ASX / Media Release

AdAlta to present at 2017 Bioshares Biotech Summit

MELBOURNE, Australia, 21 July 2017: AdAlta Limited (ASX: 1AD), the biotechnology company focused on advancing its lead i-body candidate towards clinical development today announced Chief Executive Officer Sam Cobb will be presenting at and attending the forthcoming Bioshares Biotech Summit conference in New Zealand to raise awareness with potential investors.

The Bioshares meeting will be held July 21-22 2017, in Queenstown, New Zealand, and attendees will hear from over 30 speakers, including analysts and biotech representatives who will discuss current biotech industry trends and the hottest areas of medical product development.

CEO Sam Cobb, will present data on AdAlta's lead candidate, AD-114, for the treatment of fibrosis and explain what sets the AD-114 technology apart from other drugs in market or being developed for the treatment of fibrosis.

Details of the presentation:

Session Title: Insights to Fibrosis Drug Discovery & Development

Session Details: This session will open with an overview of deal making and trends in the fibrosis drug space, which will be followed by presentations that explain what differentiates the approaches taken by four ASX-listed companies working in the field, concluding with a panel discussion.

Session Date/Time: July 21, 2017 from 1:10 PM to 4:15PM

A copy of the AdAlta presentation is attached with this cover note 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 focused on using its proprietary technology platform to generate i-bodies, a new class of protein therapeutics, with applications as therapeutic drugs to treat disease.

I-bodies are a promising, novel class of drugs that offer a new and more effective approach to treating a wide range of human diseases. They are identified and developed using our proprietary technology platform.

We have pioneered a technology that 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, now known as i-bodies, for use in treating serious diseases.

AdAlta is developing its lead i-body candidate, AD-114, 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 also plans to continue further drug discovery and development directed towards other drug targets and diseases with its i-body technology platform.

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

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AdAlta

next generation protein therapeutics

i-bodies – a new class of protein therapeutics to treat fibrosis

BIOSHARES JULY 2017

Sam Cobb, CEO and Managing Director

AdAlta Limited (ASX:1AD)

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About AdAlta

AdAlta is a drug discovery and development company using its powerful technology platform to generate a promising new class of protein therapeutic, known as i-bodies, for treating a wide range of human diseases.

Corporate and investment summary

Investment highlights

- Initial focus on treating fibrosis – high unmet medical need
- Advanced lead fibrosis drug candidate AD-114 significant pre-clinical validation in multiple models
- Fully funded for phase 1 development of lead fibrosis drug and i-body pipeline
- Orphan drug designation USA FDA
- Early commercialisation potential
- Experienced team with strong track record of drug development and ability to deliver

Capital structure	
ASX code	1AD
Shares on issue*	101,110,890
Share price (18 July 17)	AU$0.235
Market capitalisation	AU$23.5m
Escrowed shares (Aug 18)	24.1m
Current cash (30 March 17)	AU$7.47m
Trading range	AU$0.325 to $0.165
Average daily volume	51,354

Major Shareholders	%
Yuuwa Capital LP	53.5
Platinum Asset Management	7.91
Citycastle Pty Ltd	5.25
La Trobe University	3.01
Robin Beaumont	1.87
Other shareholders	28.46
Total	100%

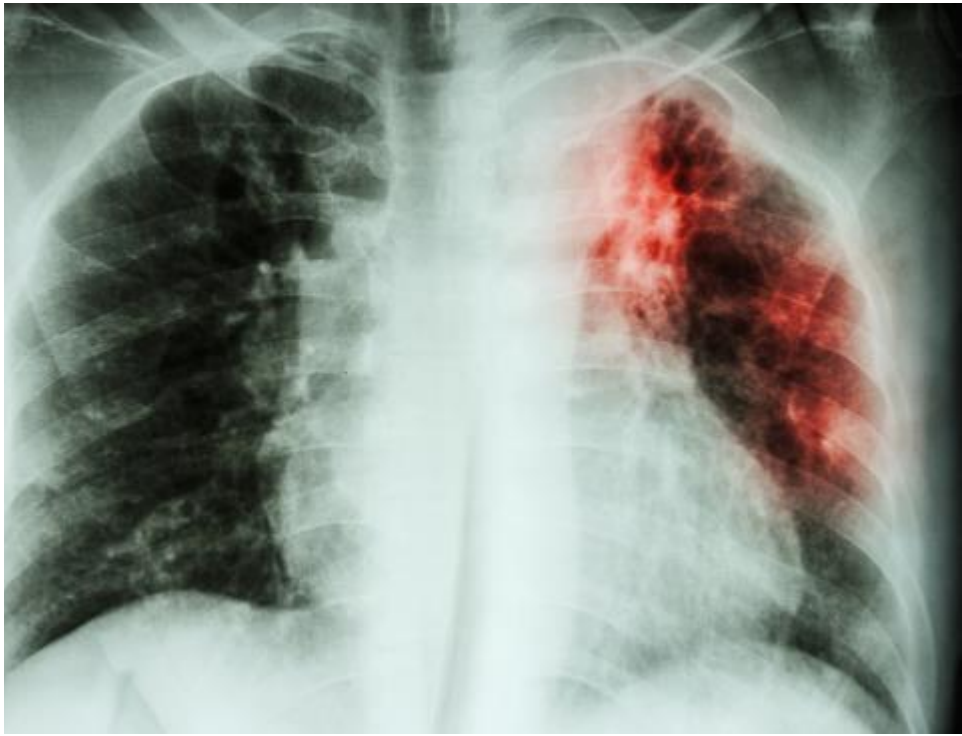
Fibrosis

A major problem

- Fibrosis can occur in many tissues of the body as a result of inflammation or damage
- As a result, collagen builds up and can result in scarring of vital organs such as the lung, liver, skin, eye, heart and kidney
- **Leads to irreparable damage and eventual organ failure**
- Fibrosis is prevalent in 45% of all diseases

There is no clinically satisfactory therapeutic approach to fibrosis

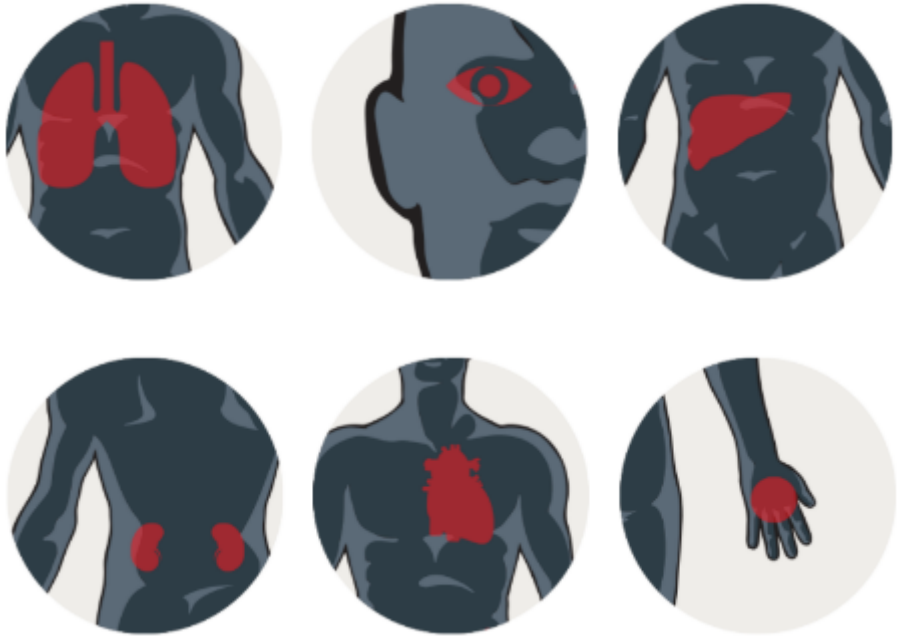
It is an area of high unmet need



AD-114 has broad application in treating fibrosis

AdAlta data shows that AD-114 can improve fibrosis across a range of fibrotic diseases

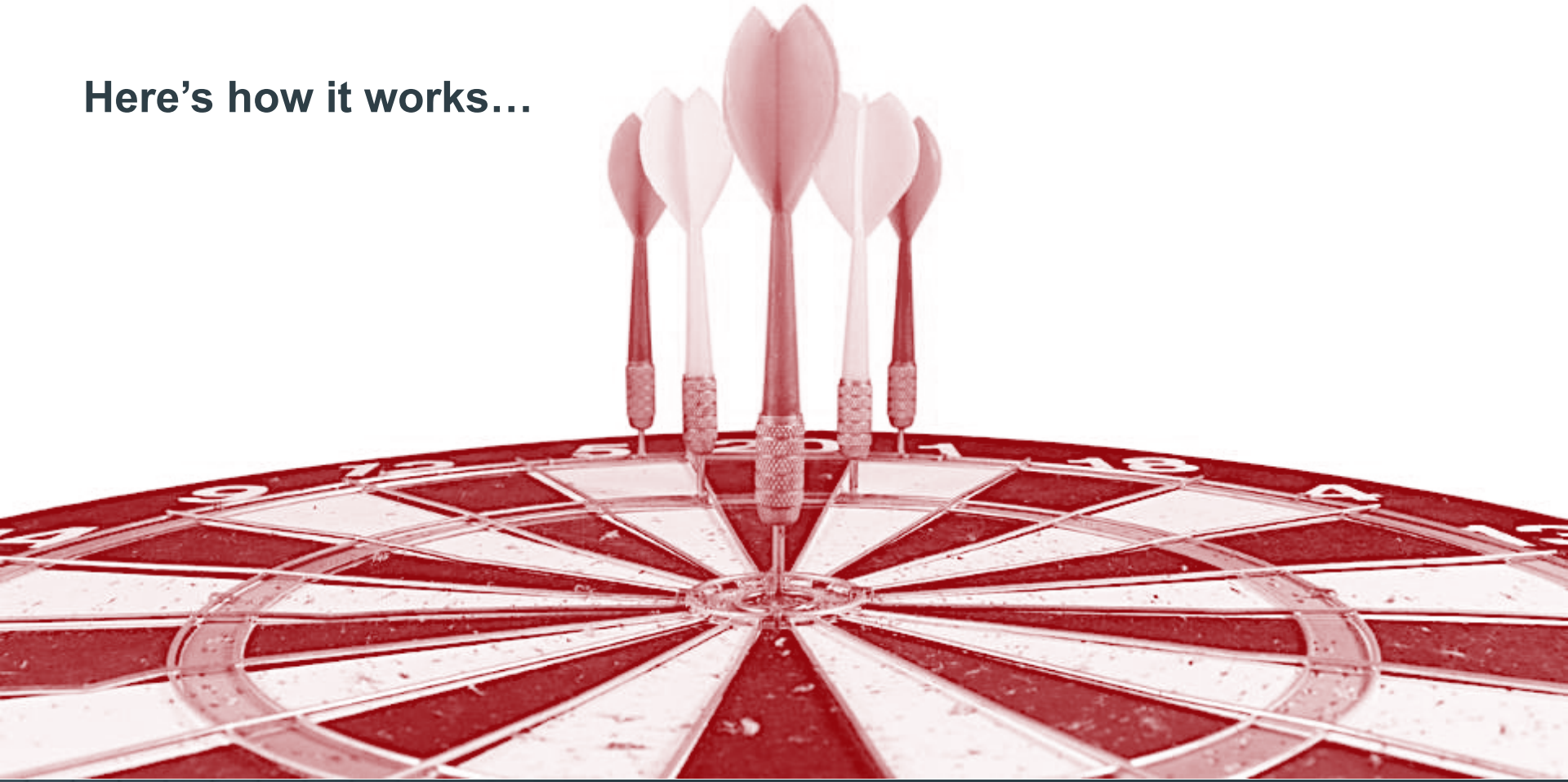
- **LUNG:** Idiopathic Pulmonary Fibrosis
- **EYE:** Wet Age Related Macular Degeneration
- **LIVER:** NASH
- **SKIN:** Hypertrophic scar
- **KIDNEY:** Chronic Kidney Disease



AD-114 has demonstrated broad anti-fibrotic and anti-inflammatory effects in several animal models of disease and with human tissues

AD-114 targets inflammation and fibrosis in multiple ways

Here's how it works…



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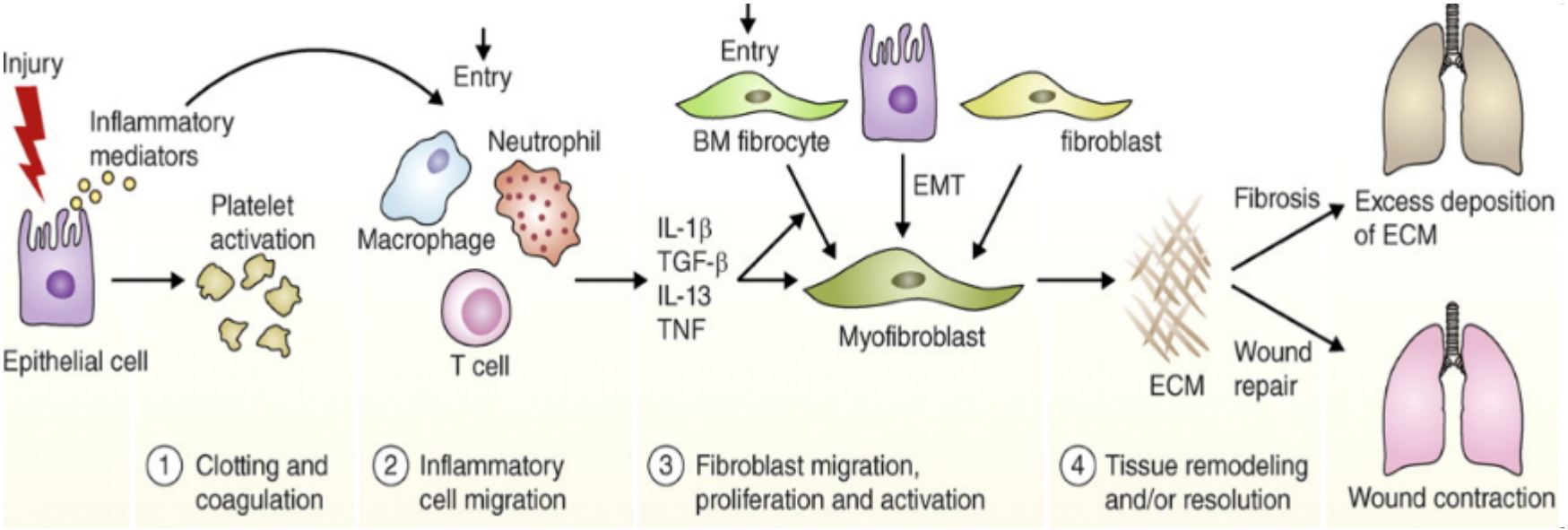
Multiple cell types and complex process



Source: J. Exp. Med. 2011, Vol. 208 No. 7 1339-1350

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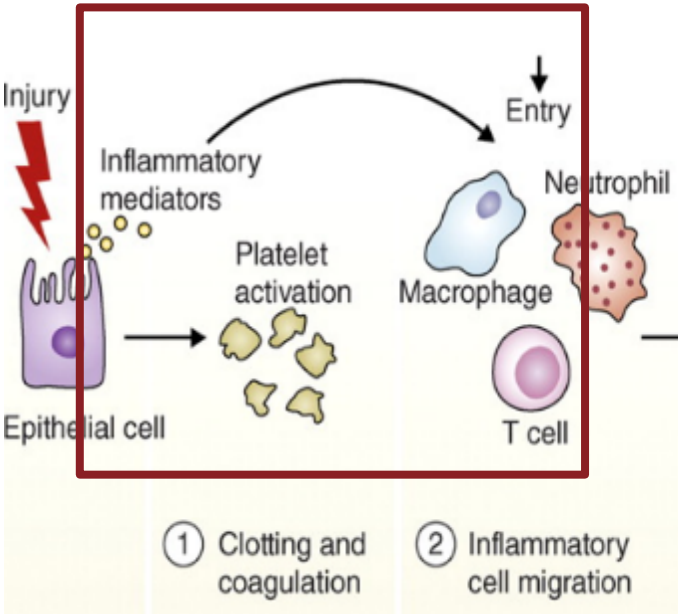
Multiple cell types – an explanation



In a number of animal models:

- ✓ AD-114 reduces inflammatory cell migration *in vitro* and *in vivo*
- ✓ AD-114 reduces anti-inflammatory effects in a number of animal models

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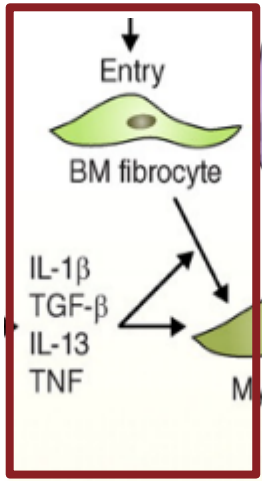
Multiple cell types – an explanation



③ Fibroblast migration, proliferation and activation

In damaged mouse lungs of fibrosis:

- ✓ AD-114 blocks fibrocyte recruitment

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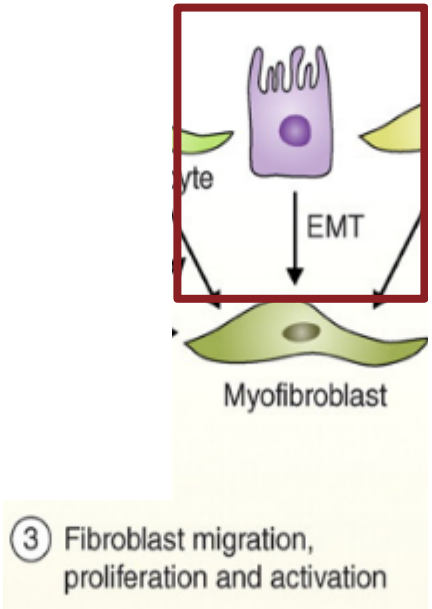
Multiple cell types – an explanation

In human lung and kidney tissue:

- ✓ AD-114 reduces epithelial–mesenchymal transition (EMT) *in vitro*



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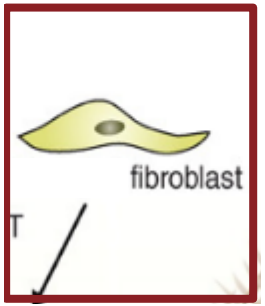
Multiple cell types – an explanation

In human diseased lung tissue:

- ✓ AD-114 reduces fibroblast migration



③ Fibroblast migration, proliferation and activation

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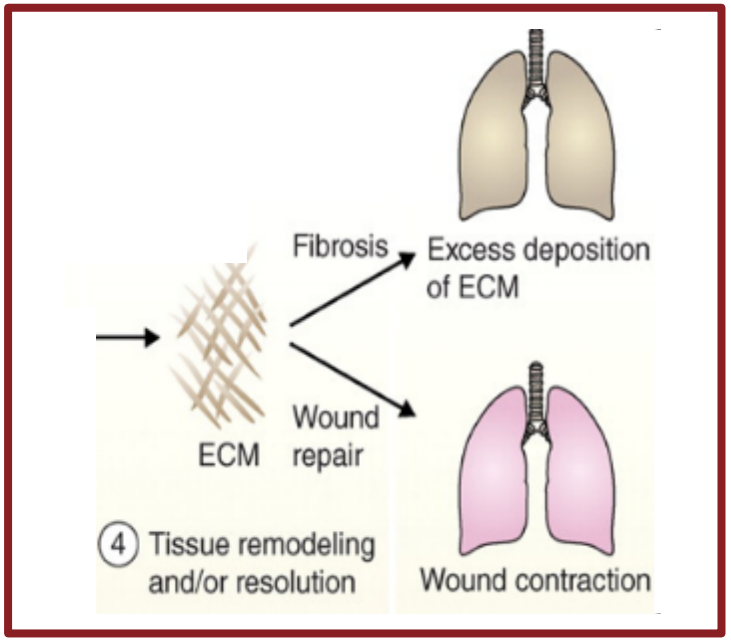
Multiple cell types – an explanation

In a number of fibrosis animal models and human tissues:

- ✓ AD-114 reduces collagen and extracellular matrix (ECM) deposition



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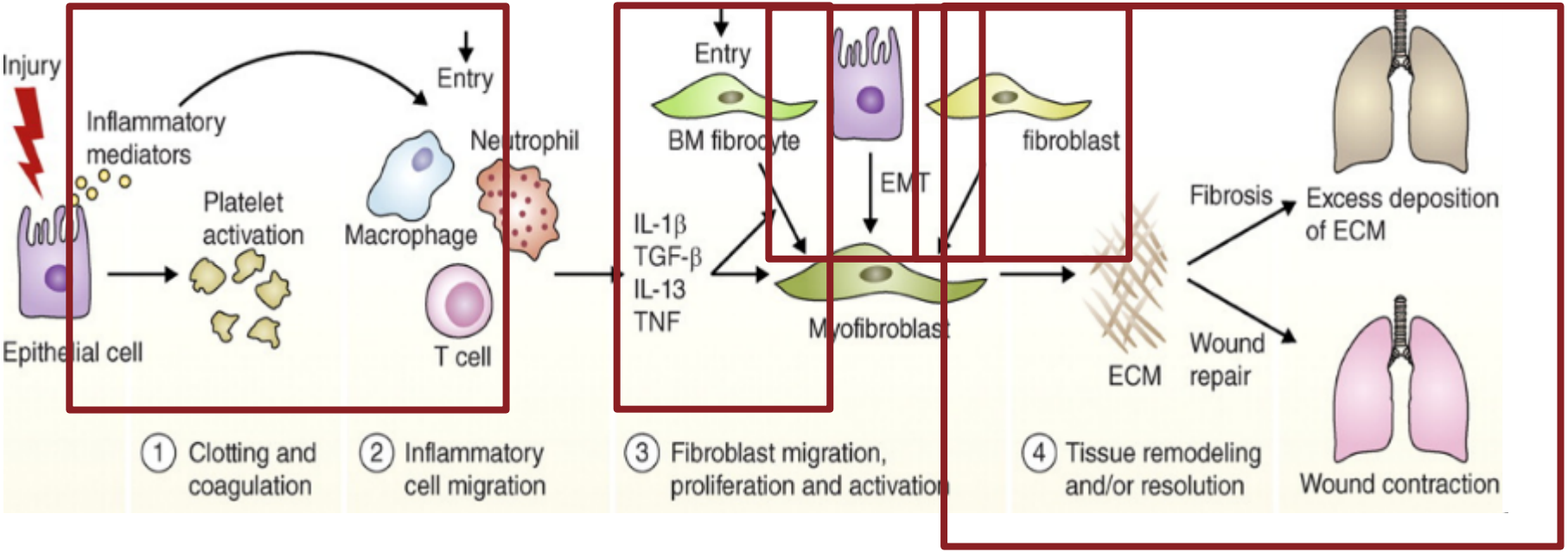
Multiple cell types and complex process



Source: J. Exp. Med. 2011, Vol. 208 No. 7 1339-1350

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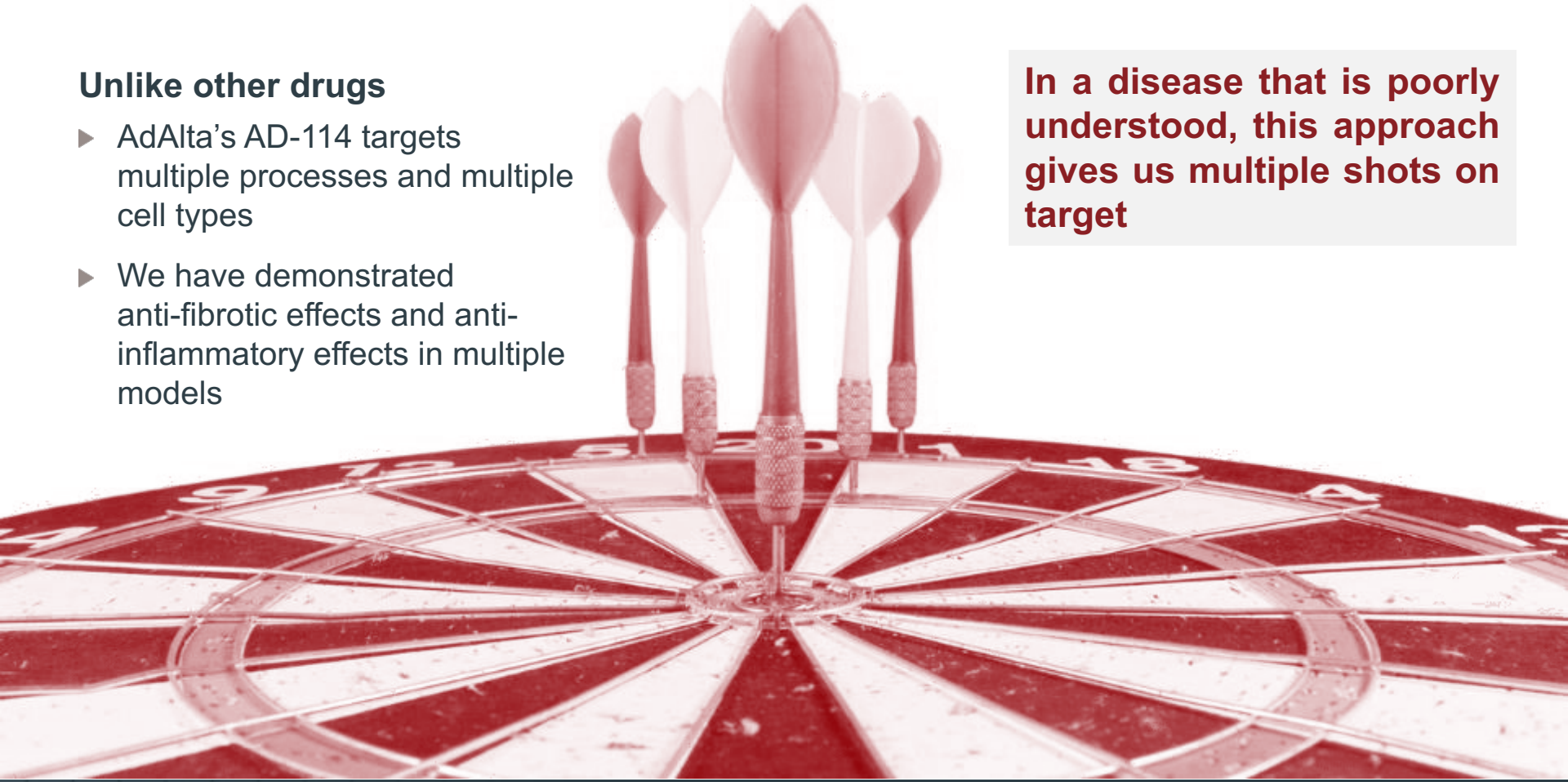
In summary: one drug, broad anti-fibrotic action

Unlike other drugs

- AdAlta's AD-114 targets multiple processes and multiple cell types
- We have demonstrated anti-fibrotic effects and anti-inflammatory effects in multiple models



In a disease that is poorly understood, this approach gives us multiple shots on target

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AD-114 binds to CXCR4

- CXCR4 is a GPCR chemokine receptor present on bone marrow derived cells including T cells, B cells and leukocytes (monocytes) and blood cells
- CXCR4 is low or absent in healthy tissues but shown to be expressed in over twenty three types of cancer, including breast, ovarian, melanoma, and prostate cancer

CXCR4 has also been shown to be involved in fibrosis = AdAlta's focus

Evidence supporting CXCR4 as a fibrosis target

Significant literature to support hypothesis of the involvement of CXCR4 in fibrosis

The SDF-1/CXCR4 ligand/receptor pair is an important contributor to several types of ocular neovascularization

Raquel Lima e Silva,* Jikui Shen,* Sean F. Hackett,* Shu Kachi,* Hideo Akiyama,* Katsuji Kiuchi,* Katsutoshi Yokoi,* Maria C. Hatara,* Thomas Lauer,* Sadia Aslam,* Yuan Yuan Gong,* Wei-Hong Xiao,* Naw Htee Khu,* Catherine Thut,† and Peter A. Campochiaro*,1

A potential role of SDF-1/CXCR4 chemotactic pathway in wound healing and hypertrophic scar formation

Leah Campeau[1], Jie Ding[1], Edward E Tredget[1,2]

CXCR4 dysfunction in non-alcoholic steatohepatitis in mice and patients

Hédia Boujedidi*†1, Olivier Robert*†1, Alexandre Bignon*††, Anne-Marie Cassard-Doulcier*†, Marie-Laure Renoud†, Hélène Gary-Gouy†, Patrice Hemon†, Hugo Tharinger†, Sophie Prévot*§, Françoise Bachelerie*††, Sylvie Naveau*†‖, Dominique Emilie*††¶2, Karl Balabanian*†† and Gabriel Perlemuter*†‖

CXCR4 Antagonism Attenuates the Development of Diabetic Cardiac Fibrosis

Po-Yin Chu[1], Ken Walder[2], Duncan Horlock[1], David Williams[1], Erin Nelson[1], Melissa Byrne[1], Karin Jandeleit-Dahm[3], Paul Zimmet[4], David M. Kaye[1]*

Chemokine receptor Cxcr4 contributes to kidney fibrosis via multiple effectors

Amy Yuan,[3] Yashang Lee,[3] Uimook Choi,[1] Gilbert Moeckel,[2] and Anil Karihaloo[3]

[1]Laboratory of Host Defense, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland; [2]Department of Pathology, Yale School of Medicine, New Haven, Connecticut; and [3]Department of Medicine, Section of Nephrology, Yale School of Medicine, New Haven, Connecticut

Submitted 12 March 2014; accepted in final form 19 December 2014

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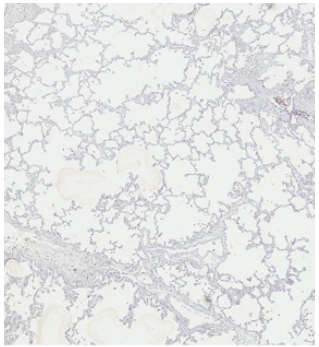
CXCR4 increased in disease

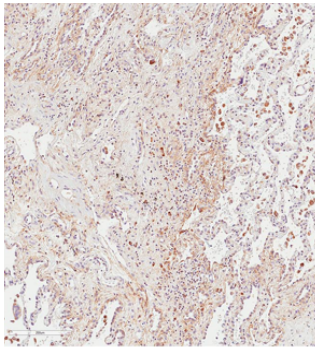
- Demonstrated CXCR4 expression increased in a number of human fibrotic diseased tissue (represented by brown staining)
- For example:
  - IPF diseased lung tissue in upper right panel compared with normal lung tissue
  - Diabetic diseased kidney tissue in lower right panel - compared with normal kidney tissue

In each of the diseased tissue examples, CXCR4 is shown to be increased

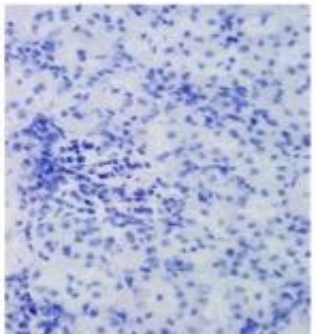
Normal human lung tissue



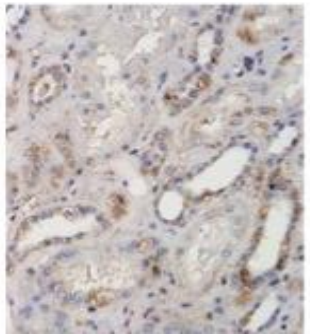
Diseased lung tissue



Normal human kidney tissue



Diseased kidney tissue



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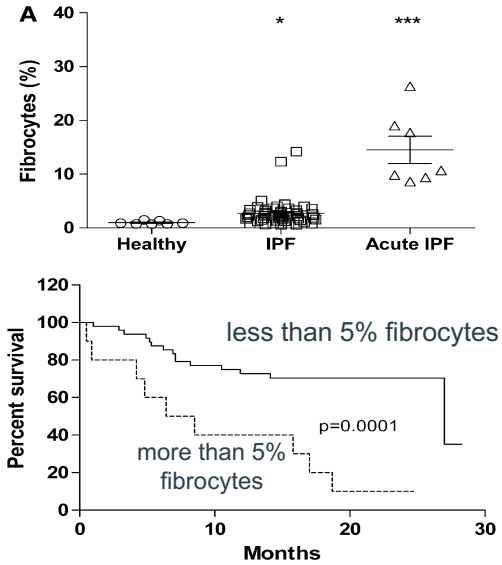
CXCR4 as a biomarker in IPF

- Fibrocyte cells (CXCR4 positive cells) were elevated in stable IPF patients, and further increased during acute exacerbations
- Fibrocytes (CXCR4 positive cells) not only correlated with lung function but were an ***independent predictor of early IPF patient mortality***
  - 7.5 months with more than 5% fibrocytes
  - 27 months with less than 5% fibrocytes

CXCR4 may have a role in predicting disease progression

Fibrocyte numbers predict mortality



REF: Moeller, et al. Am J Respir Crit Care Med Vol 179. pp 588–594, 2009

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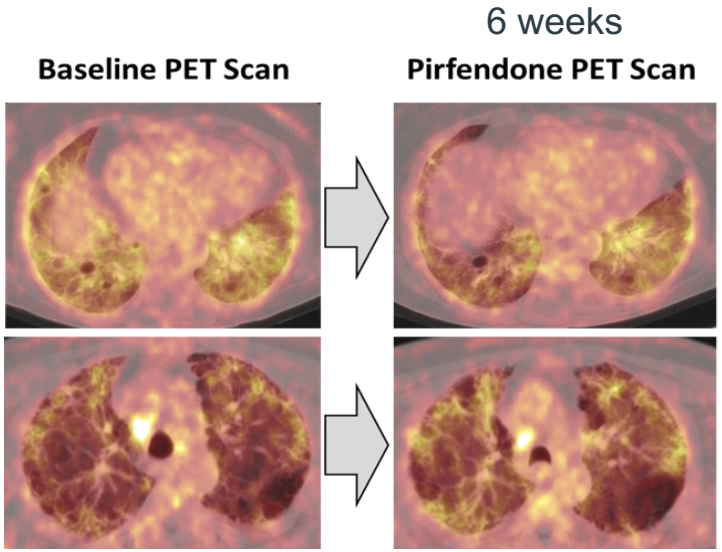
CXCR4 as a biomarker in IPF

- Strong CXCR4 expression from PET imaging agent, correlated with areas of honeycombing (associated with IPF) and with clinical parameters known to be predictive of outcome in IPF
  - Patient A (top panels) had lower expression of CXCR4 at 6 weeks and responded to Pirfenidone treatment with lung function improvement
  - Patient B (bottom panels) had a high expression of CXCR4 at 6 weeks and did not respond to Pirfenidone treatment, with no lung function improvement



REF: Prasse A, et al. American Journal of Respiratory and Critical Care Medicine 2017;195:A7678

CXCR4 imaging may have a role in monitoring disease progression and may predict response to treatment with Pirfenidone

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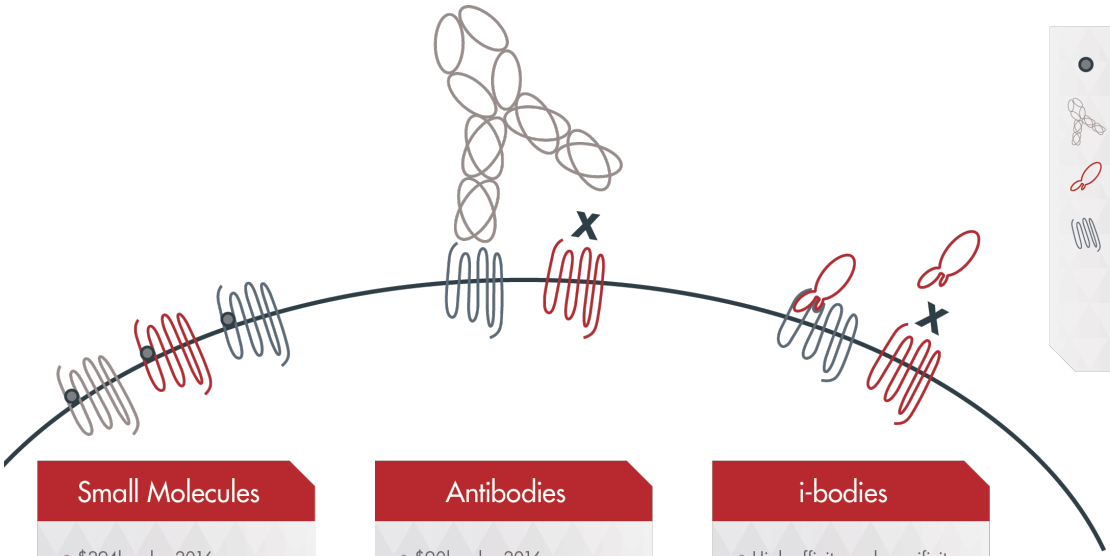
AD-114 safe in non-human primates

- **PK determined both subcutaneously and intravenously**
  - T ½ ~24hrs either route of administration
- **Ascending dose range finder study up to 30mg/kg**
  - AD-114 was well tolerated and there were no adverse effects with increased doses demonstrated by hematology / blood evaluation readouts
  - No study mortalities or clinical signs relating to the increasing doses of AD-114 were observed
  - Importantly, unlike other CXCR4 antagonists, AD-114 does not mobilise stem cells
    - This result is a potential advantage of AD-114 for long term treatment in diseases such as fibrosis
    - This data demonstrates that the long loop of the i-body has a unique activity and AD-114 is differentiated from competing CXCR4 antagonist products

Long loop of i-body: novel CXCR4 mechanism

- High affinity and specificity like an antibody with lower toxicity expected (does not bind to any other GPCR) unlike CXCR4 small molecule AMD3100
- Long loop binds deep in groove like a small molecule
- Combining advantages of antibodies and small molecules, AD-114 has unique pharmacology



Small Molecules
- $294b sales 2016
- Can have poor side effects due to lack of specificity
- Due to their small size, small molecules can bind difficult to access targets, such as G-protein coupled receptors (GPCRs)

Antibodies
- $90b sales 2016
- High affinity to target, low toxicity due to specificity
- Due to antibodies being large in size, binding only occurs on the outside of the GPCR

i-bodies
- High affinity and specificity of antibodies
- Due to long loop can bind difficult to access targets with unique pharmacology like a small molecule

Source: Global Data

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AD-114 key advantages compared to existing IPF treatments

Human tissue *In vitro* activity	No effect on normal tissue	Effect on diseased / IPF tissue
i-body AD-114	✔	✔
Nintedanib (Boehringer)	✗	✔
Pirfenidone (Roche)	✔	✗
Other CXCR4 drug (Sanofi-AMD3100)	✔	✗

- AD-114 has greater *in vitro* efficacy compared to the only approved therapies Nintedanib and Pirfenidone for IPF treatment
  - Existing IPF treatments have limited efficacy; either no effect or slow down disease progression i.e. no cure
- Novel mechanism of action compared with other drugs targeting the GPCR chemokine receptor CXCR4
- Very specific for diseased tissue and no effects on normal tissue

Novel mechanism of action for fibrosis treatment enabling a "first in class" therapy

Orphan drug status with US FDA allows for R&D tax credits, new drug application fee waivers and a seven year period of market exclusivity

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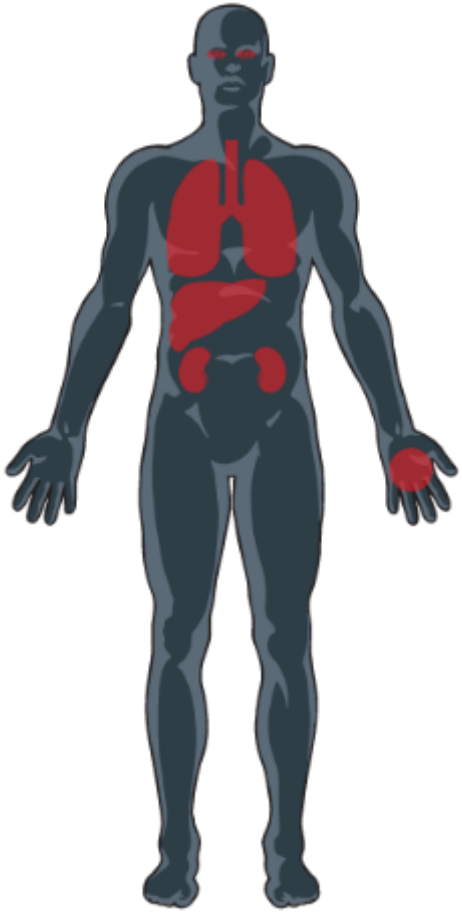
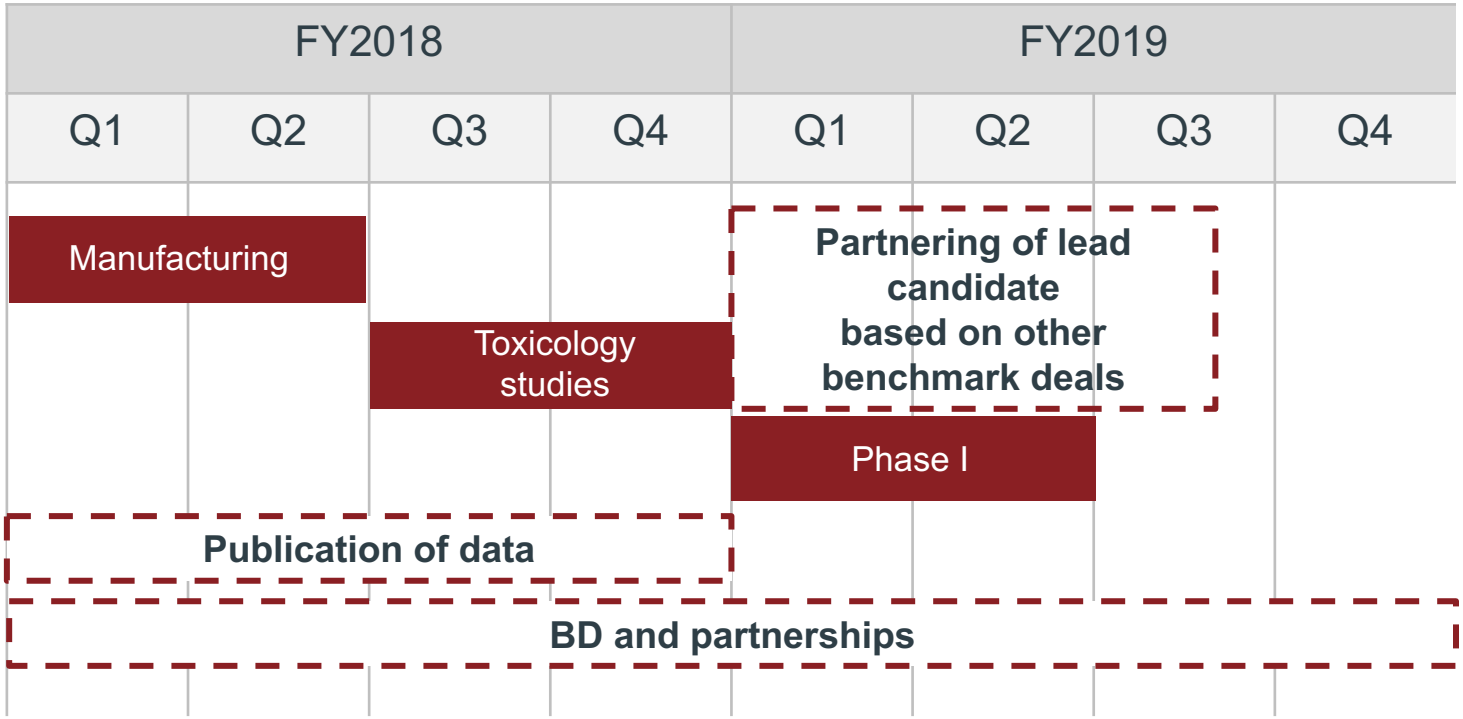
AD-114 efficacy and safety

- **Efficacy**
  - Lung: IPF
    - Animal models
    - Human IPF tissue
    - Biomarker assessments (Alfred Health & others)
  - Broad fibrotic application with demonstration in other animal models and human tissues
    - Eye: wet-AMD
    - Liver: NASH
    - Kidney: CKD
    - Skin: HT Scarring



- **Safety**
  - NHP studies:
    - PK: IV and SC
    - Dose range finder
    - Multi dosing studies
  - PK-PD assays developed demonstrating target engagement
  - Cytokine analysis (20 human blood donors)

AD-114 development: key milestones



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AdAlta / AD-114 summary

- IPO August 2016 raised $10M to meet major milestones: phase I clinical trials of AD-114 in lung fibrosis and development of i-body pipeline
- Initial focus on treating Idiopathic Pulmonary Fibrosis (IPF) and other fibrotic diseases - high unmet clinical need
- CXCR4 is a novel target for treatment of fibrosis, increased in disease
- AD-114 has significant pre-clinical validation demonstrating broad anti-fibrotic and anti-inflammatory effects as well as safety

Early commercialisation opportunity, with experienced management and Board to drive AD-114 development and secure technology platform partnerships / product licensing deals