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ASX RELEASE **3 October 2022**

AMP886 Activity in Acute Myeloid Leukemia (AML)

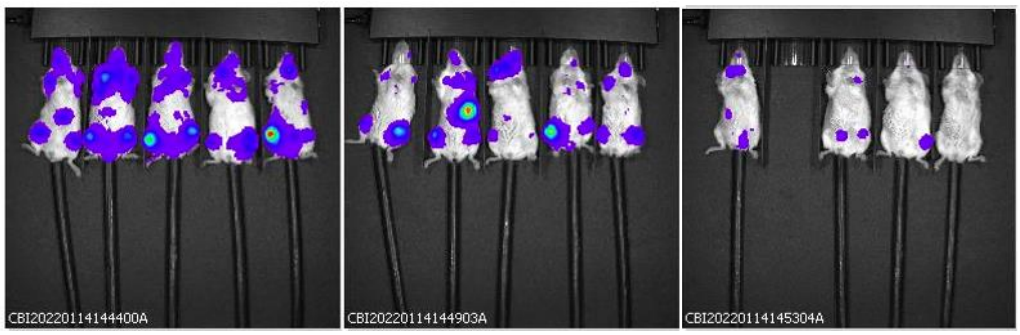
- Amplia's AMP886 inhibits AML in a well-established disease model
- AMP886 enhances efficacy of venetoclax therapy
- New opportunity to expand Amplia's pipeline into AML

Amplia Therapeutics Limited (ASX: ATX) ("Amplia" or the "Company") is pleased to report new data which demonstrates the potential of its second focal adhesion kinase (FAK) inhibitor AMP886 for the treatment of acute myeloid leukemia (AML). AML makes up about 1% of all cancer diagnoses and is a heterogeneous cluster of cancers that affect blood and bone marrow, with around 900 new cases diagnosed in Australia each year, about 50 of which are in children. Adults over 60 are the most commonly affected and have the poorest prognosis. Optimal treatment for AML depends on the disease subtype for each patient as well as their age/fitness but typically requires use of relatively toxic chemotherapies. Poorer response rates in older patients and high rates of relapse after treatment are also typical of AML.

Amplia's drug candidate AMP886 potently inhibits both FAK and Fms-like tyrosine kinase 3 (FLT3). FLT3 is mutated in around a third of AML patients and is a clinically validated target for chemotherapy. There is a poor duration of response to current FLT3 inhibitors which often leads to an aggressive disease relapse and very poor patient outcomes. Emerging research supports combing inhibition of FLT3 and FAK to overcome disease rebound following to FLT3 monotherapy,[1] and that this approach may have a beneficial impact in AML patients.[2]

Amplia has now shown that AMP886 inhibits AML in an industry-standard MV4-11 disease model recognised to carry the FLT3 mutation. Figure 1 shows that 21 days after inoculation with MV4-11 cells, oral doses of AMP886 significantly reduced the tumour burden in mice

Figure 1: Dose dependent reduction in tumour growth measured via bioluminescence of AML MV4-11 cells following treatment with AMP886



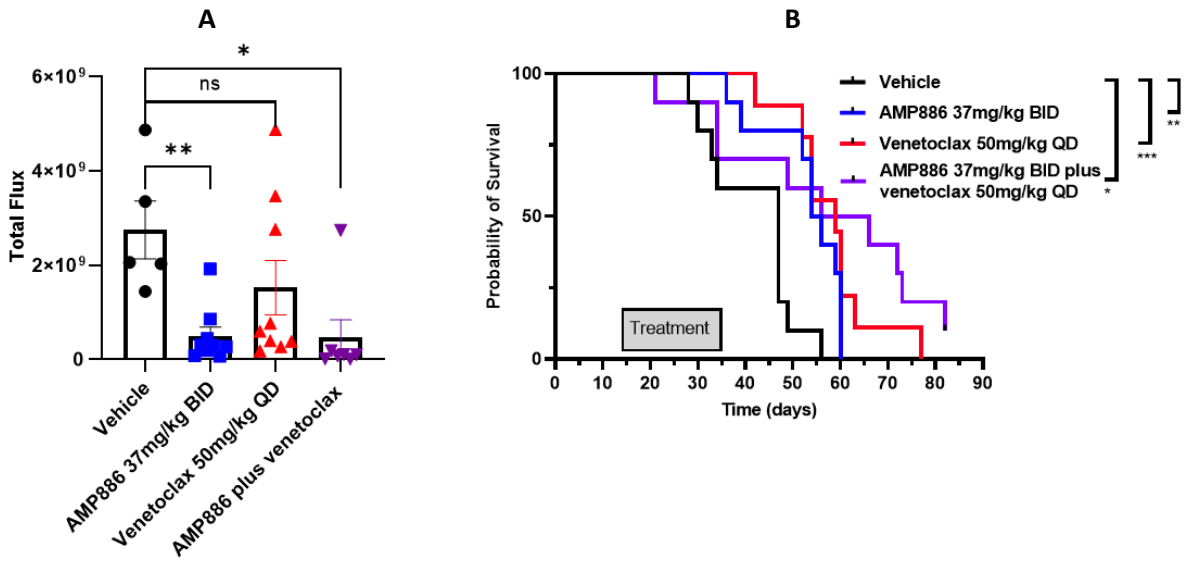
Vehicle (0 mg/kg AMP886) AMP886 37.5 mg/kg AMP886 75 mg/kg

[1] Allert, C. et al. Protein tyrosine kinase 2b inhibition reverts niche-associated resistance to tyrosine kinase inhibitors in AML. Leukemia (2022). https://doi.org/10.1038/s41375-022-01687-x

[2] Carter BZ, et al. Focal Adhesion Kinase as a Potential Target in AML and MDS. Mol Cancer Ther. (2017). https://doi.org/10.1158/1535-7163.MCT-16-0719.

In a second experiment, we measured the efficacy of AMP886 combined with venetoclax, a B-cell lymphoma 2 (BCL-2) inhibitor that is approved as part of combination therapy, typically for older patients with AML. Our rationale for adding AMP886 to venetoclax was based on recent reports showing that combination of FLT3 inhibitors and venetoclax may have clinical potential in patients with relapsed or refractory AML.[3]

The data show (Figure 2a) that AMP886 is more effective in reducing AML cell growth than venetoclax alone. Figure 2b shows that while both AMP886 and venetoclax improve survival in the MV4-11 model of AML, the combination of the two drugs tended to further enhance survival.



*p< 0.01, **p<0.0001 *p< 0.01, **p=0.006, ***p=0.002

Figure 2: (A) Inhibition of MV4-11 AML in mice treated with AMP886, venetoclax or combined AMP886 and venetoclax; (B) Kaplan-Meier survival curves of mice bearing MV4-11 AML when treated with AMP886, venetoclax or combined AMP886 and venetoclax

Amplia's CEO, Dr John Lambert, commented that "The impressive results we are reporting today tell us that there may be a clinical rationale to include AMP886 as part of new treatment regimens for unmet needs in AML. With an eye to expanding Amplia's clinical development pipeline, further experiments are already underway with AMP886 to build on this data and establish a scientifically solid foundation for initiation of formal development of AMP886."

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

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For Further Information

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[3] Wang X. et al. Combinatorial Inhibition of Focal Adhesion Kinase and BCL-2 Enhances Antileukemia Activity of Venetoclax in Acute Myeloid Leukemia. Mol Cancer Ther. (2020) https://doi: 10.1158/1535-7163.MCT-19-0841.

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).