ASX Announcement

AdAlta presentation to AusBiotech Conference

MELBOURNE Australia, 30 October, 2019: AdAlta Limited (ASX: 1AD), is pleased to release a copy of the presentation that will be delivered today at the AusBiotech 2019 conference.

AusBiotech is the leading annual conference for Australia's life sciences sector and provides a forum for industry leaders to exchange ideas on a range of topics to further advance the standing of the sector both nationally and globally.

At the conference, AdAlta's Chief Scientific Officer Dr Mick Foley will present as part of a panel session addressing the topic: "Anti-fibrotic drugs: A hot area for big pharma". He will give an overview of fibrosis and speak about AdAlta's work in this therapeutic area.

Presentation details:

Date: 30th October 2019
Event: AusBiotech 2019
Location: Melbourne Convention and Exhibition Centre
Time: 1.30pm

A copy of the AdAlta presentation is attached and will also be made available on the Company's website at www.adalta.com.au.

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Notes to Editors

About AdAlta

AdAlta Limited is an Australian-based drug development company headquartered in Melbourne. The Company is using its proprietary technology platform to generate a promising new class of protein therapeutics, known as i-bodies, that have the potential to treat some of today's most challenging medical conditions. The technology mimics the shape and stability of a crucial antigen-binding domain, that was discovered initially in sharks and then developed as a human protein. The result is a range of unique compounds, capable of uniquely interacting with previously difficult to access targets such as G-protein coupled receptors and ion channels that are implicated in many serious diseases.

AdAlta is currently preparing for its phase 1 clinical studies for its lead i-body candidate, AD214. The clinical program is expected to commence in early 2020 following completion of the current toxicity study, clinical trial design finalisation and manufacture of clinical product. AD214 is being developed for the treatment of Idiopathic Pulmonary Fibrosis (IPF) and other human fibrotic diseases, for which current therapies are sub-optimal and there is a high-unmet medical need. The Company is also in collaborative partnerships to advance the development of its i-body platform. It has recently announced an agreement with UK-based research organisation, Excellerate Bioscience to collaborate on an undisclosed target of commercial interest and an agreement with GE Healthcare for diagnostic imaging agents against several drug targets, including Granzyme B.

AdAlta plans to continue further drug discovery and development directed towards other drug targets and diseases.

Further information can be found at: www.adalta.com.au.

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AusBiotech 2019

Australia's Life Sciences Conference

30 October - 1 November 2019

Melbourne Convention and
Exhibition Centre, South Wharf

HOST INDUSTRY BODY

AusBiotech
AUSTRALIA'S BIOTECHNOLOGY ORGANISATION

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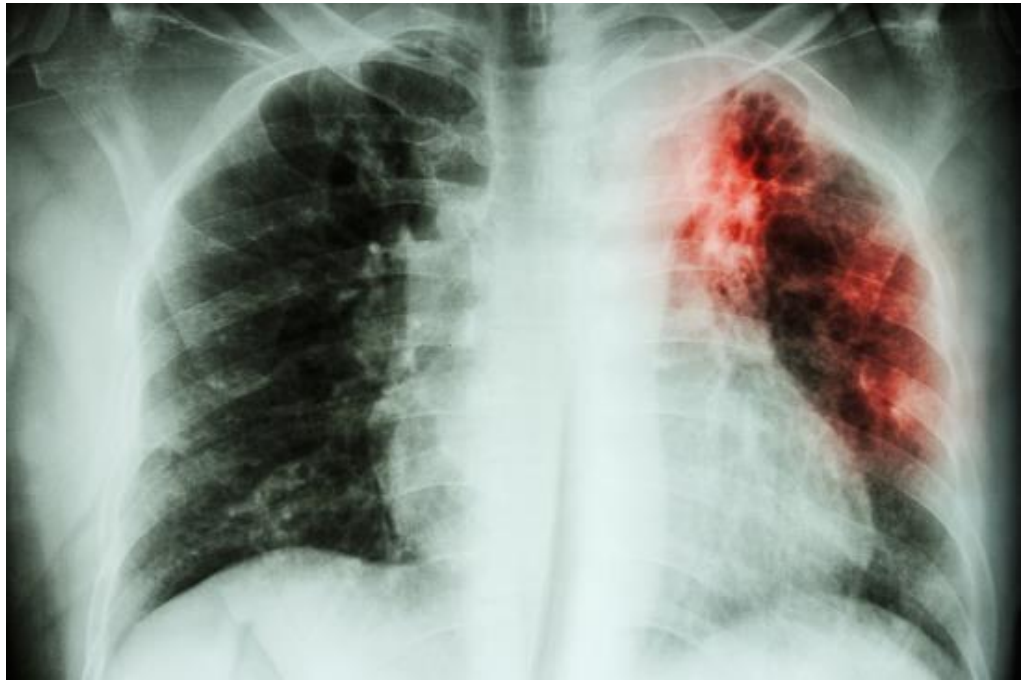
Anti-fibrotic drugs:
A hot area for big pharma

Dr Mick Foley	AdAlta
Gary Phillips	Pharmaxis
Dr Nina Webster	Dimerix
Prof Darren Kelly	Certa Therapeutics

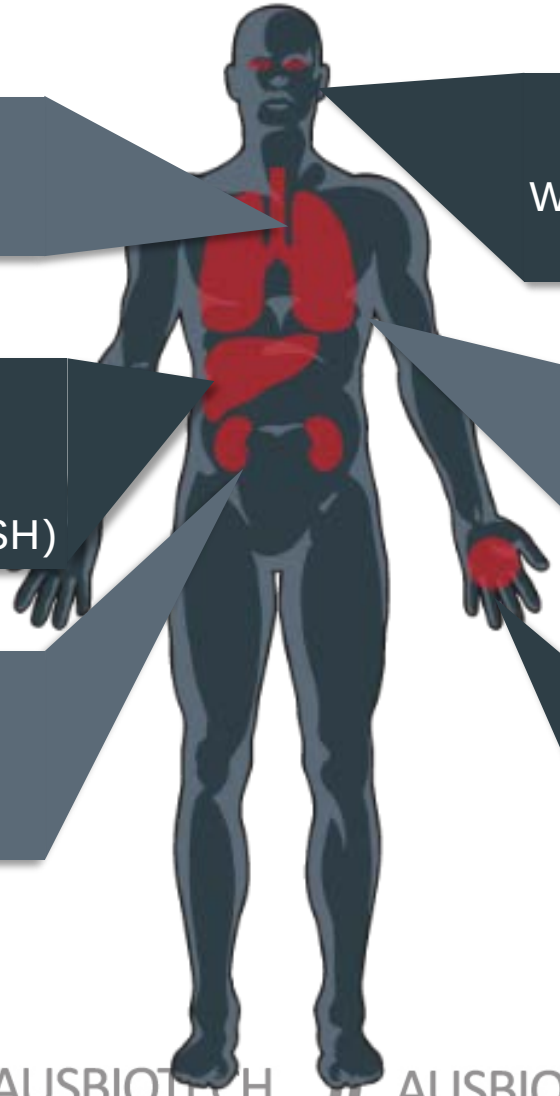
Fibrosis: significant unmet medical need

- Fibrosis is the build-up of connective tissue
  - Results from repeated inflammation and uncontrolled wound healing
  - Causes scarring of vital organs such as the lung, liver, skin, eye, heart and kidney
  - Leads to irreparable damage and eventual organ failure
  - Fibrosis contributes to the pathophysiology of 45-50% of diseases



There is no clinically satisfactory therapeutic approach to fibrosis

Fibrosis: multiple indications



Heart

Cardiac Fibrosis

Eye

Wet age-related macular degeneration
Proliferative vitreoretinopathy

Liver

Cirrhosis
Non-alcoholic steatohepatitis (NASH)

Lung

Idiopathic Pulmonary Fibrosis
Interstitial Lung Disease

Kidney

Chronic Kidney Disease
End-Stage Renal Disease

Skin

Scleroderma
Hypertrophic Scar

Global market interest in fibrosis treatments

Fibrosis assets acquired at an early stage – typically based on Phase I results

Date	Company	Target	Indication	Acquired by	Deal value (US$)	Development Stage
Oct-19	**Pliant Therapeutics**	**PLN-1474 (+3 candidates)**	**Liver (NASH)**	**Novartis**	**$80m upfront**	**Phase I**
Jul-19	**Bridge Biotherapeutics**	**BBT-877**	**Lung (IPF)**	**Boehringer Ingelheim**	**$50.75m upfront + $1.1B in milestones**	**Phase I**
July-19	Yuhan Corp	YH-25724	Liver (NASH)	Boehringer Ingelheim	$59m upfront + $1.2B in milestones	Preclinical
Sep-18	Samumed	SM04646	Lung (IPF)	United Therapeutics	$10m upfront + $340min milestones	Phase I
Apr-18	Ionis Pharmaceuticals	AZD-2963	Liver (NASH)	AstraZeneca	$44m upfront + $444m in milestones	Preclinical
Feb-18	Oraxion Therapeutics	ORX-301	Kidney (FSGS)	Undisclosed US Biopharm	Up to $185m	Preclinical
Sep-15	Adheron Therapeutics	SDP051	Lung (IPF)	Roche	$105m upfront, plus $475m in milestones	Phase I

Source: GlobalData

Australia batting above its average

pharmaxis

- PXS-4728A acquired in Phase I by BI for NASH
- $45m upfront and up to $317m in milestones
- Deal expanded to second indication of diabetic retinopathy

fibrotech therapeutics

- FT011 inhibited fibrosis of kidney and heart
- Fibrotech acquired by Shire in July 2014
- Upfront payment of US$75m
- Assets being developed as Certa therapeutics

CERTA THERAPEUTICS

Dimerix

- DMX-200 addresses 3 mechanisms of kidney fibrosis
- Phase IIa: all endpoints met
- Recruitment completed for Phase II in diabetic kidney disease and FSGS

AdAlta
next generation protein therapeutics

- AD-214 shows anti-fibrotic and anti-inflammatory effects in multiple fibrosis models
- Demonstrated safety up to 100mg/kg in 4-week tox study
- First in human study commences Q1 2020

AdAlta

next generation protein therapeutics

Targeting CXCR4 using an i-body:

A novel approach to fibrosis

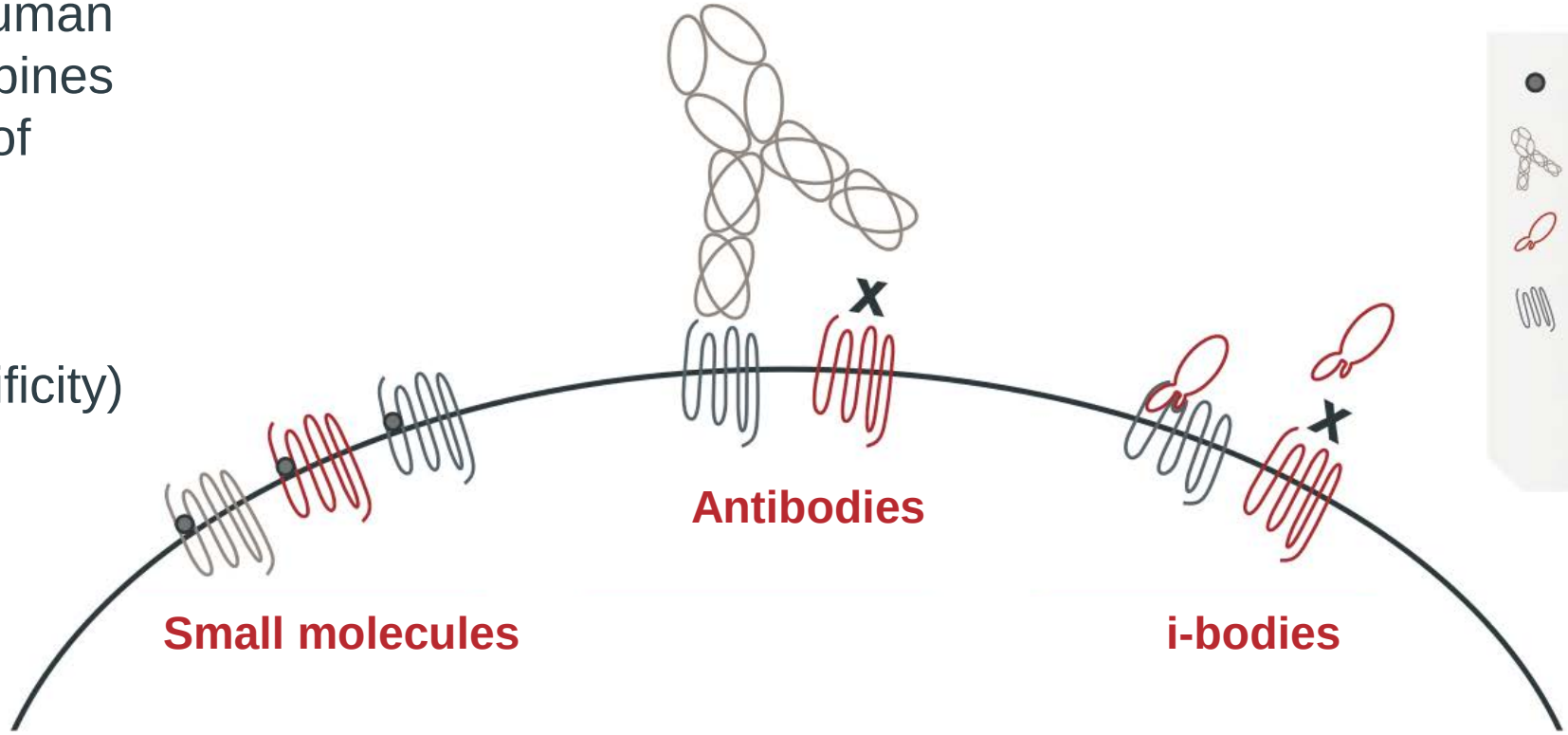
Ausbiotech 2019

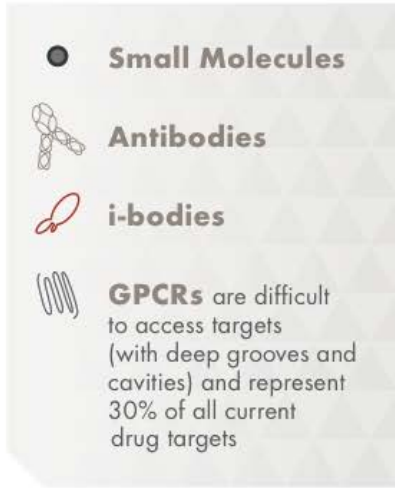
Mick Foley, CSO

AdAlta Limited (ASX:1Ad)

i-body technology

- AdAlta is developing a new technology platform that produces unique proteins known as i-bodies.
- An i-body is a human protein that combines the advantages of small molecules (stability) and antibodies (high affinity and specificity) in one powerful treatment.





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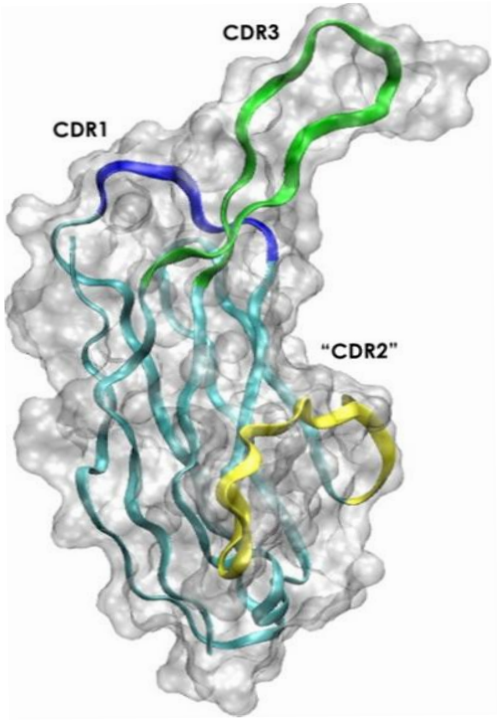
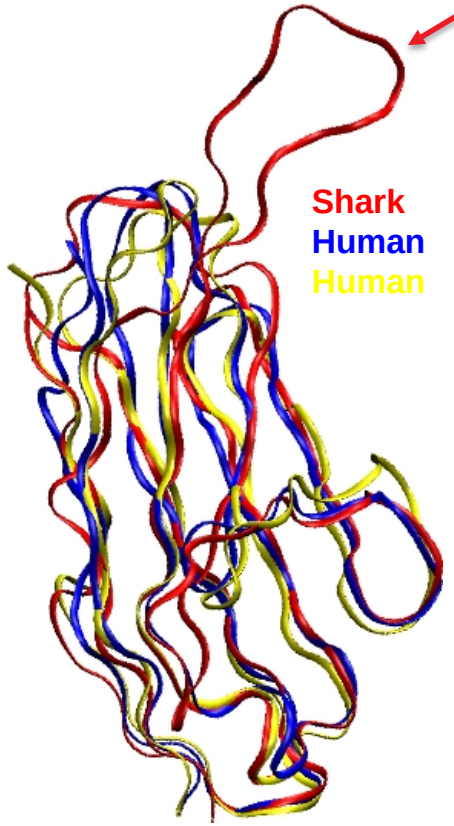
i-bodies: human single domains

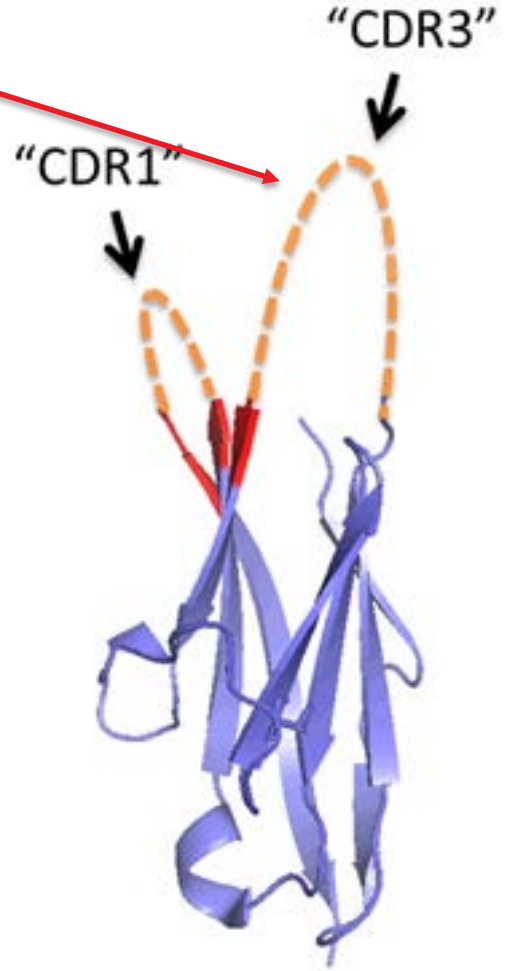


VNAR



Ribbon Overlay

NCAM Domain 1



i-body library

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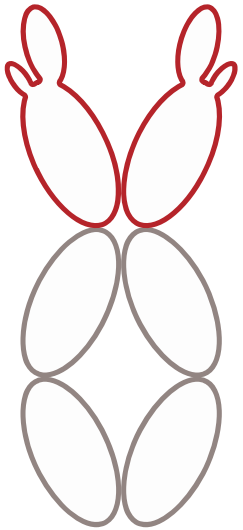
CXCR4 in fibrosis and other disease states

- Member of the C-X-C family of chemokine receptors
- Several established functions
  - Maintaining stem cells in bone marrow (approved CXCR4 antagonist is used to mobilise stem cells but cannot be used chronically due to cardiotoxicity)
  - HIV-1 uses CXCR4 as a co-receptor for viral entry into host cells
  - CXCR4 has been associated with more than 23 types of cancers
- CXCR4 has more recently been recognised as a critical player in pathophysiology of fibrosis several tissues such as:
  - Lung
  - Kidney
  - Heart
  - Eye
  - Skin

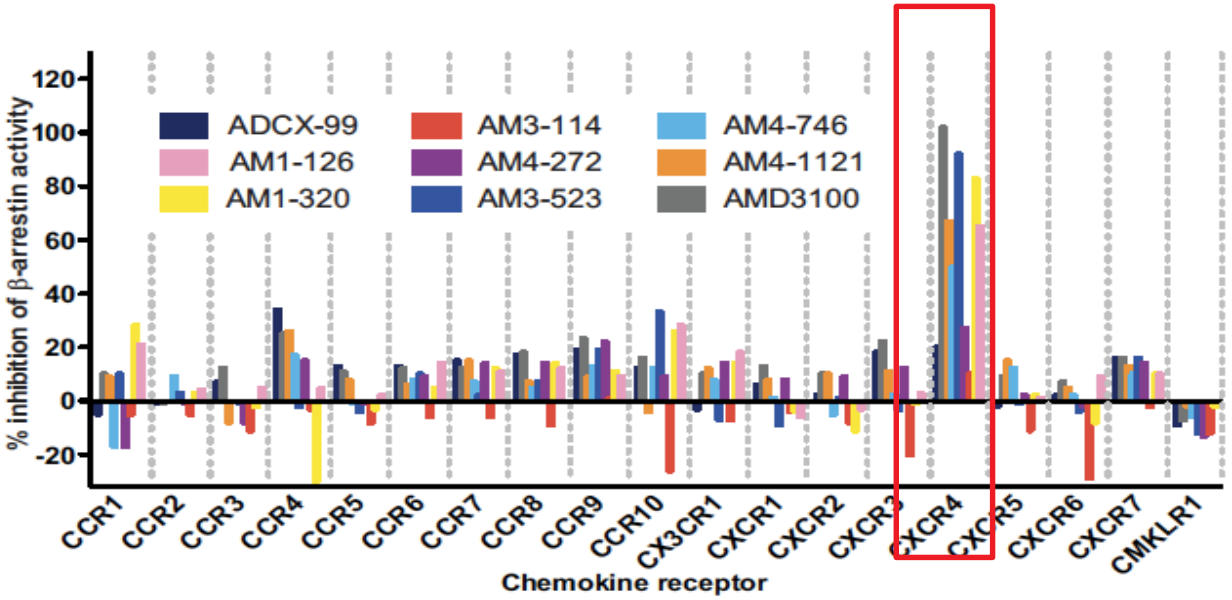
A CXCR4 i-body with high affinity and specificity

AD-114
Binds to CXCR4 on the cell surface and has anti-fibrotic activity



AD-214
Binds to CXCR4 on the cell surface and has anti-fibrotic activity. Fc Fragment extends half life

Highly specific: AD-114 tested against 167 GPCRs and selectively binds to CXCR4



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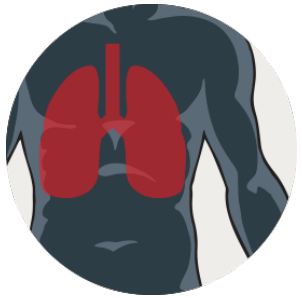
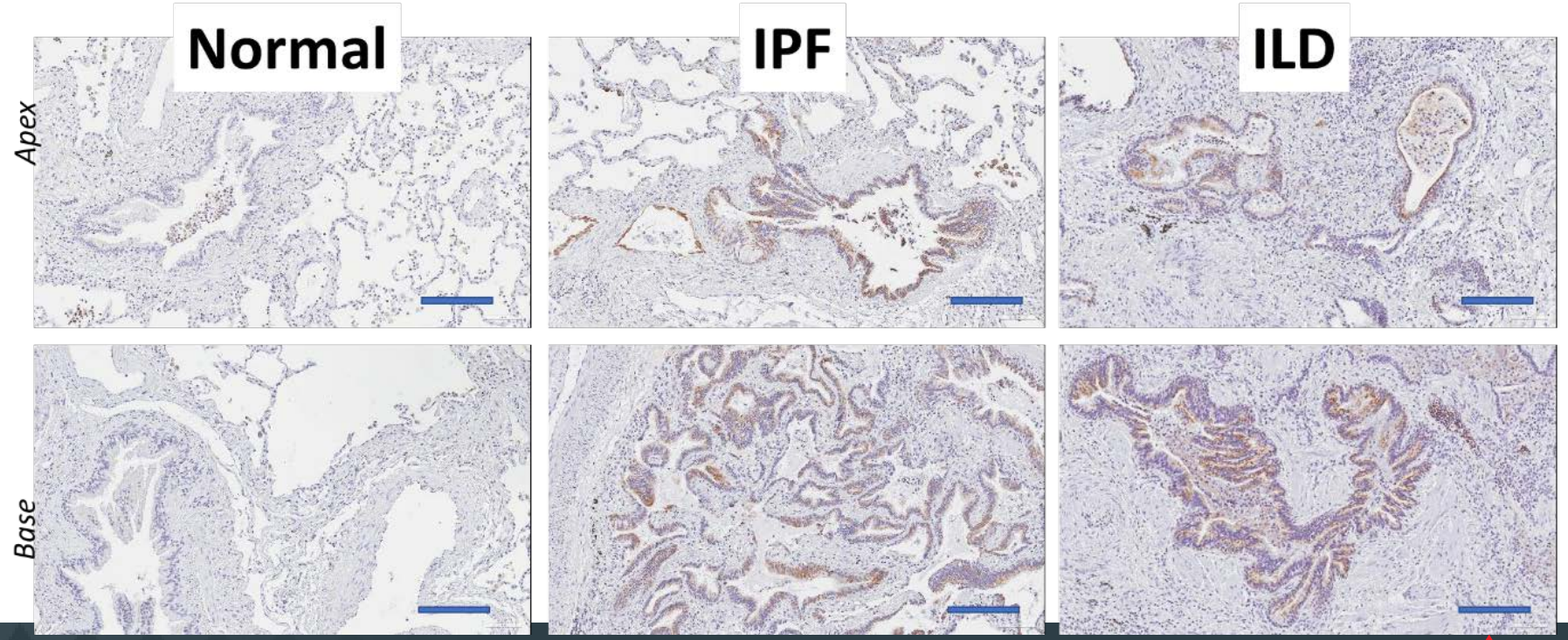


Lung

IPF

CXCR4 and lung fibrosis

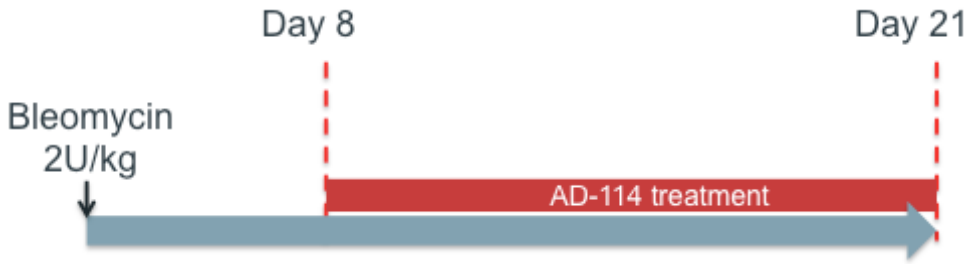
- CXCR4 expression is significantly increased in the lung tissue of patients with two lung fibrosis conditions: Idiopathic Pulmonary Fibrosis (IPF) and Interstitial Lung disease (ILD) compared to healthy controls
- CXCR4 expression not only in circulating immune cells but significantly upregulated in the fibrotic airway epithelium

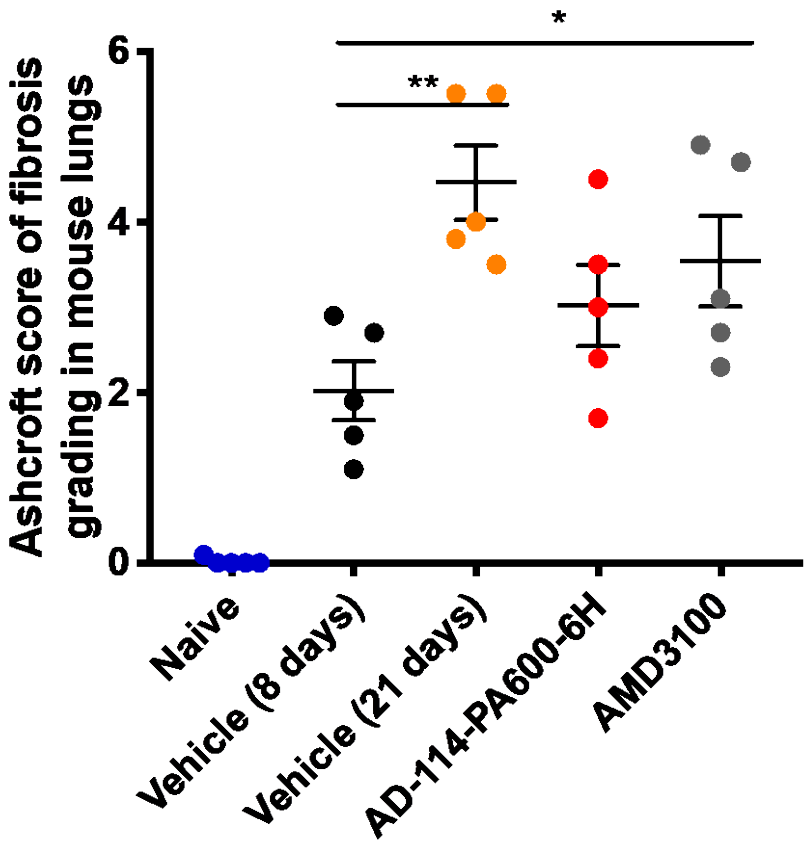


AdAlta

AD-114 prevents fibrosis in Bleomycin-induced mouse model of lung fibrosis

- The progression of the lung fibrosis was ameliorated with AD-114, with no significant difference seen from the Day 8 group (start of treatment) and the i-body treated group
- AMD3100 (CXCR4 antagonist) also reduced the the Ashcroft score compared to the Bleomycin treated mice, however due to toxicity AMD3100 cannot be used long term.

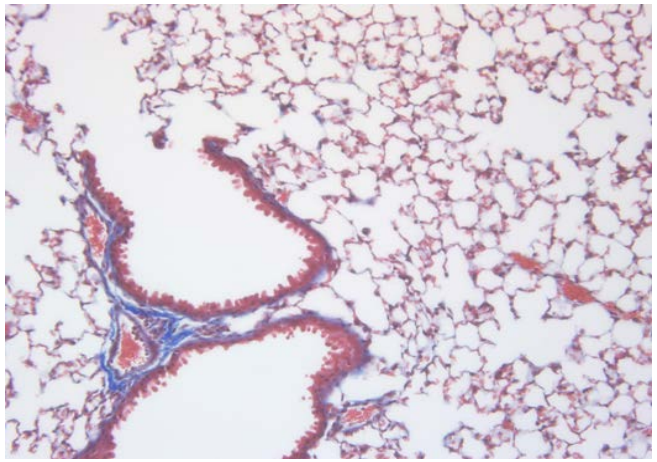




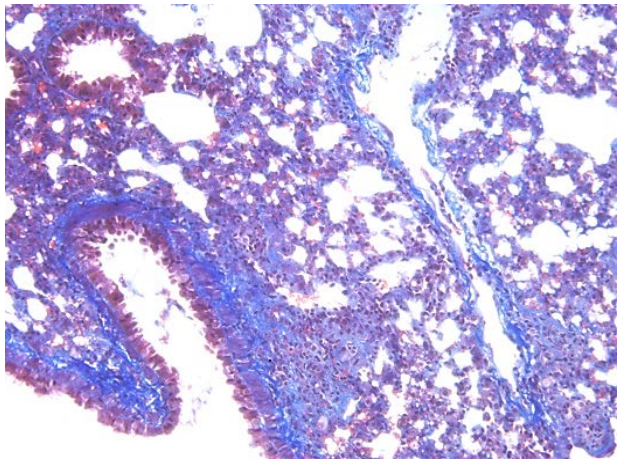
Griffiths et al, Scientific Reports (2018) 8:3212

AdAlta

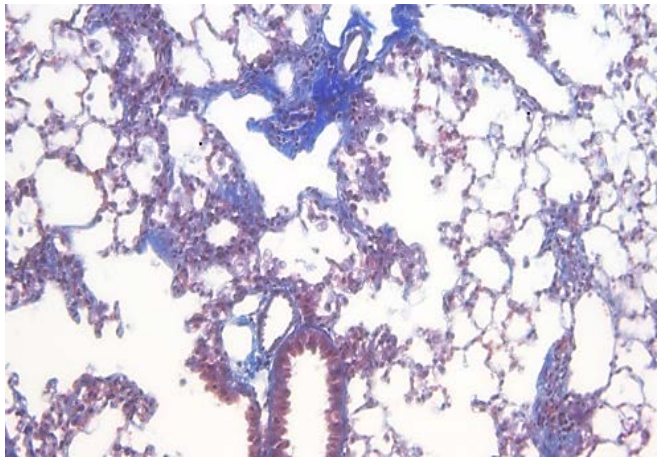
AD-114 prevents fibrosis in Bleomycin-induced mouse model of lung fibrosis



Normal
lung tissue



IPF lung tissue
(lung disease mouse model)



IPF lung tissue + AD-114
dosed for 13 days
(dosing started at day 8 in lung disease model)

AD-114 reduces collagen content and inflammatory cell infiltration in the Bleomycin mouse model and demonstrates a similar architecture to that of the normal lung

Griffiths et al, Scientific Reports (2018) 8:3212

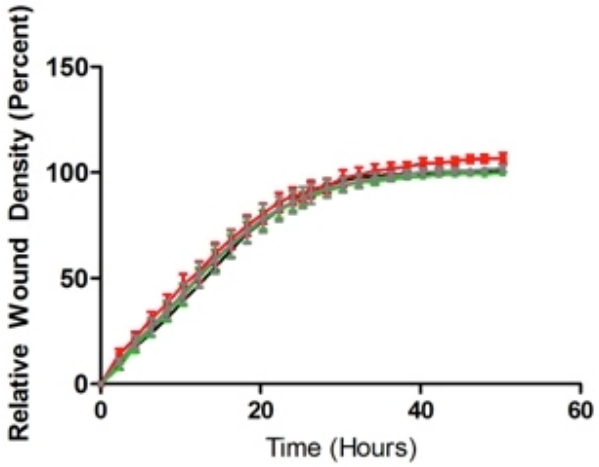
AD-114 reduced migration/invasion of IPF lung fibroblasts

AD-114 specifically inhibited migration of slow and rapid IPF fibroblast migration but did not have any effect on normal fibroblasts.

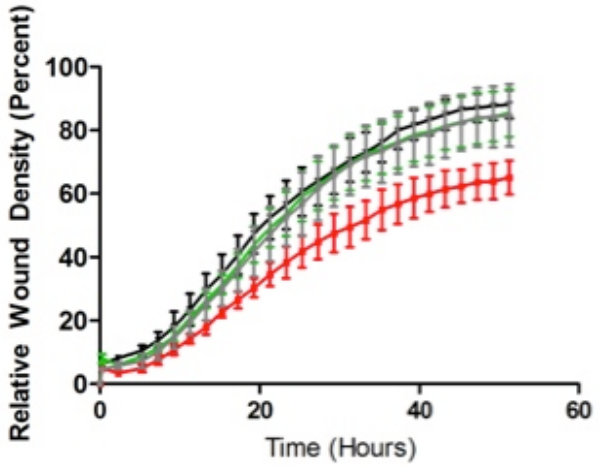
AD-114 has greater *in vitro* efficacy in this assay compared to the only approved therapies Nintedanib and Pirfenidone for IPF treatment.

MIGRATION	No effect on normal fibroblasts	Inhibits slow IPF progressors	Inhibits fast IPF progressors
i-body AD-114	✓	✓	✓
Nintedanib (Boehringer)	✗	✓	✓
Pirfenidone (Roche)	✓	✗	✗
Other CXCR4 drug (Sanofi)	✓	✗	✗

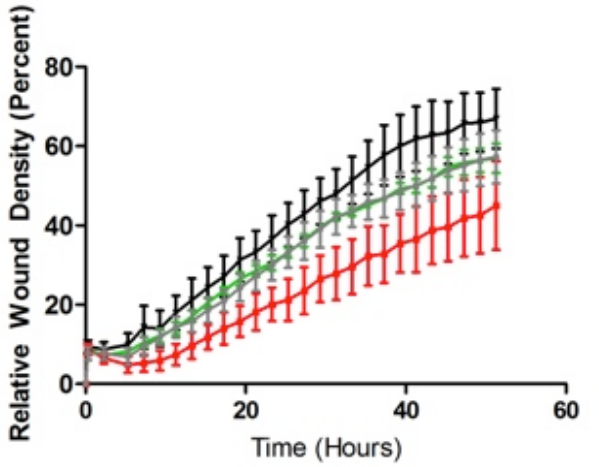
Normal fibroblasts

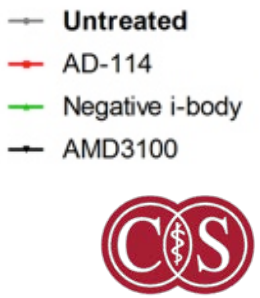


Slow/stable IPF progressor fibroblasts



Rapid IPF progressor fibroblasts





CEDARS-SINAI

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Kidney

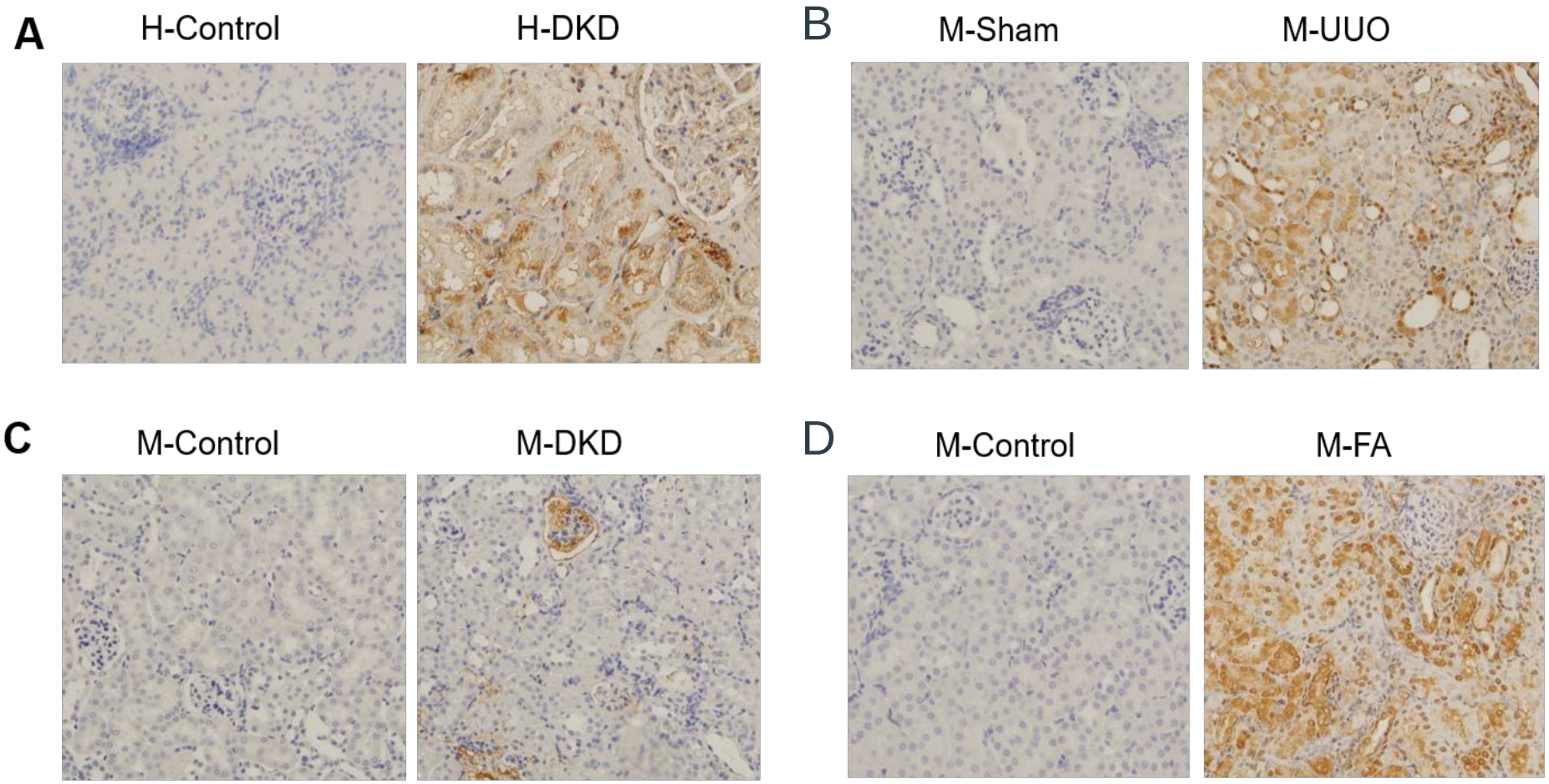
RENAL FIBROSIS

CXCR4 and and kidney fibrosis

- Kidney fibrosis may be caused when the kidneys stop working and eventually transplantation is required.
- Fibrosis may occur at any stage from the onset of chronic kidney disease (CKD) to end-stage renal disease (ESRD).
- CXCR4 is increased in human diabetic kidney tissue
- The i-body to CXCR4 has been tested in mouse models of kidney fibrosis through a collaboration with Carol Pollock at the University of Sydney

Kidney Fibrosis

CXCR4 is upregulated in fibrotic kidneys of animal and human patients



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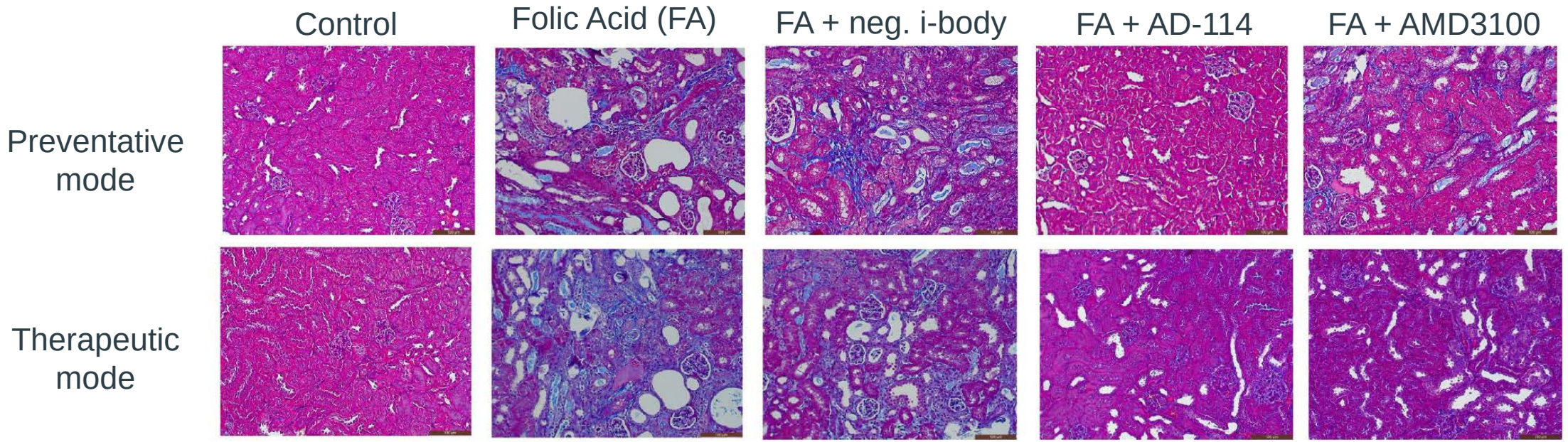
AD-114 in Mouse model of Kidney Fibrosis

- AD-114 treatment, both preventative and therapeutic, reduced collagen and improved the tissue architecture of folic acid-treatment mice to look similar to control mice
- AD-114 had a greater effect than that observed with AMD3100



Blue staining represents collagen, a hallmark of fibrosis

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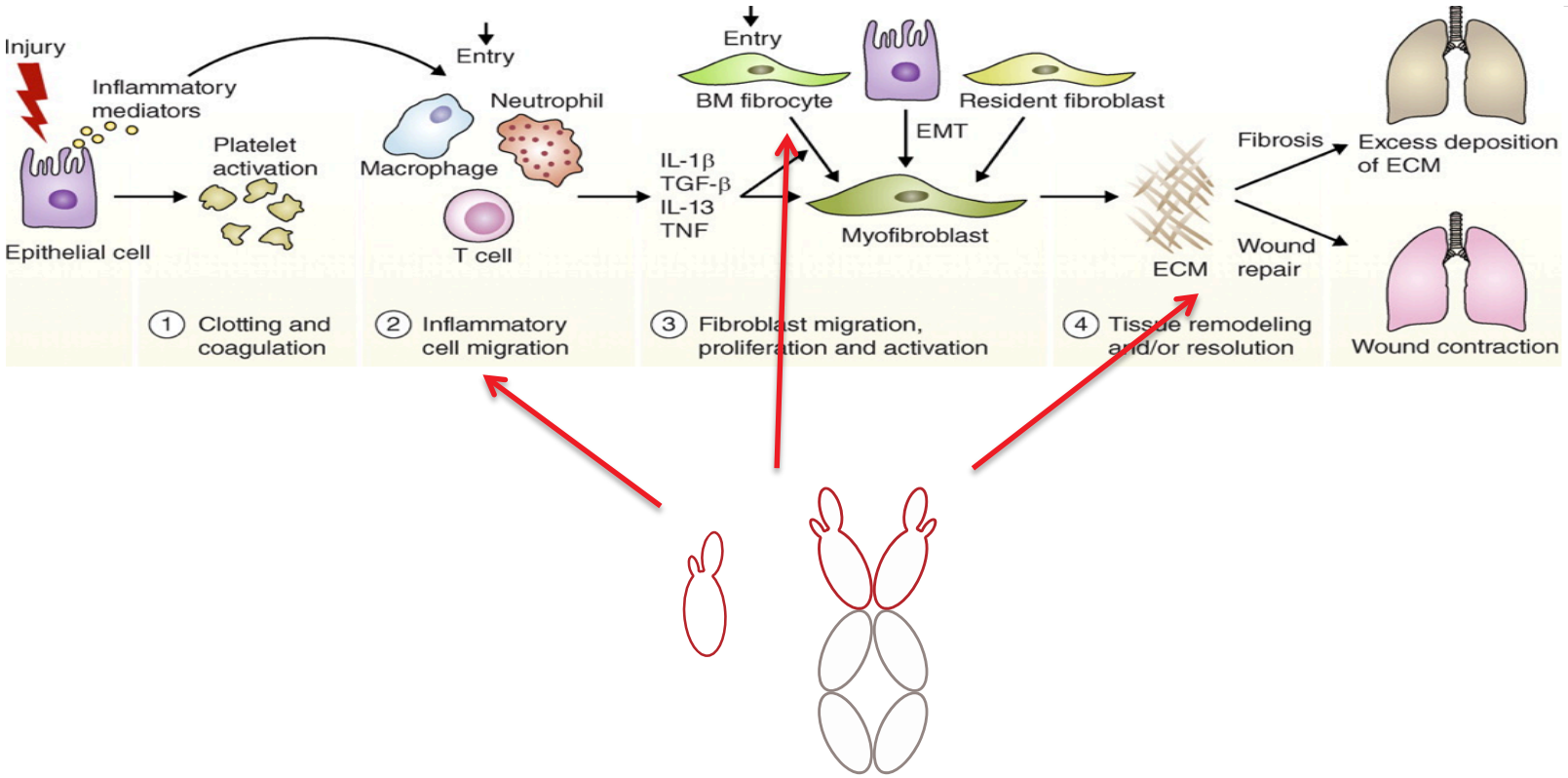
CXCR4 binding i-body products inhibit key features of the fibrogenic pathway with novel MOA



Adapted from Wynn JEM 2011

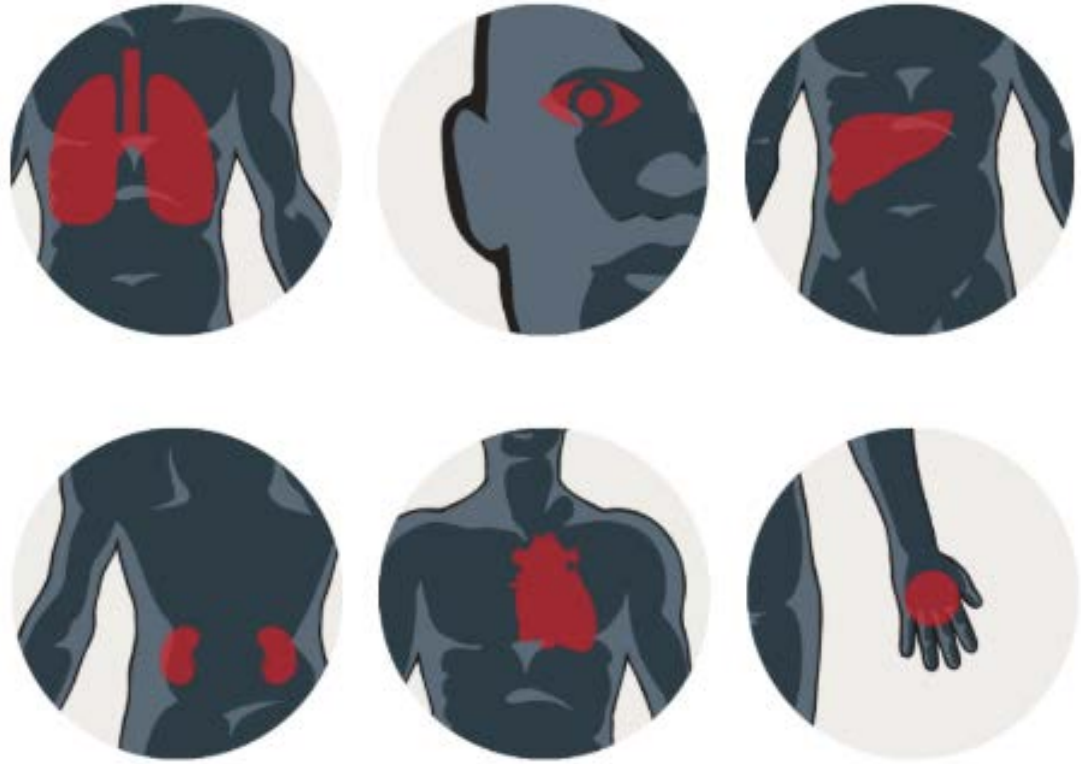
AD-114 & AD-214

AdAlta

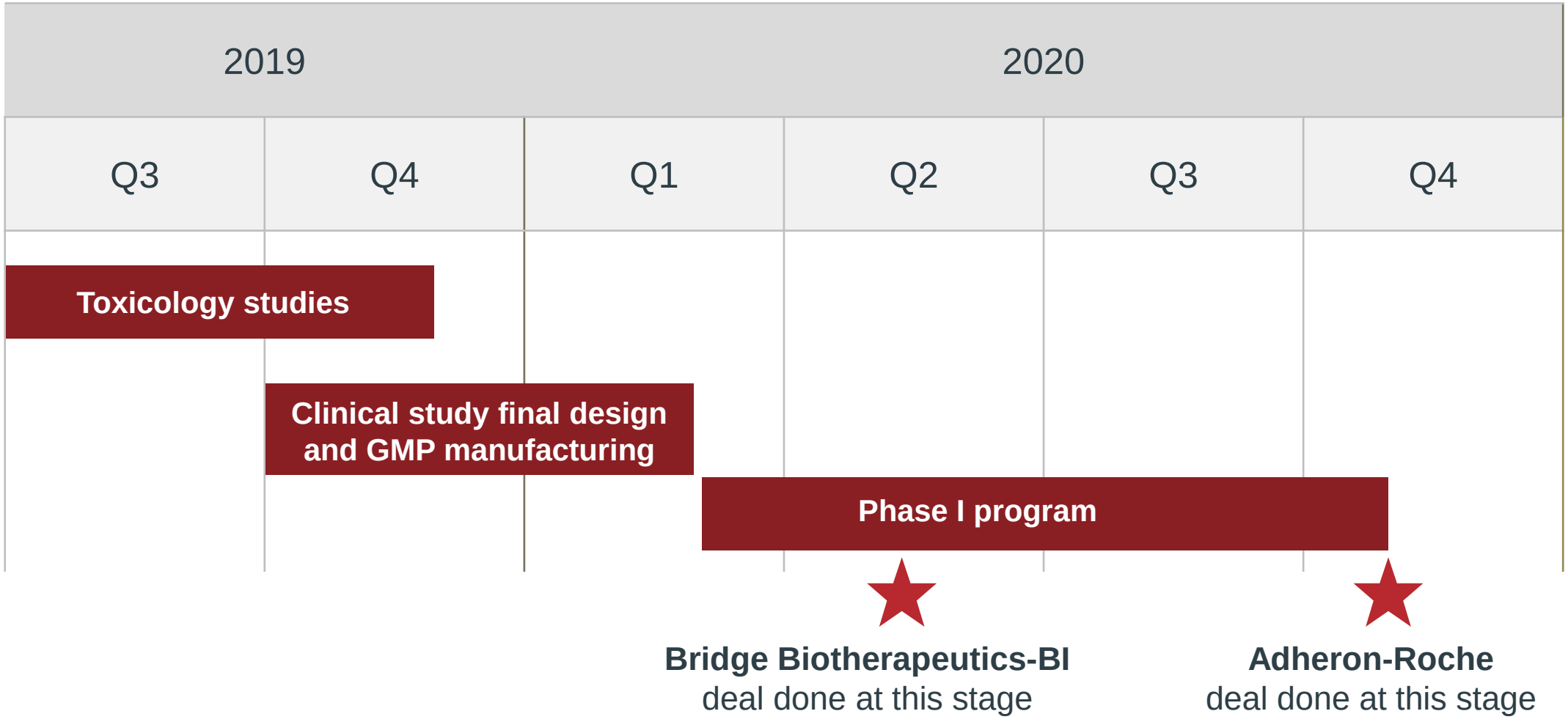
An i-body to CXCR4 has broad application in treating fibrosis

AdAlta data shows that an i-body to CXCR4 can improve fibrosis across a range of fibrotic diseases

- **LUNG:** Idiopathic Pulmonary Fibrosis
- **KIDNEY:** Chronic Kidney Disease
- **EYE:** Wet Age Related Macular Degeneration
- **LIVER:** NASH
- **SKIN:** Hypertrophic scar



AD-214 development: key milestones



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next generation protein therapeutics

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