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AMPLIA'S FAK INHIBITORS REDUCE FIBROSIS IN ANIMAL MODEL OF NASH

- AMP945 produces a statistically significant reduction in liver fibrosis
- Further evidence of broad-spectrum antifibrotic action of Amplia's FAK inhibitors
- New data is expected to support Amplia's future development and partnering programs

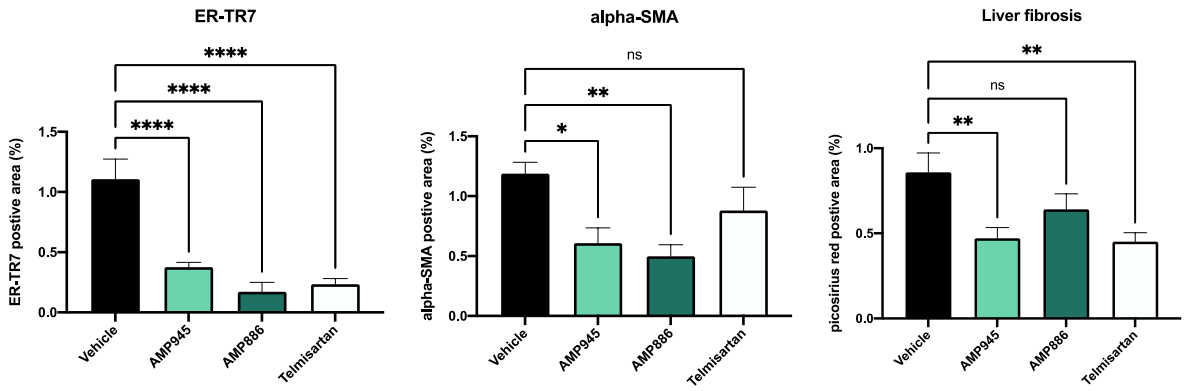
Melbourne, Australia: Amplia Therapeutics Limited (ASX: ATX), ("Amplia" or the "Company"), a company developing new drugs for the treatment of cancer and fibrosis, is pleased to announce it has received data from a successful pre-clinical study showing that both of Amplia's proprietary Focal Adhesion Kinase (FAK) inhibitors, AMP886 and AMP945, reduce fibrosis arising from the liver disease, Non-Alcoholic Steatohepatitis (NASH). These data highlight the key role that FAK plays in the underlying pathophysiology of multiple fibrotic diseases and is expected to open up new development and partnering opportunities for the Company in the future.

NASH occurs when an accumulation of fat in the liver (called Non-Alcoholic Fatty Liver Disease, or NAFLD) causes inflammation. This inflammation eventually leads to the build-up of fibrotic scar tissue throughout the liver that can then lead to cirrhosis, and then primary liver cancer (HCC, or hepatocellular carcinoma). It is estimated that approximately 5% of adults in the United States have NASH. However, despite this significant unmet need, attempts to develop an effective therapeutic for NASH have met with little success to date.

Both Amplia's FAK inhibitors, AMP886 and AMP945, were tested by SMC Laboratories Inc ("SMC", Tokyo, Japan) using their proprietary STAM™ mouse model for NASH. This model replicates the progression from fatty liver, to fibrotic liver, and then to liver cancer, and is considered to be the animal disease model that best recapitulates the pathological attributes of NASH. Consequently, the STAM™ model has been used by a number of companies trialling new therapeutic approaches to treat NASH.

The Figure below shows that AMP945 delivered a statistically significant reduction in liver fibrosis. In addition, both AMP886 and AMP945 produced a significant reduction in ER-TR7 and Alpha-SMA, two key activators of liver fibrosis. Mice were treated with a negative control (vehicle), AMP886 or AMP945, or a positive control drug called Telmisartan. ER-TR7 is a marker for fibroblasts which are the cells that lay down collagen in fibrotic tissue. Alpha-SMA is a marker for the activation of fibroblasts.

Figure. Amplia's FAK Inhibitors Reduce Fibrosis in a NASH Model



These data further support Amplia's view that FAK plays a critical role in the pathology of many fibrotic diseases. The Company has now demonstrated anti-fibrotic activity for its FAK inhibitors in animal models of lung fibrosis and NASH and in a range of *in vitro* studies. On the back of these positive data, Amplia intends to conduct further exploratory, non-clinical studies evaluating the use of AMP886 and AMP945 as potential treatments for liver fibrosis and other fibrotic diseases. Data from these studies are expected to support Amplia's future development and partnering programs.

"It is very encouraging to see such compelling data from using AMP886 and AMP945 in an industry-recognised model of NASH. We look forward to further exploring these promising findings as we continue to build the dossier of non-clinical data for our FAK inhibitors." said John Lambert, CEO of Amplia. "Having completed dosing in our Phase 1 trial of AMP945 this month, our primary focus remains on progressing AMP945 into Phase 2 clinical trials for pancreatic cancer and lung fibrosis. However, in parallel, as we have communicated previously, we will continue to expand our data set looking at other potential commercial opportunities."

This ASX announcement was approved and authorised for release by the Board of Amplia Therapeutics.

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About Amplia Therapeutics Limited
Amplia Therapeutics Limited is an Australian pharmaceutical company advancing a pipeline of Focal Adhesion Kinase (FAK) inhibitors for cancer and fibrosis. FAK is an increasingly important target in the field of cancer immunology and Amplia has a particular development focus in pancreatic and ovarian cancer. FAK also plays a significant role in a number of chronic diseases, such as idiopathic pulmonary fibrosis (IPF).