ABN 16 165 160 841

ASX RELEASE **7 August 2025**

Bioshares Biotech Summit Presentation – Targeting Pancreatic Cancer and Beyond

Amplia Therapeutics Limited ("ATX" or "the Company") releases its Bioshares Biotech Summit Presentation which Managing Director Dr Chris Burns will present at the Bioshares Biotech Summit in Hobart today.

- End -

This ASX announcement was approved and authorised for release by the Managing Director.

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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 Twitter (@ampliatx) and LinkedIn.

Amplia THERAPEUTICS

TARGETING PANCREATIC CANCER AND BEYOND

August 2025

ampliatx.com | @ampliatx

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EXECUTIVE SUMMARY

Developing a pipeline of small molecule inhibitors of FAK

Lead drug **narmafotinib** is best-in-class FAK inhibitor in development

Promising efficacy, durability and tolerability in Phase 2a ACCENT clinical trial in pancreatic cancer

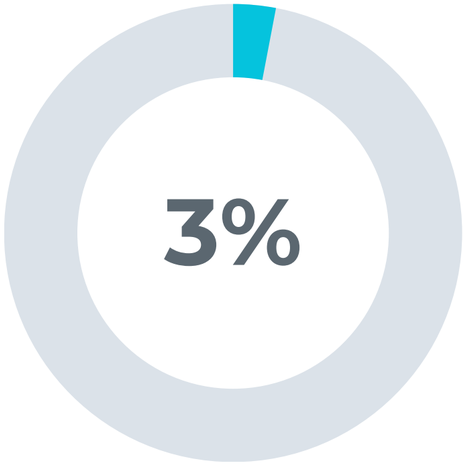
US trial of narmafotinib in pancreatic cancer to start imminently

FAST-track and **Orphan Drug Designation** granted from US FDA

METASTATIC PANCREATIC CANCER

Limited treatment options; poor patient outcomes

5Y Survival



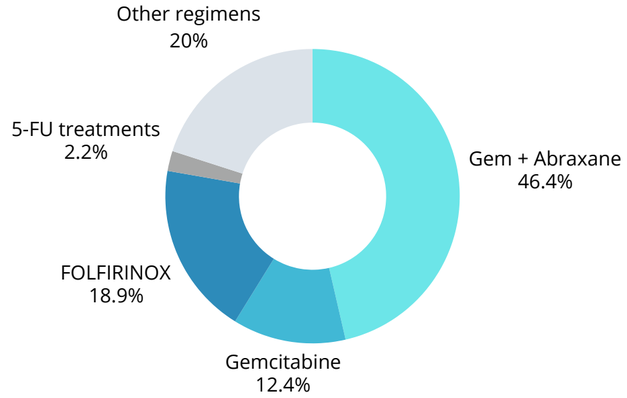
Highly aggressive with multiple genetic drivers

>50% pancreatic cancer patients **diagnosed with advanced** (metastatic, stage 4) disease at the time of diagnosis

Limited Treatment Options

Treatment	Median Progression Free Survival	Median Overall Survival	Tolerability
Gemcitabine + Abraxane® *(MPACT study)*	**5.5 months**	**8.5 months**	😐
FOLFIRINOX *(Prodige study)*	**6.4 months**	**11.1 months**	☹

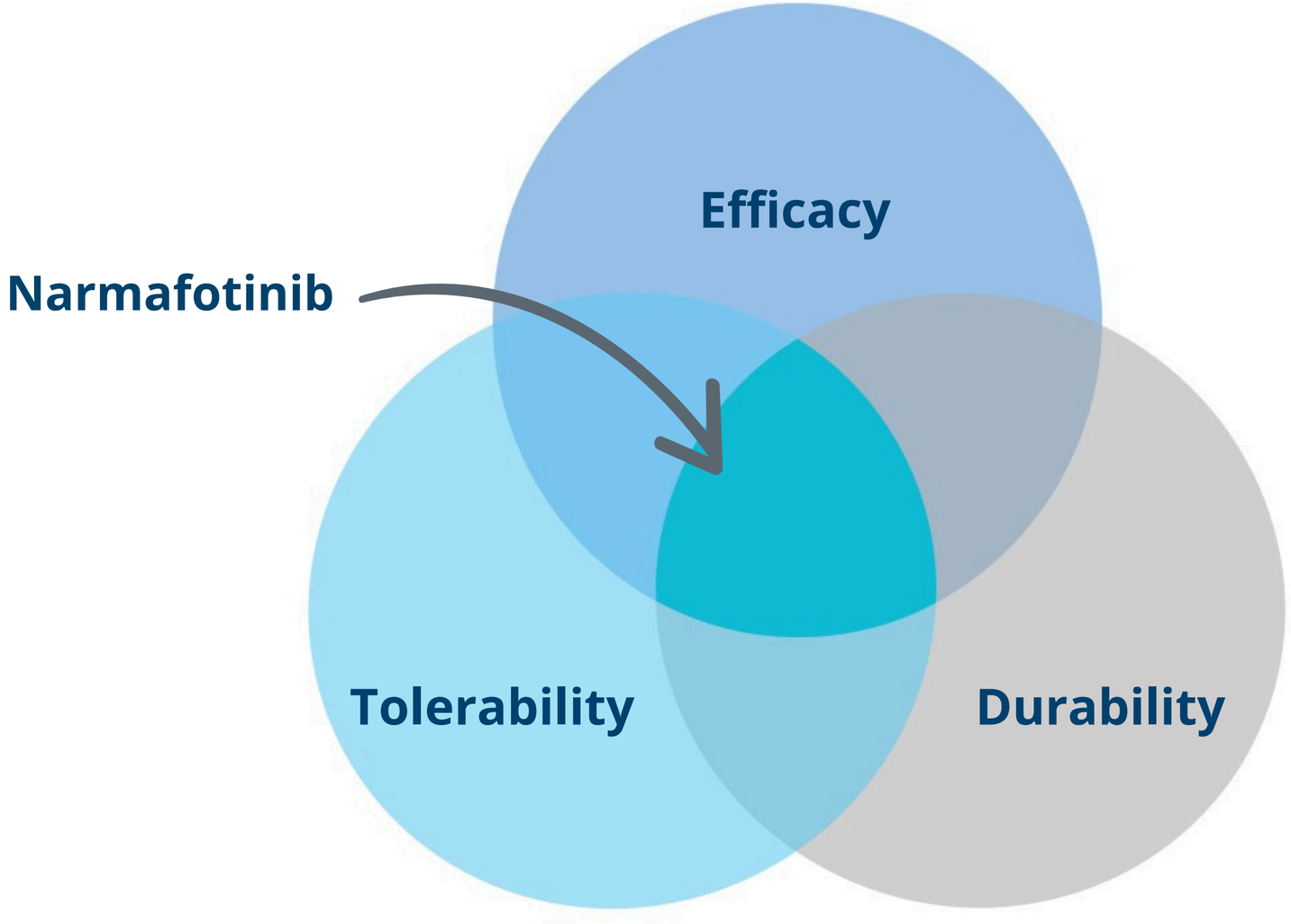
Most patients receive gemcitabine + Abraxane or FOLFIRINOX or variations of these†



MPACT Trial - Von Hoff et al., *N Engl J Med.* 2013, 369, 1691-703; PRODIGE trial - Conroy et al., *N Engl J Med.* 2011, 364, 1817-1825; † - *The Oncologist* 2022, 27, 469–475

THE AMPLIA ADVANTAGE

Three critical features



ACCENT TRIAL IN mPDAC

Phase 1b/2a study in Australia and Korea

OBJECTIVE

- To determine safety and efficacy of narmafotinib when added to standard of care in newly diagnosed patients

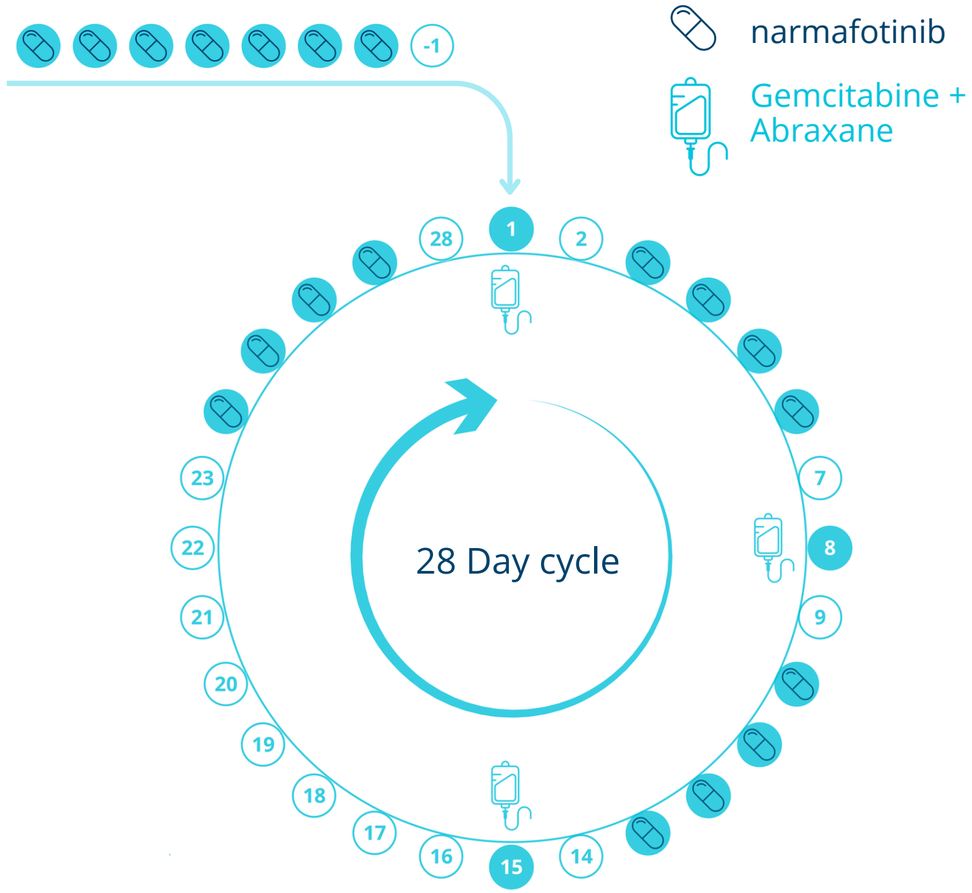
PRIMARY ENDPOINTS

- Safety, Tolerability
- ORR (RECIST 1.1)*

ADDITIONAL ENDPOINTS

- Duration on Trial
- Progression free survival (PFS)
- Overall Survival (OS)
- Disease Control Rate

Intermittent dosing schedule



* ORR - Objective Response Rate; RECIST - Response Evaluation Criteria in Solid Tumours, *Eur J Canc.* 2009, 45, 228-247. mPDAC - Metastatic Pancreatic Ductal Adenocarcinoma

ACCENT TRIAL INTERIM DATA

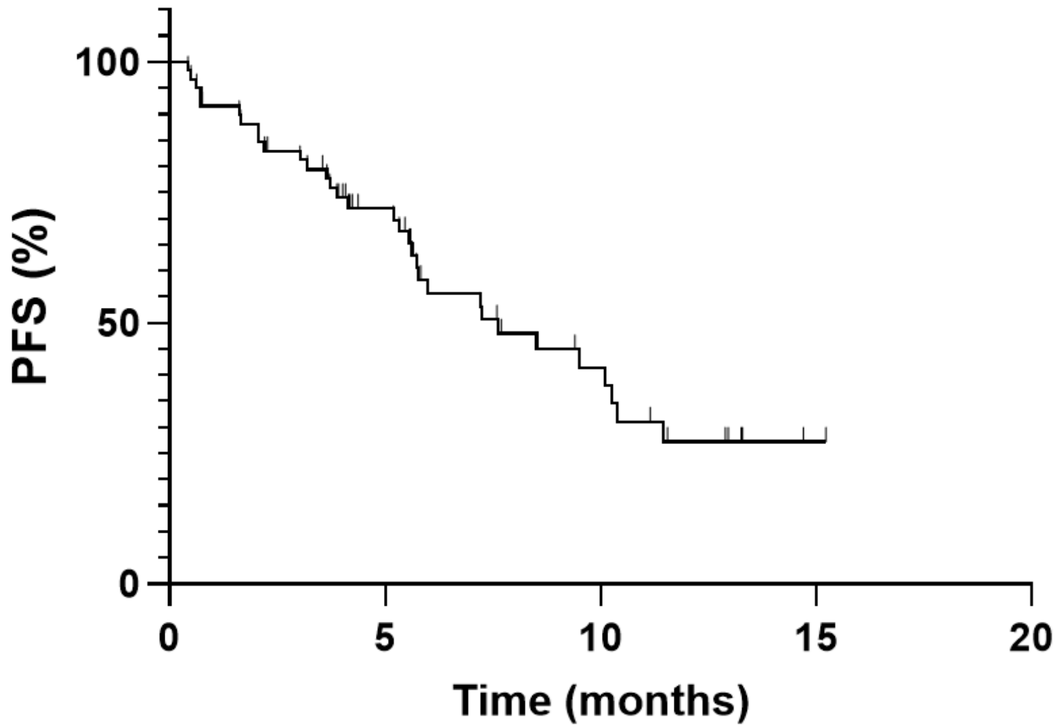
Promising evidence of efficacy, durability and tolerability

Progression Free Survival (PFS) data

- Currently determined at 7.6 months - substantially better than chemotherapy alone (5.5 months)
- Improvement over FOLFIRINOX chemotherapy (6.4 months)

	ACCENT Trial (Narmafotinib/Gemcitabine/Abraxane)	MPACT Trial (Gemcitabine/Abraxane)	PRODIGE Trial (FOLFIRINOX)
PFS	7.6 months	5.5 months	6.4 months

All ACCENT patients @ 400 mg (n = 64)



PFS data calculation included patients from Phase 1b dosed with narmafotinib (400 mg) and all patients in Phase 2a. Interim data based on data cut-off 20 Jul 2025.

ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

17 confirmed responses observed to date

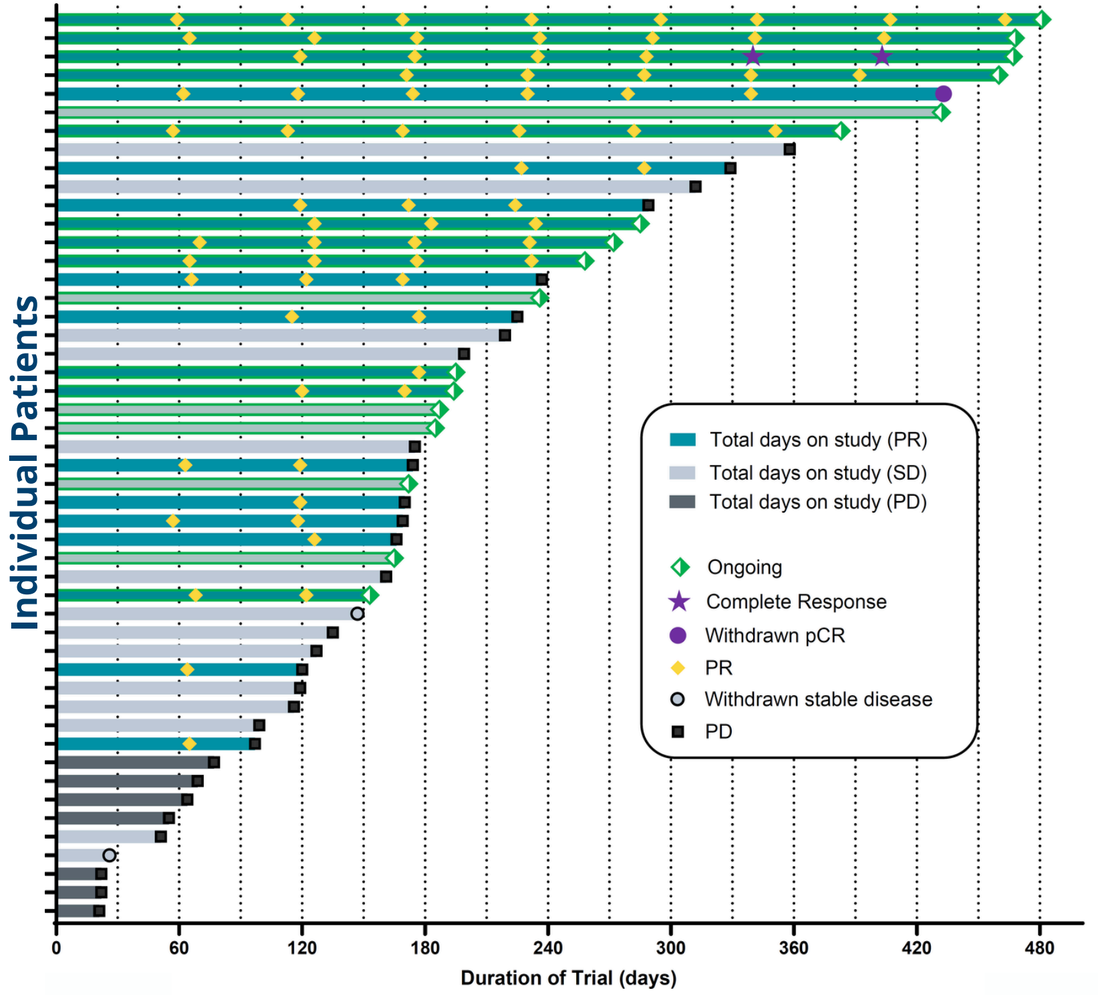
- Includes:
  - **1 confirmed complete response**
  - **1 pathological complete response**
- Indicating narmafotinib + chemotherapy is **superior** to chemotherapy alone

7 patients on study > 1 year

- Mean DoT = 201 days

At data cut-off (20 Jul 2025):

- 17 patients remain on study
- Data for 6 patients at 6 months yet to be collected



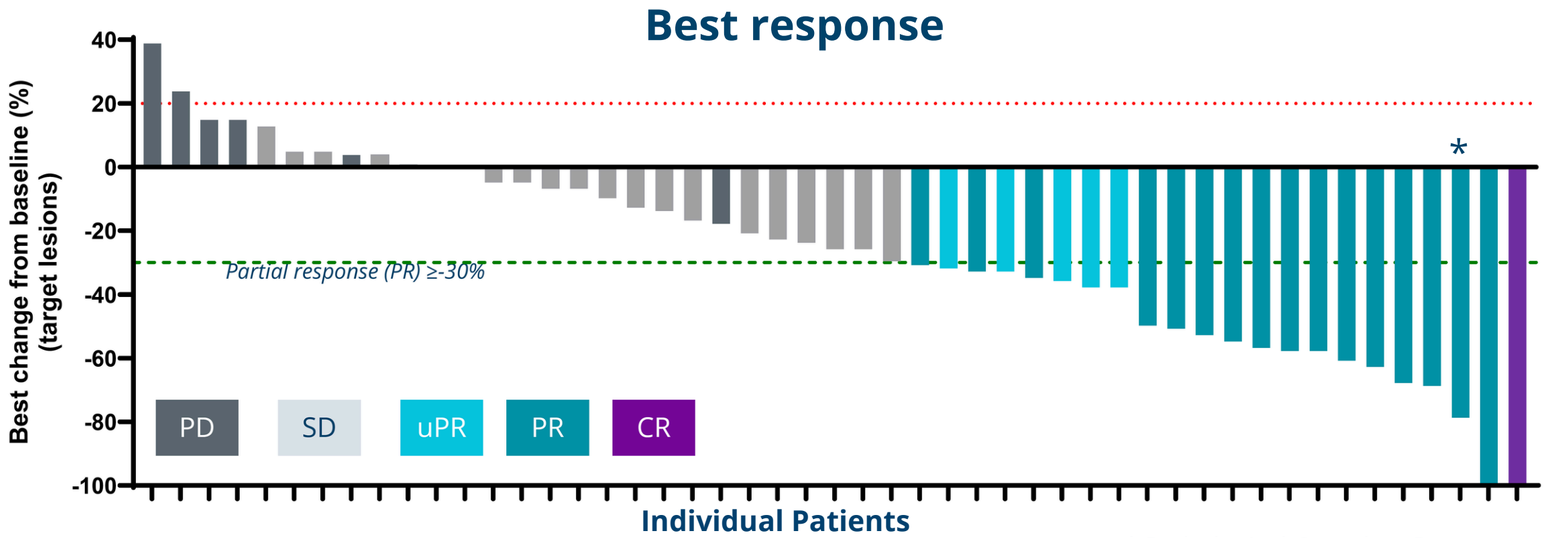
Interim data based on data cut-off 20 Jul 2025. Evaluable patients only shown. Responses are investigator read. DoT - Duration on Trial, PR - Partial Response; SD - Stable Disease, PD - Progressive Disease, pCR - pathological Complete Response.

ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

Excellent response rate observed

- **1 confirmed Complete Response**
- **16 confirmed Partial Responses**
  - **Incl. 1 patient** determined to be a **pathological Complete Response**
- Objective response rate (ORR) of 31%
- Disease control rate (DCR) of 73%



* Pathological Complete Response

Peter's pancreatic marvel: meet the luckiest man in the country

EXCLUSIVE

Test results stunned doctors in Australia ... and across the world

NATASHA ROBINSON
HEALTH EDITOR

Peter Moulding was recovering from surgery when his oncologist received the Melbourne tradie's pathology results – and the doctor couldn't believe his eyes.

"I actually called the pathologist and said: Are you sure you're looking at the right specimen?" says Prasad Cooray, an oncologist at the Jeffrisuli Pancreatic Centre at the Epworth Hospital in Melbourne.

"Because I think all of us had difficulty believing this was true."

The tissue specimens were small slices of what had appeared as "shadows" on medical imaging of Mr Moulding's pancreas. Clinicians had performed tumour resection surgery of these suspect tissues 12 months after Mr Moulding, a metastatic pancreatic cancer patient, had been signed up to a clinical trial testing a novel drug. But the shadow tissue was not cancer at all.

Mr Moulding is in remission from metastatic pancreatic cancer, having experienced what is known in medicine as a pathological complete response to treatment. That means that cancer is no longer detectable. This is vanishingly rare in metastatic pancreatic cancer, so rare that Dr Cooray is confident no oncologist in Australia he's in touch with has ever seen such a phenomenon. In the scientific literature, doctors believe only one other case of a pathological complete response in a metastatic cancer patient has been recorded worldwide.

"I've never come across a case like Peter's where there is no residual cancer left," Dr Cooray says. "So this is a highly, highly unusual finding."

'Groundbreaking'

Mr Moulding was part of a clinical trial of a drug developed in Australia known as narmafotinib, or AMP945, which has the potential to make chemotherapy much more effective because it breaks down a fibrous shield that surrounds cancer cells, making them difficult to penetrate.

This fibrous shield builds up around pancreatic cancer tumours largely owing to a protein known as focal adhesion kinase, which forms a protective environment around tumours that stops chemotherapy from reaching tumours. FAK can also act as a "survival switch" for cancer cells, switching on the activity of the FAK protein, which also contributes to the formation of the fibrous protective layer around tumours. AMP945 may be able to turn off that switch, making the cancer cells easier to kill.

When Mr Moulding, a refrigeration mechanic from outer western Melbourne, was given the opportunity to join the trial testing AMP945, he jumped at it. At the time, he didn't know the prognosis for pancreatic cancer patients was devastatingly poor. Only one in five of all patients is alive 12 months after diagnosis.

"I didn't know what stage four was, and I didn't ask," Mr Moulding says. "I just went along for the ride, basically. I just thought, well, I'll do what I've got to do, and hopefully they'll operate and fix it for me."

In fact, surgery for metastatic cancer patients – where the cancer has spread to other parts of the body – usually does not happen. Currently, the best those patients can hope for is that chemotherapy prolongs their life.

"In the pancreatic cancer space there really hasn't been any significant development in treatment for decades," Dr Cooray says. "Yes, there's been some improvement in chemotherapy drugs, but a drug that looks at the cancer from a different angle has not happened in pancreas cancer ever. So if this drug proves to be effective, this will be a groundbreaking development in pancreatic cancer."

AMP945 was developed by Amplia Therapeutics under the umbrella of Australia's Cancer Therapeutics Co-operative Research Centre – set up by the federal government in 2007 to bridge the gap between research breakthroughs and commercialisation – in conjunction with scientists from the nation's top universities and scientific institutes. The Garvan Institute of Medical Research has previously established that targeting FAK prior to treatment makes pancreatic cancer cells more sensitive to chemotherapy and reduces cancer spread by 50 per cent in mice. The drug has been shown in early studies to also have application for ovarian cancer, which also involves fibrosis.

Reason for hope

Despite the promising results in animal studies, Amplia chief executive Chris Burns said the results of the human trial so far have exceeded his company's expectations.

"To see a pathological complete response was totally unexpected. We never thought it would happen," Dr Burns says.

He says the majority of patients in the trial have had more tumour shrinkage than would have been expected with chemotherapy alone. "This is entirely consistent with breaking down that fibrotic tissue and allowing the drug to penetrate and work better," he says. "So we definitely have an indication that the fibrosis is breaking down.

"We were hoping that we could extend survival significantly, but where we see the kind of level of response that we've seen, because it's so new, we don't know what that might mean longer term. Potentially it's transformative, we could actually totally change the trajectory of this disease.

"It definitely gives patients hope that there is progress being made, that rather than pancreatic cancer being a death sentence, I would hope that we're looking at the beginning of a change that means this cancer might be a manageable disease in a few years' time."

The clinical trial for AMP945, known as the ACCENT trial, is in phase two safety and efficacy testing. Full phase two results will be analysed and will be published in about six months, and an international phase three study will follow.

As well as Mr Moulding's stunning response, one other patient in the trial has also had a complete response to treatment, meaning the disappearance of all tumour lesions that is maintained for at least 2 months. Sixteen other patients have had a partial response, where tumour shrinkage greater than 30 per cent is recorded and sustained for two or more months and where no new cancerous lesions have been detected. About 50 people in Australia and Korea were initially signed up to the trial 18 months ago, and there are 20 still remaining on the drug treatment, which is given for about four days prior to monthly rounds of chemotherapy.

No other patients have had such a stunning response as Mr Moulding, but Dr Cooray, who is planning to write up Mr Moulding's case for a scientific journal, says his observation is that most of the patients in the trial have done better than had they received standard treatment.

"We don't have the data published yet, so I don't want to be speaking prematurely but, at the same time, I don't want to dial down the excitement that goes with this pathological complete response, either. Every win we need to celebrate," he says. "This being the first pathological complete response is highly significant. I can definitely say that much. And in my career of close to 20 years, this is the first time I've come across that. And the only added variable in this case was the drug, the FAK inhibitor.

"I'm not saying all pancreatic patients will benefit from this drug. We know from other cancer types, when a targeted treatment works, there's some subgroup where it is more effective than in others ... that is part of the puzzle."

Making plans

Allan Zimet, a medical oncologist at the Jeffrisuli Pancreatic Centre, says Mr Moulding's case and that of other patients whose tumours have significantly shrunk may provide important clues as to why some patients respond to AMP945 and others don't.

Pancreatic cancer patients often have in their DNA what is known as KRAS mutations, which drive tumour mutation and progression. These mutations have been considered undruggable, but that may not be true.

"We're now in the era of personalised medicine," Dr Zimet says. "Genetic mutations may be driving the fibrosis in pancreatic cancer patients, and if you target the mutation and switch off the fibrosis, you may be able to improve the patient's outcome.

"This drug is still very much investigational, and so it's a potential pointer that this may be a good drug that may have particular activity, but it's a pointer at this stage. It's not a sort of 'lay-down misery'. It's important people understand and that we don't raise false hopes.

"But pancreatic cancer has been an orphan cancer in many ways, because people have been nihilistic about the effects of treatment. There are not many patient advocates, because our patients are too unwell for that, and their survival is not good enough for them to be involved. "So I think that a good news story like this will only help to stimulate medical research efforts further, and to look and to review and see what's special about that person who has had such a good response, and how we can we learn some deeper lessons from this."

As for Mr Moulding, the hard-working tradie has realised he has no time to waste. He has worked as a small-business owner all of his life and always intended to put off travel and taking time out until retirement. But he is now fast-tracking his plans.

"I just want to do some things that are going to make me happy and enjoy what I've got left of my time, I suppose," he says. "It would be nice to actually get off my bum and do some travelling."

As patient zero, Mr Moulding has immense gratitude for the being part of the clinical trial. "I don't know really what to say except that I'm just so happy," he says. "I was given the opportunity to have a go of it, and it's actually worked."

Peter Moulding can now see the funny side after going into remission from pancreatic cancer, an almost unprecedented event

NADIA KNANI

"I didn't know what stage four was, and I didn't ask. I just went along for the ride, basically"
PETER MOULDING
PANCREATIC PATIENT SURVIVOR

"I've never come across a case like Peter's where there is no residual cancer left"
PRASAD COORAY
ONCOLOGIST

"To see a pathological complete response was totally unexpected. We never thought it would happen"
CHRIS BURNS
AMPLIA CHIEF EXECUTIVE

Interim data based on data cut-off Jul 20, 2025. Responses are investigator read

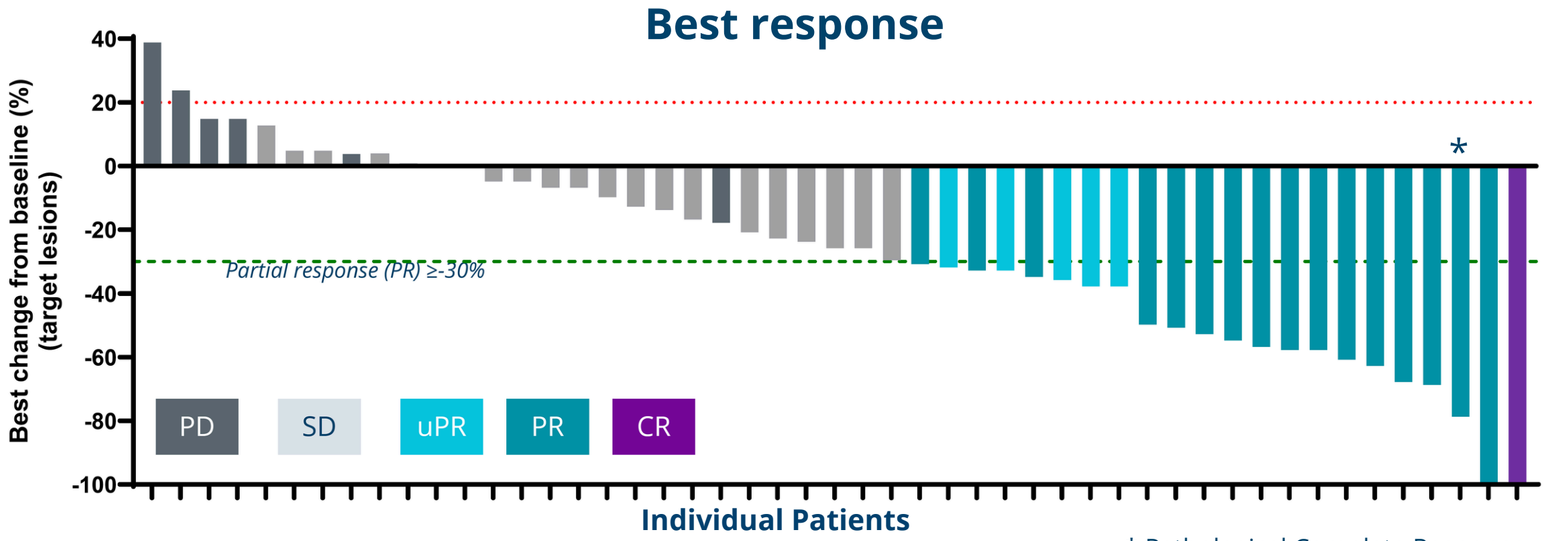
ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

Excellent response rate observed

- **1 confirmed Complete Response**
- **16 confirmed Partial Responses**
  - **Incl. 1 patient** determined to be a **pathological Complete Response**
- Objective response rate (ORR) of 31%
- Disease control rate (DCR) of 73%

	ACCENT Trial (Narmafotinib/Gemcitabine/Abraxane)	MPACT Trial (Gemcitabine/Abraxane)
CR	2%	0.2%
PR	29%	23%
SD	42%	27%
PD	16%	20%
NE	11%	30%
ORR	31%	23%
DCR	73%	50%
DOT	201 days	117 days



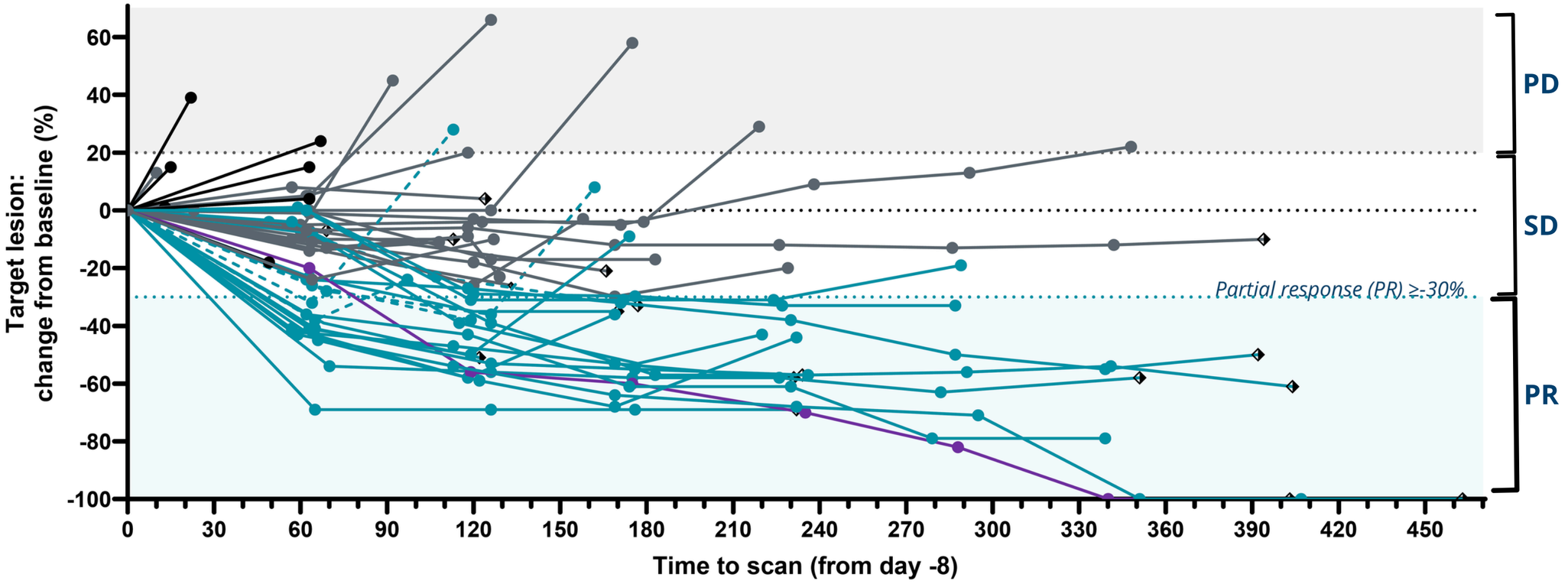
* Pathological Complete Response

Interim data based on data cut-off Jul 20, 2025. Responses are investigator read. NE - Non-evaluable

ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

Excellent durability observed



Interim data based on data cut-off Jul 20, 2025. Responses are investigator read

ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

Excellent tolerability observed to date

- Narmafotinib treatment results in negligible extra patient burden

Adverse Events (Grade 3 or above)

Adverse Event (AE) Grade ≥ 3	Narmafotinib +Gem/Abr (ACCENT; N=55)	Gem/Abr (MPACT; N=421)	Gem/Abr (NAPOLI 3; N=379)	FOLFIRINOX (PRODIGE; N=171)	NALIRIFOX (NAPOLI 3; N=370)
Neutropenia	38.2%	38%	39%	46%	24%
Anemia	9.1%	13%	18%	8%	11%
Diarrhea	5.5%	6%	5%	13%	20%
Peripheral neuropathy	3.6%	17%	6%	9%	3%
Vomiting	3.6%	NR	2%	15%	7%
Febrile Neutropenia	5.5%	3%	NR	5%	NR
Thrombocytopenia	NR	13%	NR	9%	NR
Fatigue	NR	17%	5%	24%	6%
Hypokalemia	NR	NR	4%	NR	15%
Nausea	3.6%	NR	3%	NR	12%

ACCENT and MPACT trials have very similar inclusion criteria and trial designs however no head-to-head clinical trial has been conducted. Differences exist between ACCENT and other trials in design, inclusion criteria and patient demographics. NAPOLI 3 Trial - *The Lancet* 2023, 402(10409),1272-1281

ACCENT TRIAL SUMMARY

On track to achieve trial goals

Superior Efficacy

For full 55 patient cohort

- **1 CR**
- **16 PR**

Improved PFS over Gemcitabine + Abraxane, and FOLFIRINOX

Improved Durability

7 patients on trial for >12 months

Deep and sustained response for a subset of patients

- Biomarker discovery to be initiated

Demonstrated Tolerability

Excellent tolerability profile

Minimal additional burden on the patients above standard of care

No evidence or likelihood of drug-drug interactions

FUTURE OPPORTUNITIES

FAK inhibition will enhance multiple therapeutic strategies

Narmafotinib
(FAK inhibition)

CHEMOTHERAPY
Clinical and preclinical data incl. ACCENT study

IMMUNOTHERAPIES
Preclinical data

KRAS INHIBITORS
Preclinical data, incl. NEXT&BIO collaboration

RADIOTHERAPY
Published data

ANTIBODY DRUG CONJUGATES
Published data

AMPLICITY TRIAL in mPDAC

Phase 1b/2a study in the US and Australia

OBJECTIVE

- To determine safety and efficacy of narmafotinib when added to FOLFIRINOX in newly diagnosed patients
- To identify recommended phase 2 dose (RP2D)

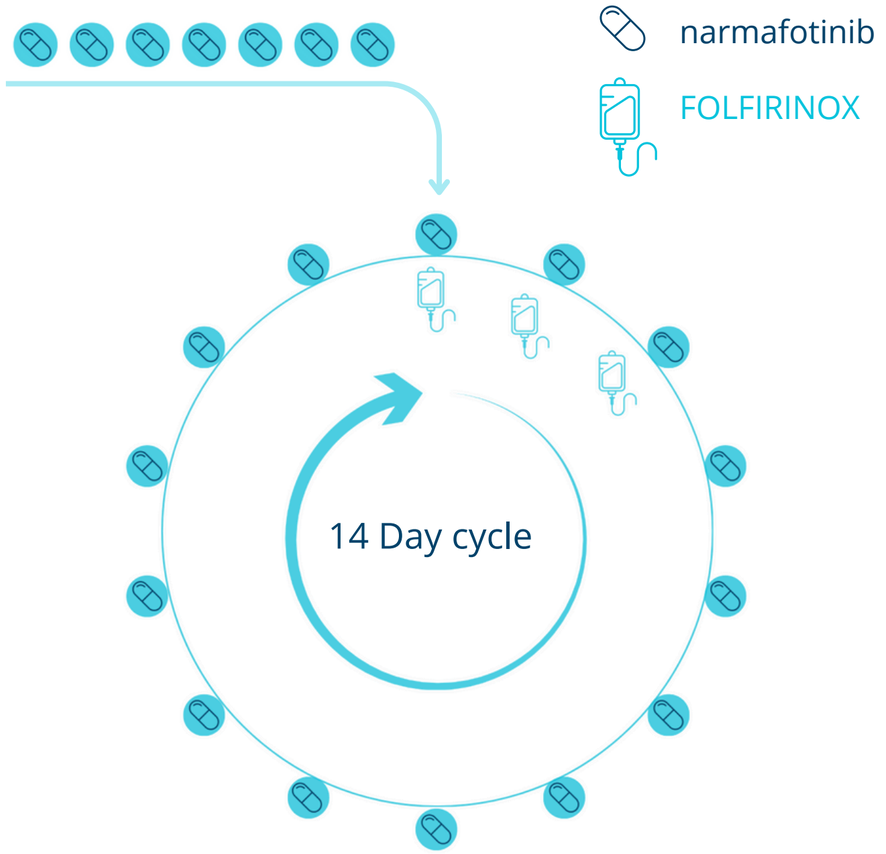
PRIMARY ENDPOINTS

- Safety, Tolerability
- RP2D

ADDITIONAL ENDPOINTS

- ORR (RECIST v1.1)
- Duration of Response
- Progression free survival (PFS)
- Overall Survival (OS)
- Disease Control Rate

Moving from intermittent to daily dosing



NARMAFOTINIB: A POTENT AND SELECTIVE FAK INHIBITOR

FAK enzyme overactive in pancreatic cancer

FAK levels are elevated in pancreatic cancer

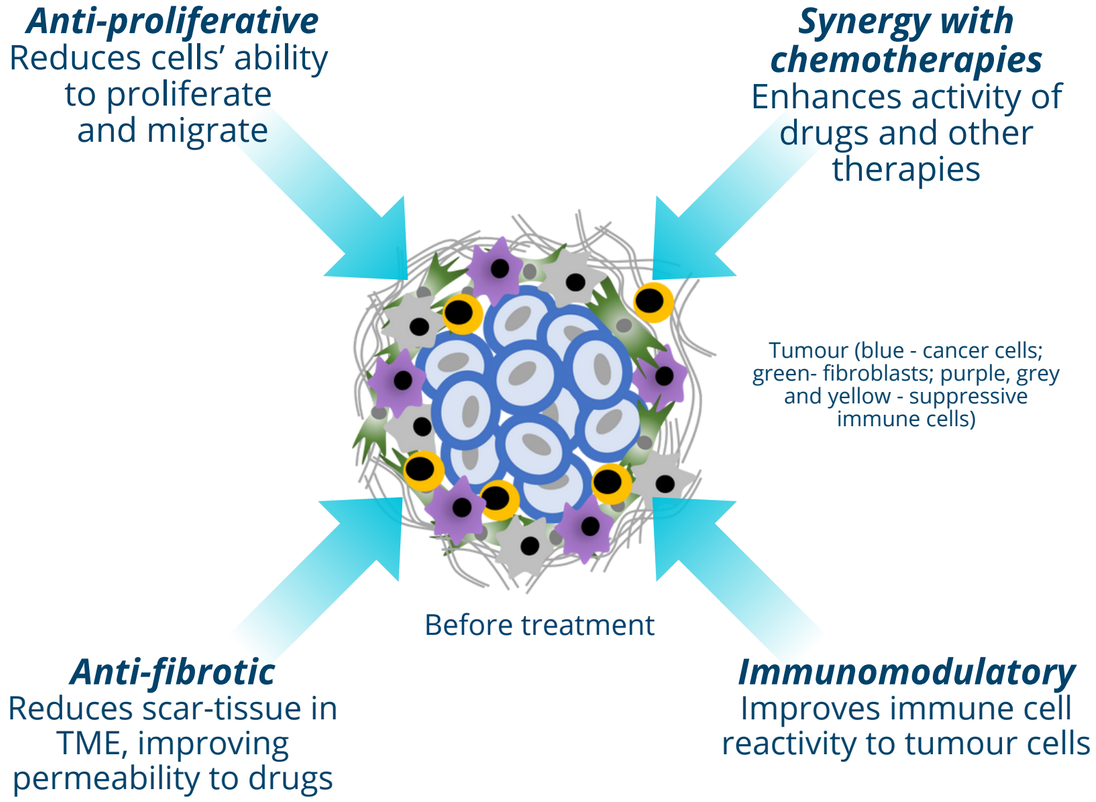
- Correlate with worse patient outcome

FAK inhibition blocks processes that support:

- Tumour growth
- Metastasis
- Treatment resistance

Demonstrated efficacy in preclinical models
of human pancreatic cancer

Benefits of FAK Inhibition



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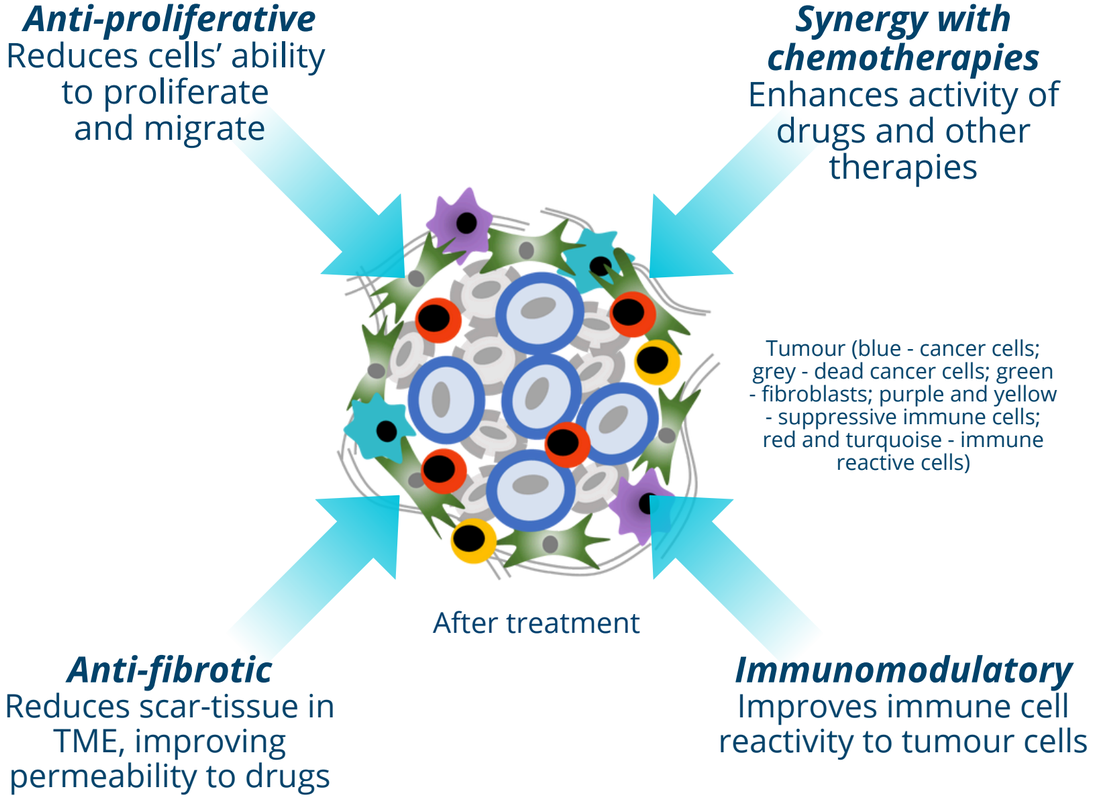
Benefits of FAK Inhibition



Modified from *Journal for ImmunoTherapy of Cancer* (2017) 5:17

NARMAFOTINIB: A POTENT AND SELECTIVE FAK INHIBITOR

Best-in-class profile

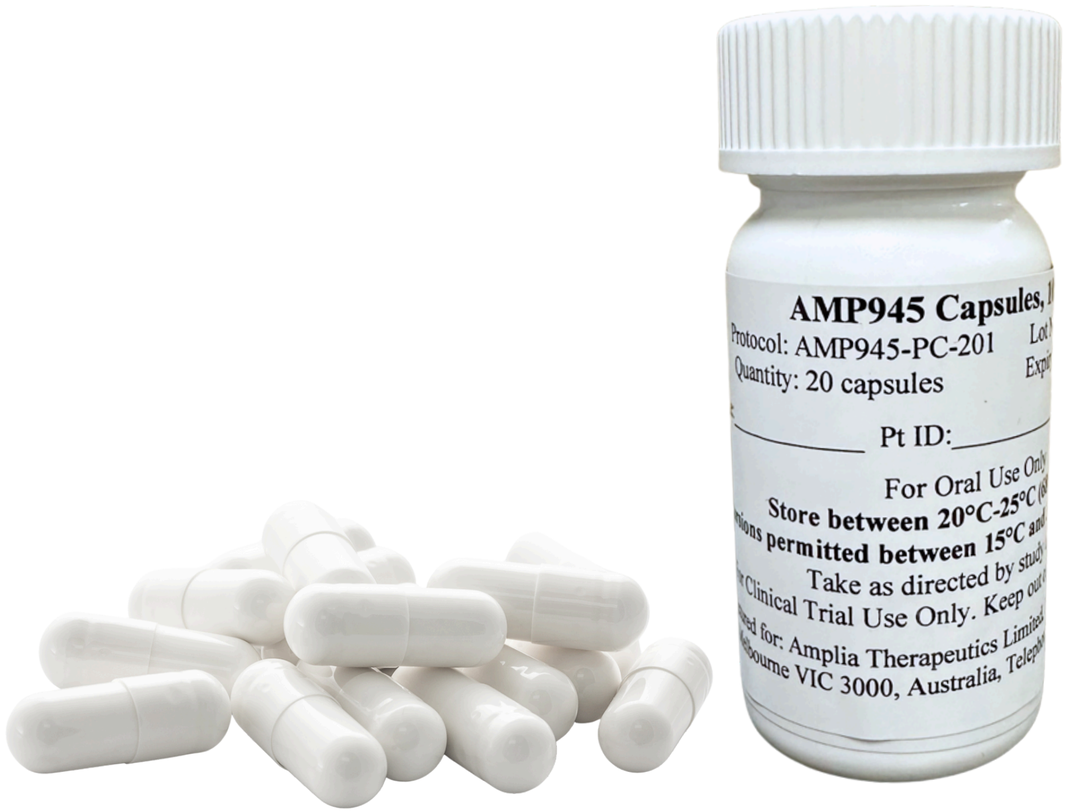
Convenient to take

- Once a day oral dosing by capsule
- Storage at room temperature

Safe to combine with other medicines

- No evidence of drug-drug interactions

Evidence of FAK target engagement in humans



UPCOMING MILESTONES

Q3 2025

ACCENT top-line data

ACCENT request FDA type C meeting - Phase 2b/3 pivotal trial design

AMPLICITY first patient dosed (part A)

ACCENT further patient updates

Q4 2025

ACCENT further patient updates

AMPLICITY first safety and efficacy data

FDA meeting and minutes **ACCENT** trial pathway

IIT funding outcome(s)

Possible EU regulatory filings

Q1 2026

ACCENT mature data (including OS)

AMPLICITY complete dose escalation

Initiate kRAS combination IIT

EU regulatory response

Q2 2026

ACCENT trial completion

AMPLICITY further patient updates

Initiate IIT in ovarian cancer

Drug product scale-up

ACCENT Phase 2b/3 trial protocol finalised

2H 2026

ACCENT full data release

AMPLICITY 2-dose comparison trial begins

IIT data updates (kRAS and Ovarian)

ACCENT Phase 2b/3 trial planning finalised

BOARD + MANAGEMENT

World-class experts

BOARD

Warwick Tong

MB ChB MPP GAICD

Chair

Robert Peach

PhD

Director

Jane Bell

LLB LLM (Lond) FAICD

Director

Chris Burns

PhD GAICD

CEO and MD

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Jason Lickliter

MBBS FRACP

CMO

Tim Luscombe

BCom CA GIA(Cert)

CFO

THANK YOU

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