ASX Announcement

AdAlta to present at the American Society of Nephrology (ASN) annual kidney week in New Orleans

MELBOURNE Australia, 31 October, 2017: AdAlta Limited (ASX:1AD), the biotechnology company advancing its lead i-body candidate towards clinical development, announces that Professor Carol Pollock from the University of Sydney has been invited to speak at the American Society of Nephrology's (ASN) Kidney Week in New Orleans, LA between 31 October and 5 November, 2017, with regard to AdAlta's lead candidate AD-114 and its potential for use as a treatment for Chronic Kidney Disease, another fibrotic condition.

ASN Kidney Week is the world's premier nephrology meeting, attended by more than 13,000 kidney professionals from across the globe.

AdAlta's CEO, Sam Cobb commented, "An increase in diabetes and obesity across the world is leading to a massive surge in the number of people diagnosed with Chronic Kidney Disease. The medical community is looking for alternative treatment options, so this early work with AD-114 is encouraging.

The research collaboration with the Kolling Institute and the University of Sydney further informs our pre-clinical package and shows that AD-114 works across a range of fibrotic disease areas. We know this data helps speak to the value of AD-114 and will be important to potential pharmaceutical partners."

Details of the presentation

Session date / time: Thursday 2 November between 10:00 am and 12:00 pm
Session title: 305-PO01 CKD: Clinical Trials and Tubulointerstitial Disorders

The session presentation entitled "*A novel i-body AD-114 supressed TGFß1 fibronectin and collagen 4 in renal proximal tubular cells via Smad and p38 pathways"* follows this cover page and will also be made available on the corporate 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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KOLLING INSTITUTE
Discovery *into* Reality™

A NOVEL PROTEIN I-BODY (AD-114) SUPPRESSED TGFβ1-INDUCED FIBRONECTIN AND COLLAGEN 4 IN RENAL PROXIMAL TUBULAR CELLS VIA SMAD AND P38 PATHWAYS

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Background:

- **Fibrosis** is the final common pathway of chronic kidney disease (CKD).
- **TGF-β** is the master regulator of renal fibrosis.
- **CXCR4** (Chemokine receptor type 4) has been demonstrated to be a central player in the development of tissue fibrosis.
- **Adalta Ltd** has developed a fully human single-domain antibody-like scaffold termed i-body AD-114 with specific high binding affinity to CXCR4.
- **AD-114** selectively blocks CXCR4 signaling and has shown anti-fibrotic effects in lung, liver and eye fibrosis.
- **The role of AD-114 in renal fibrosis** has not been studied.

Results:

1. CXCR4 is significantly upregulated in fibrotic kidneys of animal models and human tissue

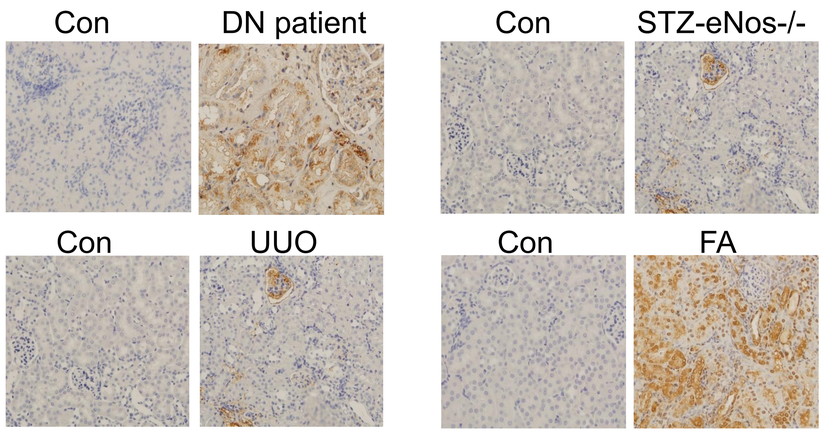

Fig 1: CXCR4 expression is examined in kidney biopsies from DN patients, STZ-eNos -/- DN mice, UUO- mice and FA-mice by immunohistochemistry (20x). DN: Diabetic nephropathy; STZ: Streptozotocin; UUO: Unilateral ureteral obstruction; FA: Folic acid.

2. AD-114 binds TGFβ-induced CXCR4 in human renal PTCs

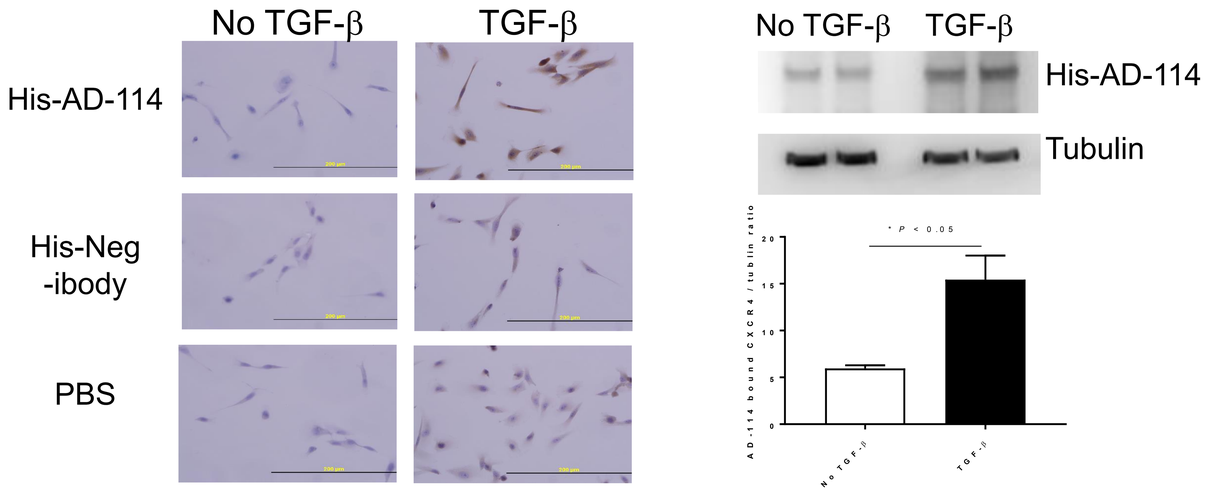

Fig 2: PTCs were incubated with/without TGFβ (2ng/ml) for 48 hours. Cells were grown on glass slides and analysed by immunocytochemistry. Cell lysates were collected for Western blot analysis (n=4). PTCs: Proximal tubular cells.

3. AD-114 inhibits secretory Fibronectin(FN) and Collagen-4 (Col-4) in Human PTCs

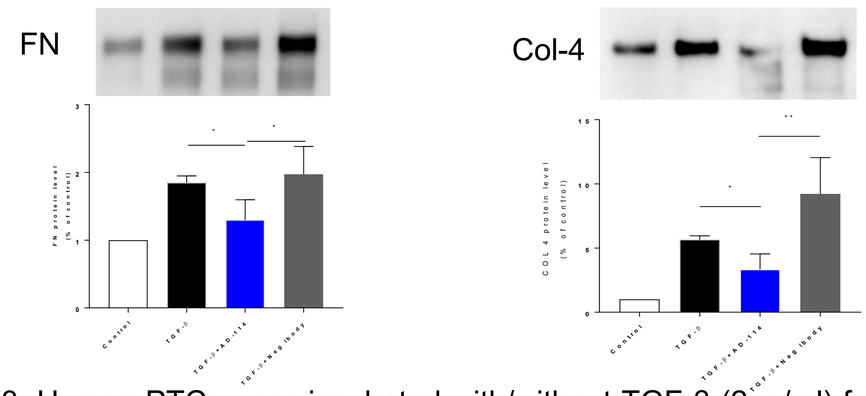

Fig 3: Human PTCs were incubated with/without TGF-β (2ng/ml) for 48 hours in absence or presence of AD-114 (3μM) for 48 hours and the supernatant was collected for Western blot analysis. *P<0.05, **P<0.01, n=4.

4. AD-114 inhibits phosphorylation of Smad2/3 and P38

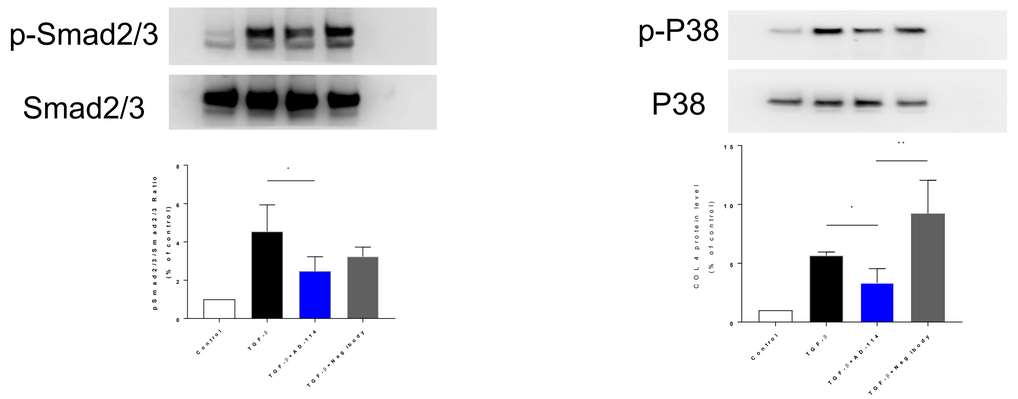

Fig 4: Human PTCs were incubated with/without TGF-β (2ng/ml) for 48 hours in absence or presence of AD-114 (3μM) for 48 hours and the cell lysis protein was collected for Western blot analysis. *P<0.05, **P<0.01, n=4.

Conclusions: Blocking CXCR4 using i-body AD-114 may be a promising therapeutic strategy for renal fibrosis.

THE UNIVERSITY OF SYDNEY
Royal North Shore Hospital
KIDNEYWEEK 2017 New Orleans, LA • Oct 31 - Nov 5
NSW GOVERNMENT Health Northern Sydney Local Health District
AdAlta next generation protein therapeutics