AdAlta
next generation protein therapeutics

ASX Announcement

AdAlta Announces Positive Pre-clinical Data Showing Lead Drug Candidate Has Broad Fibrosis Treatment Potential

MELBOURNE Australia, 9 January, 2017 - AdAlta Limited (ASX: 1AD), the biotechnology company advancing its lead i-body candidate towards clinical development today announced additional positive pre-clinical data for its lead drug candidate AD-114, a novel first-in-class drug candidate being developed to treat fibrosis, a condition prevalent in 45 to 50 per cent of all diseases.

The new data show the administration of AD-114 reduced fibrosis in therapeutic mouse models of both lung and liver fibrosis. In the mouse model of lung fibrosis, collagen deposition and collagen gene expression were both reduced in the lungs, after 14-days of i-body AD-114 treatment. Excessive collagen stiffens tissue, causing fibrosis. In the mouse model of liver fibrosis, serum alanine aminotransferase (ALT) levels and the non-alcoholic fatty liver disease (NAFLD) score were improved with the treatment of AD-114 for 21 days. Concentration of ALT in plasma is a biomarker for liver disease progression: the improvement in serum liver enzyme ALT levels suggests that the i-body ameliorated liver injury and inflammation. The NAFLD score improvement indicates AD-114 possesses hepatoprotective effects and therefore has potential for the treatment of non-alcoholic steatohepatitis (NASH).

Extensive pre-clinical AD-114 studies have demonstrated positive *in vitro* (in the lab) and *in vivo* (in animals) data. These new data validates a number of earlier studies and AdAlta has now demonstrated anti-fibrotic activity of AD-114 in animal models of lung, liver and eye fibrosis.

AdAlta Chief Executive Officer Sam Cobb said, "We are encouraged by these new pre-clinical data which supports the broad anti-fibrotic activity of AD-114 and the therapeutic use of AD-114 in the treatment of idiopathic pulmonary fibrosis and more broadly in other fibrosis diseases including liver fibrosis or NASH.

“AdAlta remains on track to meet its stated clinical development milestones and management remains focused on expediting AD-114 into Phase 1 human clinical trials for

idiopathic pulmonary fibrosis by early 2018. Our strategy is to license this drug candidate on completion of the planned Phase 1 clinical studies."

There is growing global interest in promising new treatments for fibrosis, especially for idiopathic pulmonary fibrosis, which has a 50 to 70 per cent mortality rate and affects 135,000 people in the US every year.

The data in the lung fibrosis mouse model will be used to support AdAlta's application for orphan drug status for AD-114 for the treatment of idiopathic pulmonary fibrosis (IPF). Orphan Drug status allows for significant R&D tax credits, new drug application fee waivers and a seven year period of market exclusivity from the US Food & Drug Administration (FDA) after approval. Companies pursuing orphan treatments are usually granted accelerated development and regulatory timelines as well as premium pricing.

AdAlta Chief Scientific Officer Dr. Mick Foley, said, "The response and data observed with AD-114 is important for arresting and modulating a number of fibrosis related diseases and addressing the treatment of fibrotic conditions, including those of the lung and liver.

"While idiopathic pulmonary fibrosis (IPF) is the primary indication for AD-114, these data demonstrate the broad applicability of AD-114 for the treatment of multiple fibrosis related diseases including cirrhosis of the liver."

"We believe the new data of AD-114 in animal models of lung and liver fibrosis add to a growing body of evidence to support AdAlta's application to begin a Phase I clinical study in humans aimed at validating its promise in treating fibrosis, notably IPF and other fibrotic diseases, for which current therapies have limited efficacy and where there is a high-unmet medical need.

Full details of these new studies are available on the AdAlta website ***www.adalta.com.au.*** The new data will be presented by AdAlta CEO Sam Cobb, along with other pre-clinical data on AD-114, at the Biotech Showcase 2017 conference in San Francisco at 9am on 11 January 2017 (Hilton Hotel - Room 3, Ballroom Level, Union Square, 333 O'Farrell Street, San Fran, CA 94102).

Notes to editors

About AdAlta Limited

AdAlta Limited (ASX:1AD) 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 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. AD-114 has strong pre-clinical results for IPF, demonstrating both anti-fibrotic and anti-inflammatory activity in human lung tissue and indicating greater efficacy than existing approved IPF drugs.

The i-body is a human analogue of the antigen binding domain of the shark antibody, which combines the advantages of monoclonal antibodies (high target specificity and affinity) with the beneficial stability features of small molecules. In addition to stability, the i-body has a long binding loop that is a feature of shark antibodies not present in either human or next generation antibodies. This feature enables the i-body to recognise and bind to a diverse range of different therapeutically-relevant drug targets, including those that are difficult/intractable to access by current antibody therapies. These include clinically important targets such as G-protein coupled receptors (GPCRs) and ion channels.

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*

Additional AD-114 lung fibrosis data

In a therapeutic mouse model of Bleomycin, a standard animal disease model used for the assessment of drug candidates for the treatment of pulmonary fibrosis, the administration of AD-114 markedly reduced the extent of collagen deposition in the lungs, as measured by Ashcroft score and hydroxyproline assay. Additionally, a reduction in collagen (both Col1A and Col3A) gene expression was also noted after 14-days of i-body treatment. Excessive collagen stiffens tissue, causing fibrosis. These additional pre-clinical data supports the anti-fibrotic activity of AD-114 and support the therapeutic use of AD-114 in the treatment of IPF.

This additional data will be used to support AdAlta's application for orphan drug status for AD-114 for the treatment of idiopathic pulmonary fibrosis (IPF). IPF is a chronic, lethal and

rare disease with a 50 to 70 per cent mortality rate. It currently affects at least 135,000 people in the US annually and a high-unmet medical need. Orphan Drug status allows for large R&D tax credits, NDA fee waivers, and seven years' market exclusivity from the US FDA after approval. Companies pursuing orphan treatments are usually granted accelerated development and regulatory timelines as well as premium pricing.

AD-114 liver fibrosis data

AD-114 decreased serum alanine aminotransferase (ALT) levels to 43U/L from 64U/L in the vehicle, a 33% reduction. The improvement in serum ALT levels suggests that i-body ameliorated hepatocellular injury and inflammation. Concentration of liver enzyme (ALT) in the plasma is a biomarker for liver disease progression. AD-114 also reduced the non-alcoholic fatty liver disease (NAFLD) score compared with the vehicle or diseased group. Hepatocyte ballooning was also significantly decreased compared with the vehicle or diseased group. AD-114 possesses hepatoprotective and anti-NASH effects.

Nonalcoholic steatohepatitis (NASH) is liver inflammation and damage caused by a buildup of fat in the liver, inflammation and fibrosis. NASH can get worse and the progressive fibrosis or scarring of the liver leads to cirrhosis. NASH affects 70-90% of obese or diabetic populations, and overall, NASH affects 3-5% of the US population, for which there are no treatments currently approved.

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