CORBUS PHARMACEUTICALS

OTCQB: CRBP

www.CorbusPharma.com

Developing Breakthrough Therapies for Rare Inflammatory Diseases

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," "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.

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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 "off" switch 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)
  - 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

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Our Target Indications: Current & Future

Indication	Patient numbers (USA)	Estimated Market size	Current therapies for inflammation	Drawbacks to current therapies
Current lead indications:				
Cystic Fibrosis	30,000	>$3B	Steroids, ibuprofen	Considerable side effects
Diffuse Systemic Sclerosis (Scleroderma)	50,000	>$2B	Steroids, methotrexate	Side effects, poor efficacy
Potential future indications:				
Dermatomyositis	13,000	>$1B	Steroids, mAbs	Side effects, poor efficacy
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)

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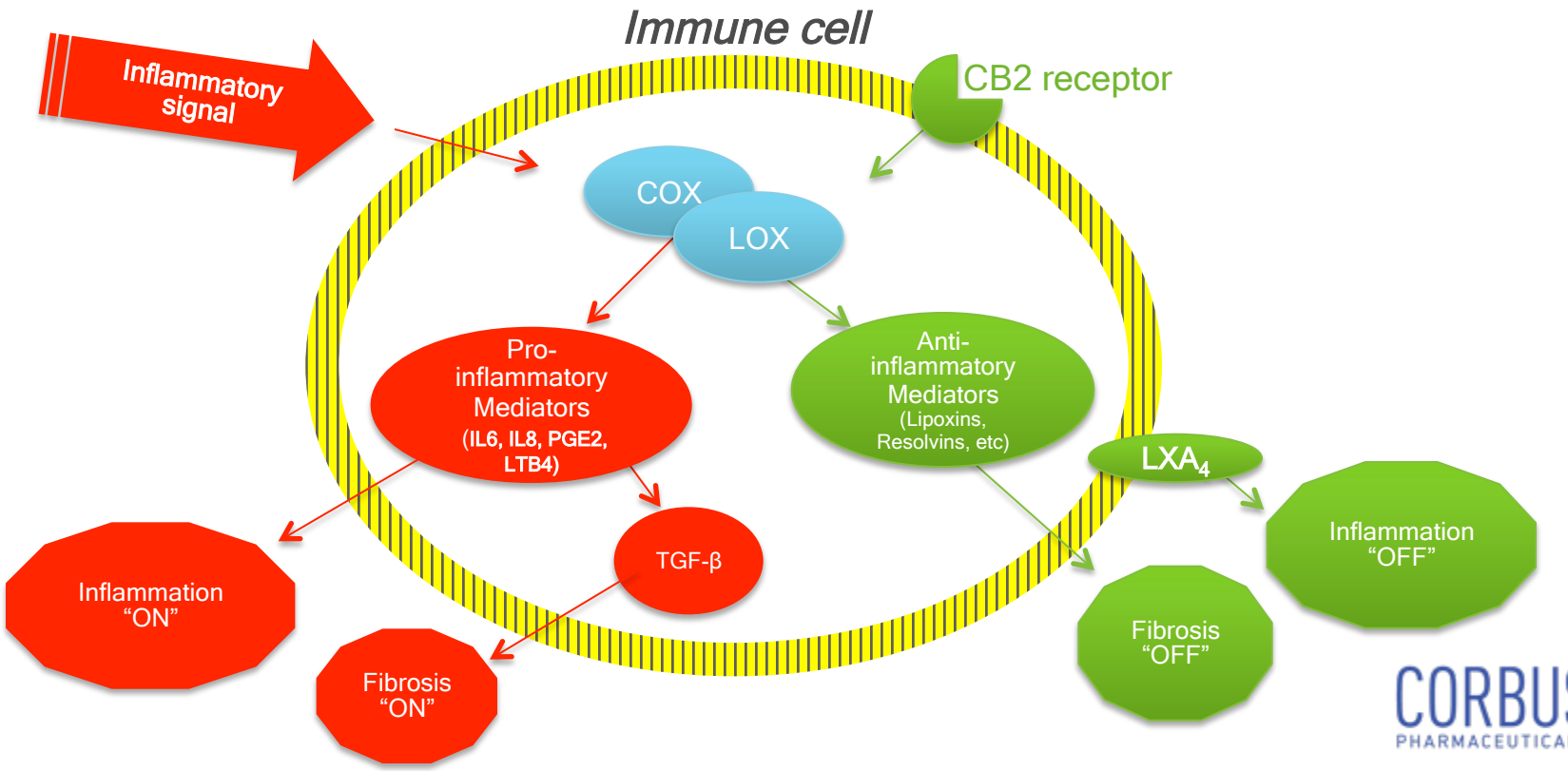
CB2 Receptor: Turns inflammation "off"

- 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



ARTHRITIS & RHEUMATISM
Vol. 52, No. 12, December 2005, pp 3680–3687
DOI 10.1002/art.21434
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EDITORIAL

Eicosanoids in Scleroderma: Lung Disease Hangs in the Balance

Bruce D. Levy

Lung disease has been recognized as a complica- fore lung involvement is detectable by high-resolution

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 a major burden on modern societies. Understanding

ECM.[1] However, the molecular mechanisms of fibroblast activation and potential counter-regulatory mechanisms, which limit the inflammatory reaction and the consecutive ECM accumulation,

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

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 chronic

nature immunology

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], Rong Yang[3], Jasim Uddin[3], William B Guggino[4], Sowsan F Atabani[1], Yasmine Belkaid[1], Yan Xu[5], Jeffrey A Whitsett[5], Frank J Accurso[6], Marsha Wills-Karp[7] & Nicos A Petasis[3]

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; TGF = 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

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

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]

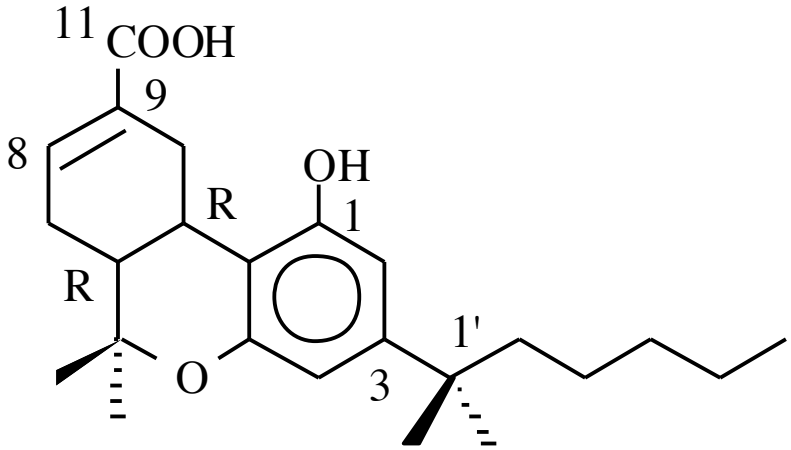
Affiliations: [1]National Children's Research Centre, Crumlin, Dublin, Ireland. [2]Our Lady's Children's Hospital, Crumlin, Dublin, Ireland. [3]Midwestern Regional Hospital, Limerick, Ireland. [4]Centre for Interventions in Infection, Inflammation and Immunity (4i), Graduate Entry Medical School, University of Limerick, Limerick, Ireland. [5]Molecular Medicine Laboratories, Royal College of Surgeons in Ireland, Beaumont Hospital, Dublin, Ireland. [6]Institut National de la Santé et de la Recherche Médicale, U845, Faculté de Médecine Paris Descartes, Paris, France.

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

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)

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=123)
- Preparing to launch two Phase 2 clinical studies in H1 2015



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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

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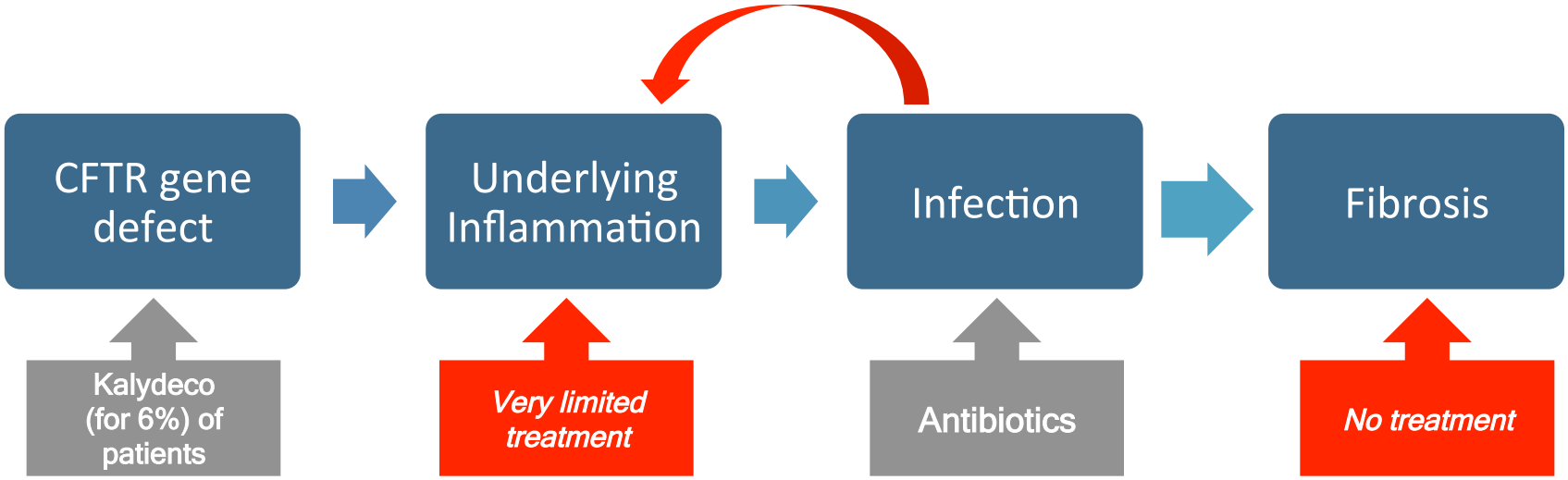
Cystic Fibrosis

Targeting inflammation at the core of the disease

Overview: Cystic Fibrosis

- 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
- Pharmacoeconomics support premium pricing (e.g. Kalydeco by Vertex priced at $320,000/yr)



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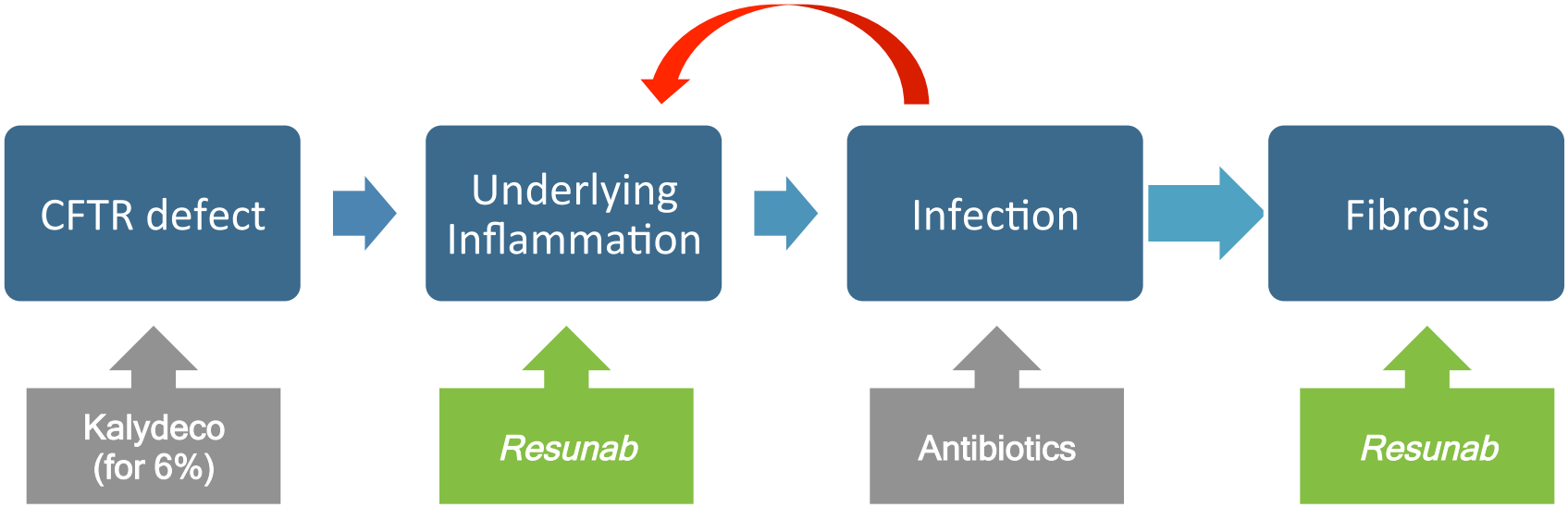
Resunab targets key CF inflammatory players

↓ TGF-β	↑ Lipoxin-A4
• Genetically linked to disease • Associated with worsening symptoms	• Absent in CF lungs • Replacement therapy effective in animal models



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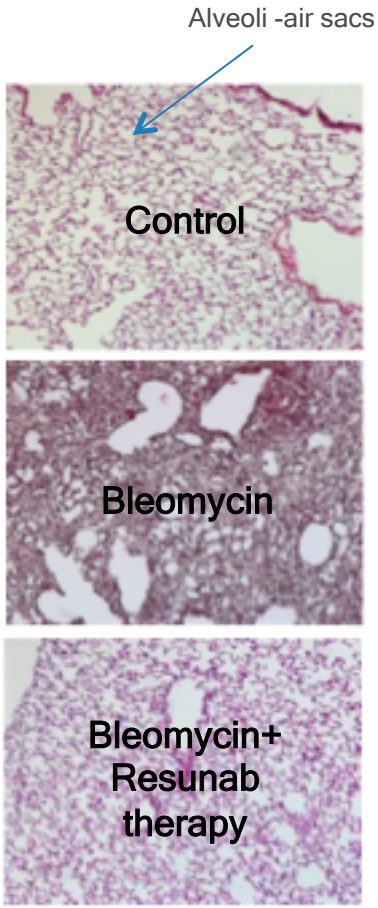
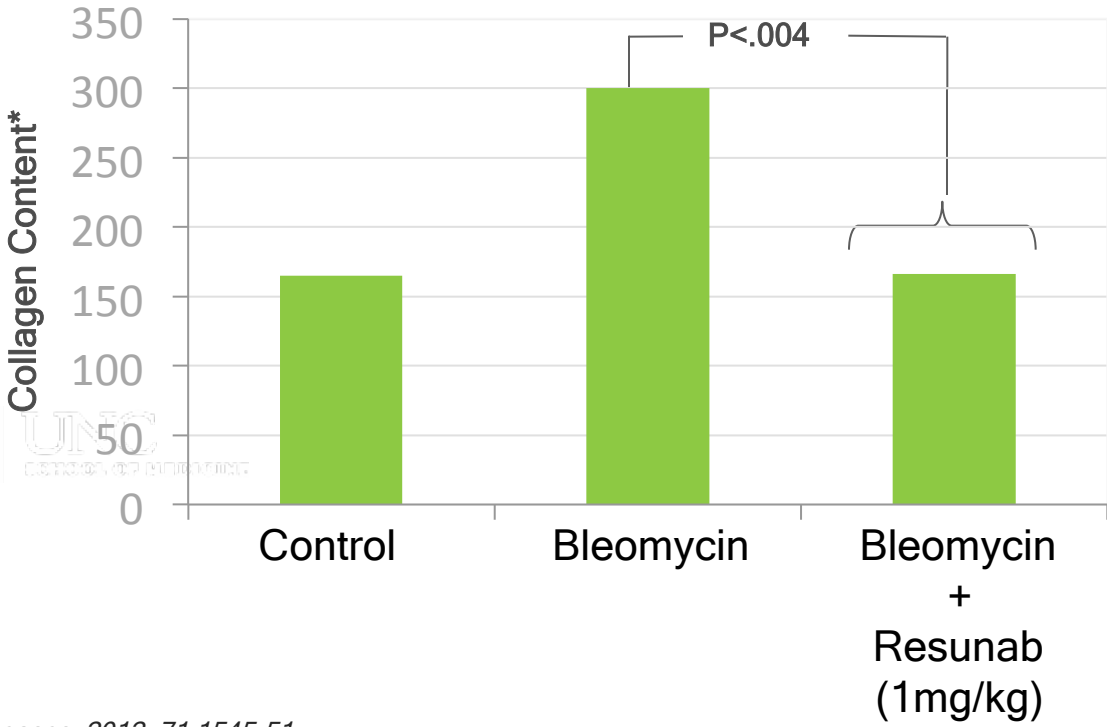
Resunab Reduces Pulmonary Fibrosis In Animal Models



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*

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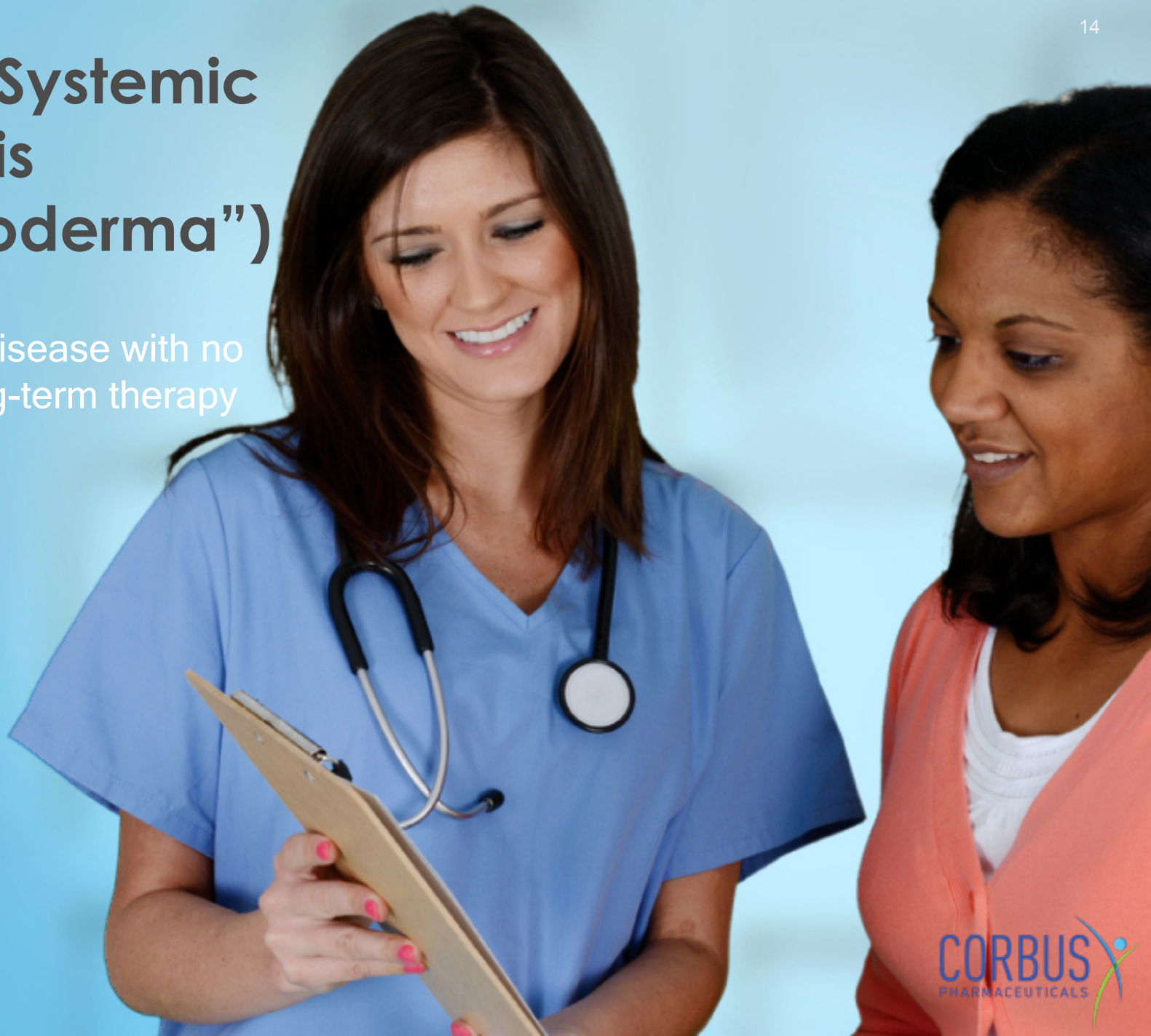
Resunab Planned Cystic Fibrosis Phase 2 Trial

- ✓ 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

	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



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Overview: Diffuse Cutaneous 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 40's, 50's and 60'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

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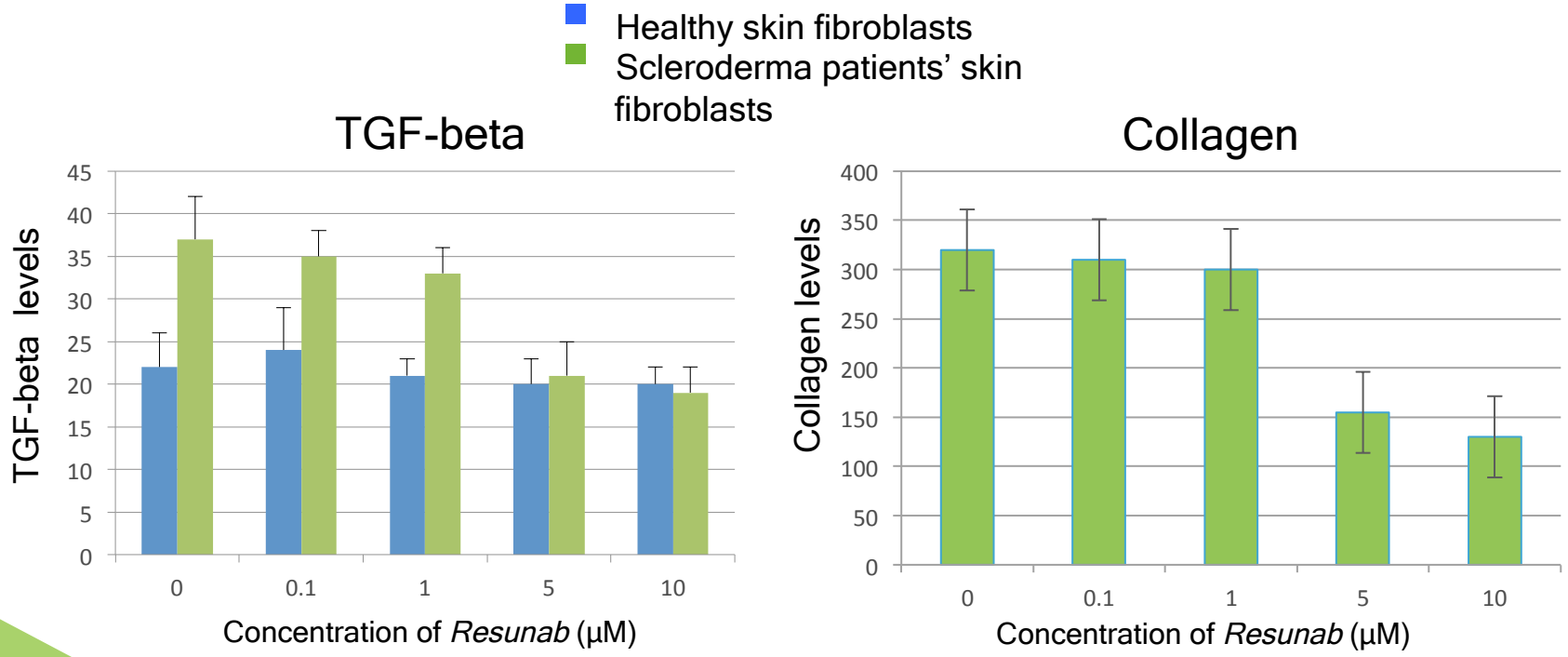
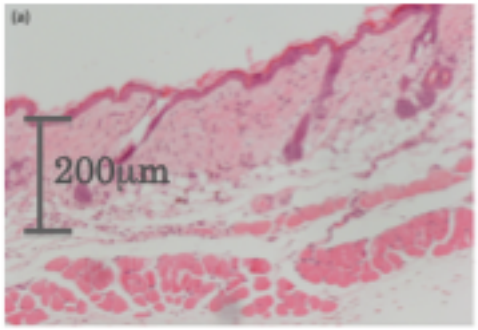
Resunab Inhibits Key Factors in SSc

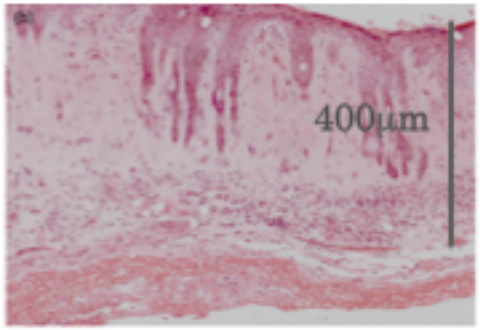
- TGF-beta plays key role in SSc progression (same in CF and IPF)
- Elevated TGF-beta levels associated with disease progression
- Strong Resunab efficacy data in animal models
- Resunab reduces TGF-beta and collagen in skin fibroblasts from SSc patients



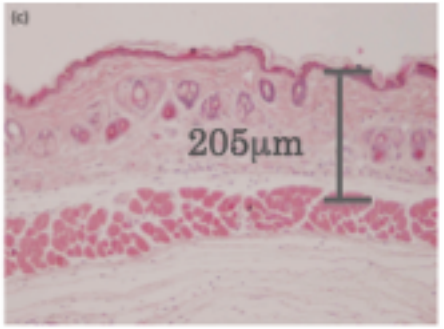
Resunab Inhibits Skin Thickening In Mouse SSc Model



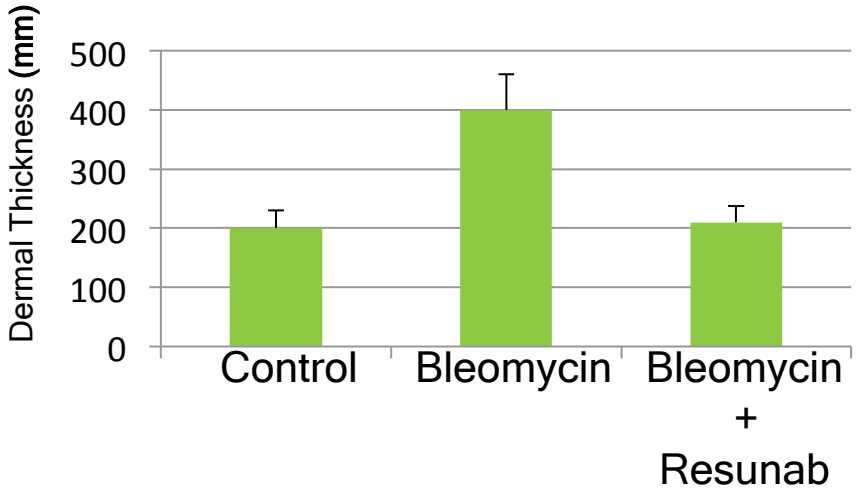
Healthy skin



Thick skin induced by Bleomycin



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



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

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Resunab: Planned SSc Phase 2 Clinical Trial

- 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 with 8-10 US sites
- **Treatment duration:** 3 months + 1 month follow-up
- **Dose response:** 5mg/day, 20mg/day and 20mg/2Xday

	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

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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), Orencia® (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

- Rheumatologist and 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

Scott Constantine, M.S. – Director, Clinical Operations

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

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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 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)

Avery W. (Chip) Caitlin

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

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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 Spiera M.D. – Hospital for Special Surgery NYC
Professor of Medicine, Head of Scleroderma and Vasculitis Center

Daniel Furst, M.D. – UCLA School of Medicine
Director of UCLA Scleroderma Program

Robert Zurier, M.D. - UMass Medical School
Ex-Chair of Rheumatology

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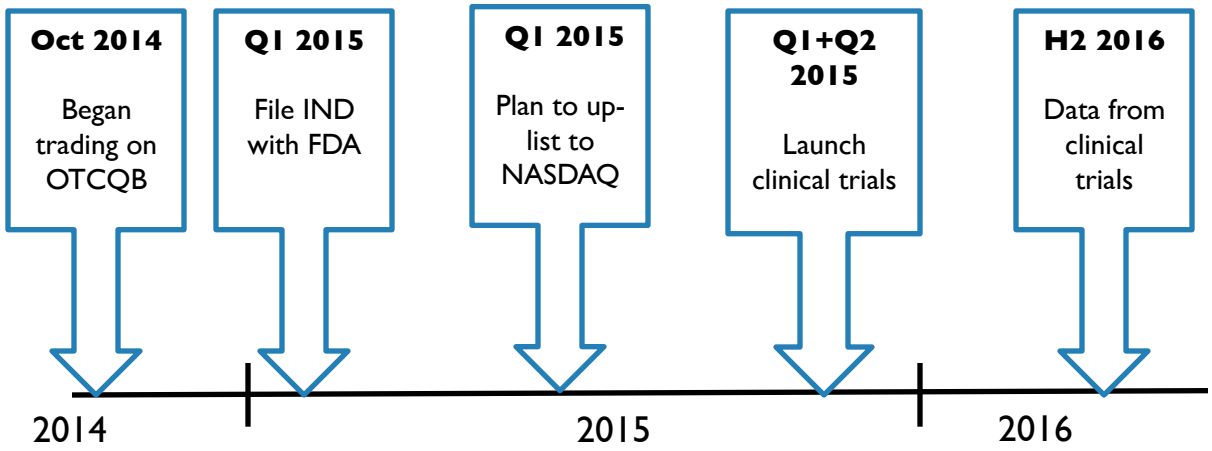
Financial Profile
OTCQB: CRBP

Stock Ticker:	OTCQB: CRBP
$74,820,000	Market capitalization as of January 8, 2015
$10,300,000	Raise from successful private placement (Q2 2014) from institutional and retail base
25,800,000	Common shares outstanding
41,500,000	Fully diluted shares outstanding (including warrants and stock options)
$11,400,000	Available from exercise of warrants callable at $2.50 per share
NASDAQ	Up-listing to NASDAQ planned by Q-1 2015



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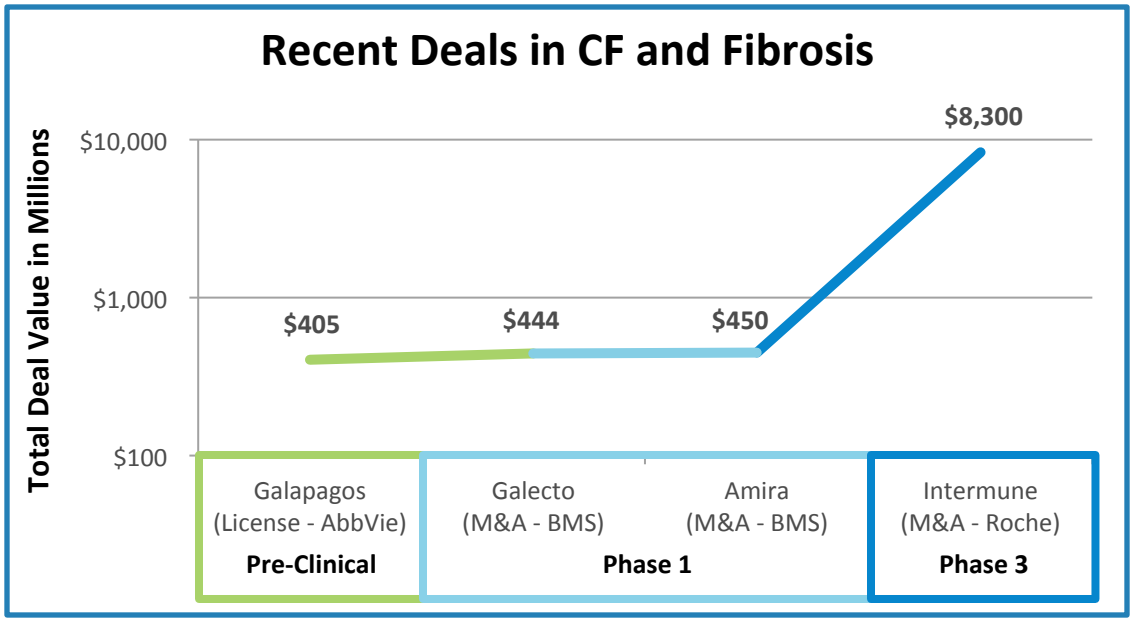
Corbus Poised for Significant Upside



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

Peer Valuation in Fibrosis Field

Company	Indication	Clinical Phase	Share Price*	Market Cap*
Corbus (CRBP)	Cystic Fibrosis / Systemic Sclerosis	Phase 2	$2.90	$75m
ProQR (PRQR)	Cystic Fibrosis	Pre-Clinical	$19.58	$457m
Insmed (INSM)	Cystic Fibrosis	Phase 3	$16.07	$798m
PTC (PTCT)	Cystic Fibrosis	Phase 3	$54.18	$1.59bn
Intercept (ICPT)	Liver Fibrosis	Phase 3	$151.04	$3.23bn
Vertex (VRTX)	Cystic Fibrosis	Approved, $800m in sales	$123.97	$29.82bn

* As of January 8, 2015

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Conclusions

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

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100 River Ridge Drive
Norwood, MA 02062
www.CorbusPharma.com

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