

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](http://www.ampliatx.com) and follow Amplia on [Twitter \(@ampliatx\)](https://twitter.com/ampliatx) and [LinkedIn](https://www.linkedin.com/company/ampliatx).

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# TARGETING PANCREATIC CANCER AND BEYOND

August 2025

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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

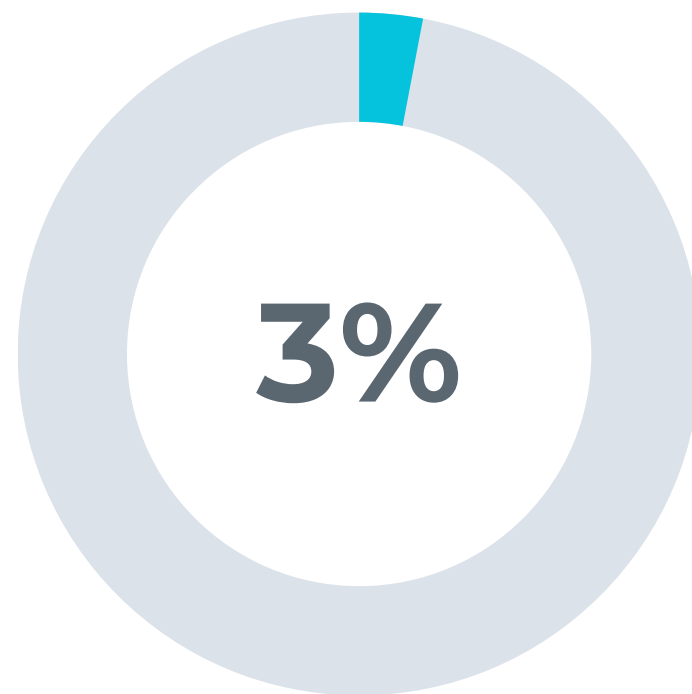


**FAST-track** and **Orphan Drug Designation** granted from US FDA

# METASTATIC PANCREATIC CANCER

Limited treatment options; poor patient outcomes

## 5Y Survival



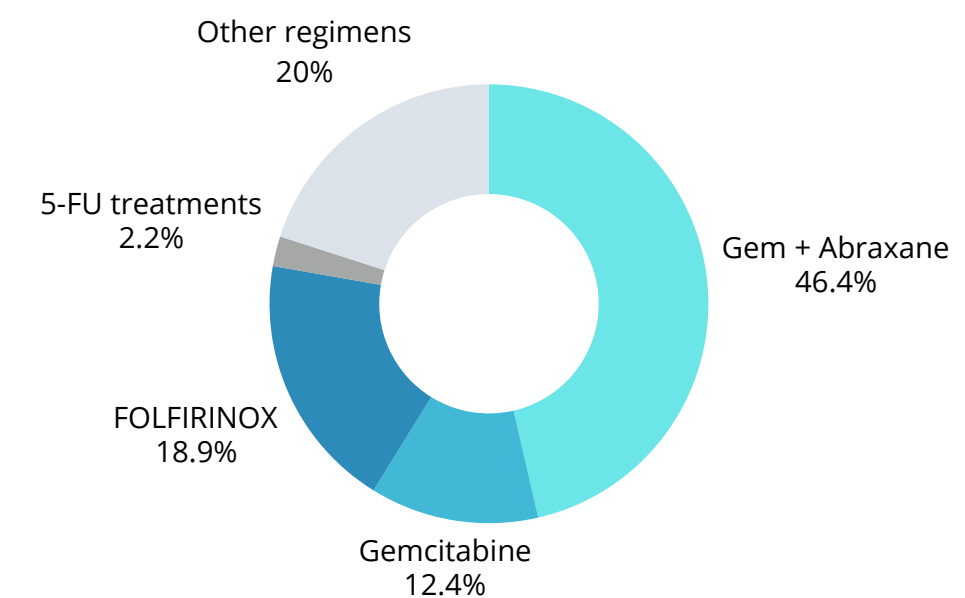
Highly aggressive with multiple genetic drivers

>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	☹️

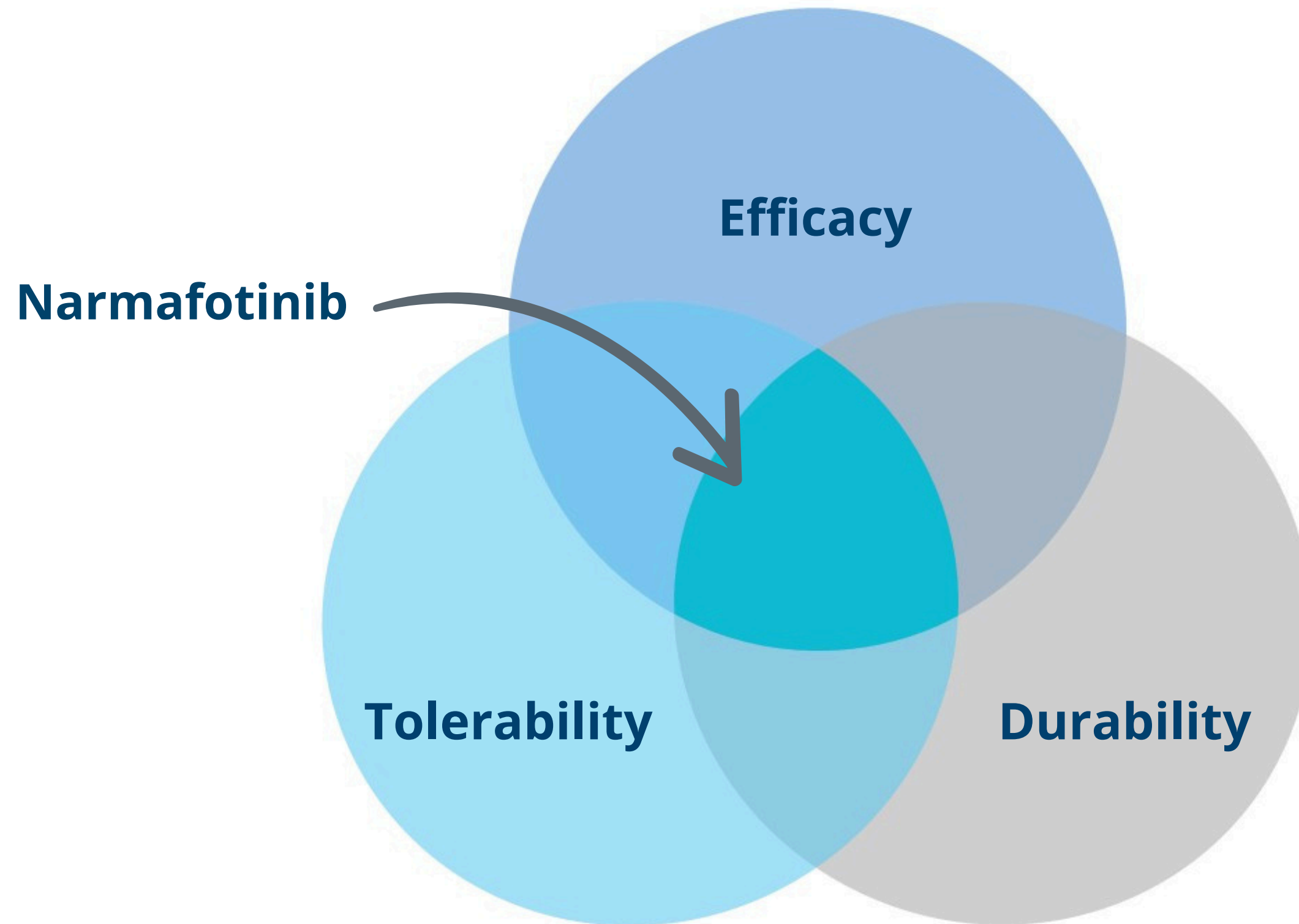
Most patients receive gemcitabine + Abraxane or FOLFIRINOX or variations of these<sup>†</sup>



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# THE AMPLIA ADVANTAGE

*Three critical features*



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# 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

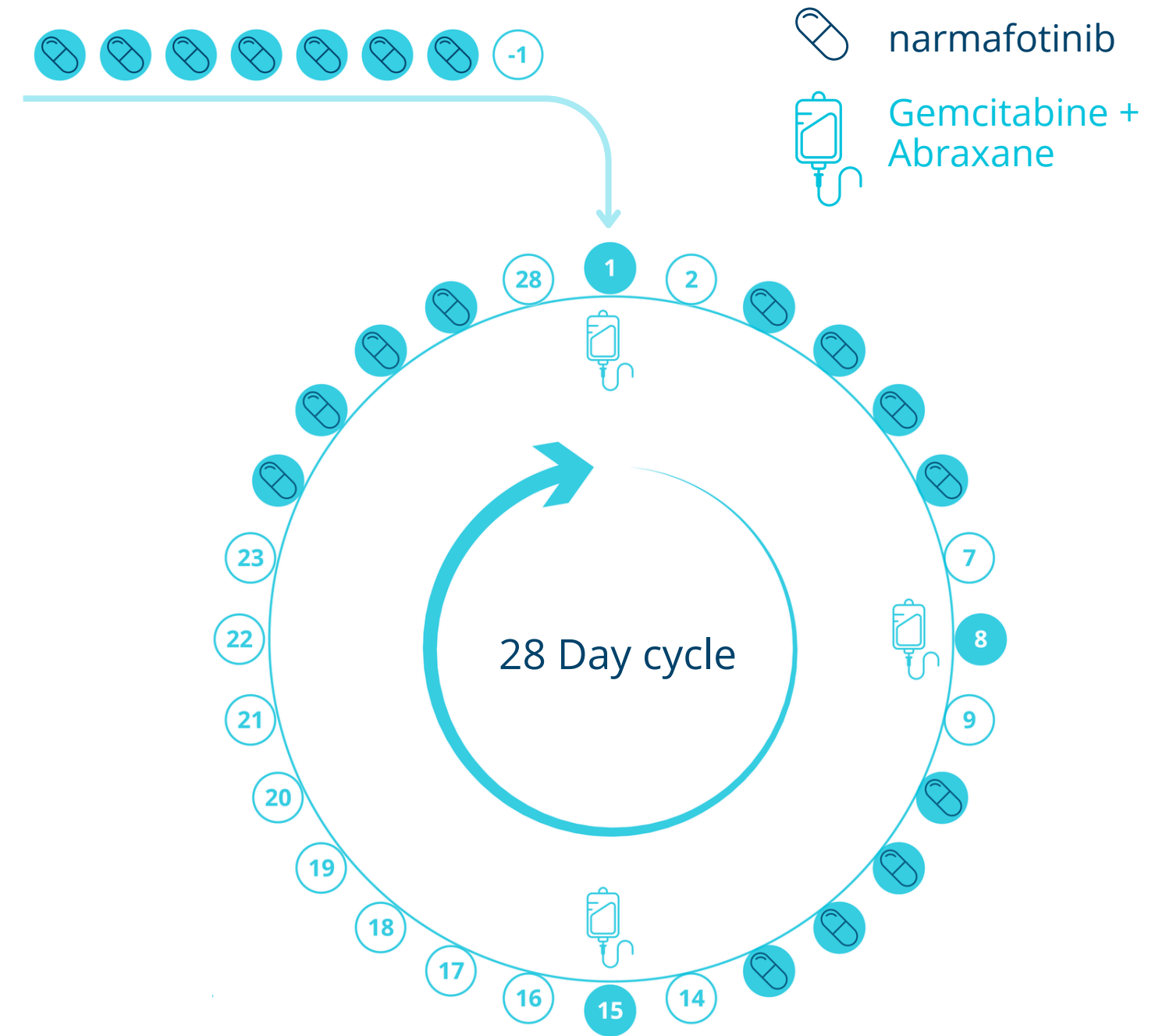
### PRIMARY ENDPOINTS

- Safety, Tolerability
- ORR (RECIST 1.1)\*

### ADDITIONAL ENDPOINTS

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

### Intermittent dosing schedule



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# ACCENT TRIAL INTERIM DATA

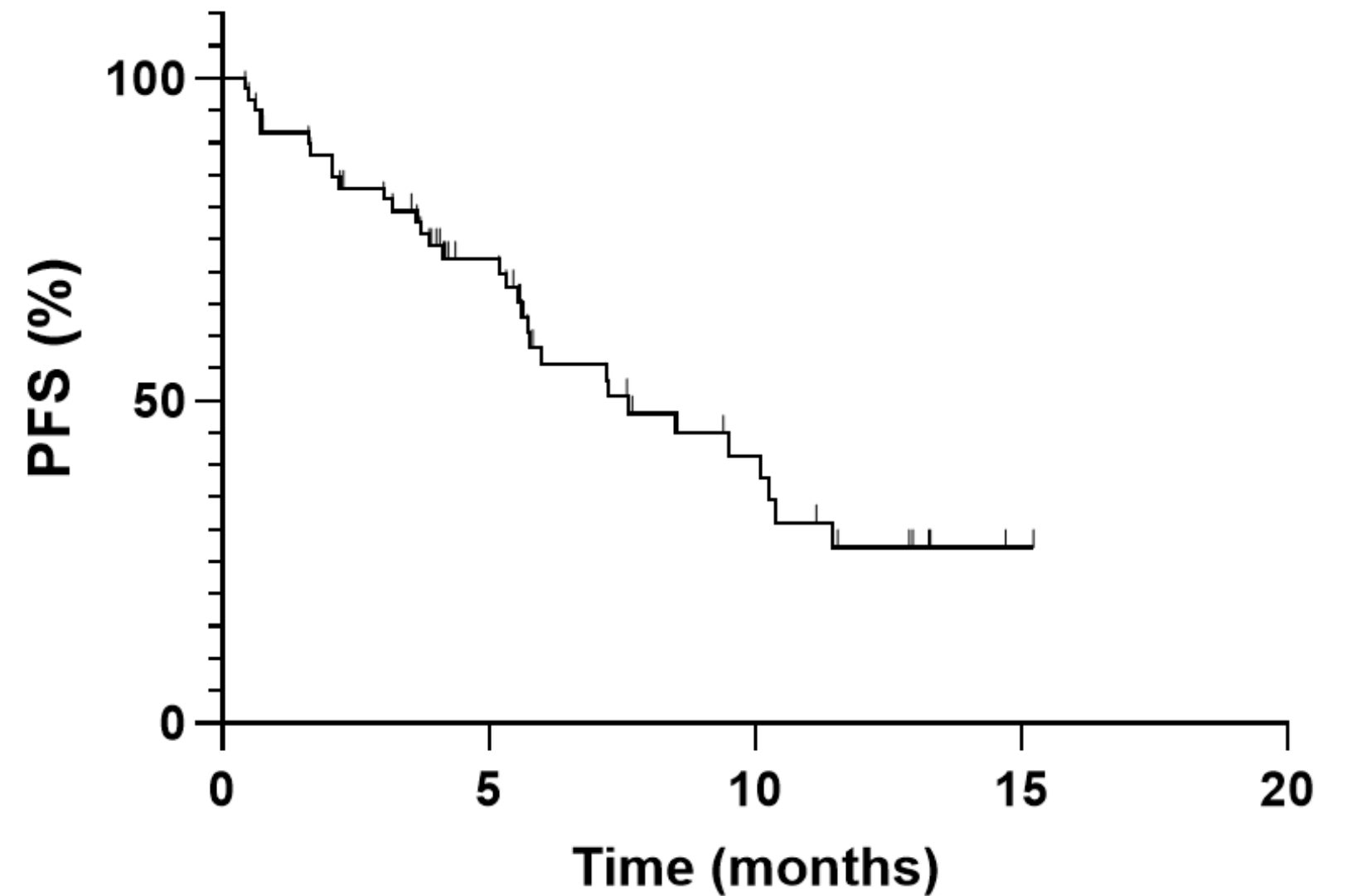
*Promising evidence of efficacy, durability and tolerability*

## 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/Abiraxane)	MPACT Trial (Gemcitabine/Abiraxane)	PRODIGE Trial (FOLFIRINOX)
PFS	7.6 months	5.5 months	6.4 months

All ACCENT patients @ 400 mg (n = 64)



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# ACCENT TRIAL INTERIM DATA

*Promising evidence of efficacy, durability and tolerability*

**17 confirmed responses** observed to date

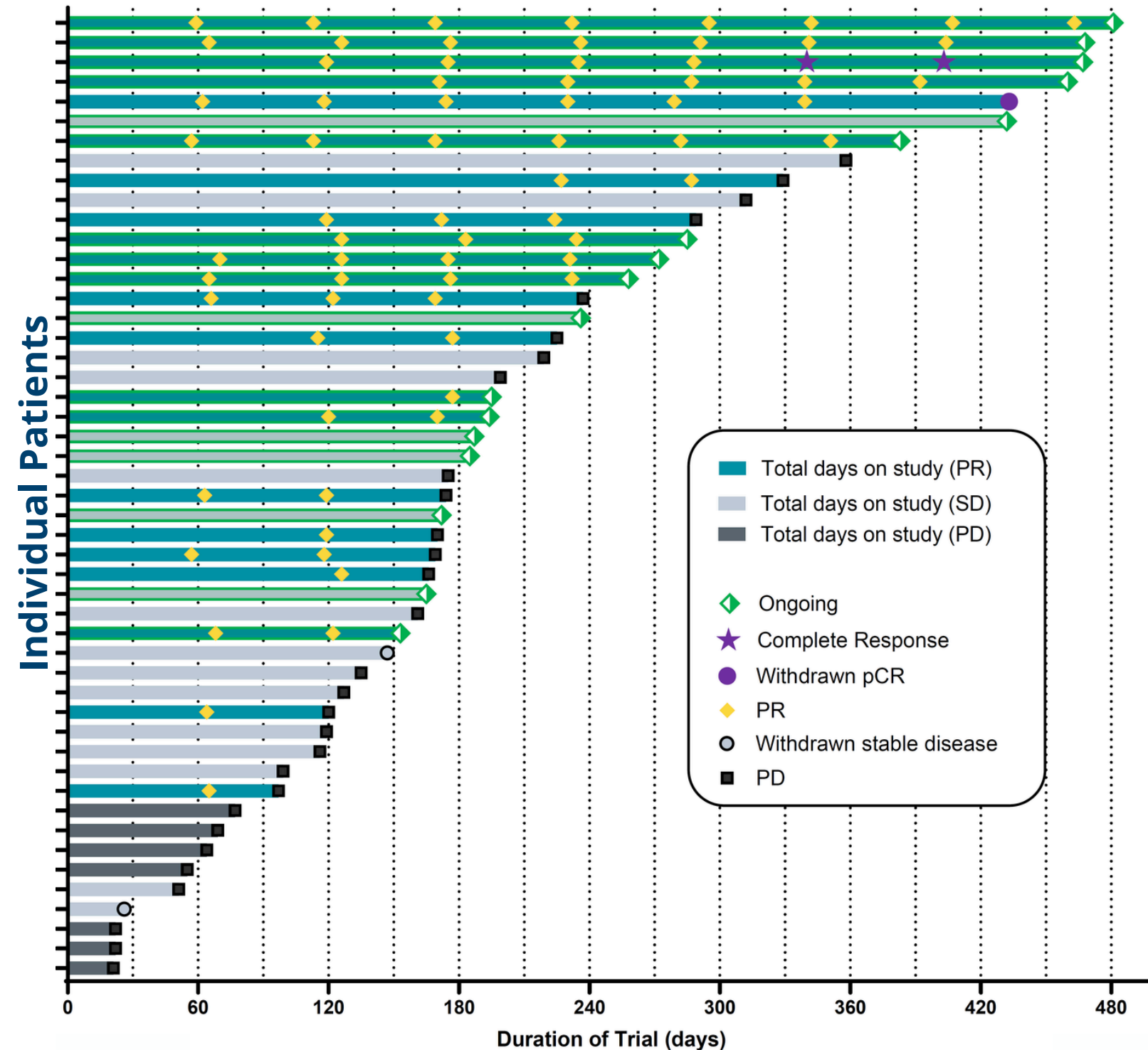
- Includes:
  - **1 confirmed complete response**
  - **1 pathological complete response**
- Indicating narmafotinib + chemotherapy is **superior** to chemotherapy alone

**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



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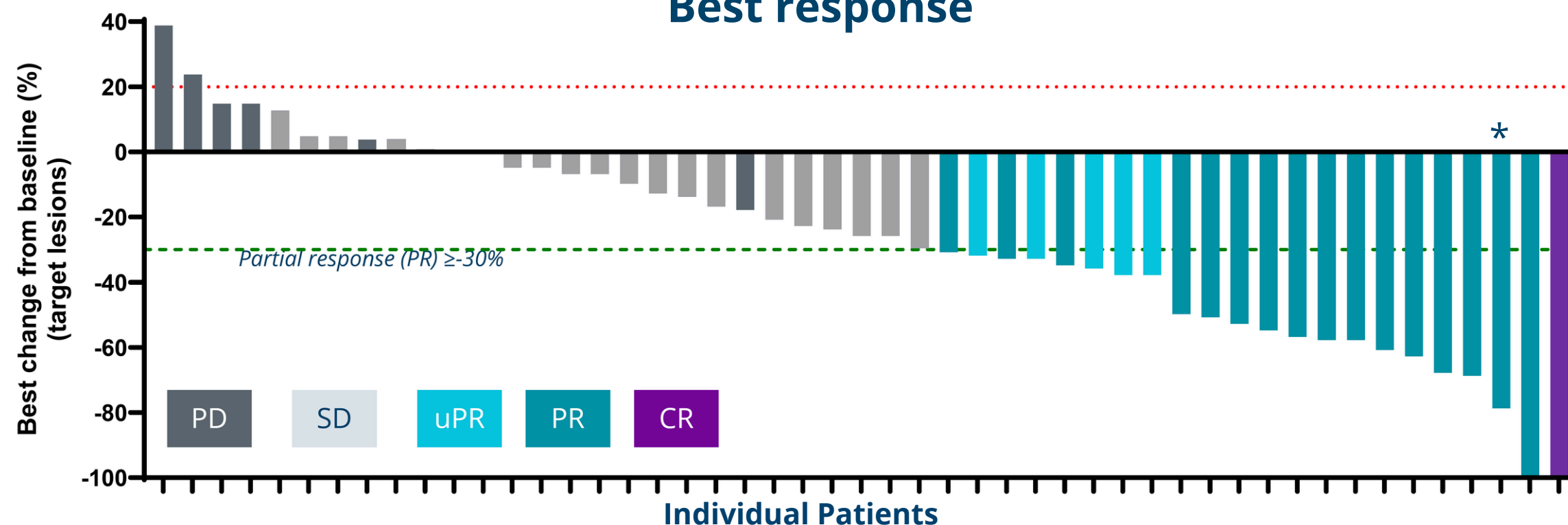
# ACCENT TRIAL INTERIM DATA

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%

### Best response



\* 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 trader'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 Jessaunt Pancreatic Centre at the Epworth Hospital in Melbourne.

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

The three 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. This 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 to 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 naramixin, 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 holds 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 tumour cells. AMP945 is designed to block the activity of the FAK protein, which also contributes to the formation of the fibrous protective layer around tumours. AMP945 was 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, he was devastated. "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'd do what I've got to do, and hopefully they'll operate and it'll be fine."

In fact, surgery for metastatic



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

**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

**I actually called the pathologist and said: 'Are you sure you're looking at the right specimen?'**  
CHRIS BURNS  
AMPLIA CEO & EXECUTIVE

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

**Making plans**  
Allan Zinat, a medical oncologist at the Jessaunt 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. "We don't have the data published yet, so I don't want to be speaking prematurely," he says. "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."

As for Mr Moulding, the hard work of the trial has resulted in a 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 boat and do some travelling."

As patient arm, 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."

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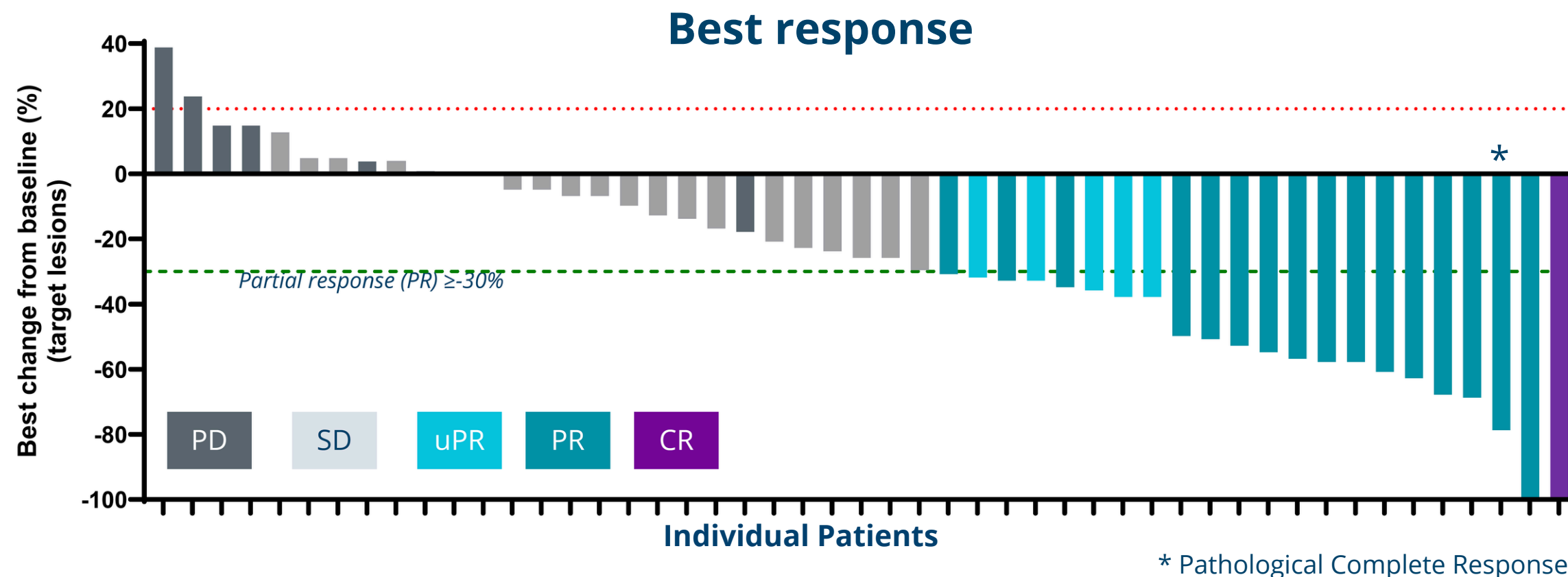
# ACCENT TRIAL INTERIM DATA

*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

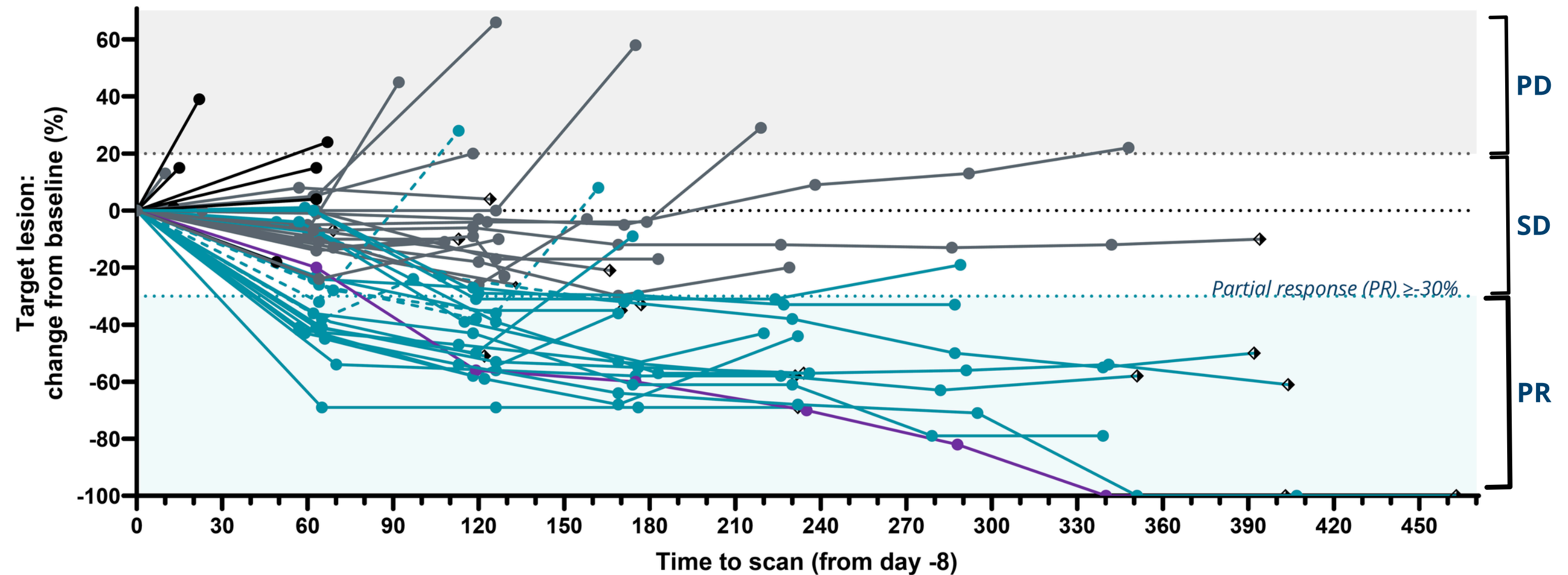


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# ACCENT TRIAL INTERIM DATA

*Promising evidence of efficacy, durability and tolerability*

**Excellent durability observed**



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# 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)
Neutropenia	38.2%	38%
Anemia	9.1%	13%
Diarrhea	5.5%	6%
Peripheral neuropathy	3.6%	17%
Vomiting	3.6%	NR
Febrile Neutropenia	5.5%	3%
Thrombocytopenia	NR	13%
Fatigue	NR	17%
Hypokalemia	NR	NR
Nausea	3.6%	NR

Gem/Abr (NAPOLI 3; N=379)	FOLFIRINOX (PRODIGE; N=171)	NALIRIFOX (NAPOLI 3; N=370)
39%	46%	24%
18%	8%	11%
5%	13%	20%
6%	9%	3%
2%	15%	7%
NR	5%	NR
NR	9%	NR
5%	24%	6%
4%	NR	15%
3%	NR	12%

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# 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

### RADIOOTHERAPY

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)

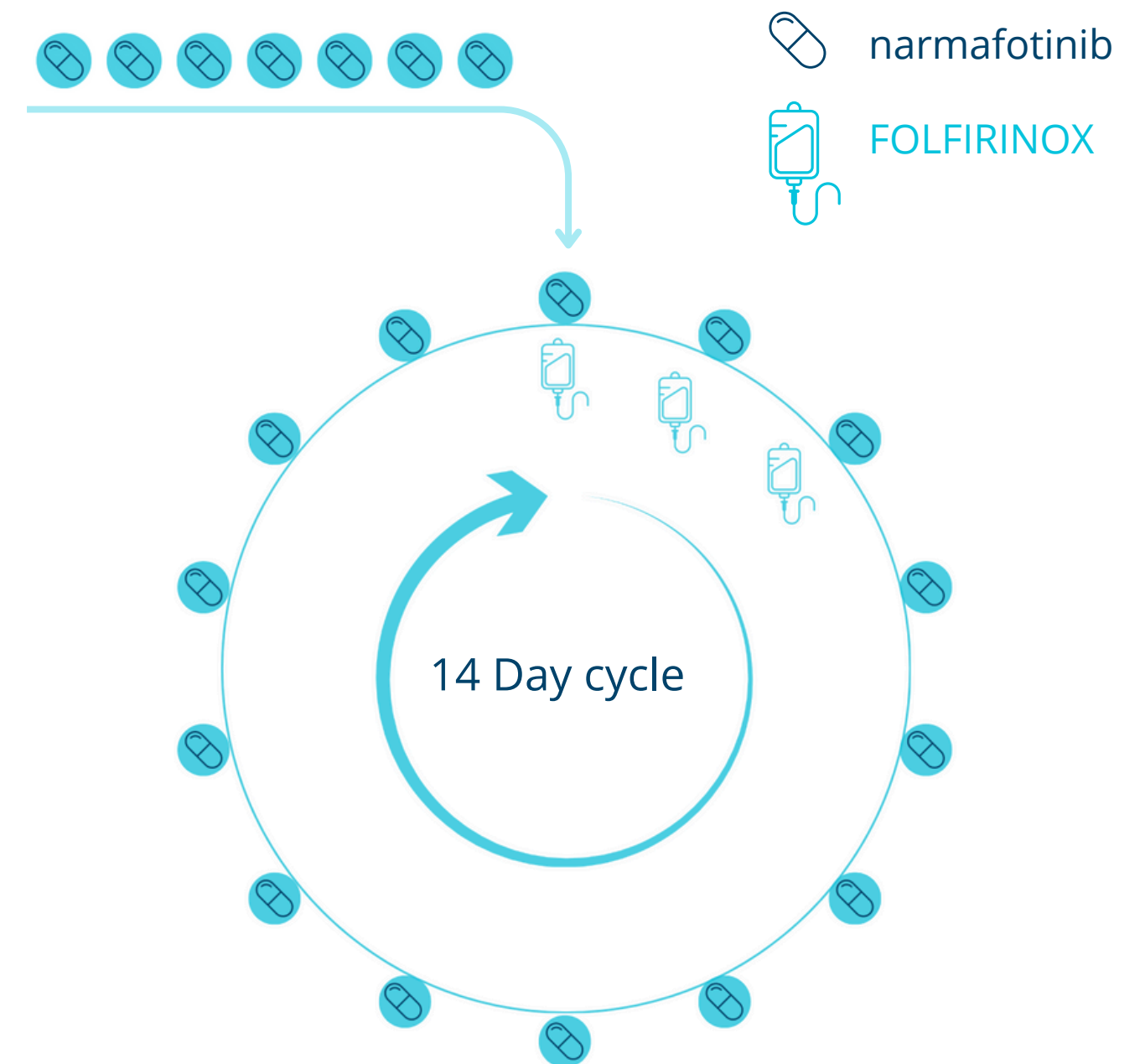
### PRIMARY ENDPOINTS

- 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:

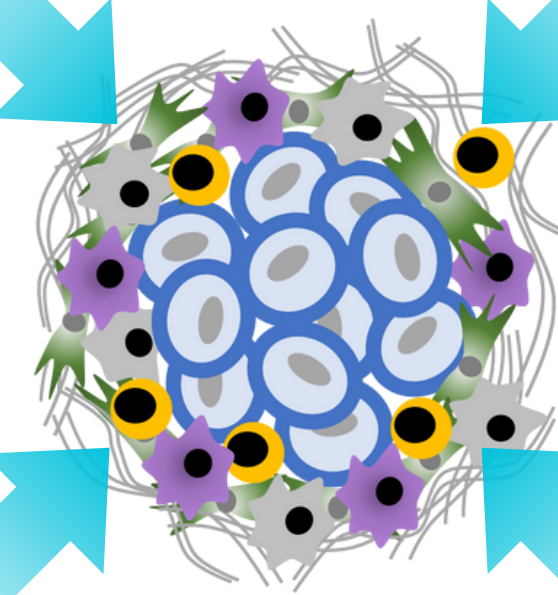
- Tumour growth
- Metastasis
- Treatment resistance

## Demonstrated efficacy in preclinical models of human pancreatic cancer

### Benefits of FAK Inhibition

**Anti-proliferative**  
Reduces cells' ability to proliferate and migrate

**Synergy with chemotherapies**  
Enhances activity of drugs and other therapies



Before treatment

Tumour (blue - cancer cells; green- fibroblasts; purple, grey and yellow - suppressive immune cells)

**Anti-fibrotic**  
Reduces scar-tissue in TME, improving permeability to drugs

**Immunomodulatory**  
Improves immune cell reactivity to tumour cells

# NARMAFOTINIB: A POTENT AND SELECTIVE FAK INHIBITOR

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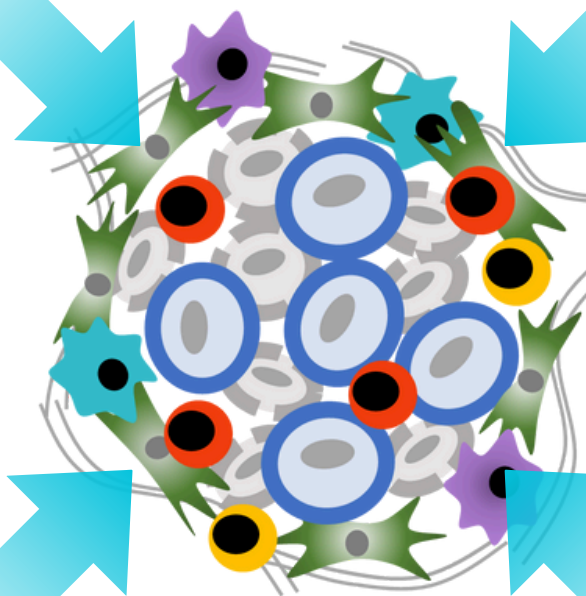
- Tumour growth
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## Demonstrated efficacy in preclinical models of human pancreatic cancer

### Benefits of FAK Inhibition

**Anti-proliferative**  
Reduces cells' ability to proliferate and migrate

**Synergy with chemotherapies**  
Enhances activity of drugs and other therapies



After treatment

Tumour (blue - cancer cells; grey - dead cancer cells; green - fibroblasts; purple and yellow - suppressive immune cells; red and turquoise - immune reactive cells)

**Anti-fibrotic**  
Reduces scar-tissue in TME, improving permeability to drugs

**Immunomodulatory**  
Improves immune cell reactivity to tumour cells

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

# NARMAFOTINIB: A POTENT AND SELECTIVE FAK INHIBITOR

*Best-in-class profile*

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## Convenient to take

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

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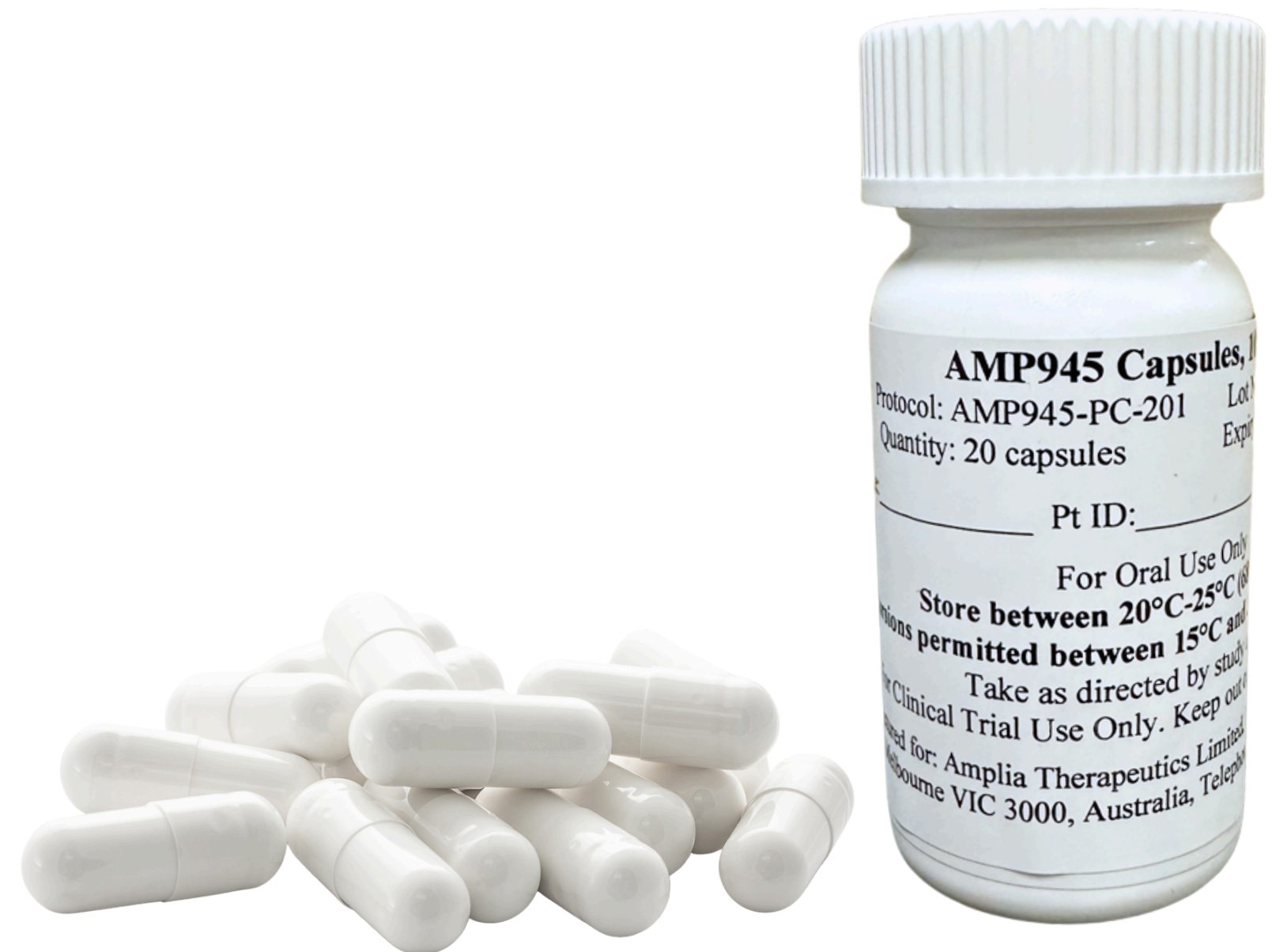
## Safe to combine with other medicines

- No evidence of drug-drug interactions

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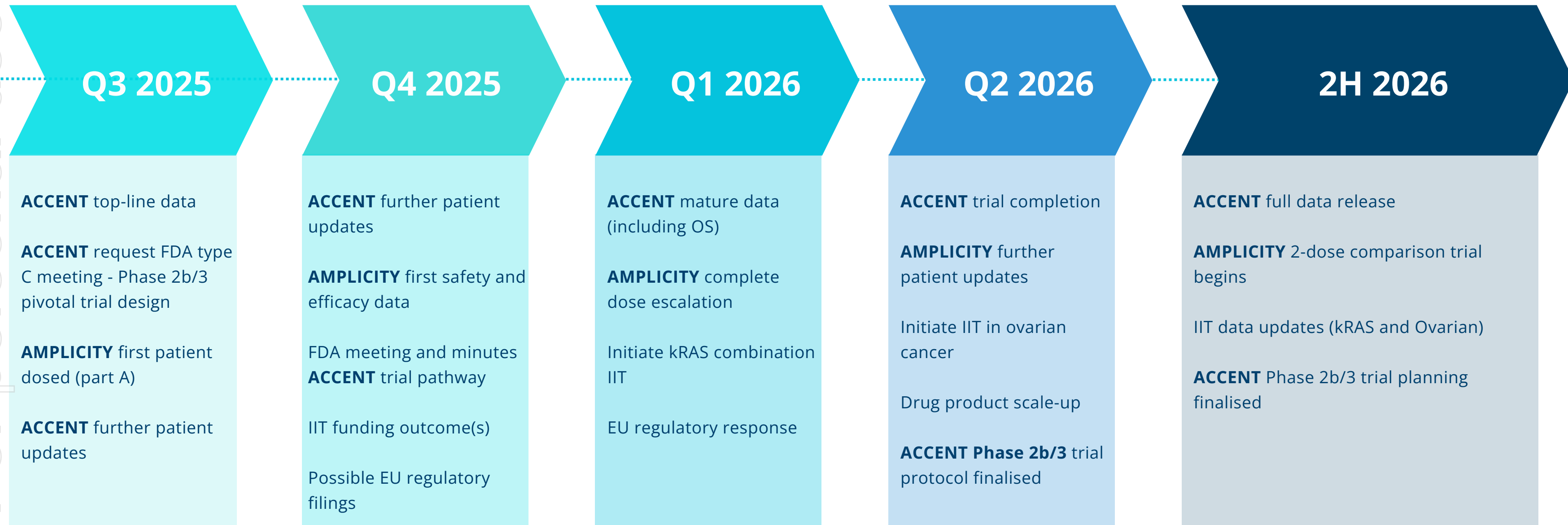
Evidence of FAK target engagement in humans

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# UPCOMING MILESTONES

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# BOARD + MANAGEMENT

## World-class experts

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MB ChB MPP GAICD  
Chair



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PhD  
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**Jane Bell**  
LLB LLM (Lond) FAICD  
Director



**Chris Burns**  
PhD GAICD  
CEO and MD



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COO



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## THANK YOU

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