUBLICATIONS

ARTHRITIS & RHEUMATISM
Vol. 52, No. 12, December 2005, pp 3683–3687
DOI 10.1002/art.21434
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EDITORIAL

Eicosanoids in Scleroderma: Lung Disease Hangs in the Balance

Bruce D. Levy

EXTENDED REPORT

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

Gerhard Krönke,[1,2] Nicole Reich,[1] Carina Scholtysek,[1,2] Alfiya Akhmetshina,[1] Stefan Uderhardt,[1,2] Pawel Zerr,[1] Katrin Palumbo,[1] Veronika Lang,[1] Clara Dees,[1] Oliver Distler,[3] Georg Schett,[1] Jörg H W Distler[1]

ABSTRACT

Background Idiopathic and inflammation-dependent fibrotic diseases such systemic sclerosis (SSc) impose

ECM.[3] However, the molecular mechanisms of fibroblast activation and potential counter-regulatory mechanisms, which limit the inflammatory

www.nature.com/clinicalpractice/rheum

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 (SLD) is a leading cause of morbidity and mortality in patients with systemic sclerosis. Although the pathogenesis of SLD is not clear, excessive fibrosis and inflammatory cell infiltration are the main histologic features of this disorder. Leukotrienes and lipoxins are two functionally different classes of lipoxygenase-derived eicosanoids. Leukotrienes are potent proinflammatory mediators and directly and indirectly stimulate fibroblast chemotaxis, proliferation, and collagen synthesis. Lipoxins counter-regulate the proinflammatory actions of leukotrienes and activate resolution of the inflammatory response. In addition, lipoxins inhibit growth-factor-induced fibroblast proliferation and collagen synthesis. Studies using bronchoalveolar lavage have revealed that there is an overproduction of proinflammatory and profibrotic leukotrienes in the lungs of patients with SLD, and that leukotriene levels correlate with inflammatory indices within the lungs. Moreover, the increased levels of leukotrienes in these patients are not balanced by an upregulation of anti-inflammatory and antifibrotic lipoxins. Unopposed actions of leukotrienes might, therefore, induce chronic inflammation and fibrosis in the lungs of SLD patients. Accordingly, pharmacologic correction of a leukotriene–lipoxin imbalance using leukotriene inhibitors or lipoxin analogs might be a new approach to the treatment of SLD.

KEYWORDS leukotrienes, lipoxins, lipoxygenase, scleroderma interstitial lung disease, systemic sclerosis

REVIEW CRITERIA

To identify relevant publications for inclusion in this article we searched the PubMed database from the beginning of January 2000 through to the end of April 2006 using the following search keywords: "systemic sclerosis", "interstitial lung disease", "lung fibrosis" and "fibrosis", together with "lipoxygenase", "leukotriene" and "lipoxin". Both abstracts and papers were included in the search. In addition, relevant publications published before the beginning of January 2000 were identified by searching the reference lists of the articles found by the PubMed search. The search was limited to English-language papers.

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INTRODUCTION

Scleroderma interstitial lung disease (SLD) is a frequent complication, and the leading cause of death, in systemic sclerosis. Histologically, SLD 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.[1] Although the pathogenesis of interstitial lung disease is not fully understood, studies over the past 10 years point to early injury and inflammation that leads to dysregulated tissue repair and fibrosis. The mechanisms responsible for the progression of lung fibrosis are complex, and include fibroblast activation, alterations in immune regulation and response, chronic inflammation, and microvascular and epithelial injury.

A number of proinflammatory and profibrotic mediators have been implicated in the pathogenesis of interstitial lung disease, including cytokines, chemokines and growth factors, such as interleukin (IL) 4, IL-13, CC-chemokine ligand (CCL) 2 (also known as monocyte chemoattractant protein 1), transforming growth factor β (TGF-β), platelet-derived growth factor (PDGF), and many others.[2] In addition to the well-established role of stimulatory pathways, the importance of natural counter-regulatory mechanisms, which are responsible for the preservation of tissue function through the limitation of inflammatory and fibrotic responses, is being recognized in the development of interstitial lung disease. There is growing evidence that

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 profibrotic leukotrienes and a deficiency of antifibrotic prostaglandins. The relevance of an eicosanoid imbalance is pertinent 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; cysLT = cysteinyl leukotriene; COX = cyclooxygenase; cysLT1 = cysteinyl leukotriene receptor 1; EP = E prostanoid receptor; IL = interleukin; IP = I prostanoid receptor; IPF = idiopathic pulmonary fibrosis; 5-LO = 5-lipoxygenase; LT = leukotriene; PG = prostaglandin; TCF = transforming growth factor; Th = T helper

As a result of both research advances and therapeutic disappointments 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 fibroproliferative responses to lung injury that result in tissue remodeling.[1] Such responses are thought to involve the apoptotic loss of alveolar epithelial cells; recruitment, expansion, and activation of mesenchymal cells; and deposition of excess matrix proteins such as collagen, particularly by α-smooth muscle actin-positive myofibroblasts. These processes in turn are driven by a profibrotic milieu that is characterized by oxidant stress, growth factors such as transforming growth factor (TGF)-β, T-helper (Th) type 2 immune response polarization, and abnormalities in the coagulation cascade.[1] Although the prototypic fibroproliferative lung diseases are diffuse disorders of the pulmonary parenchyma, such as idiopathic pulmonary fibrosis (IPF), many aspects of their pathobiology are shared by remodeling diseases involving other compartments of the lung. Examples include airway remodeling in patients with asthma and bronchiolitis obliterans, and vascular remodeling in patients with

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

Christopher L Karp[1], Leah M Flick[1,8], Kiwon W Park[2,8], Samir Softic[1,8], Todd M Greer[1], Raquel Keledjian[3], Dong Yang[3], Jasim Uddin[3], William B Guggino[4], Sowsan F Atabani[1], Yasmine Belkaid[1], Yan Xu[5],
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ORIGINAL ARTICLE
CYSTIC FIBROSIS

Reduced 15-lipoxygenase 2 and lipoxin A_4/leukotriene B_4 ratio in children with cystic fibrosis

Fiona C. Ringholz[1], Paul J. Buchanan[1], Donna T. Clarke[1], Roisin G. Millar[1], Michael McDermott[2], Barry Linnane[1,3,4], Brian J. Harvey[5], Paul McNally[1,2] and Valerie Urbach[1,6]

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ABSTRACT Airway disease in cystic fibrosis (CF) is characterised by impaired mucociliary clearance, persistent bacterial infection and neutrophilic inflammation. Lipoxin A_4 (LXA_4) initiates the active resolution of inflammation and promotes airway surface hydration in CF models. 15-Lipoxygenase (LO) plays a central role in the "class switch" of eicosanoid mediator biosynthesis from leukotrienes to lipoxins, initiating the active resolution of inflammation. We hypothesised that defective eicosanoid mediator class switching contributes to the failure to resolve inflammation in CF lung disease.

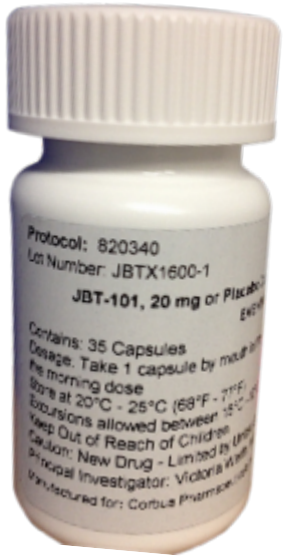
Using bronchoalveolar lavage (BAL) samples from 46 children with CF and 19 paediatric controls we demonstrate that the ratio of LXA_4 to leukotriene B_4 (LTB_4) is depressed in CF BAL ($p<0.01$), even in the absence of infection ($p<0.001$).

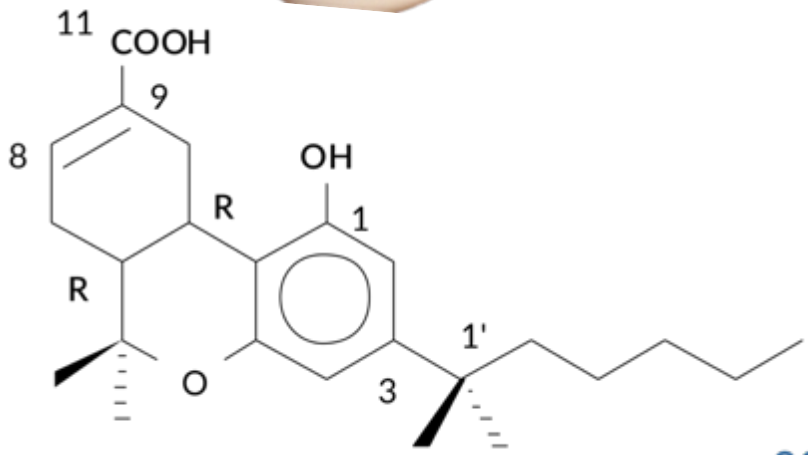
Furthermore, 15-LO2 transcripts were significantly less abundant in CF BAL samples ($p<0.05$). In control BAL, there were positive relationships between 15-LO2 transcript abundance and LXA_4/LTB_4 ratio ($p=0.01$, $r=0.66$) and with percentage macrophage composition of the BAL fluid ($p<0.001$, $r=0.82$), which were absent in CF.

Impoverished 15-LO2 expression and depression of the LXA_4/LTB_4 ratio are observed in paediatric CF BAL. These observations provide mechanistic insights into the failure to resolve inflammation in the CF lung.

RESUNAB™ (JBT-101, AJULEMIC ACID)

- Oral small molecule (MW 400 Da) with once or twice a day dosing
- Preferentially binds and activates cells through CB2 > CB1 (Ki ratio12:1)
- Reduced penetration of blood-brain-barrier (30%)
- Safely tested in 126 subjects for up to 7 days
- Adverse events dose-dependent at ≥ 150 mg/day consistent with class
- Expected therapeutic doses of 5 mg/day (range 0.5 – 40 mg/day)
- 13-wk tox studies in rats and dogs support 3 month clinical testing
- CMC: 5 kg batches GMP material with 2 year stability
- FDA: Open IND in 3 clinical trials, Orphan and Fast Track status in SSc





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CYSTIC FIBROSIS: TARGETTING INFLAMMATION

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CYSTIC FIBROSIS:

CF is a life-threatening, genetic disease that primarily affects the lungs and digestive system. CF is characterized by chronic lung inflammation that leads to lung damage and fibrosis.

30,000
PATIENTS IN THE USA

75,000
PATIENTS WORLDWIDE

40 YEARS
AVERAGE LIFE EXPECTANCY OF CF PATIENTS

KEY TAKE-AWAYS

- Need for safe, chronic inflammation-targeting drug is unmet and recognized
- Inflammation and fibrosis play key role in CF morbidity and mortality
- Pharmacoeconomics are proven and favorable

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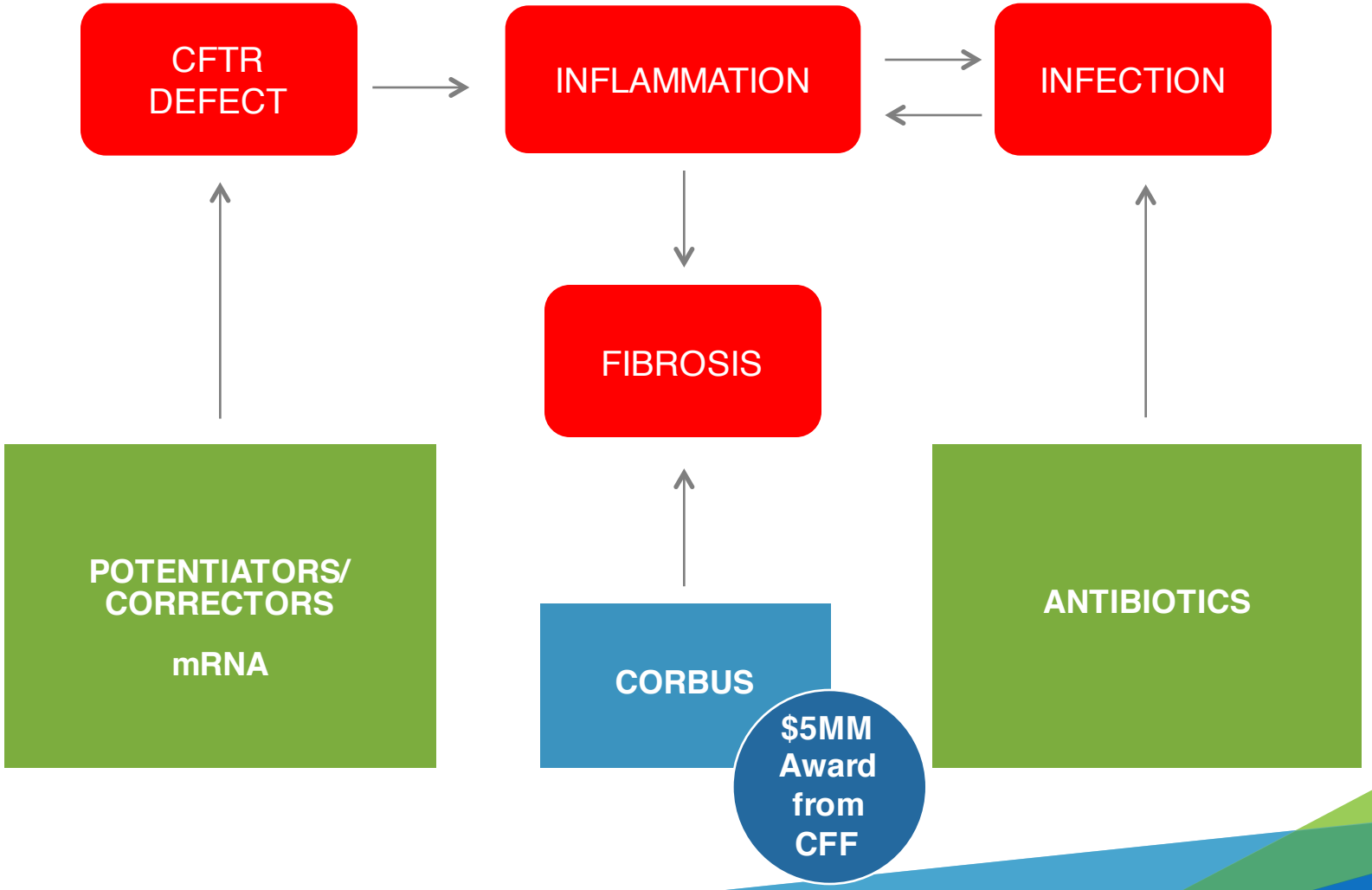
CORBUS IS UNIQUELY POSITIONED WITH RESUNAB TO RESOLVE INFLAMMATION IN CF



COMPETITIVE LANDSCAPE IN CF INFLAMMATION

COMPANY	TARGET (DRUG)	STATUS
Celtaxsys	LTB4 inhibitor (CTX-4430)	Entering Phase 2
Boehringer Ingelheim	LTB4 receptor antagonist (BIIL 284 BS)	Failed in Phase 2 due to increased pulmonary exacerbations
Merck	CystLT inhibitor (Singulair)	Failed in Phase 2 for CF
AZ	LOX inhibitor upstream of LTB4 (Zyflo)	Never officially tested in CF but poor effect in asthma
J&J	Similar to Celltaxysis (JNJ-40929837)	Never tested in CF but failed in asthma Phase 2a

Resunab is unique in its focus on **resolving** inflammation and halting fibrosis, combined with promising safety data

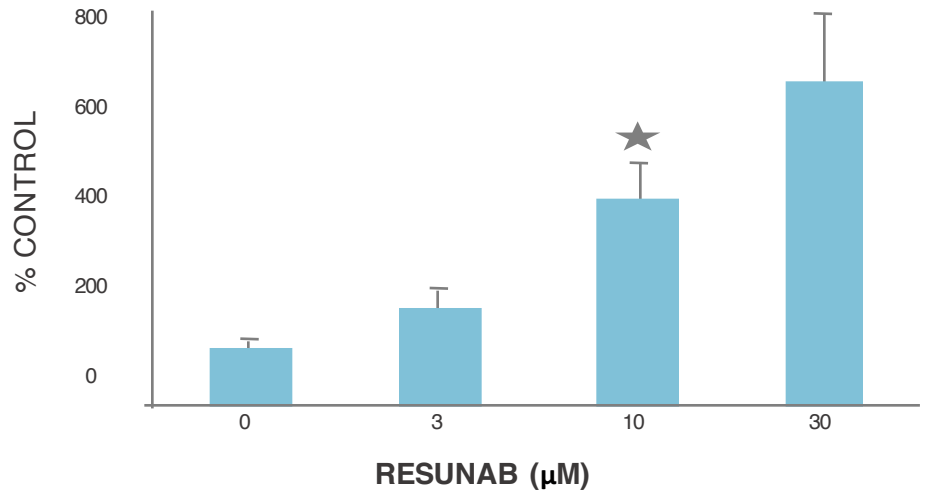
RESUNAB FAVORS PRODUCTION OF SPMs & INHIBITS PRO-INFLAMMATORY MEDIATORS IMPORTANT IN CF

LIPOXIN A4

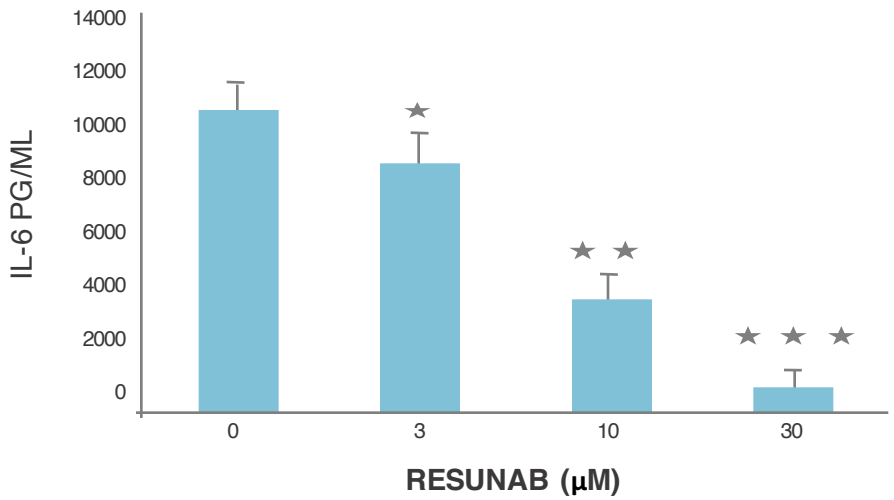
- Reduced in CF lungs
- Replacement therapy effective in animal models

IL-6, IL-8, IL-1, Type I IFNs, TNFα

- Associated with worsening symptoms



LIPOXIN A4 Zurier et al., FASEB J 2009; 23(5)



IL-6 Monocyte-derived macrophages ±Resunab, stimulated with LPS .
* p = 0.03, ** p = 0.01, *** p = 0.005 versus no Resunab1

Parker et al. Rheumatol Int 2008:28;631-635

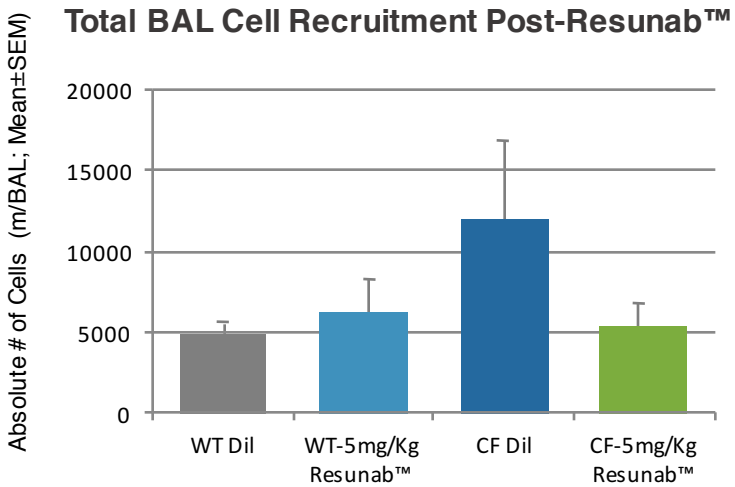
RESUNAB RESOLVES LUNG INFLAMMATION IN PSEUDOMONAS AERUGINOSA INFECTED CF MOUSE MODEL

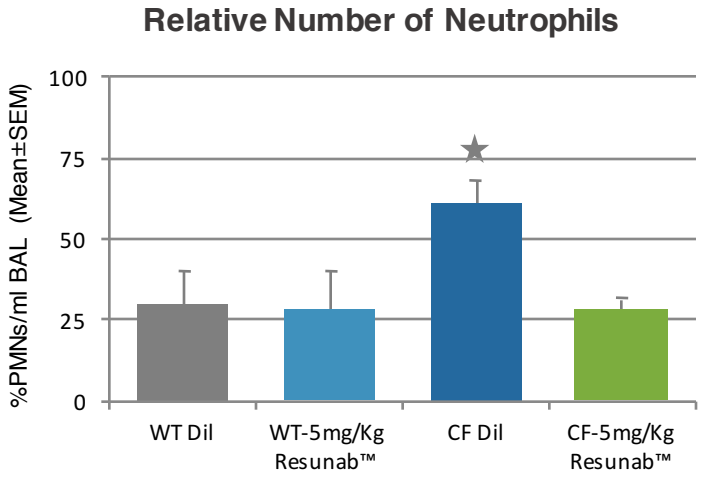
DESIGN

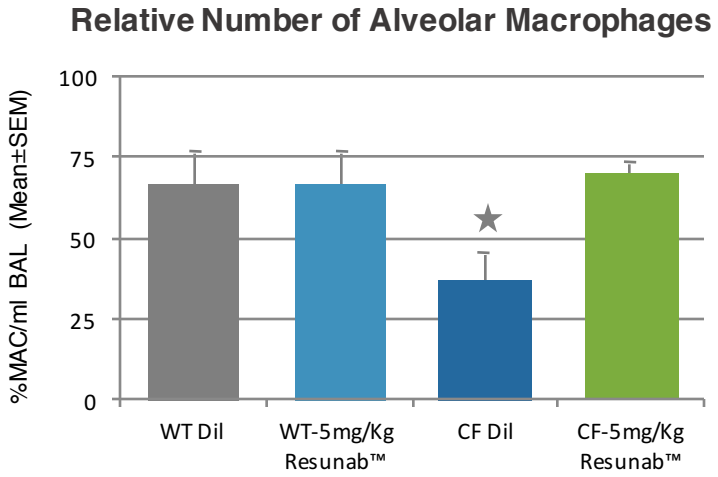
- Wild type or CFTR gut corrected mice
- All infected with *Pseudomonas aeruginosa* (10^5 viable-CFUs on agarose beads)
- Oral Resunab at 1 or 5 mg/kg bid starting 24 hours post infection
- Mice sacrificed on Day 10

RESULTS

- Total BAL cells were increased at Day 10 in CF mice
- Resunab normalized the increase in BAL cells
- Resunab induced a shift from neutrophil-predominant to macrophage-predominant cells in the lungs of CF mice, consistent with resolution of inflammation







Bonfield, Tracey; Tepper, Mark 2015

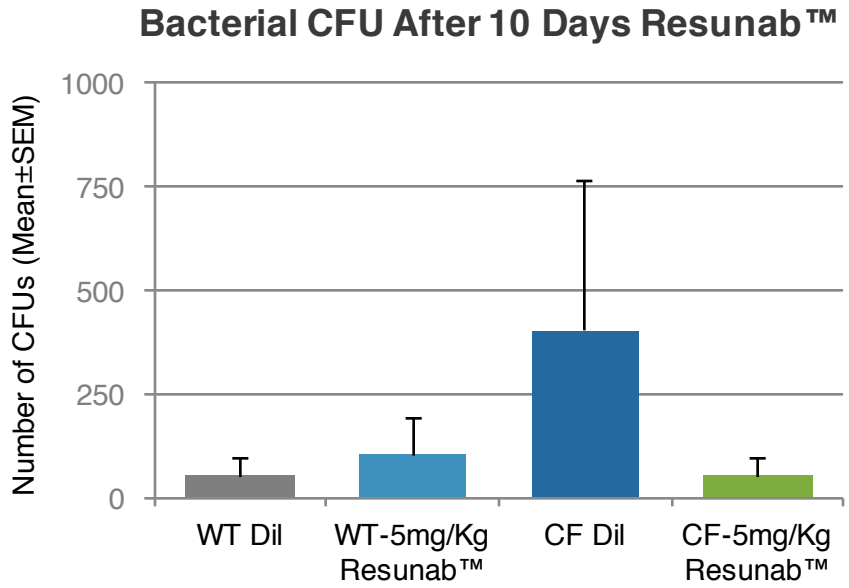
RESUNAB ENHANCES RESOLUTION OF LUNG INFECTION IN CF MICE INFECTED WITH PSEUDOMONAS

DESIGN

- Wild type or CFTR gut corrected mice
- All infected with *Pseudomonas aeruginosa* (10^5 viable-CFUs on agarose beads)
- Oral Resunab at 1 or 5 mg/kg bid starting 24 hours post infection
- Mice terminated on Day 10

RESULTS

- Total CFUs were increased at Day 10 in CF mice
- Resunab normalized the increase in CFUs in the lungs of CF mice at Day 10, consistent with resolution of inflammation



Bonfield, Tracey; Tepper, Mark 2015

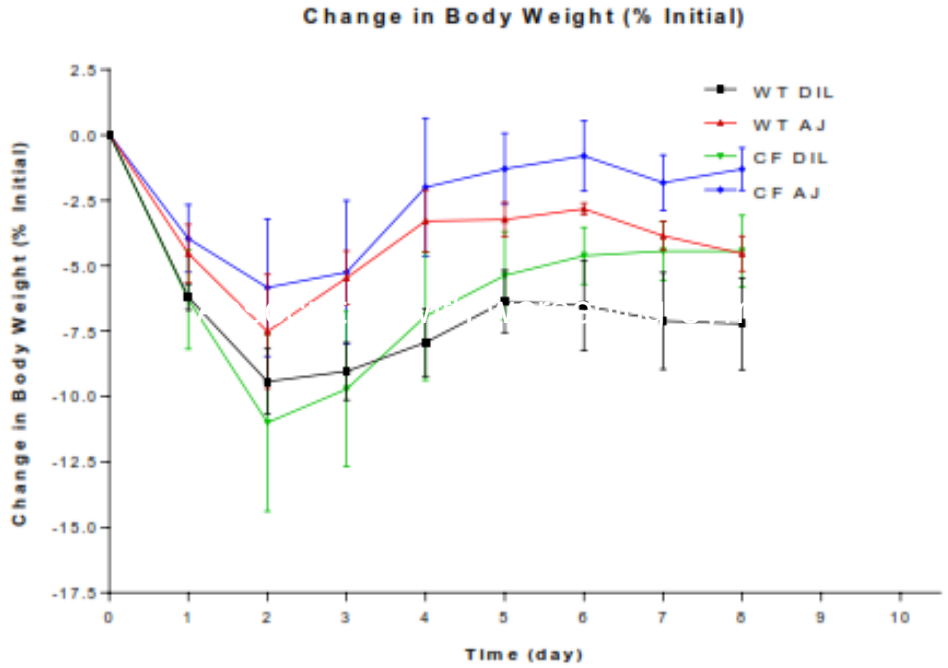
RESUNAB REDUCES WEIGHT LOSS AND IMPROVES SURVIVAL IN CF MICE INFECTED WITH PSEUDOMONAS

DESIGN

- Wild type or CFTR gut corrected mice
- All infected with *Pseudomonas aeruginosa* (10^5 viable-CFUs on agarose beads)
- Oral Resunab at 1 or 5 mg/kg bid starting 24 hours post infection
- Mice terminated on Day 10

RESULTS

- Weight decreases during Pseudomonas lung infection in mice
- Resunab reduces weight loss in both wild type and CF mice
- Resunab improves survival rate in CF mice



GROUP	SURVIVAL RATE DAY 10
WT	5/5 (100%)
WT + Resunab	5/5 (100%)
CF	3/5 (60%)
CF + Resunab	5/5 (100%)

Bonfield, Tracey; Tepper, Mark 2015

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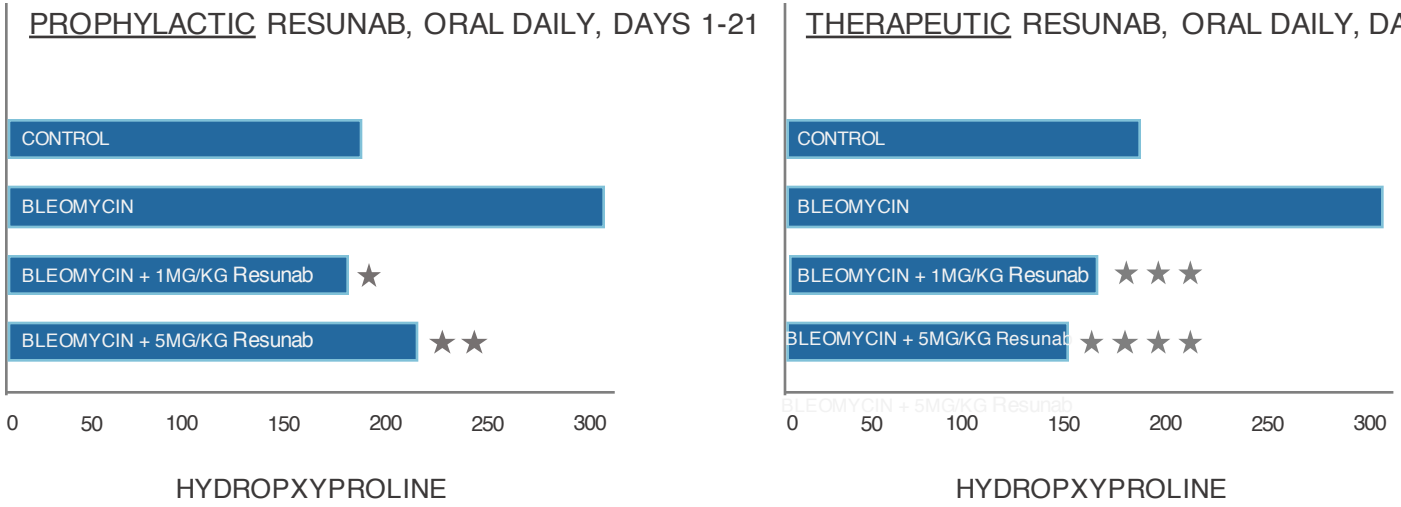
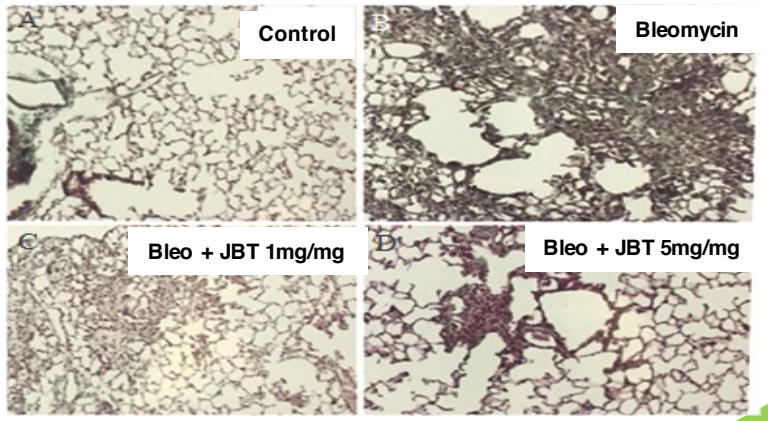
PROPHYLACTIC AND THERAPEUTIC RESUNAB INHIBIT COLLAGEN DEPOSITION IN BLEOMYCIN-INDUCED LUNG FIBROSIS

- Bleomycin intratracheal injection, Day 1. Mice sacrificed after 21 days.
- Resunab by gavage, Days 1-21 (prophylactic) or Days 8-21 (therapeutic)





*p = 0.002, **p = 0.004, ***p = 0.001, ****p = 0.0001, bleomycin + Resunab treatment versus bleomycin

RESUNAB PLANNED CYSTIC FIBROSIS PHASE 2 TRIAL

	Q1 2015	Q2 2015	Q3 2015	Q4 2015	Q1 2016	Q2 2016	Q3 2016	Q4 2016
IND open with FDA		✓						
Study launches								
First patient dosed								
Study duration								
Last patient dosed								
Study data released								

- Double blind randomized placebo control study in the US and EU
- Primary endpoints: Safety/tolerability
- Secondary endpoints: Pharmacokinetics and efficacy (FEV1, Lung Clearance Index, CFQ-R Respiratory Domain)
- Exploratory endpoints: Metabolipidomic profile for MOA, biomarkers of disease activity in blood and sputum, biomarkers of inflammation, and microbiota in the lungs
- Patient number: 70 adults with CF in ~20 sites US & EU
- Treatment duration: 3 months + 1 month follow-up
- Dose response: 1 mg/day, 5 mg/day, 20 mg/day and 2x20 mg/day

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DIFFUSE CUTANEOUS SYSTEMIC SCLEROSIS "SCLERODERMA":

RELIEF FOR A DISEASE WITH NO APPROVED THERAPY

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SCLERODERMA

Chronic inflammatory disease causing fibrosis of skin and internal organs

70,000
PATIENTS IN THE USA

80%
FEMALE PATIENTS

40-60 YEARS
AVERAGE AGE OF PATIENTS

LUNG FIBROSIS

COMMON CAUSE OF DEATH -
50% MORTALITY IN 10 YEARS

KEY TAKE-AWAYS

- Current therapy involves steroids and immunosuppressive agents
- No effective and safe long-term therapy available
- No FDA approved drugs
- Pipelines often target Idiopathic Pulmonary Fibrosis (IPF) in conjunction with SSc

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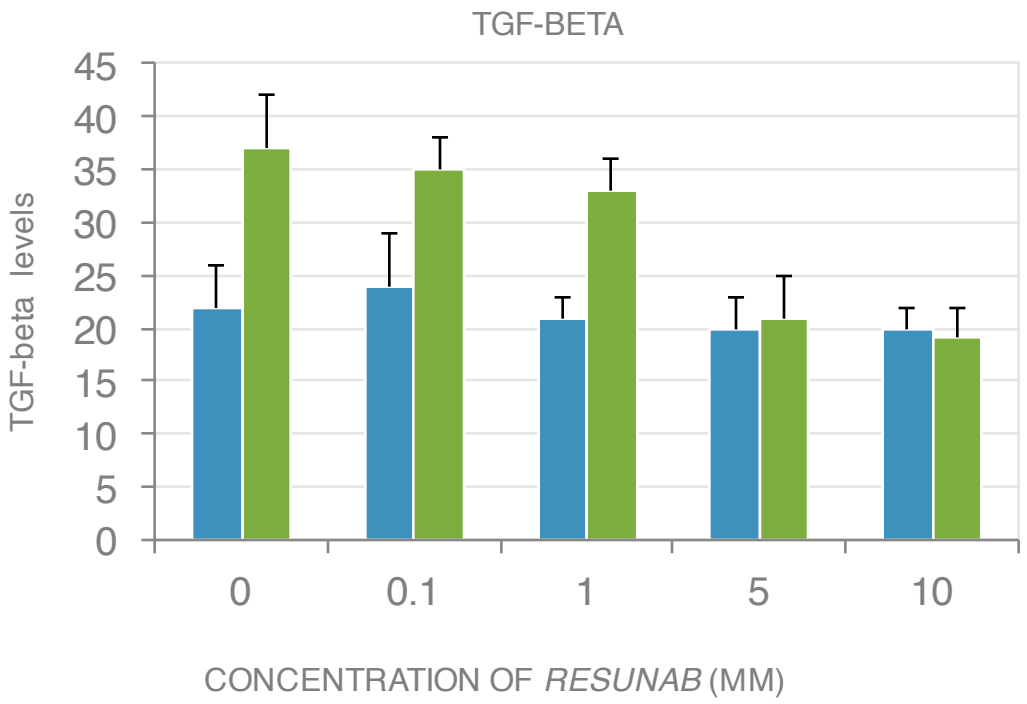
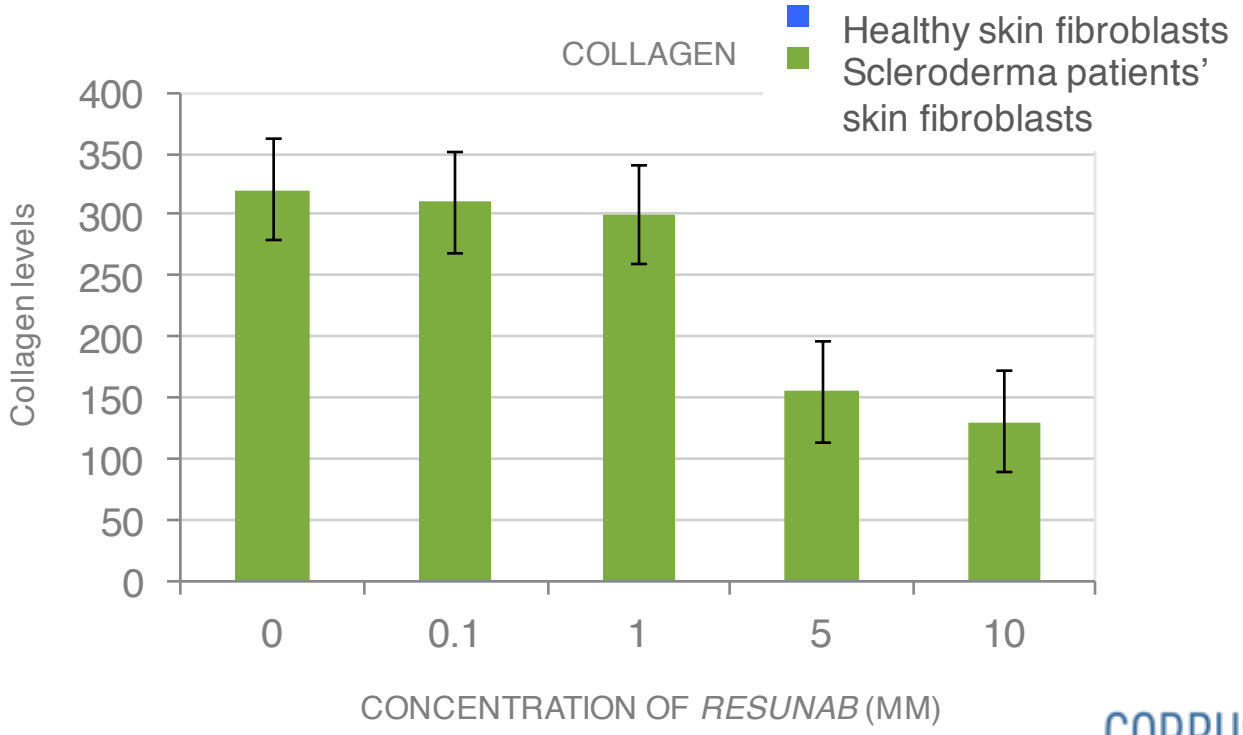
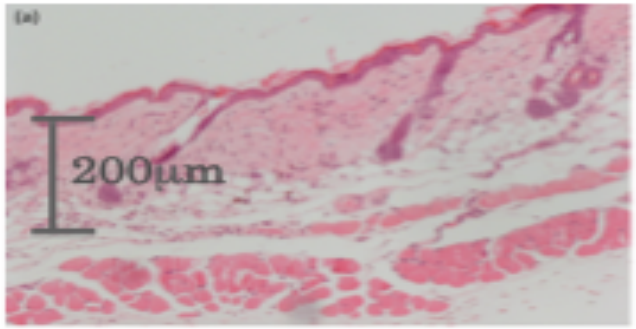
RESUNAB INHIBITS KEY FACTORS IN SCLERODERMA

- 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 SSc animal models
- Resunab reduces TGF-beta and collagen in skin fibroblasts from SSc patients

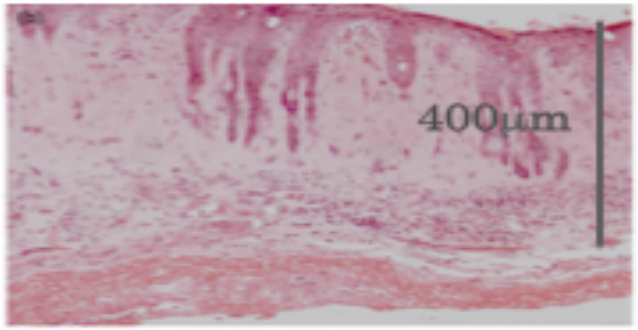




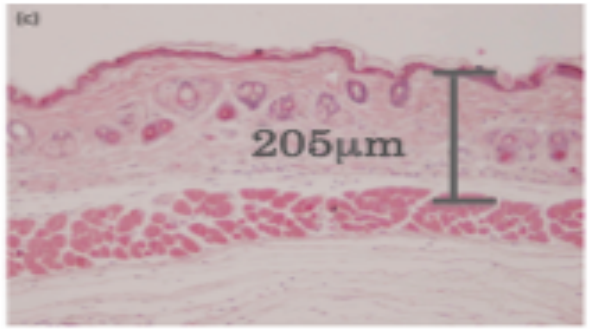
RESUNAB INHIBITS SKIN THICKENING IN MOUSE SSC MODEL



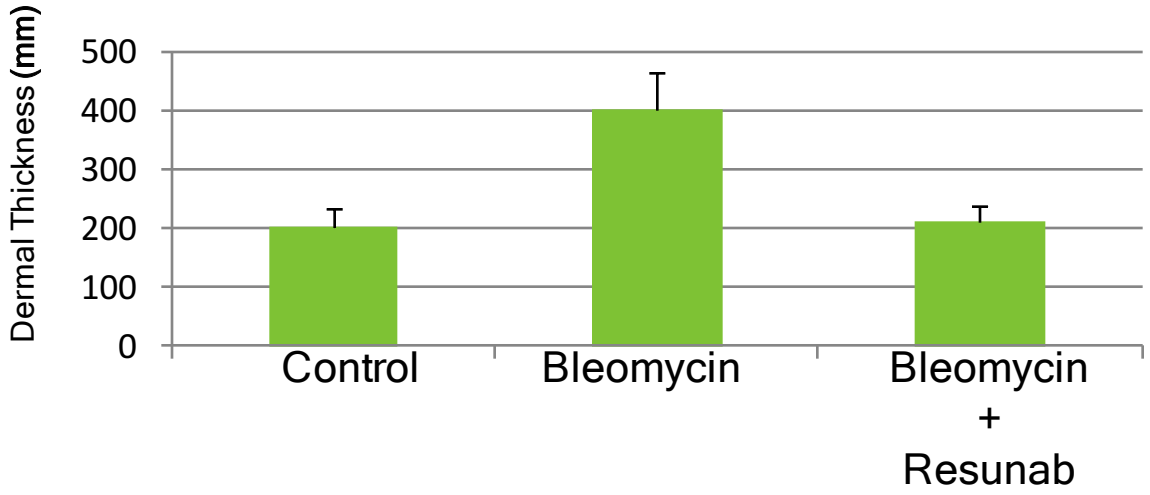
Healthy skin



Thick skin induced by Bleomycin



Near normal skin after oral *Resunab* taken once daily for four weeks

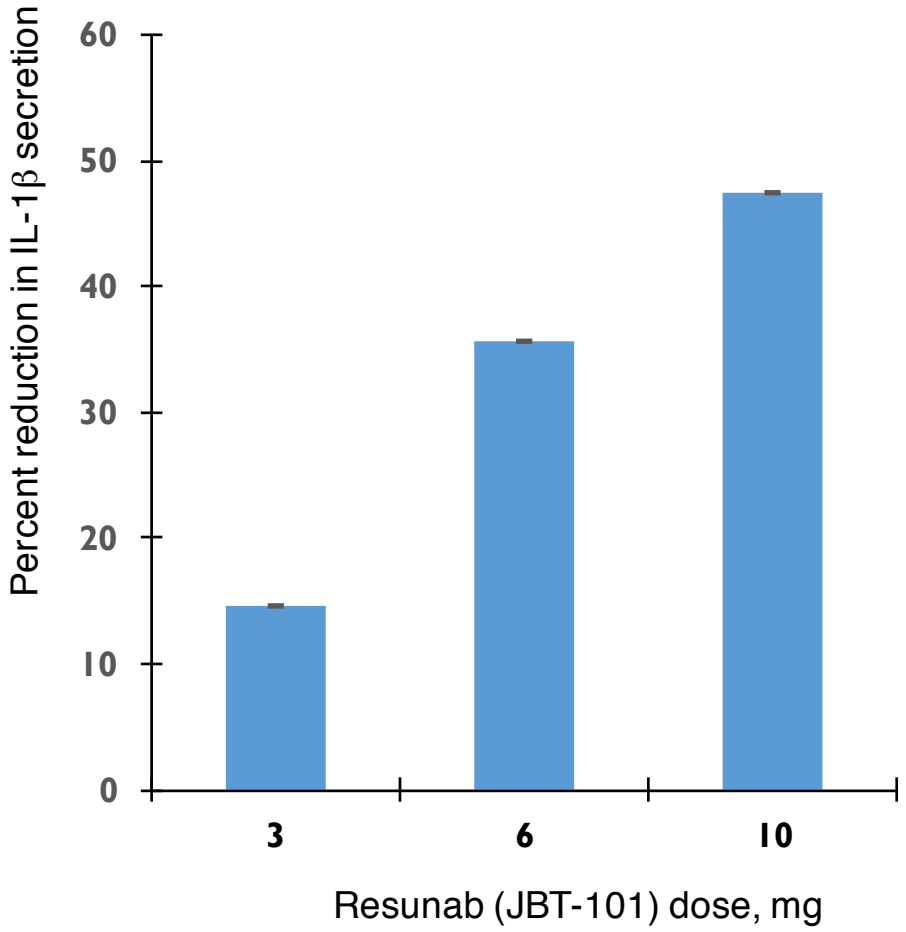


Gonzales et.al., Annals of Rheumatic Diseases, April 4, 2012

CORBUS PHARMACEUTICALS

PRELIMINARY EVIDENCE OF MOA IN HEALTHY HUMAN SUBJECTS

Three healthy volunteers received single doses of 3, 6, and 10 mg Resunab (JBT-101). Five hours following each dose, PBMC were isolated and stimulated with IL-1α, then IL-1β secretion was measured after 18 hours incubation. Percent inhibition was determined relative to baseline values prior to JBT-101.



RESUNAB: PLANNED SSC PHASE 2 CLINICAL TRIAL

	Q1 2015	Q2 2015	Q3 2015	Q4 2015	Q1 2016	Q2 2016	Q3 2016	Q4 2016
IND open with FDA	✓							
Study launch			✓					
First patient dosed								
Study duration								
Last patient dosed								
Study data released								

- Double blind placebo control randomized study in USA under IND from FDA
- Primary end points: Safety/tolerability + Change in clinical outcomes (CRISS)
- Secondary end points: Metabolipodomic profile + biomarkers of disease activity & inflammation + quality of life (QOL)
- Patient number: 36 adults with SSc at 8-10 US sites
- Treatment duration: 3 months + 1 month follow-up
- Dose response: 5mg/day, 20mg/day and 20mg/2Xday

DERMATOMYSITIS: CRITICAL UNMET IN RARE DISEASE

DERMATOMYOSITIS

is a connective tissue disease characterized by inflammation of skin and muscles

25,000
PATIENTS IN THE USA 

NO FDA
APPROVED THERAPIES
FOR DERMANTOMYSITIS

KEY TAKE-AWAYS

- Treated with steroids and immunosuppressive therapies but with significant toxicities
- **Phase 2 study in 22 patients underway** at University of Pennsylvania
- NIH is funding the study
- Data read out expected in early 2017

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

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)

SCOTT CONSTANTINE M.S.
Director, Clinical Operations

Expertise in CF and Pulmonary diseases trials. Former Director, Clinical Research & Operations of Insmed and Clinical Program Scientist at PTC Therapeutics (PTCT)

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), Orencia® (RA, 2013 sales of $1.4B), Rebif® (MS, 2013 sales of $2.59B), Gonal-F® (Fertility, 2013 sales of $815MM)

BARBARA WHITE M.D.
CHIEF MEDICAL OFFICER

Board-certified Rheumatologist and clinical immunologist. Previously held positions in industry: SVP and Head, R&D for Stiefel a GSK company, VP and Head of Inflammation Clinical Development at UCB and MedImmune/AstraZeneca, and Director, Medical Affairs, Amgen

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
Chairman of the Board of the Metropolitan Washington, D.C. Chapter of the Cystic Fibrosis Foundation

AVERY W. (CHIP) CAITLIN
CFO Celldex Therapeutics (CLDX) since 2000
Raised over $600MM financing
20 years experience in industry: Repligen (CFO) and Endogen (CFO)

DAVID HOCHMAN
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 Insmed, 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)

WORLD-CLASS SCIENTIFIC ADVISORS

ETHAN BURSTEIN, PH.D.
ACADIA PHARMACEUTICALS INC.
Senior Director of Biosciences

SUMNER BURSTEIN, PH.D.
UMASS MEDICAL SCHOOL
Professor of Biochemistry and Pharmacology; inventor of Resunab

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CASE WESTERN RESERVE MEDICAL SCHOOL
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Director of UCLA Scleroderma Program

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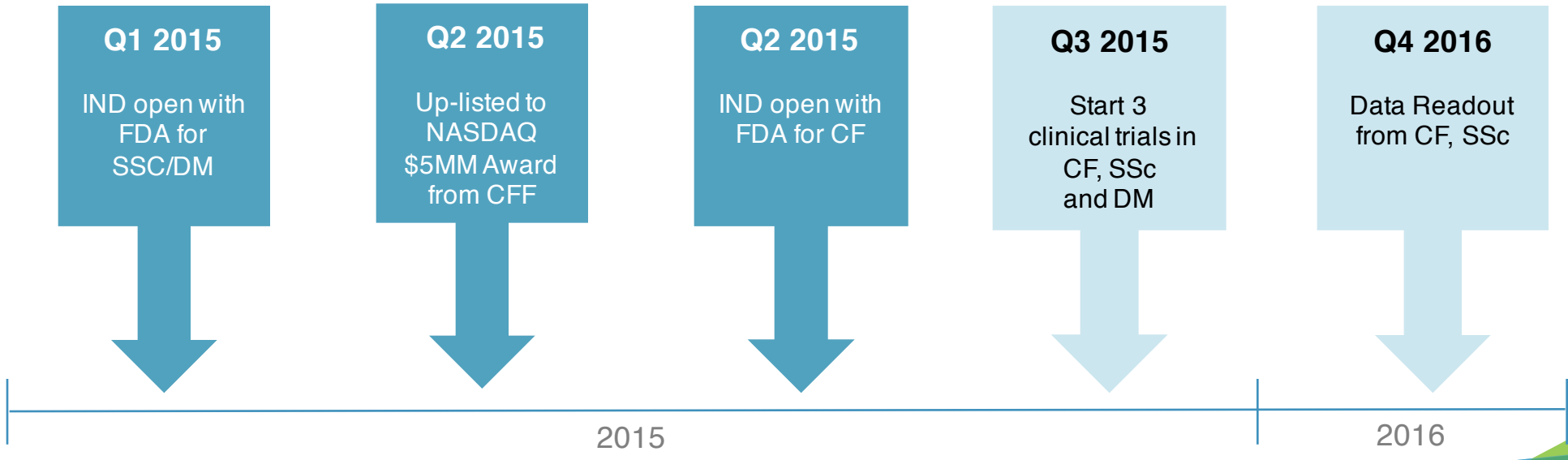
Director of CET&RI; Professor of Anesthesia, Perioperative and Pain Medicine, Infection and Immunity

ROBERT ZURIER, M.D.
UMASS MEDICAL SCHOOL
Ex-Chair of Rheumatology

FINANCIAL PROFILE I NASDAQ: CRBP

Stock Ticker:	NASDAQ: CRBP
$72 MM	Market capitalization as of 8/28/2015
37,535,000	Common shares outstanding after warrant exercises (called on 7/27/2015)
43,350,000	Fully diluted outstanding
$14.2MM	Cash as of 08/28/2015 not including additional $3.8MM from CFF award
862,000	Average daily trading volume



MILESTONES Q3+Q4 2015

ANTICIPATED DATE	MILESTONE
August 26th	Completed warrant call:100% of callable warrants exercised
August 19th	Fast Track status granted for systemic sclerosis
Aug 31st	Systemic sclerosis (SSC) Phase 2 clinical trial launched
Q3	Launch CF Phase 2 clinical trial
Oct 8-10	North American CF Conference: pre-clinical data presented
Oct 20-21	The 14th Annual BIO Investor Forum
Q4	All 3 Phase 2 clinical trials (CF, SSC and DM) are enrolling and dosing patients
Q4	Anticipated Orphan designation for CF
Q4	Anticipated Fast Track designation for CF

CONCLUSIONS

- Experienced, seasoned team with proven track record
- Lead Product Resunab is a novel, promising clinical stage oral drug
- Conducting Phase 2 trials in three separate rare diseases
- Data read out from Phase 2 trials in Q4 2016
- $5MM Award from CFF and NIH funding for DM
- Near-term milestones can drive valuation

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