9 MARCH 2026

ASX RELEASE

POSTER PRESENTATION DEMONSTRATING COMBINATION BENEFIT OF NARMAFOTINIB IN PRECLINICAL KRAS-MUTATED CANCER MODELS

HIGHLIGHTS

- *Amplia's lead drug narmafotinib enhances the activity of kRAS inhibitors in multiple preclinical cancer models presented at US conference*
- *Data showing narmafotinib activity in preclinical models of pancreatic cancer, lung cancer and ovarian cancer are presented*
- *The development of kRAS inhibitors for different types of solid tumours is currently an area of intense global activity*

Melbourne, Australia: Amplia Therapeutics Limited (ASX:ATX; OTCQB:INNMF), ("Amplia" or the "Company"), announces that compelling data describing new clinical opportunities for its lead drug narmafotinib was presented at the *AACR Special Conference in Cancer Research: RAS Oncogenesis and Therapeutics* in Los Angeles, California on Friday March 6.

The poster presentation discloses preclinical data demonstrating that the Company's best-in-class FAK inhibitor narmafotinib enhances the activity of a new class of drugs called kRAS inhibitors in various models of cancer. In particular, the data indicates that narmafotinib blocks resistance pathways that can emerge with kRAS inhibitor treatment, thereby enhancing efficacy and durability of response.

A copy of the poster is included with this announcement.

Inhibitors of mutant kRAS proteins are an exciting new class of drug in development for the treatment of lung, colon and pancreatic cancer, amongst others. There are currently over 50 different kRAS inhibitors undergoing clinical studies across the globe. Despite promising mid-stage clinical data, however, side-effects of these drugs can be significant and treatment-emergent resistance is commonplace.

Dr Chris Burns, CEO of Amplia, commented, "We are excited to present our research findings at this specialist conference focused on RAS inhibition in cancer. We believe there is significant clinical potential in combining narmafotinib with kRAS inhibitors and will be discussing our findings with pharma and biotech companies actively working in this space."

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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 and Amplia has a particular development focus in fibrotic cancers such as pancreatic and ovarian cancer. FAK also plays a significant role in a number of chronic diseases, such as idiopathic pulmonary fibrosis (IPF). For more information visit www.ampliatx.com and follow Amplia on X (@ampliatx) and LinkedIn.

About Narmafotinib

Narmafotinib (AMP945) is the company's best-in-class inhibitor of the protein FAK, a protein over-expressed in pancreatic cancer and a drug target gaining increasing attention for its role in solid tumors. The drug, which is a highly potent and selective inhibitor of FAK, has shown promising data in a range of preclinical cancer studies. Narmafotinib is currently undergoing a clinical trial (the ACCENT trial) where it is dosed in combination with the chemotherapies gemcitabine and Abraxane in first-line patients with advanced pancreatic cancer. The trial has already achieved its primary endpoint in achieving a confirmed response rate of 35%, superior to 23% reported in the benchmark MPACT study for gemcitabine and Abraxane alone. An interim median PFS of 7.7 months has also been reported. A second trial – AMPLICITY – has recently opened and is being run under an IND at sites in Australia and the US, investigating the combination of narmafotinib with the chemotherapy FOLFIRINOX in advanced pancreatic cancer patients.

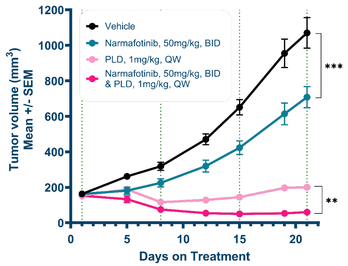
Combination benefits of Focal Adhesion Kinase Inhibitor Narmafotinib and KRAS inhibitors

Terrie-Anne Cock[1], Sarah Kinkel[1], Han Kyul Cho[2], Jiho Park[2], Braydon Meyer[3], Kendelle Murphy[3], David Herrman[3], Paul Timpson[3], Sang Hyub Lee[4], Kyung Min Lee[4], Jin Ho Choi[4], Jason Lickliter[1], ***Christopher J. Burns[1]***

[1]Amplia Therapeutics, Melbourne, Australia; [2]NEXT&BIO, Seoul, Korea; [3]Garvan Institute of Medical Research, Sydney, Australia, [4]Seoul National University Hospital, Seoul National University College of Medicine, Seoul, Korea

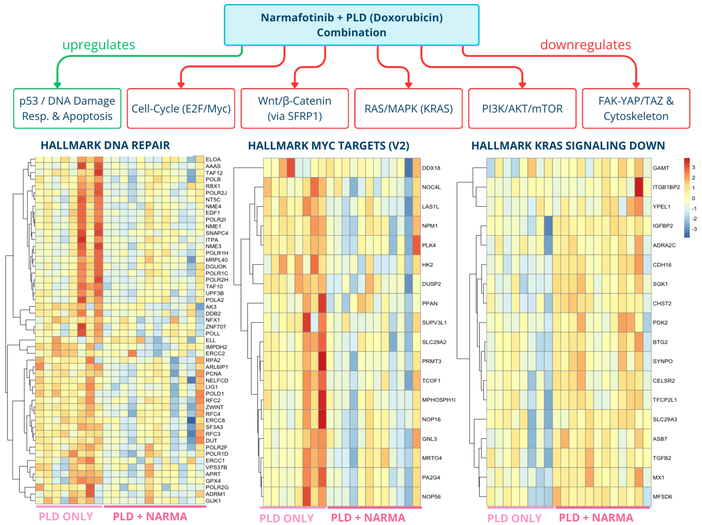
Improved responsiveness to chemotherapy in preclinical models

Enhanced tumor reductions in ovarian cancer TOV-21G *KRAS^{G13C}* xenograft tumors treated with narmafotinib combined with pegylated liposomal doxorubicin (PLD) reduces KRAS signaling

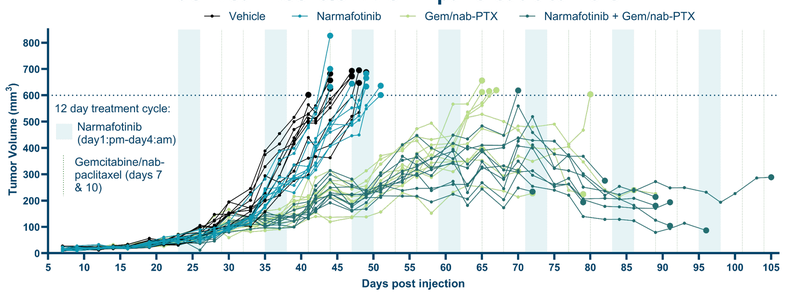


Expression profiling showed that narmafotinib monotherapy enhances p53 signaling pathways, while downregulating Wnt signaling

The addition of narmafotinib to PLD further disrupts the RAS/ERK/MAPK signaling axis and dampens the downstream Myc responsive proliferation programs



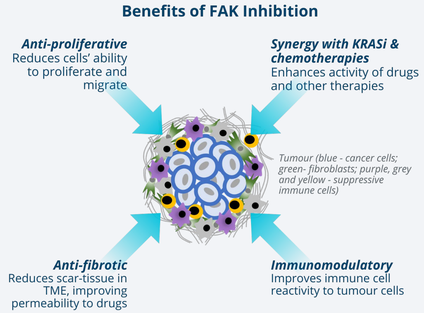
Narmafotinib and gem/nab-PTX combination prolonged inhibition of patient derived TKCC10lo *KRAS^{G12V}* pancreatic tumors



1. J Clin Oncol 44, 735(2026) Volume 44, Number 2_suppl
2. Von Hoff DD, et al. N Engl J Med. 2013;369(18):1691-703
3. Zhang B, et al. Adv Sci (Weinh). 2021;8(16):e2100250.

**p < 0.05, ** p < 0.01, ***p < 0.001, ****p < 0.0001*

NARMAFOTINIB: FAK inhibitor in Phase 2 PDAC

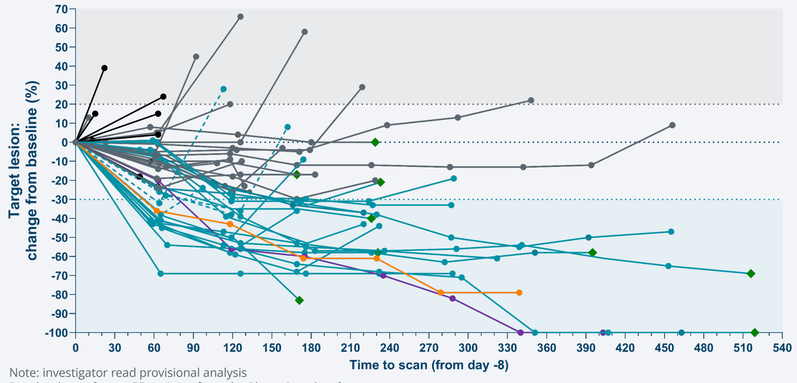


Best-in-class profile

- Highly potent and selective
- Oral once-a-day dosing
- Good safety and tolerability
- Patient population not limited by genetics
- Ability to combine with SOC therapies for multiple oncology indications

Narmafotinib in Phase 2 development for mPDAC in combination with two 1L SOC chemotherapies

1. **FOLFIRINOX (AMPLICITY trial NCT07026279)**
2. **Gem/nab-PTX (ACCENT trial NCT05355298)**
   - **Clinically favorable safety and tolerability** profile in combination with chemotherapy
   - **Durable and sustained clinical efficacy**



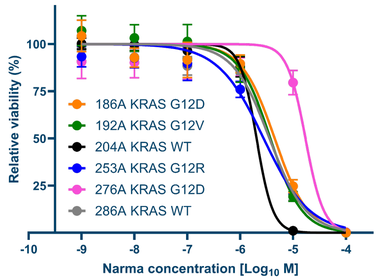
Note: investigator read provisional analysis
Results shown for n = 55 patients from the Phase 2a cohort[1].

- **Confirmed objective response rate of 35% (19/55 patients),** including **1 complete response** and **1 pathological CR**
- **Median PFS 7.7 m** compares favorably to MPACT benchmark study (5.5 m)[2]
- The combination was **well tolerated** with no evidence of increased grade ≥3 AEs compared to chemotherapy alone

Narmafotinib-related AEs in ≥ 5% of patients, n (%)		
Severity of Treatment-Related AEs	**Any grade n (%)**	**Grade ≥ 3 n (%)**
Nausea	16 (29.1)	2 (3.6)
Diarrhea	9 (16.4)	2 (3.6)
Vomiting	8 (14.5)	1 (1.8)
Fatigue	6 (10.9)	0
Gastroesophageal reflux disease	4 (7.3)	0
Constipation	3 (5.5)	0

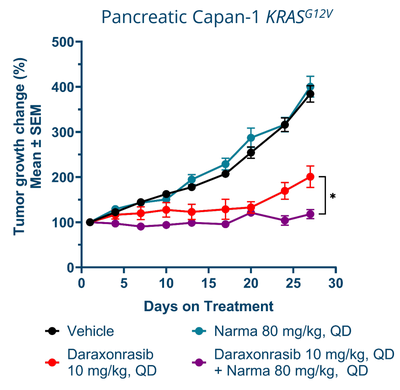
Combination benefit of narmafotinib with different KRASi

Narmafotinib treatment alone inhibits pancreatic cancer patient-derived organoid growth irrespective of KRAS mutation



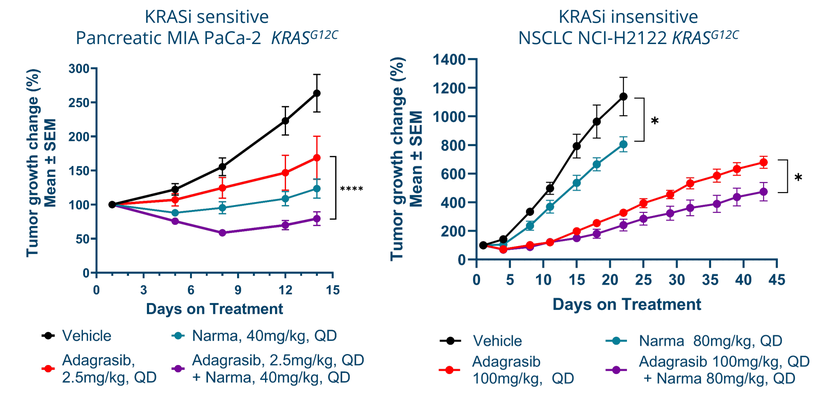
PDOs were stabilized for 96h followed by a 96h treatment period. Viability was measured by CellTitre-Glo®2.0.

Narmafotinib combined with pan-RAS inhibitor improves tumor growth inhibition



Combination treatment with narmafotinib enhanced tumor responsiveness to KRAS^{G12C} inhibitor in both sensitive and insensitive xenograft models

KRAS^{G12C} inhibition has been suggested to hyperactivate FAK signaling, which can lead to fibrosis and reduce the effectiveness of treatment[3]



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